Nutrition, Education and Development: The Case of Vitamin D Milk

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Abstract

Micronutrient deficiencies that reduce the health of children risk impeding human capital investments critical for economic development. While the developed world has largely eliminated the most pernicious of these deficiencies, they remain widespread in poorer countries. This study looks at the effects of the introduction of fortified milk, which contributed to the decline of one such micronutrient deficiency in the United States: vitamin D. At the time of vitamin D milk's introduction in the early 1930s, vitamin D deficiency, manifested most prominently in the form of rickets, affected large numbers of children. Using previously unexamined historical sources, I compile and introduce an original dataset describing the rollout of vitamin D fortified milk across the United States throughout the decade. I then use this dataset to examine the impact of fortified milk on schooling. The gradual expansion of vitamin D milk, along with natural variation in susceptibility to vitamin D deficiency due to geographic and racial factors, permits the identification of fortification's impact from other regional and temporal trends. Using a difference-in-difference-in-difference (DDD) estimator, I find that the availability of vitamin D milk increased schooling for the group at highest risk for vitamin D deficiency: African-American children from cities with low sunlight. A variety of sensitivity tests supports the validity of the results. They indicate that large scale food fortification initiatives merit further consideration from economists and policy makers concerned with achieving development outcomes.

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I. Introduction

Micronutrient deficiencies afflict more than 2 billion people globally, the majority of whom reside in developing countries (World Health Organization 2007). These nutritional shortfalls can impede physical and cognitive development in children and reduce overall health levels. Consequently, the potential for widespread micronutrient deficiencies to impede human and economic development has received increased attention in recent years.

In the developed world, however, micronutrient deficiencies have generally been eliminated as major public health threats. In the United States, for example, scientific developments in nutritional sciences during the early part of the 20th century translated into improvements in micronutrient status at a relatively rapid pace. Micronutrient deficiency maladies prominent during the pre-war period, including goiter and cretinism (iodine), pellagra (niacin), beriberi (thiamin), scurvy (vitamin C), rickets (vitamin D), and certain types of vision impairment (vitamin A), became extremely rare as the century progressed. Much of the credit for reducing the prominence of these disorders is due to the fortification or enrichment of foods with essential micronutrients (Bishai and Nalubola 2002; Park et al 2001). However, despite the critical role of these nutritional interventions, their impacts on measures of human capital remain relatively understudied.

This paper examines the effects of one particular nutritional innovation in the United States during the 1930s: vitamin D milk. During the 1920s, vitamin D deficiency, manifested most prominently in the skeletal disorder known as rickets, was among the most widespread diseases afflicting children (Backstrand 2002). Due to the body's dependence on sunlight for synthesizing vitamin D, and the fact that darker skin pigmentation impedes the body's ability to convert ultraviolet rays into vitamin D, black children in Northern cities suffered disproportionately from this malady (Weick 1967). However, within two decades after the discovery in the early 1920s of vitamin D's pivotal role in preventing rickets, the disease ceased to be a major public health problem.

Much of the credit for the decline of rickets is due to the widespread fortification of milk with vitamin D (Rajakumar 2003; Harrison 1966). Introduced in a few cities in the early 1930s, vitamin D milk was available in most areas of the country by the end of the decade. By the end of the war, the milk was virtually ubiquitous.

I examine the causal impact of the introduction of fortified milk during the 1930s on a measure of human capital investment. The gradual expansion of vitamin D milk, along with natural variation in susceptibility to vitamin D deficiency due to geographic and racial factors, permits the

identification of fortification's impact from other regional and temporal trends. I look specifically at how variation in the availability of vitamin D milk affected school attendance among African American children in low-sunlight cities, the group who suffered most severely from vitamin D deficiency.

This work is rooted in the evolving literature on health, nutrition, and economic development.[2] Economists have recently focused on the causal role of health improvements on human capital investment and, subsequently, economic development. Glewwe and Miguel (2007) cite recent progress in establishing causal evidence for a positive impact of nutrition on child health, and health on educational outcomes. Many of these studies examine the products of randomized evaluations. Most notably, in a long term follow up to a nutritional supplementation trial from the 1960s, Maluccio et al (2009) show that supplements increased the educational attainment of rural Guatemalan girls and raised labor market outcomes for both genders.

While such studies provide valuable evidence, the external validity of their results remain questionable. In particular, it is not always clear how a similar intervention operationalized as a large-scale public health initiative might perform. For policy makers interested in boosting human capital levels via efforts aimed at increasing nutritional status, external validity concerns are non-trivial. In contrast, the results here stem directly from such a large scale effort.

The findings here are of interest for a variety of reasons. Vitamin D deficiency remains a problem in many parts of the world, including a recent resurgence in the United States. Even in countries situated in areas that receive high quantities of sunshine, cultural attitudes towards dress that encourage covering large swaths of the body leave the population at risk.[3] The problem is particularly acute for women because mothers transmit vitamin D status both in utero and through the vitamin D content of breast milk (Wharton and Bishop 2003; Andrian et al 2002). Further, recent research has linked vitamin D deficiency with a host of health outcomes beyond rickets. The fortification of milk thus affords an opportunity to examine the effects of an in increase in the vitamin on the general population.

In poor countries, access to nutrients via fortified foods remains limited. For example, despite Sub-Saharan Africa's high rates of iron deficiency anemia—associated with cognitive impairment and decreased productivity (Horton and Ross 2003)—residents of only seven African countries have reliable access to iron-enriched flour (FFI 2010). By establishing a causal link at the aggregate level between a large-scale fortification program and child education, this paper hopes to

[2] See Alderman et al (2008) for an excellent summary.

[3] Despite nearly equatorial latitudes, studies in Jordan, Lebanon, Iran, Saudi Arabia and Turkey have found high levels of vitamin D deficiency among veiled women (Lips 2007).

spur more interest in researching the potential for similar programs to achieve development outcomes. Indeed, the return to remedying deficiencies in several micronutrients—e.g. iron, vitamin A, iodine and zinc—potentially exceeds the benefit of reductions in vitamin D deficiency (Horton 2008).

While other studies with similar structures, such as Bleakley (2007), have examined the impact of interventions that lower the burden of infectious disease on children, this study looks specifically at micronutrient status. Since the negative externalities of infectious disease (particularly hookworm) leave an easily justifiable role for external intervention, the proper role of community-wide public health initiatives (public or private) in boosting nutritional status is less clear. Recently, Field, Robles and Torero (2009) and Feyrer, Politi and Weil (2008) have found significant human capital payoffs to interventions aimed at eliminating iodine deficiency, the former focusing on contemporary Tanzania and the latter on salt iodization in the United States in the 1920s. This study seeks to build on this literature by describing the effects of an intervention aimed at vitamin D.

Finally, from a strictly historical perspective, the fortification of milk has not been previously detailed in a systematic way. I have compiled an original dataset using various historical and archival sources in order to describe, for the first time, the spread of fortified milk throughout the 1930s.

Results indicate that the availability of vitamin D milk did increase schooling for the population most at risk for vitamin D deficiency: African American children from cities with low levels of sunshine. The positive and significant impact is robust to a variety of different specifications. Further, I find no link between contemporaneous adult labor market outcomes and vitamin D milk, which lends further credence that the schooling effects are not artifacts of unobserved factors simultaneously influencing labor market conditions and the availability of fortified milk.

In Section II, I describe vitamin D deficiency in more detail and trace the development of fortified milk. In section III, I describe the data. Section IV details the rollout of vitamin D milk throughout the country. The identification strategy and empirical model are described in Section V. Section VI contains the estimation results, and I conclude in Section VII.

II. Vitamin D Deficiency and the Fortification of Milk with Vitamin D

A. Vitamin D Deficiency

Humans can obtain vitamin D from exposure to sunlight or consumption of foods or supplements containing the vitamin (Holick 2007). However, few foods, mainly oily fish, contain large amounts of vitamin D. Consequently, prior to the availability of supplements or fortified foods, sunlight constituted the most important vitamin D source (Carson 2008). The production of vitamin D from sunlight exposure varies by several factors, including season, latitude and climate. Most importantly, darker skin pigmentation can severely limit the body's ability to synthesize vitamin D from solar UV-B irradiation (Wharton and Bishop 2003).

Vitamin D originally came to prominence when medical researchers in the 1920s discovered that deficiencies in the vitamin led to the skeletal disorder known as rickets. The vitamin was soon well known among the medical community for its critical role in bone growth. By allowing the body to absorb calcium and phosphorus, vitamin D permits proper mineralization and growth of the skeleton (Holick 2007). Common rachitic symptoms at the time included flat feet, bow-leggedness, dental carries and extreme pain and muscle weakness (Moore 1928; Weick 1967). Aside from its crippling impact on those afflicted with the disease, rickets can cause skeletal deformities in the pelvis that lead to complications during childbirth (Loudon 1986).

As rickets has markedly receded over the past century, researchers have turned their attention to the role of vitamin D in a host of other biological functions and disorders. Guallar et al (2010) characterize the recent growth in vitamin D related research and attention as an "explosion". Recent interest has been piqued in part because of a reemergence in the presentation of classical, nutritional rickets, albeit at a much smaller scale (Wagner et al 2008; Rajakumar and Thomas 2005). The new cases generally occur among dark skinned, breast-fed infants (Kreiter et al 2000; Welch et al (2000); Misra et al 2008).

As noted by Andiran, Yordam and Ozon (2002), however, rickets and its adult form, osteomalacia, are only the most overt signs of vitamin D deficiency. Recent research has also this micronutrient deficiency to diabetes, cancer, autoimmune disorders and osteoporosis in adults (Calvo, Whiing and Barton 2004). Other work has connected vitamin D deficiency with hypocalcemic seizures, growth failure, lethargy, irritability, and a predisposition to respiratory infections during infancy for children (Wagner et al 2008). Vitamin D deficiency has also been implicated in mental health disorders, such as schizophrenia and depression (Holick 2007).

While the exact extent of vitamin D deficiency in the early 20th century United States is unknown, the available evidence suggests that it was quite prevalent, particularly in the urban

areas of the North and among African-Americans and others groups with dark skin pigmentation. Hess (1929), who studied rickets primarily in New York, surmised that it was the most common malady afflicting children in Northern industrial cities. Writing in a 1925 article in *The Scientific Monthly,* Craig and Belkin (1925) note that rickets was "so common that most babies experience a mild degree of it at some period, especially in winter". The use of X-Ray imaging to diagnose the disease reinforced this notion, as children that did not exhibit the most severe symptoms, like bowleggedness, were found to still demonstrate rachitic skeletal structure.

Statistics regarding the prevalence of rickets in the early 20th century are derived primarily from small, regional studies.[4] Moore (1900), for example, found that 318 of 400 babies in a Boston hospital were rachitic. A study in New Haven in 1923 using X-Ray for diagnosis led investigators to conclude that rickets was nearly universal in the region (Weick 1967). Other studies in the mid-1920s using radiographic diagnosis in Baltimore, Boston, New Orleans, and New York likewise found some form of rickets to affect the vast majority of young children (Greenbaum et al 1926; Wyman and Weymuller 1924; Supplee 1933; Weick 1967).

While mild forms of rickets were considered common in all populations, black children, whose darker skin pigment put them at higher risk, were most severely affected by vitamin D deficiency. In rural Maryland, a 1924 study examining rickets as defined by obvious skeletal changes found a 30 percent rate among white children under 2 and a 70 percent rate for blacks of the same age (Knox and Zenati 1926). Autopsies on babies at Johns Hopkins University hospital between 1926 and 1942 confirmed a higher relative prevalence among African American children, as well (Follis et al 1943).

Symptoms and complications from vitamin D deficiency likely affected schooling in a variety of ways. For severe, disabling cases of rickets, physical disfigurement posed practical barriers to attendance. Public schools at the time offered little accommodation for the disabled. Compulsory education laws exempted disabled children, and some schools specifically excluded them (Wright and Wright 1999). In schools that did permit enrollment, poor education quality offered little incentive to attend, as the mental and physically disabled were often grouped together and segregated from normal children (Wright and Wright 1999).

Pain and muscle weakness, common symptoms in rachitic children, can similarly disrupt educational outcomes. Vitamin D deficiency also leaves affected children more vulnerable to secondary illness, particularly reparatory infections like pneumonia and tuberculosis (Wharton and Bishop 2003). The connection between rickets, health and schooling did not go unnoticed at the

[4] See Weick (1967) for a more comprehensive summary of these studies

time. In a study investigating educational attainment by African Americans in New York City, Payne (1928) attributes a large share of lagging attendance and promotion rates among blacks to poor child health, particularly a 95 percent prevalence rate of rickets in Harlem. Likewise, Poull (1938) cites delays in mental development during infancy and early childhood among the rachitic. Further, health problems unknown to be connected with vitamin D deficiency at the time, like lethargy or autoimmune disease, likely also diminished affected children's ability to attend school, as well as their perceived payoff from education.

B. Developments Leading to Fortification of Milk with Vitamin D

Despite speculation and limited experimentation on the link between sunlight, cod liver oil and rickets, it was not until the end of World War I that scientists embarked on the path that would lead to the discovery of the etiology and cure of rickets. The final pieces of the rickets puzzle were assembled by American scientists. In 1922, Johns Hopkins University scientist E.V. McCollum showed that rats fed a diet of cod-liver oil that had been treated to remove vitamin A were still protected from rickets by the remaining nutrient (McCollum 1967). That nutrient was dubbed vitamin D, and would later be fully isolated and characterized in 1931. More importantly, almost immediately after McCollum published his findings, Columbia University's Thomas Zucker would patent a procedure for isolating the anti-rachitic element of cod-liver into a stable concentrate.[5] This concentrate would later be used to fortify foodstuffs with vitamin D, first poultry feed, and then bread and milk.

While McCollum had been able to distil the antirachitic element of cod liver oil, University of Wisconsin biochemist Henry Steenbock demonstrated that vitamin D could be added to substances by means of irradiation with ultraviolet light. Steenbock published these findings in 1924,[6] and filed for a patent, which he donated to the University of Wisconsin, on the process that same year.[7] The University of Wisconsin then formed the Wisconsin Alumni Research Foundation (WARF) in order to manage the patent.

The first commercial food product to be irradiated with vitamin D was Quaker Oats brand rolled oats, whose company had acquired the first license agreement with WARF in February of 1927 (Rajakumar et al 2007). In 1928, WARF licensed the production of a pharmaceutical product known as Viosterol (irradiated ergosterol) as an alternative to the unpleasant cod-liver oil taken

[5] Patent 1563134 was filed by Zucker in November, 1923, and granted in 1925.

[6] "The induction of Growth Promoting and Calcifying Properties in a Ration by Exposure to Ultra-Violet Light" *The Journal of Biological Chemistry*, 1924, 61: 405-22 (with A. Black); "The Induction of Growth Promoting and Calcifying Properties in a Ration by Exposure to Light" *Science*, 1924, 60: 224-25.

[7] Working separately but at almost the same time as Steenbock, Alfred Hess also demonstrated that light rays could increase vitamin D content.

routinely as a curative for rickets. Steenbock and WARF rejected the vast majority of other potential licensees, and by 1930, the only non-Viosterol irradiated products on the market were Quaker Oat's Farina and Muffets (i.e. shredded wheat) and Fleischman's yeast cakes (UWAHSC Blowney 1930).[8] These products, however, did not receive widespread distribution and were still considered experimental in 1931 (UWAHSC Craig 1931). Bread fortified with vitamin D, both with cod-liver oil concentrate via the Zucker process and WARF's irradiation would appear in 1932, but it was the fortification of milk that would give the American public widespread exposure to a prophylaxis for vitamin D deficiency (Henderson 1932; Zucker 1933).

C. Fortified Milk

The discovery of vitamin D confirmed that both breast milk and cow's milk were naturally poor sources, and the idea of remedying that shortfall soon took hold. As early as 1925, Cowell (1925) had exposed milk to an ultraviolet lamp and demonstrated that it could be a viable antirachitic product. The desirability of employing fluid milk as the primary agent for fortification was self-evident to many of the health researchers working in the field during the 1920s and 30s. Tobey (1936) counted at least 30 clinical trials of vitamin D milk between 1925 and 1935. The reason for the enthusiasm was twofold: milk was almost universally consumed by children, and nutritionists at the time believed it to be the most nutrient rich food available for children (i.e. "the perfect food"), containing vitamins thought to work in harmony with Vitamin D for healthy skeletal development (particularly calcium and phosphorus).[9]

As was the case with the addition of iodine to salt in the 1920s, the fortification of milk began with a push from concerned medical professionals and was subsequently adopted by private companies looking for a competitive edge for their products. Though, the initial lobbying and research from the medical community led to voluntary implementation by a small number of manufacturers, subsequent industry-wide adoption was then driven primarily by consumer demand (Bishai and Nalubola 2002; Markel 1987). Mandatory fortification laws for fluid milk were never enacted.

The first commercially available vitamin D milk was introduced in 1931 and known as "Yeast Milk" or "Metabolized Milk". Production of the milk, licensed by WARF under Steenbock's patent, was accomplished by feeding cows irradiated yeast. However, it never grew beyond a niche

[8] All citations marked UWAHSC refer to archival materials from the University of Wisconsin Archives Henry Steenbock Collection and are listed in a special section of the references.
[9] For expressions of this viewpoint, see Gowen et al (1926), Supplee (1933), Tobey (1932) and Weckel (1941).

product, due mainly to its cost and reliance on intervention at the farm level.[10] With the availability of other methods of improving the vitamin D content of milk, only small scale producer-distributors continued to market yeast milk in the 1930s (Roadhouse and Henderson 1950).

The analysis of this paper will focus on the two principal methods of fortifying fluid milk with vitamin D that rose to prominence during the 1930s.[11]

1) Concentrate Fortified (Vitex) Milk[12]

The appearance of Vitex milk in Detroit in February of 1932 marked the beginning of the mainstream adoption of fortified milk. The National Oil Products Company (NOPCO) of Harrison, NJ used Zucker's process to produce a cod liver oil concentrate they branded Vitex under an exclusive license deal with Columbia University's University Patents Inc. Dairies purchased the Vitex concentrate, added the substance to fluid milk, and mixed in the concentrate until it achieved a uniform distribution in the milk. These milks generally contained 400 USP per quart.

Columbia University's decision to allow NOPCO virtually free reign in marketing its product provided a crucial step in raising vitamin D milk from the purview of the medical profession to the mass market. Unlike the more circumspect WARF, NOPCO brazenly marketed its products to dairies and medical professionals.

The method of adding Vitex to milk implied an almost purely marginal cost structure. Consequently, since dairies using Vitex did not need to make any large initial investments in order to begin producing Vitamin D milk, the Vitex option was attractive to small dairies hoping to improve their market position and profitability by adding a premium product.

2) Irradiated Milk

The second major vitamin D milk to appear on the market had the most immediate initial impact. Dairies produced irradiated milk by exposing a thin film of flowing milk to intense ultra-violet radiation, usually via a carbon-arc lamp. Milk produced in this manner contained 135 USP of Vitamin D per quart, though Steenbock was adamant that research by Hess and others demonstrated that irradiated milk actually delivered a therapeutically equivalent dose of vitamin D as Vitex milk's 400 USP per quart (UWAHSC Minnesota Milk Company 1933).

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[10] Steenbock himself, as well as other public health officials, were never enthusiastic about the process due to the difficulty in controlling the vitamin D content of milk fortified in this manner. Peak yeast milk production in 1939 saw only 4,000 cows fed irradiated yeast nationally in four different states (Roadhouse and Henderson 1950; AMA Council on Food 1939)

[11] A fourth method, directly irradiating cows with ultra-violet rays proved largely inefficient and impractical.

[12] Vitex was not the only brand of concentrate fortified milk, though it was far and away the most successful. Its main competitor in the concentrate field was known as Clo-D. During the next decade, concentrates from irradiated ergosterol would supplant other types of vitamin D milk to become the standard fortification method (Roadhouse and Henderson 1950).

Following the successful development of commercial irradiation equipment, Borden's introduced the first consumer irradiated milk product in Detroit in January of 1933 (UWAHSC Milk Report #8 1935). Unlike Vitex, which appealed to small dairies, the initial investment required for an irradiator meant that only large producers found irradiated milk a profitable venture. However, for those with large enough sales, the minimal marginal cost—due mainly to limited energy consumption from the irradiators—made irradiated milk an obvious and much cheaper choice. While Vitex costs would soon come down, the lower cost per quart for large producers of irradiated milk helped facilitate its rapid expansion (Roadhouse and Henderson 1950).

III. Data

This study links data on the rollout of fortified milk across the United States during the 1930s with individual data on educational attainment and demographic and socioeconomic controls taken from historical census files. Due to the structure of fluid milk distribution during the 1930s, the availability of vitamin D milk varied at the city level. For that reason, the city level is the primary geographic unit of analysis. Because the census does not identify cities below a certain population threshold, I also undertake secondary analysis at aggregated county groupings known as "State Economic Areas" (SEAs).[13]

I assemble data on the spread of vitamin D milk from two primary sources: 1) The Henry Steenbock Collection of the University of Wisconsin Archives and 2) The roster of products accepted into the American Medical Association (AMA) "Seal of Approval" program. Other sources of data on Vitamin D milk include dairy industry trade periodicals, public health journals, newspapers, and USDA reports.

Due to the University of Wisconsin's prominent role in developing the science and technology behind vitamin D fortification of milk, as well as its active role in managing the licenses it granted to dairies to market the product, the university's archives contains the most comprehensive information on the vitamin D milk's origins and proliferation. For all dairies that produced vitamin D milk under a license from WARF, files from the archive identify the dairy's location and date of the licensing agreement. For 74 percent of these dairies, a reliable estimate of the average quantity of vitamin D milk produced daily is either stated directly or can be imputed using royalty payment invoices.[14] Dairies with missing information on quantity and royalties are assigned quantity values based on state averages.

[13] The census identifies 97 cities consistently between the 1930 and 1940 census
[14] Reliable quantity data is available for 74 percent of WARF dairies in the dataset.

Data on the timing and location of dairies producing Vitex milk is taken from American Medical Association (AMA) Council on Foods, which distributed a "Seal of Acceptance" to vitamin D milk products. The AMA published a list of newly approved dairies in the *Journal of the American Medical Association* (*JAMA*) until the end of 1935.[15] In 1939, the AMA published a full listing of vitamin D dairies in a separate volume. Consequently, data on vitamin D milk produced by dairies not licensed by WARF is missing for 1936 to 1938.[16] In addition, quantity data for non-WARF dairies is not known. For estimates that use quantity data, non-WARF dairies are assigned a quantity of 2,000 quarts daily, which a Vitex official reported to be the average output of a Vitex dairy (Post 1934).

Data on the location of dairies distributing vitamin D milk are matched with data on insolation, which measures the amount of solar energy (i.e. direct sunlight) an area receives.[17] Because the body produces vitamin D by synthesizing cholesterol and sunlight, insolation is a good measure of the geographic variation in susceptibility to vitamin D deficiency (Carson 2008). Reliable and consistent measurements of insolation during the 1930s do not exist for most of the United States, so I calculate the mean insolation from 1980-1999 using data from the National Aeronautics and Space Administration.[18] I normalize this data, and create a dummy variable equal to one for cities with below average insolation. This categorization is both a natural breakpoint, and, due to the geographic distribution of US cities, consistent with current medical knowledge of the effect of sunshine on vitamin D synthesis. In particular, due to the zenith angle of the sun, UV-B exposure above 37° latitude is nearly absent for much of the year, while vitamin D synthesis is much greater below this parallel (Holick 2004). In the normalization performed here, all cities characterized as having above average insolation fall below 37° latitude, and all cities with below average insolation are north of that point.[19] Consequently, the dummy variable categorization follows the accepted medical standard regarding the interaction of geography and vitamin D.

Micro data come from the *Integrated Public Use Microdata Series* (IPUMS). Primary analysis uses the one percent sample from the United States Census of 1930 and 1940. Sensitivity analysis includes samples from 1920 through 1950. For the main analysis, I focus on native born children

[15] It is unclear why *JAMA* stopped publishing its list of new vitamin D milk dairies, though space considerations due to the accelerated pace of the adoption of the technology likely played a role.
[16] In the estimation, dairies first listed in the 1939 volume are considered to be beginning in 1939.
[17] Insolation is an acronym for Incident Solar Radiation
[18] Insolation data are taken at the county centroid, so the city measurement corresponds to its county's centroid. Data are available from http://eosweb.larc.nasa.gov/cgi-bin/sse/sse.cgi?
[19] Louisville, KY, the sunniest city still considered 'below average', sits at 38° latitude. The least sunny city considered 'above average' is Knoxville, TN, located just below 36° latitude.

aged from five to fourteen. Including older children does not substantially alter the results.[20] In addition, I omit children who have migrated within the previous five years in order to reduce hidden variation in length of exposure to vitamin D milk among children from the same city or SEA.

To determine the impact of the introduction of vitamin D milk on education, I use a binary school attendance measure from the census as an outcome variable.[21] Children reporting that they attended school during the months before the census are coded as 1, and those responding that they did not attend coded as 0. The census data also includes data on race, age, and the occupation of the parent.

In addition to individual level controls, I also include aggregate variables that may have influenced schooling decisions. A full listing of controls is given in table 1. Note that New Deal spending relief is missing for several cities identified by IPUMS. Therefore, in order to preserve observations, the New Deal variable is used primarily for sensitivity analysis.

Summary statistics provided in table 2 give an initial indication of the effects of the introduction of vitamin D milk. Schooling increased on average for high sunlight areas regardless of access to vitamin D milk. However, schooling increases in low sunlight areas were negligible in cities without early access to vitamin D milk, but greater than one percentage point in cities with vitamin D milk. Though the standard deviation is quite high, that contrast is even starker for African American schooling rates. The differential rates of growth in schooling provide motivating evidence for a vitamin D milk effect that varies by race and geography.

IV. The Spread of Fortified Milk

Table 3 shows the increase in the number of milk plants nationally between 1932 and 1935. The rapid increase during 1934 and 1935 grew vitamin D milk from a niche to a mainstream product in several cities. The rollout of vitamin D milk across the country is displayed in figures 1 through 3. Vitamin D milk established itself early in the upper Midwest, particularly Michigan—the state that originated iodized salt. Both WARF and Vitex made their milk available at approximately the same time in many of the state's markets (i.e. Detroit, Battle Creek, Flint, Grand Rapids and the Lansing area).

Throughout the decade, the availability of vitamin D milk spread from a handful of cities, primarily in the upper Midwest, to include large swaths of the Northeast and significant cities in the South and West of the country. However, despite the near ubiquity of vitamin D milk in America's

[20] The chosen age group is meant to be large enough to allow substantial variation in vitamin D milk exposure within an area while maintaining a focus on younger children likely to be most affected by the effects of vitamin D deficiency.

[21] Other measures of education are not consistently available in both 1930 and 1940.

population centers by the end of the decade, significant heterogeneity in the timing and intensity of take up prevailed even within regions.

Some areas of the country had virtually no exposure to Vitamin D milk. On the East Coast, Maryland, Vermont and Rhode Island were completely bereft, with regulatory hurdles particularly important in the first. Baltimore's ban on any cap markings signifying that the milk underneath contains vitamin D effectively precluded the entrance of vitamin D milk in the city, as dairies were unable to charge a premium for a product with an identical label as their non-premium brand (AJPHNH 1938). In contrast, vitamin D milk's market penetration in Portland, Maine topped the rest of the country (Standard Brands 1936).[22]

With the exception of California and the Northwest, little Vitamin D milk reached the thinly populated states of the West. Additionally, San Francisco required residents to have a prescription in order to obtain vitamin D milk, though the milk was available without prior medical approval in Oakland ("Milk and Dairy Products" 1938). Some areas in the Deep South also saw little access to fortified milk. Birmingham, for example, did not have fortified milk during the entire decade due to regulations that required vitamin D milk producers to pay the cost of regulatory supervision of the product ("Milk and Dairy Products" 1938).

The absence of legal mandates requiring fortification stands out among the notable features of vitamin D milk's growth during the 1930s. As with iodized salt, private businesses supplied milk based on voluntary expected consumer demand. A large element in creating that demand, however, was the involvement of the medical and public health community. Vitamin D milk was thus able to 'piggy back' on the recommendations of physicians and health researchers who continued to publish work on the special milk's desirability throughout the decade. Indeed, both Vitex and irradiated milk prominently displayed their respective University affiliations in advertisements, and were quick to capitalize on new academic publications that recommended use of their products.

V. Empirical Modeling

A. Identification Strategy

The identification of the impact of vitamin D fortification from other unobserved factors derives from both the temporally and regionally disparate nature of the rollout of vitamin D milk, as well as the geographic and racial differences in pre-intervention susceptibility to Vitamin D deficiency. Vitamin D is primarily obtained via exposure to sunlight, and the effectiveness of such

[22] Residents of Benton Harbor, with a metropolitan area of just over 26,000, appear to have consumed the highest proportion (approximately 50 percent) of vitamin D milk for cities with a population above 25,000 (*Bulletin of the Dairy Research Bureau* 1937).

absorption is reduced by skin pigmentation (Holick 2007).[23] Consequently, darker-skinned individuals living in areas with low solar radiation are at highest risk of deficiency. Therefore, they should have the most to gain from the introduction of vitamin D milk. Further, the introduction of vitamin D milk developed gradually, occurring in different regions of the country at different times.

The structure of the vitamin D milk situation suggests a natural difference-in-difference-in-difference (DDD) modeling approach. The treatment effect of vitamin D milk is measured by the difference in outcomes between racial groups and cities of different latitude after the milk's introduction. In this sense, whites in areas with high sunlight act as controls to blacks from places with low insolation. This strategy does not require particularly strong assumptions of the randomization in the spatial and temporal rollout of vitamin D milk. Even if the timing and location of the introduction is systematically related to an unobserved variable that also impacts the outcome of interest, the impact of the unobserved factor would need to systematically differ by insolation and racial group in order to bias the results.

To illustrate what would be required to threaten the identification strategy, consider an extreme hypothetical. Suppose an unobserved component of the WARF license agreement forced each new dairy licensee to build a school in the same year that they begin distributing vitamin D milk.[24] Further, suppose that this clause applied only to Northern cities classified as high insolation areas. This scenario would still only violate the exogeneity assumption in the estimation if the school benefitted one of the racial groups more than the other. Further, the bias would only be positive if blacks benefitted more than whites; otherwise the bias would be attenuating. In fact, I have found no evidence of any such systematic relationship between vitamin D milk's introduction and other educational or health interventions.

The timing of the introduction of vitamin D milk does not appear systematically related to unobserved variables that may have also influenced the outcome under study. The rollout occurred gradually and touched nearly every region of the US throughout the 1930s (see figures 1-3). The most important factors appeared to be the market structure of the dairy industry and the attitude of local health authorities towards interventions that affected the town's milk supply.

The most likely scenarios that might pose a challenge is that cities that permitted or encouraged dairies to adopt vitamin D milk did so due to a concomitant change in leadership or attitude towards health measures among residents. For example, the installation of more forward

[23] Larger amounts of melanin in the outer layer of the epidermis interferes with the conversion of 7-dehydrocholesterol to vitamin D caused by exposure of the skin to ultraviolet radiation.
[24] This is a strict hypothetical scenario. No such terms existed or were implied in any license agreements observed in the archives.

looking health officials could influence both the timing of vitamin D milk's introduction and the adoption of unobserved public health measures that improve outcomes. Alternatively, an unobserved factor that stimulates consumer demand for healthier products or interventions could attract vitamin D milk and other goods and services that alter health outcomes. Though these scenarios are unlikely—the rollout of vitamin D milk was more intertwined with patters of regional technology diffusion in the dairy industry and attitudes of local regulators towards milk safety and marketing—they imply that the impact of vitamin D milk could be confounded by other similarly timed initiatives without differencing at the geographic and racial levels.

Finally, the intervention under examination here—the introduction of vitamin D milk—is measured at the aggregate level. The results here provide estimates of the average impact of *access* to this technological innovation in nutrition in the private market. While individual data that might elucidate the precise mechanism underlying the relationship between fortified milk and schooling do not yet exist, the aggregate measure here given here is of interest precisely because it occurs at the policy level. In a randomized control trial, where investigators, for example, might provide vitamin D milk and make observations at the individual level, it is not always clear how a similar intervention operationalized as a large-scale public health initiative might perform. In contrast, the estimates here provide a causal estimate of the average impact of the availability of a nutritional intervention on schooling nationwide.

B. Econometric Specification

In order to estimate the effect of access to vitamin D milk on school attendance, I estimate the following difference-in-difference-in-difference (DDD) model:

$$\begin{aligned} A_{icjt} = {} & \beta_1 D_{cjt} * S_j * R_{icjt} + \beta_2 D_{cjt} * S_j + \beta_3 D_{cjt} * R_{icjt} + \beta_4 S_j * R_{ijt} \\ & + \beta_5 D_{cjt} + \beta_6 R_{icjt} + \delta_t + \delta_j + \delta_c + X_{ijt}\Gamma + Z_{jt}\Psi + \varepsilon_{ijt} \end{aligned} \quad (1)$$

Schooling attendance for person i of birth year cohort c in place (city/SEA) j and year t is given by A_{icjt}, where t can take the values 1930 and 1940. The chosen vitamin D exposure variable is given by D_{cjt}, S_j denotes the insolation dummy, R_{icjt} denotes a dummy variable for race equal to one for African Americans, δ_j is a geographic fixed effect, δ_t are time (year) dummies, δ_c cohort fixed effects, X_{ijt} is the vector of individual control variables, and Z_{jt} the regional control variables (see table 1 for list of control variables).

The estimation considers a range of definitions for the vitamin D exposure variable. The main results consider D_{cjt} as a dummy variable equal to one if any vitamin D milk is available in

area *j* prior to a specified age cutoff. Because vitamin D status plays its most critical role in musculoskeletal development during very early childhood, I consider a variety of age cutoffs ranging from zero (in utero) to five. Thus, under this treatment definition, a child born in 1934 in a city where vitamin D milk was first available in 1936 would be considered treated (i.e. $D_{cjt} = 1$) for an age cutoff of 2, but untreated for an age cutoff of 1 or 0 (i.e. fetal exposure only).

The dummy variable specification yields the most straightforward interpretation of the DDD parameter β_1. In addition, the simple dummy variable specification is less prone to error from missing or imputed data. However, a simple dummy may miss potentially important underlying variation in the extent of the milk's availability. For this reason, I also include specifications of the vitamin D exposure variable that model D_{cjt} as either the number of dairies or the imputed quantity of vitamin D milk available to children below the specified age cutoff. While the former provides an indication of the penetration of vitamin D milk in a city, it is unclear how the marginal effect of an extra dairy changes the accessibility of milk as the number of dairies increases. In contrast, the treatment dummy approach is relatively nonparametric. And while it is desirable to have a measure of the actual quantity of vitamin D milk available in the city, the high degree of imputation due to missing data adds significant measurement error to estimates using this specification.

VI. Results

A. Schooling

This section presents the results of estimating versions of equation (1) under various specifications. Using the 1930-40 census, and restricting the geographic unit of analysis to the city level, tables 4 presents the main results of the paper. In table 4, the effect of vitamin D milk's availability, specified as a dummy variable equal to one if a dairy in an individual's city supplied vitamin D milk prior to a given age cutoff, is displayed for various values of the cutoff. Using the full set of control variables in table 1 except New Deal relief, the DDD parameter is positive and significant for all age cutoffs with the exception of age 5, where the estimate is positive but not as precisely estimated.

The magnitude of the DDD parameter declines as the age cutoff increases. This pattern suggests vitamin D milk's availability had its greatest impacts on infants and the unborn. Given that maternal vitamin D status during pregnancy plays a critical role in protecting infants from Vitamin D deficiency even after birth, this is not an implausible result (Wharton and Bishop 2003). Alternatively, because those with early access to vitamin D milk also accumulated more childhood

years with the milk, the average duration of exposure to fortified milk increases as the age cutoff for the treatment group declines. The pattern of results might therefore indicate the importance of prolonged intake, rather than a critical age threshold.

For blacks in cities with below average sunshine, the estimates imply that the availability of vitamin D milk in utero led to an approximately 5 percentage point increase in school attendance. The increases for ages 1, 3 and 5 are 3.5, 2.3 and zero, respectively.

To gauge the sensitivity of the results to the addition of control variables, table 5 displays estimates of the DDD parameter under a variety of different controls. The different specifications have little effect on the point estimates. Across all age cutoffs, the addition of control variables slightly reduces the magnitude and increases the standard errors of the DDD parameter. The reduction in sample size resulting from the inclusion of the New Deal relief variable, which is missing for 32 cities in the sample, accounts for part of this reduction in precision. However, for all age cutoffs except age five, the parameter remains positive and significant.

As a further check on the robustness of the results, I vary the samples and geographic units for the estimates in table 6. In rows A and B, SEA replaces the city as the unit of analysis. While the city-level estimates are restricted to cities with over 100,000 residents in 1940, which are consistently identified in both 1930 and 1940 IPUMS data, the use of SEA permits estimation over all areas of the United States. However, as noted earlier, dairy plants generally served very localized areas. Consequently, aggregating to the SEA-level introduces considerable noise into the effect of vitamin D milk, as observations from outlying areas with little true exposure are grouped with city dwellers. The implications of the aggregation can be seen particularly in row B, which includes identical controls to the main results in table 4. The DDD parameter estimates remain positive and of comparable magnitude to the baseline specification. However, the increase in the standard errors causes the estimated coefficients to fall just outside the conventional range of significance.

In rows C and D of table 6, I estimate variants of equation (1) using the census years 1920 through 1950. In row C, I replicate the baseline estimates from table 4 using available control variables.[25] The DDD parameter estimates remain almost unchanged from the 1930-40 estimation, with the exception of the zero (i.e. fetal) age cutoff, which is nearly halved. The diminishing of the estimate for only those with fetal exposure calls into question whether the relatively larger coefficients on the in-utero estimates in table 4 reflect the true importance of maternal vitamin D status and intake during pregnancy. This change induced by expanding the sample indicates that the results from the 1930-40 regressions, in which only very young children (between 5 and 8) in

[25] Table 1 indicates variables available for the 1920-50 sample.

cities with vitamin D milk access prior to 1936 had such milk available during gestation, may be driven by relatively large effects in a few cities. In the 1920-50 sample, children of all ages, including cities that had a relatively late introduction of vitamin D milk, have the potential to fall in the treatment group for all age cutoffs, including age zero. Likewise, the use of the expanded sample reduces disparities in average duration of exposure across the different treatment groups defined by age cutoff. Indeed, point estimates across the cutoff ranges are more similar to each other in this expanded sample.

In order to determine if trend differences account for the estimated vitamin D milk effects, I incorporate a city-year trend term to the estimates using the expanded 1920-50 sample. The results are reported in row D of table 6. With the exception of the age one cutoff, adding the trend term only slightly alters the DDD parameter point estimates across all the cutoffs. All estimates remain positive and significant.

I also use alternative measures of vitamin D milk availability to examine its impact on schooling. In table 7, I replace the dummy variable specification of the milk treatment variable D_{cjt} with two alternatives: the number of dairies producing vitamin D milk, and the imputed quantity of vitamin D milk available, the latter expressed in 1000s of quarts daily. For both specifications, all the estimates of the DDD parameter remain positive. However, the parameter is significant at the 10 percent level for number of dairies only for the age cutoffs of zero (fetal) and 1. The marginal impact of an additional dairy on schooling attendance for blacks in low sunlight cities ranges from 2.6 percentage points (age 0 cutoff) to 1.9 percentage points (age 3 cutoff).

For quantity, the parameter does not reach conventional significance levels for any age cutoff. This should not be surprising, as the poor quality of quantity data introduces sizeable amounts of measurement error. The marginal impact of 1000 additional quarts daily of vitamin D milk is approximately 2 percentage points for all age cutoffs. However, the comparability of the results for quantity and number of dairies is largely an artifact of the high degree of imputation in the former.

During the 1930s, Vitamin D milk often sold at a premium of usually one or two cents per quart. The cost differential suggests that the distribution of the benefits from vitamin D milk's emergence may have skewed towards wealthier families. Unfortunately, the IPUMS samples from this time period do not have income data that permit the estimation of a precise income gradient. Instead, I split the sample based on the Duncan socioeconomic score (SEI) of father's occupation,

which proxies the socioeconomic status of the child's family.[26] I then estimate equation (1) for those above and below the mean score using the baseline specification that generated the results in table 4.

Table 8 displays the results of splitting the sample based on the implied status of father's occupation. The estimates of the DDD parameter imply that the positive effect of vitamin D milk exposure does indeed appear to be driven by better-off families. In both magnitude and precision, the DDD estimate for both the 1930-40 IPUMS sample and the expanded 1920-50 IPUMS sample reveal positive and significant effects for the sample above the mean SEI. The below-mean SEI sample estimates are much smaller—significantly so for the age cutoffs zero and one—and less precisely estimated. While the lack of income data precludes a more formal test, the results in table 8 are consistent with the idea that better-off African American children from the North captured relatively higher benefits from vitamin D milk's introduction than their less wealthy peers.

B. Contemporaneous Labor Market Outcomes

One potential threat to the identification strategy is that unobserved shifts in the labor market for Northern blacks influenced the potential profitably of vitamin D milk and hence the timing of the milk's availability. This argument supposes that a positive shock to the labor market for Northern blacks may have caused the attractiveness of schooling to increase while concomitantly increasing consumer demand—through simple income effects, for example—for vitamin D milk.[27]

To determine the validity of this explanation, I estimate a version of equation (1) on working aged males using the 1930 and 1940 census. In these regressions, the treatment variable D_{cjt} is equal to one for all individuals in the 1940 census if vitamin D milk became available in their city prior to the census year, and zero otherwise. While ideal proxies for labor market conditions, such as individual wage data, do not exist, the IPUMS files contain constructed variables on occupational prestige. I use two of these variables, occupational income score (*occinc*), which assigns an approximate income to the respondent's stated occupation, and the percentage of people in the respondent's occupation that completed a year of college, (*edscor*).[28] These variables give an indication of the availability of higher paying jobs in the city. I also include unemployment as an outcome variable. If the pattern of vitamin D milk's influence on these labor market outcomes for adults mimic the results from the schooling regressions, it is possible that the latter are being

[26] Children with unemployed or missing fathers have a score of zero.
[27] Note that this objection fails to explain the timing of vitamin D milk availability in cities where regulatory hurdles were the primary determinant of vitamin D milk's availability (e.g. Baltimore).
[28] The unemployed receive a score of zero.

driven by unobserved changes simultaneously influencing economic conditions and vitamin D milk's availability.

The results in table 9 do not indicate that vitamin D milk's availability influenced contemporaneous labor market outcomes. The signs of the DDD parameter—negative for the occupation scores and positive for unemployment—are the opposite of what would be expected if positive labor market changes hastened vitamin D milk availability's availability and raised schooling simultaneously. Furthermore, none of the estimated parameters are significant at conventional levels. While better measures of labor market conditions, such as wage income, may provide a better test of this hypothesis, they are unavailable. In particular, the occupation-based measures used here would not account for strictly intraoccupational shocks. However, it is unlikely that such shocks would not at least carry over to unemployment, which does not appear to be negatively influenced by vitamin D milk (last row of table 9). The available evidence thus gives no indication that the schooling results suffer from this simultaneity weakness.

VII. Conclusion

This paper investigated the effect of a micronutrient intervention on a measure of human capital investment. I look specifically at the impact of vitamin D milk's availability during the 1930s on the school attendance of children. Using the fact that African American children in low sunlight cities were at a higher risk of suffering from vitamin D deficiency, the evidence presented here shows that vitamin D milk's introduction positively impacted school attendance. The overall magnitude and precision of the effect is fairly robust to different specifications of the estimating equations and the composition of the sample.

While some evidence indicates that fetal exposure to vitamin D milk has the largest influence on subsequent school attendance, estimation using a wider sample cannot distinguish a critical time period. The range of estimates, however, does emphasize the importance of intervention in the earlier stages of childhood.

This study contributes to both the historical questions surrounding the effectiveness of food fortification programs and the contemporary discussion of the role of nutritional interventions in improving health outcomes. For the former, I have compiled an original dataset from historical sources that describes, for the first time, the spatial and temporal rollout of vitamin D milk. Further, the data analysis shows that blacks in low sunlight cities with access to vitamin D milk demonstrated educational gains in the form of higher rates of school attendance. That finding implicates a potentially important but overlooked role for nutritional differences in explaining historical racial education gaps.

In terms of the debate over the causal pathways between nutrition and education, this study demonstrates that remedying micronutrient deficiencies can yield increases in human capital investment in the form of schooling. Further, these effects can be detected from a large scale fortification program. In large swaths of the developing world, where micronutrient deficiencies remain at high levels, development economists and policy makers should not overlook the importance of investigating the addition of micronutrients to the general food supply. Indeed, the development economics literature has paid relatively scant attention to large-scale food fortification initiatives.

Despite the seemingly positive effects of vitamin D milk's introduction in the United States, these results are not necessarily generalizable to all places where segments of the population suffer from a micronutrient deficiency. Vitamin D milk appeared during a time when alternative remedies were unpleasant and the deficiency was widespread. On the production side, producers both had the will, due to very competitive market conditions in the milk industry, and the way, since the technology had developed fortification methods that were cost effective for large and small dairies.

This study should, at the very least, draw the attention of policy makers to regulations and market conditions that impede the adoption of food fortification in places with obvious needs. Baltimore's regulations on cap markings, for example, likely impaired the health and educational outcomes of African American children during this era, and potentially impacted long-term outcomes for its residents. A follow up study that more closely investigates the determinants of vitamin D milk adoption should serve to clarify specific areas of focus.

Future research will also attempt to focus more closely on the mechanisms underlying vitamin D milk's impact on schooling. In particular, follow up studies will focus on health outcomes. The large, recent literature on vitamin D has relied mainly on observational studies and the identification of plausibly exogenous increases in vitamin D status due to fortified milk's rollout can contribute to the debate concerning vitamin D's importance to health status. Further, by clarifying the particular areas of health impacted by vitamin D milk's introduction, this work can identify priority areas nutritional interventions aimed at improving human capital investment.

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Table 1: Control Variables

	Level	Source
Gender	Individual	IPUMS Census
Race	Individual	IPUMS Census
Age	Individual	IPUMS Census
Socioeconomic score of Father's Occupation (Duncan)	Individual	IPUMS Census
Per Capita Federal New Deal Relief Aid*	City; County	Fishback, Haines and Kantor (2007)
Population	County	ICPSR 3
Real Manufacturing Wages (total)†	County	ICPSR 3/Lleras-Muney (2001)
Educational Expenditures per Capita†	County	Lleras-Muney (2001)
Crop Value	County	ICPSR 3
Doctors per Capita†	City	Lleras-Muney (2001)

*Variable only available in limited number of cities.

†Variable only available for 1930 and 1940 sample

Table 2: Summary Statistics

		Vitamin D Milk Introduced Before 1936		Vitamin D Milk Introduced After 1936	
	Whole Sample	Below Average Sunlight	Above Average Sunlight	Below Average Sunlight	Above Average Sunlight
Cities	99	35	21	20	23
Change in Manufacturing Wages (1000s), 1930-40	-15309	-21097	-316	-12227	-2319
	(28775)	(33663)	(4641)	(13855)	(2485)
Change in Crop Values (1000s), 1930-40	-1034	256	-7385	-325	-1104
	(5746)	(691)	(13226)	(678)	(2760)
White School Attendance, 1930	0.877	0.882	0.871	0.875	0.853
	(0.033)	(0.025)	(0.051)	(0.028)	(0.039)
Black School Attendance, 1930	0.856	0.866	0.847	0.86	0.798
	(0.086)	(0.084)	(0.087)	(0.089)	(0.066)
Change in Overall School Attendance, 1930-40	0.016	0.014	0.023	0.002	0.035
	(0.025)	(0.019)	(0.034)	(0.033)	(0.024)
Change in White School Attendance, 1930-40	0.014	0.013	0.021	0.002	0.031
	(0.026)	(0.018)	(0.033)	(0.033)	(0.032)
Change in Black School Attendance, 1930-40	0.019	0.02	0.032	-0.019	0.045
	(0.083)	(0.068)	(0.098)	(0.128)	(0.061)

Geographic unit is the city. Sample is non-migrant, native born children ages five to fourteen. Standard deviation in parentheses. See table 1 for data sources for variables.

Table 3: The Rollout of Vitamin D Milk, 1933-1935

	Jan 31, 1933	June 1, 1933	Jan 1, 1934	Jan 1, 1935
Number of Dairies Producing Vitex Milk	1	29	110	210
Number of Dairies producing WARF's Irradiated Milk	1	3	35	125

Source: University of Wisconsin Archives Henry Steenbock Collection Trustee Report 29, 2/30/1934; Milk Report 8, 4/20/1935; "Producing Zucker Vitamin D Milk", Milk Plant Monthly (1933); *Journal of the American Medical Association* volumes 100-105.

Table 4: The Effect of Vitamin D Milk on Schooling Using a Dummy Variable for Treatment

Age Cutoff	0	1	3	5
Treatment Dummy X Black X Insolation Dummy	0.112**	0.0784**	0.0518*	0.0234
	(0.0493)	(0.0385)	(0.0268)	(0.0209)
Treatment Dummy X Insolation Dummy	0.00910	0.0139	0.0117	0.0125
	(0.0457)	(0.0335)	(0.0184)	(0.0128)
Black X Insolation Dummy	0.0189*	0.0179*	0.0171	0.0201*
	(0.00968)	(0.0100)	(0.0104)	(0.0108)
Treatment Dummy X Black	-0.0998**	-0.0589	-0.0367	-0.0151
	(0.0451)	(0.0357)	(0.0245)	(0.0189)
Treatment Dummy	0.0347	0.0117	0.00519	-0.0197
	(0.0427)	(0.0316)	(0.0173)	(0.0124)
Black	-0.0192**	-0.0198**	-0.0198**	-0.0220**
	(0.00789)	(0.00813)	(0.00855)	(0.00885)
Observations	109,093	109,093	109,093	109,093
Sample	1930-40	1930-40	1930-40	1930-40
Geographic Unit	City	City	City	City
Controls	All	All	All	All
Birth Year Cohorts	Yes	Yes	Yes	Yes

Dependent variable is school attendance. Robust standard errors clustered at city level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Columns indicate age by which child must be exposed to vitamin D milk to be considered treated. Treatment is a dummy variable equal to one if vitamin D milk available in child's city prior to age cutoff. All regressions include controls for age, sex, Duncan's socioeconomic score of father's occupation, total crop values, manufacturing wages, population and per capita educational expenditures of county where city is located, doctors per capita in city, and fixed effects for city, birth year cohort and time. Sample consists of all native born children aged five to fourteen in the 1930 and 1940 IPUMS that did not migrate in the five years previous to the census and reside in cities consistently identified by IPUMS in 1930 and 1940.

Table 5: Sensitivity Analysis of DDD Parameter for the Effect of Vitamin D Milk on Schooling Using a Dummy Variable for Treatment

	Age Cutoff	0	1	3	5	Observations
A	Individual Controls Only	0.153***	0.109***	0.0707***	0.0408**	111,229
		(0.0431)	(0.0353)	(0.0243)	(0.0186)	
B	Individual Controls and Cohort Fixed Effects	0.115**	0.0809**	0.0522**	0.0243	111,229
		(0.0462)	(0.0361)	(0.0251)	(0.0199)	
C	Individual Controls, Cohort FE and New Deal Relief	0.103**	0.0745*	0.0503*	0.0199	97,483
		(0.0510)	(0.0404)	(0.0278)	(0.0227)	
D	All Variables	0.102*	0.0773**	0.0496*	0.0195	97,197
		(0.0515)	(0.0388)	(0.0278)	(0.0226)	

Dependent variable is school attendance. Robust standard errors clustered at city level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Table reports estimates of the DDD parameter only. Rows and columns refer to DDD estimates for separate regressions. Columns indicate age by which child must be exposed to vitamin D milk to be considered treated. Vitamin D milk exposure specified as a dummy variable equal to one if vitamin D milk is available in child's city prior to age cutoff. Treatment group consists of African American children exposed to vitamin D milk by age cutoff and living and cities with below average solar radiation. All regressions include controls for age, sex, Duncan's socioeconomic score of father's occupation, Difference-in-Difference interaction terms shown in Table 4, and fixed effects for time and city. In Row A, no additional controls are used. In Row B, fixed effects for birth year are added. In Row C, per-capita federal new deal relief aid is added to the row B controls. In Row D, total crop values, manufacturing wages, population and per capita educational expenditures of county where city is located, and doctors per capita in city, is added to the row C controls. Sample consists of all native born children aged five to fourteen in the 1930 and 1940 IPUMS that did not migrate in the five years previous to the census and reside in cities consistently identified by IPUMS in 1930 and 1940. Dependent variable in all regressions is school attendance.

Table 6: DDD Parameter Estimates for the Effect of Vitamin D Milk on Schooling Under Different Samples Using a Dummy Variable for Treatment

	Census Years	Geographic Unit	DDD Parameter Estimate by Age Cutoff				Birth year Cohorts	Regional Controls	City-Year Trend
			0	1	3	5			
A	1930-40	SEA	0.0920**	0.0686**	0.0457**	0.00238	No	No	-
			(0.0449)	(0.0341)	(0.0198)	(0.0149)			
B	1930-40	SEA	0.0497	0.0331	0.0274	0.0248	Yes	Yes	-
			(0.0363)	(0.0288)	(0.0190)	(0.0162)			
C	1920-50	City	0.0495*	0.0814**	0.0574**	0.0349*	Yes	Yes	No
			(0.0254)	(0.0370)	(0.0255)	(0.0206)			
D	1920-50	City	0.0553**	0.0468*	0.0405**	0.0265*	Yes	-	Yes
			(0.0266)	(0.0237)	(0.0178)	(0.0158)			

Dependent variable is school attendance. Robust standard errors clustered at city level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Table reports estimates of the DDD parameter only. Rows and columns refer to DDD estimates for separate regressions. Columns indicate age by which child must be exposed to vitamin D milk to be considered treated. Vitamin D milk exposure specified as a dummy variable equal to one if vitamin D milk is available in child's city prior to age cutoff. Treatment group consists of African American children exposed to vitamin D milk by age cutoff and living and cities with below average solar radiation. All regressions include controls for age, sex, Duncan's socioeconomic score of father's occupation, Difference-in-Difference interaction terms shown in Table 4, and fixed effects for time and geographic unit. In Row A, no additional controls are used, and geographic unit is SEA. In Row B, fixed effects for birth year and regional controls (total crop values, manufacturing wages, population, per capita educational expenditures and doctors per capita) aggregated to the SEA level. In Row C, geographic unit is the city. Controls in Row C include fixed effects for birth year, population and total SEA crop value. In Row D, controls are identical to row C, and a cityXyear time trend is added. Sample for rows A and B consists of all native born children aged five to fourteen that did not migrate in the five years previous to the census. Sample in rows C and D consists of all native born children aged five to fourteen in cities consistently identified by the census between 1920 and 1950.

Table 7: DDD Parameter Estimates for the Effect of Vitamin D Milk on Schooling Under Different Specifications of the Treatment Variable

Sample	1930-40	1920-50	1930-40	1920-50	1930-40	1920-50	1930-40	1920-50
Age Cutoff	0	0	1	1	3	3	5	5
Number of Dairies	0.0234*	0.0244*	0.0109	0.0126*	0.00461	0.00496	0.00291	0.00317*
	(0.0137)	(0.0140)	(0.00750)	(0.00736)	(0.00362)	(0.00361)	(0.00192)	(0.00189)
Imputed Quantity (1000s quarts daily)	0.00203	0.00179	0.00285	0.00342	0.00104	0.00123	0.00167	0.00167
	(0.00329)	(0.00358)	(0.00345)	(0.00344)	(0.00194)	(0.00191)	(0.00111)	(0.00119)
City-Year Trend	-	Yes	-	Yes	-	Yes	-	Yes

Dependent variable is school attendance. Robust standard errors clustered at city level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Table reports estimates of the DDD parameter for different specifications of the vitamin D milk treatment variable. Rows and columns refer to DDD estimates for separate regressions. Columns indicate age by which child must be exposed to vitamin D milk to be considered treated. Vitamin D milk exposure specified as number of dairies or quantity of milk available in child's city prior to age cutoff. Treatment group consists of African American children exposed to vitamin D milk by age cutoff and living and cities with below average solar radiation. Geographic unit in all regressions is the city. All regressions include controls for age, sex, Duncan's socioeconomic score of father's occupation, Difference-in-Difference interaction terms shown in Table 4, and fixed effects for time and geographic unit. For all regressions using the 1930-1940 sample, controls include total crop values, manufacturing wages, population and per capita educational expenditures of county where city is located, doctors per capita in city, and fixed effects for city, birth year and time. For regressions using the 1920-50 sample, controls consist of fixed effects for birth year, population, total crop value and a cityXyear time trend. The 1930-40 sample consists of all native born children aged five to fourteen that did not migrate in the five years previous to the census in cities consistently identified by the census in those year. The 1920-50 sample consists of all native born children aged five to fourteen in cities consistently identified by the census between 1920 and 1950.

Table 8: Estimate of the DDD Parameter by Father's Occupational Status Using a Dummy Variable for Treatment

Census Years		DDD Parameter Estimate				Observations
	Age Cutoff	0	1	3	5	
1930-40	All	0.112**	0.0784**	0.0518*	0.0234	109,093
		(0.0493)	(0.0385)	(0.0268)	(0.0209)	
	SEI Above Mean	0.202***	0.137***	0.0742*	0.0676*	68,367
		(0.0613)	(0.0490)	(0.0399)	(0.0354)	
	SEI Below Mean	0.0499	0.0335	0.0325	0.00858	40,726
		(0.0535)	(0.0433)	(0.0275)	(0.0216)	
1920-50	All	0.116**	0.0764*	0.0529**	0.0307	148,449
		(0.0495)	(0.0385)	(0.0257)	(0.0198)	
	SEI Above Mean	0.207***	0.126**	0.0583	0.0409	94,763
		(0.0679)	(0.0487)	(0.0403)	(0.0350)	
	SEI Below Mean	0.0561	0.0333	0.0399	0.0274	53,686
		(0.0574)	(0.0459)	(0.0283)	(0.0207)	

Dependent variable is school attendance. SEI refers to Duncan's socioeconomic score of father's occupation. Geographic unit is the city in all regressions. Robust standard errors clustered at city level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Table reports estimates of the DDD parameter only for regressions using samples above or below mean SEI. Columns indicate age by which child must be exposed to vitamin D milk to be considered treated. Vitamin D milk exposure specified as a dummy variable equal to one if vitamin D milk is available in child's city prior to age cutoff. Treatment group consists of African American children exposed to vitamin D milk by age cutoff and living and cities with below average solar radiation. All regressions include controls for age, sex, Duncan's socioeconomic score of father's occupation, Difference-in-Difference interaction terms shown in Table 4, and fixed effects for time and geographic unit. In the 1930-40 sample, controls and sample are identical to table 4 (see notes below table). For the 1920-50 sample, controls and sample are identical to Row D of table 6.

Table 9: Contemporaneous Adult Labor Market Outcomes Using a Dummy Variable for Treatment

	DDD Parameter
Dependent Variable	
Occupational Income Score	-0.452
	(0.447)
Occupational Education Score	-38.77
	(49.26)
Unemployment	0.0300
	(0.0211)

Robust standard errors clustered at city level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Table reports estimates of the DDD parameter only. Each row represents a different regression. Geographic unit in all regressions is the city. All regressions include controls for age, sex, Difference-in-Difference interaction terms shown in Table 4, and fixed effects for time, birth year and geographic unit. Sample consists of all males aged 18 to 49 in the 1930 and 1940 IPUMS that did not migrate in the five years previous to the census and reside in cities consistently identified by IPUMS in 1930 and 1940. Unemployment regression excludes those reported not to be in the labor force.

Figure 1

Dairies Distributing Vitamin D Milk, 1933

County Level Data

Figure 2

Dairies Distributing Vitamin D Milk, 1935

County Level Data

Figure 3

Dairies Distributing Vitamin D Milk, 1939

County Level Data