SECOND TRIMESTER SUNLIGHT AND ASTHMA

Evidence from Two Independent Studies

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ABSTRACT
One in 12 Americans suffers from asthma, and its annual costs are estimated to exceed $50 billion. Yet the root causes of the disease remain unknown. A recent hypothesis posits that maternal vitamin D levels during pregnancy affect the probability the fetus later develops asthma. Employing two large-scale studies, we test this hypothesis using a natural experiment afforded by historical variation in sunlight, a major source of vitamin D. Specifically, holding the birth location and month fixed, we see how exogenous within-location variation in sunlight across birth years affects the probability of asthma onset. We show that this measurement of sunlight correlates with actual exposure, and consistent with preexisting results from the fetal development literature, we find substantial and highly significant evidence in both data sets that increased sunlight during the second trimester lowers the subsequent probability of asthma. Our results suggest policies designed to augment vitamin D levels in pregnant women, the large majority of whom are vitamin D insufficient, could be very cost effective and yield a substantial surplus.

KEYWORDS: asthma, sunlight, vitamin D, natural experiment
JEL CLASSIFICATION: I12, I18, J13

I. Introduction

The worldwide incidence of asthma imposes staggering physical and financial costs. Asthma afflicts more than 300 million individuals worldwide and kills more than 250,000 people annually (Cruz et al. 2007). The resulting cost—measuring both direct treatment expenditures and indirect productivity loss—is enormous, estimated at about $56 billion annually in the United States alone (Barnett and Nurmagambetov 2011). Simultaneously, asthma's root causes appear varied and remain poorly understood. A recent hypothesis with growing support speculates that low maternal vitamin D levels during pregnancy increase the probability that the unborn child later develops asthma (Weiss and Litonjua 2011). If validated, this hypothesis presents a low-cost way to reduce asthma incidence,

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resulting in both improved quality of life for millions of individuals and reductions in net health-care spending.

To assess this hypothesis, we examine the impact of local weather conditions while in utero on the probability of developing asthma later in life. Specifically, our approach holds fixed the location and month of birth, and then analyzes how exogenous within-location variation in sunlight levels across birth years affects the probability of asthma onset. Though we cannot observe maternal vitamin D levels directly, we can capitalize on the well-documented connection between vitamin D levels and sunlight. Namely, Americans obtain over 90 percent of their vitamin D from sunlight exposure (Holick 2004), and we provide evidence that the sunlight variation we are exploiting correlates with actual exposure, but not with other factors that might affect asthma incidence. Our reduced-form analysis cannot rule out every alternative pathway through which sunlight might be affecting asthma incidence. However, for reasons we discuss in greater depth below, we believe that the direct vitamin D channel is a likely path.

Employing two independent data sets, we find highly significant evidence that an increase in sunlight in an individual's location during the second trimester in utero reduces his/her probability of becoming asthmatic. This result is consistent with preexisting research from the fetal development literature, which has found substantial evidence suggesting vitamin D is particularly important during this period for asthma pathogenesis. Further consistent with past research on vitamin D and asthma, we find evidence of differential effects by race—namely, the effects of sunlight seem larger for blacks, though within each study the differences are not significant.

We report the results from our two data sets separately. Our first data set (Study I) draws on individual-level data on the state, month, and year of birth for more than 260,000 Americans. We find that doubling the amount of sunshine[1] during the second trimester lowers the probability of an asthma diagnosis by 1.15 percentage points, which at a mean incidence of 11.49 percent represents a 10.0 percent reduction. In our second data set (Study II), we look for an effect of sunlight on a separate, more cost-relevant margin—hospital discharges from asthma—on the county level using data on more than 2.1 million births aggregated into about 3,000 birth month–county cohorts. Here we find that doubling the amount of sunlight during the second trimester reduces the per capita rate of asthma emergencies by 2.21 percentage points, which is a 21.3 percent reduction at the mean.[2] Both sets of results pass several robustness checks detailed in the Online Appendix (see http://www.mitpressjournals.org/doi/suppl/10.1162/ajhe_a_00073).

Current estimates are that nearly three out of every four adults are either vitamin D deficient or insufficient (Ginde, Liu, and Camargo 2009).[3] Among pregnant women, multiple smaller-scale studies have reported rates that are if anything slightly higher (Bodnar et al. 2007; Holmes et al. 2009; Johnson et al. 2011). Our results suggest that these numbers

1 This is about the difference between the average summer in Nevada and that in New Hampshire.

2 This estimate is significantly different from the previous one, but this is understandable given that the data sets look at two different margins. We report the results here in terms of doubling sunlight for sake of comparison, but we describe each result in more detail in Section IV.

3 Deficiency is defined as less than 10 ng/ml blood; insufficiency is 10–29 ng/ml.

may be more costly than previously known, and that policies designed to boost maternal vitamin D levels during the second trimester—through either increased usage of (or adherence to) vitamin D supplements or increased sun exposure—may greatly enhance welfare, on the order of billions of dollars in surplus gained.[4]

The rest of the paper is organized as follows. Section II provides the background from both the medical and the economics literature. Section III describes our two data sets in greater detail. Section IV presents our results and Section V concludes.

II. Background

Our paper builds off of two separate literatures. Thus, we provide the relevant background for the two sequentially before discussing the connection between sunlight and vitamin D in more detail.

A. PRIOR MEDICAL LITERATURE

Many studies in both animals and humans point to a role for maternal vitamin D levels during pregnancy in asthma pathogenesis.[5,6] One stream of this literature has tried to find evidence of a clinical effect. Multiple studies on human subjects in different parts of the world have found associations between low maternal vitamin D levels during pregnancy and asthma incidence in the child (Camargo et al. 2007; Devereux et al. 2007; Erkkola et al. 2009; Miyake et al. 2010; Belderbos et al. 2011). By design, however, these studies can only report associations, and other associational studies report either no correlation or a positive one between low maternal vitamin D levels and later asthma incidence (e.g., Gale et al. 2007). Separately, in a controlled study on animals, rats born to mothers who were experimentally deprived of vitamin D exhibit several lung abnormalities similar to those found in human asthmatics (Gaultier et al. 1984). Within this literature, how much circulating maternal vitamin D in humans is required for healthy fetal lungs and exactly how lung health varies with different vitamin D levels remain open questions.

A second stream of literature has been more developmentally focused and tries to determine when in a human pregnancy maternal vitamin D levels are most important. Human lung development occurs in five stages: embryonic (roughly 4–7 weeks post conception), pseudoglandular (7–17 weeks), canalicular (17–26 weeks), saccular (27–36 weeks), and alveolar (36 weeks to about 2 years old) (Burri 1984; Weiss and Litonjua 2011).

4 A year's worth of vitamin D supplements typically costs less than $20 online. In addition, exposure to as little as ten minutes of direct sunlight a day—far less time than it takes to sunburn—is adequate for most individuals to obtain their daily recommended dosage (Holick 2007). In contrast, the average asthma-related hospitalization lasts three days and can cost between $12,000 and $24,000 (Milet et al. 2007).

5 There have also been studies recently that look at treating current asthmatics with vitamin D (e.g., Castro et al. 2014). This is a related, but distinct, hypothesis from those we consider in this paper.

6 Indeed, the evidence is sufficiently strong that two randomized-controlled trials have been started to look at maternal vitamin D supplementation during pregnancy and asthma outcomes. These studies are the Maternal Vitamin D Supplementation to Prevent Childhood Asthma (VDAART) and Vitamin D Supplementation during Pregnancy for Prevention of Asthma in Childhood (ABCvitaminD); their ClinicalTrials.gov identifiers are NCT00920621 and NCT00856947, respectively.

Several studies find suggestive evidence that maternal vitamin D levels could be important during part of the pseudoglandular and canalicular stages.[7,8] Not only is vitamin D present in fetal lung tissue during much of this period (Brun, Thomasset, and Mathieu 1987), but it is also known to affect either the growth or the gene expression of several types of cells that are active in the lungs at this time (Lunghi et al. 1995; Stio et al. 1997; Bossé, Maghni, and Hudson 2007). At week 11 of the pregnancy, many vitamin D–related genes that are known to be involved in lung development and differentially expressed in asthmatics are suddenly activated (Kho et al. 2013). These studies illuminate a pathway through which vitamin D deficiency could lead to the abnormal airway development associated with asthma (Weiss and Litonjua 2011). Based on these results, we look for evidence within weeks 11–26 post conception, a period that overlaps relatively closely with the second trimester.

B. PRIOR ECONOMIC LITERATURE

A growing literature within economics employs economic methods to address questions in health. Economists' interests in welfare and health costs, combined with the amenability of many health questions to econometric analysis (especially when randomized trials are hard to conduct because of ethical or other constraints), helps to explain this increase. For example, recent economics papers have focused on and provided deep insights into HIV prevalence, chronic disease, long-term mortality, infant health, and even respiratory ailments (respectively, Oster 2005; Fogel 2004; Costa 2003; Currie et al. 2013; Schlenker and Walker 2011).

Our paper also relates to the literature within both economics and medicine on the long-run impacts of events early in life. Much of that research shows that outcomes of considerable interest—such as educational achievement, income, and many health conditions—are strongly influenced by events early in life, including nutrition (e.g., Grossman 1972; Bhattacharya and Currie 2001; Banerjee et al. 2010; Clay and Troesken 2006; Heckman 2006; Bleakley 2007; Fletcher, Green, and Neidell 2010; Bharadwaj, Løken, and Neilson 2013) or in utero (e.g., Black, Devereux, and Salvanes 2007; Almond, Edlund, and Palme 2009; Black et al. 2014). More closely relevant to this paper are the negative effects of temperature (Barreca, Deschenes, and Guldi 2015) and hurricanes (Zahran et al. 2014) on birth outcomes.[9]

Our paper relates to several prior economic analyses that employ weather as a proxy variable for a variable of interest. For example, Maccini and Yang (2009) report on

7 It is worth emphasizing that the fetal supply of vitamin D is completely determined by the mother's (e.g., Heaney et al. 2003), and so statements about vitamin D in fetal lungs during this period reflect the mother's supply.

8 We note that other studies have also reported results consistent with the third trimester (e.g., Glasgow and Thomas 1977; Phokela et al. 2005). We do not find evidence in support of this, but note that there may well be an effect during that period that is not large enough to observe in our data.

9 From a theoretical perspective, Almond and Currie (2011) adapt a model from Heckman (2007) to show how the empirical evidence of the "fetal origins hypothesis" can fit within a standard economics modeling framework. Namely, using a constant elasticity of substitution production function with health investments at different ages as inputs, they show that complementarities between health shocks and later investments could amplify small initial differences across individuals.

several positive long-term outcomes for women who were exposed to greater rainfall early in life, where the presumed causal channel operates through increased agricultural production and hence income. Jensen (2000) uses rainfall as a proxy for agricultural conditions to study investments in children, and Oster (2004) uses temperature as a proxy for economic conditions to study the prevalence of witchcraft trials. Similar to these studies and many of the aforementioned fetal origins papers, we are unable to observe our variable of interest (maternal vitamin D levels) directly.[10] Thus we estimate our model using sunlight instead, which we provide evidence is correlated. Clearly, direct measurement would be ideal, but our approach still enables us to provide evidence of the hypothesized effect.

Finally, the paper that relates most closely to ours is Trudeau, Conway, and Menclova (2016), which explores the consequences of relative sunlight levels on birth weight. These authors use a similar (though actually less restrictive) specification in their analysis, and find that increased sunlight has a positive but diminishing effect on birth weight for black mothers and a negative effect for white mothers. The effect sizes they observe are similar in magnitude to those found for maternal smoking, participation in SNAP and WIC, and environmental factors such as air pollution. Hence, whereas we find no negative effects of sunlight on our outcome variables, Trudeau, Conway, and Menclova (2016) point to a possible risk (for whites) of increased maternal sunlight exposure.

C. SUNLIGHT AND VITAMIN D

As mentioned earlier, humans obtain the vast majority of their vitamin D supply from sunlight (in fact, a nickname for vitamin D is "the sunshine vitamin"). Though there are many dietary sources of vitamin D—such as several fish species or a range of fortified dairy products—it would take about 30 glasses of vitamin D–fortified milk to produce the same amount of vitamin D as one receives from an average of 5–10 minutes of direct sunlight exposure (Holick 2007).[11] A common definition of a sufficient vitamin D serum level is greater than or equal to 30 ng/ml. We note that there may be adverse effects if an individual's vitamin D level exceeds 150 ng/ml, though importantly sunlight exposure cannot produce such toxicities—the body naturally shuts off vitamin D synthesis long before this amount (Holick 2007).[12]

Further evidence on the role of sunlight in vitamin D production is provided by seasonal variation. In the Northern hemisphere vitamin D levels are known to follow strong

10 A few data sets do contain information on vitamin D levels, the largest of which is the NHANES (National Health and Nutrition Examination Survey), but even that data set does not have enough geographical and seasonal variation for our purposes.

11 There is substantial seasonal and latitudinal variation in the number of minutes of direct sun exposure necessary to produce a daily dose of vitamin D. For example, for darker-skinned individuals in the winter in northern areas (e.g., Minneapolis) no amount of sunlight would be sufficient. (See http://zardoz.nilu.no/~olaeng/fastrt/VitD-ez_quartMEDandMED_v2.html for more detailed calculations.) That said, employing fixed effects for the birth location-season, as in this paper's regressions, controls for these differences in average sunlight levels.

12 For this reason, we suspect the negative effect observed in Trudeau, Conway, and Menclova (2016) is due to a correlate of sunlight other than vitamin D.

TABLE 1. Overview of our two studies

	Study I	Study II
Unit of observation	Individual	Cohort
Level of observation	Each individual's state, month, and year of birth	Each cohort's county, birth month, and year of birth
Weather data	Hours of sunlight, at the state, month, and year level	Average intensity of sunlight at the county, month, and year level
Outcome variable	The individual's self-reported asthma condition	Per capita number of emergency department discharges for asthma for that cohort
Years	1914–87	1999–2009
N	264,701 individuals	2.1 million people grouped into 3,036 birth month–county–year cohorts

seasonal cycles of being highest in the summer and lowest in the winter as the number of sunlight hours varies (with a reverse pattern in the Southern hemisphere; Chen 1999). Isolated data from small-scale studies have similarly found seasonal fluctuations in the vitamin D level of both pregnant women and their newborns (Disanto et al. 2013; Bodnar et al. 2007).[13] Finally, on a separate but related note, rickets (which is caused by vitamin D deficiency) was documented more than a century ago to follow similar seasonal cycles (Kassowitz 1897). For identification purposes, the variation in sunlight we exploit is within-location rather than seasonal, but we include this discussion to simply convey a sense of the tight connection between sunlight and vitamin D.

III. Data

This paper presents two separate studies. Table 1 provides an overview of each. The text then elaborates on the description of each data set. We employ two analyses, first to see whether our main effect replicates, and second to capitalize on the distinct advantages each possesses. Study I has the advantage of having individual-level data, with sunlight aggregated by state, over a national sample. Study II, by contrast, operates at the more granular county level for potential sunlight exposure, uses more up-to-date data in a narrower time frame, and has a more definitive indication of asthma from emergency department discharges. We note that there is no data overlap (with respect to either time frame or data sources) between the two studies. This creates a highly independent replication, albeit employing the same identification strategy.

13 There is some suggestive evidence that the actual temperature of the human skin may affect vitamin D synthesis, and so other factors such as humidity may indirectly affect vitamin D levels (Tsiaras and Weinstock 2011). We leave future exploration of this possibility and how it may affect asthma incidence in humans to future work.

A. STUDY I

Individual-level data on the state and month of birth (as well as health status and controls) for more than 260,000 Americans were obtained from the 1997–2008 National Health Interview Survey (NHIS). These data were merged with historical weather data from the National Oceanic and Atmospheric Administration (NOAA).

An adult from the NHIS data was coded as asthmatic if he/she responded affirmatively to the question, "Have you EVER been told by a doctor or other health professional that you had asthma?" and similarly for a child below 18 years old if a knowledgeable adult in the household answered affirmatively to an equivalent question about the child. Negative responses were coded as a zero; all other responses were omitted (e.g., refused to answer, not ascertained, don't know).[14] The publicly available NHIS data provide information on each individual's year and month of birth, as well as demographic data such as gender and ethnicity. The restricted-use data we used included each individual's state of birth, which was the lowest geographic level available. Thus, for every individual in our sample we knew his/her state, month, and year of birth.

Historical sunlight data were collected from 240 NOAA weather stations located around the country. The data range from 1891 to 1987, though some stations do not cover the entire time period.[15] Each station measured the total number of hours of sunlight at its location each month. For example, we can see in our data that Asheville, North Carolina, had 188 hours of sunlight in January 1977. For states with more than one weather station reporting in any month, the average within-state correlation in sunlight hours was quite high (0.86), and so monthly data were simply averaged in such cases. In Section 2 of the Online Appendix, we provide figures to demonstrate the variation in sunlight at the level of identification (Figures A.1 and A.2).

B. STUDY II

Our second data set looks at birth month–county–year cohorts, not individual data. Sunlight data came from the National Solar Radiation Database (NSRD) at the US Department of Energy, asthma hospital discharge data from the Healthcare Cost and Utilization Project (HCUP), and birth records from the Centers for Disease Control and Prevention (CDC) Vital Statistics.

The sunlight data show for each 10 kilometer × 10 kilometer grid cell a measure of sunlight intensity at hourly intervals (though for our analysis we use the monthly average) for 1998–2009.[16] While available for fewer years, this measure is superior to that of Study

14 The NHIS relies on self-reported asthma status. Our second study measures asthma using hospital discharge data.

15 The average duration of a weather station was just above 60 years, and the vast majority were reporting over continuous stretches.

16 For each 10 kilometer × 10 kilometer grid cell, Geostationary Operational Environmental Satellites measure solar radiation reflected back from any cloud cover. Ground radiation can then be calculated from subtracting reflected radiation from total radiation, presumably using latitude and day of the year to calculate total radiation (Perez et al. 2002). These ground radiation estimates are then integrated over an hour, since the most granular data available are at the hour-day-month-year level. Any gaps in the satellite data

I since it captures both more hours of sunlight and more or less sunlight within the same number of hours.

To map the NSRD to counties, since there is minimal sunlight variation within a county for a particular month and year, we matched the coordinates of the population-weighted county centroid to the 10 kilometer × 10 kilometer gridded cell that contains it. As for Study I, Section 2 of the Online Appendix contains visual depictions of the variation in sunlight used in this study at the level of identification (Figures A.3 and A.4).

We were able to obtain hospital discharge data from the HCUP for two states: New Jersey and Arizona. Specifically, we used the State Emergency Department Database, which records visits to the emergency room that did not result in an admission.[17] The discharge data for these states contain the patient's county of residence, birth month, and birth year, information critical for our analysis.[18] We limit our sample to children aged 0–10 years old when discharged, and we assume that their county of residence is also the county of birth.[19] Discharges are coded as "asthma" if the diagnosis code they have falls into that category as defined by the clinical classifications software.

As mentioned, this study aggregates data to the county–birth month–birth year cohort. We thus normalize the discharge data by the number of live births in each county in the respective birth month and birth year, using vital statistics data from the CDC.[20]

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are corrected using a long fill method with data from other years for that time and location, smoothing endpoints. The units for each hour are watt-hours per square meter, or equivalently an average instantaneous rate of watts per square meter.

17 Emergency department visits that resulted in an admission are recorded in the inpatient discharge files. Unfortunately, the inpatient discharge file for New Jersey does not contain month of birth. As a result, this analysis uses the emergency department databases, which include more than 80 percent of the total discharges with an asthma diagnosis. There are two possible biases here, depending on whether insufficient vitamin D creates more marginal cases or more serious cases. If it is the more marginal cases, then dropping the most severe cases would have no effect on this paper's results. If it is the more severe cases, then dropping them would bias our estimate downward, thus implying that our substantial and significant coefficient would be a lower bound. Either way, Study II includes the vast majority of the discharge observations.

18 Unfortunately, there is no information about birth day within the birth month in the HCUP files, which restricts the granularity of sunlight data to the month level.

19 We limit the sample to ages 0–10 to minimize the effects of cohort differences across time (e.g., changes in maternal smoking habits). With regards to our county of birth assumption, we note that even if there were significant movement, it would enter our regressions as classical measurement error as the movement should not be correlated with whether a county was having a more or less sunny month relative to its mean. The measurement error would not be classical only if individuals moving somewhere somehow caused that place to be relatively sunnier than before. Furthermore, this treatment is superior to using the location of the hospital, which would mix families from urban counties with families from rural counties that traveled to that urban hospital. Additionally, since this paper is interested in sunlight over the entire pregnancy, county of residence is significantly more indicative of long-term sun exposure than is the location of the hospital.

20 Unfortunately, the publicly available data cover only counties with populations exceeding 100,000. For birth years 1999–2009, data are publicly available for 18 of New Jersey's 21 counties, but only 5 of Arizona's 15 counties. Consequently, we omitted the other 3 New Jersey and 10 Arizona counties from the analysis. This should not bias the results of this analysis, since there is not likely to be a relationship between time-invariant (over the decade in Study II) county characteristics (e.g., population, urban/rural) and within-county-month

Whereas Study I focuses on incidence of asthma (without any measure of exacerbations), Study II fundamentally covers exacerbations of asthma, without any real measure of incidence across the population. A high per capita asthma emergency discharge rate could be caused by more individuals in that cohort having asthma and therefore more individuals having emergencies, or it could be caused by the same types of individuals who would have had asthma in a different cohort having more severe asthma and therefore more emergencies. In this way, the two studies complement each other, and show that the relationship between sunlight in utero and asthma is robust to the choice of outcome measure. Given this background, Table 2 provides means and general summary statistics for our two data sets.[21,22]

C. CONSTRUCTION OF THE SUNLIGHT VARIABLE

Neither data set enables us to trace the weather back to the exact day of birth. Given this coarseness in our data, we cannot determine the precise weeks of the pregnancy to which a sunlight measure pertains. However, we can assess a range. For example, in the Online Appendix, Section 3, we estimate our model at the month level (i.e., we include months on the right-hand side instead of trimesters). Suppose we found that the month two months prior to birth was significant. For individuals born on the first day of their month of birth this would be the second to last month of the pregnancy, but for those born on the last day of the month, it would be the third to last. We thus bound our results accordingly and in that case would say that we find evidence of significance within the last two to three months of the pregnancy.

For both studies, our trimester measurements for sunlight sum three adjacent months. Thus, our third trimester starts with the sunlight from one month prior to the birth month and adds to it the amounts in the two preceding months. We define the other trimesters correspondingly. We start one month prior to the birth month so that our second trimester measurement aligns better with the late pseudoglandular and canalicular stages.

We also stress that neither of our data sets contains information on the length of the pregnancies at hand. This is a further drawback of our data, because if individuals were born very prematurely, we may be systematically mislabeling their trimesters. Namely, if our samples contained many premature births, then what we label as the second trimester on average may actually be an earlier period in the pregnancy. To partially counter this timing issue, we note that there is scant medical evidence for an asthma–vitamin D

annual sunlight variation. Furthermore, there is a concern that less populated counties will have a count of emergency asthma or allergy emergency discharges censored at zero, which would bias a linear regression model. Omitting those counties helps avoid that bias.

21 For Study I, our full sample consists of 595,093 people, and analyzing the means of our variables of interest across the entire data set and those for whom we have asthma data reveals that though they are qualitatively similar, there are significant differences (Table A1 in the Online Appendix). Our restricted sample is still described as the "principal source of national asthma prevalence data for the United States" (National Center for Environmental Health 2014), but those significant differences could be due to some unobserved selection problem, and if so present a potential caveat to our results.

22 We unfortunately do not have measurements of socioeconomic status in either data set. We mention this as a weakness of our analysis.

TABLE 2. Summary statistics for Studies I and II

Study I		Study II	
N	264,701	*N*	3,036
		N (total births)	2,119,857
Percentage asthmatic	11.5%	Per capita rate of asthma emergencies	0.104[1]
Average year of birth	1956.06	Average year of birth	2004
Range of birth years	1914–87	Range of birth years	1999–2009
Range of ages	9–85	Range of ages	0–10
Percentage female	56.0%	Average cohort share female	48.8%
Major ethnicities		Major ethnicities (average cohort share)	
White	81.2%	White	79.1%
African American	15.7%	African American	13.0%
Other	2.8%	Other	n/a
Hours of sunlight:		Sunlight intensity (W/m^2):	
Mean ± std. dev.		Mean ± std. dev.	
1st trimester	673.74 ± 196.79	1st trimester	182.88 ± 65.99
2nd trimester	676.58 ± 197.28	2nd trimester	182.42 ± 65.49
3rd trimester	668.82 ± 197.24	3rd trimester	182.10 ± 65.55

Notes: [1]The per capita rate of asthma emergencies is calculated by the count of 2005–09 emergency department discharges with an asthma diagnosis for a particular birth month–birth year–county cohort, divided by the number of individuals born in that cohort. This rate is larger in magnitude than one might expect since multiple discharges for the same individual are counted separately, and since it pools multiple years. Data limitations make it impossible to track the same individual across discharges and years, which would be necessary to calculate an individualized rate.

connection prior to the second trimester. Apart from the timing concern, we cannot rule out the possibility that the causal channel we are observing is that vitamin D deficiency induces slightly premature births, and that premature births are a risk factor for asthma. Published studies have found associative evidence supporting both of these claims (e.g., Bodnar, Platt, and Simhan 2015). Given the effect sizes observed in these studies, however, we believe such a channel cannot explain the full magnitude of our results.

IV. Methodology and Results

We begin at a very broad level by comparing the distributions of normal and asthmatic births across the calendar year utilizing no controls, employing the data set from Study I. The distributions differ significantly (Pearson's chi-squared test, $\chi^2 = 25.73$, df $= 11$, $p = 0.0071$), with asthmatic births more common roughly during the summer.

Given the seasonal variation in weather across most of the nation, these data suggest a potential connection between sunlight and asthma. However, other factors could contribute to or produce this pattern. For example, it is known that seasonality of birth correlates with race (e.g., Bound and Jaeger 2000; Buckles and Hungerman 2013; Currie and Schwandt 2013). Therefore, to the extent that race is correlated with asthma incidence, racial differences in the season of birth could conceivably produce this pattern. The same logic holds for other factors that have been shown to correlate with season of birth, such as socioeconomic status or the degree to which pregnancies are planned.

To study our research question in greater detail, we now turn to our main results. They capitalize on the aforementioned natural experiment to rigorously test the sunlight hypothesis. Our approach fixes the birth location and month, and then assesses how within-location sunlight variation across years affects the probability of asthma.[23] We report results as linear probability models with our binary measure of asthma as the dependent variable and measures of sunshine and our controls as independent variables.

A. STUDY I

Given the individual-level data, this study's regressions take the following form:

$$ASTHMA_{ismy} = \alpha + \lambda_{sm} + \rho_y + \gamma \ln(SUN_{smy}) + X_i'\beta + \varepsilon_{ismy},$$

where $ASTHMA_{ismy}$ is the asthma status of individual i born in state s in month m and year y; λ_{sm} are state of birth interacted with month of birth fixed effects; ρ_y are year of birth fixed effects (which also captures the effect of aging, as the longer one is alive the more time one has to receive as asthma diagnosis); $\ln(SUN_{smy})$ is the log-transformed measurement of sunlight; and the X_i are individual controls for race and gender.[24]

In words, here is an example of how this specification proceeds. Individuals born in July in Georgia in 1978 received a certain exposure to sunlight in utero; individuals born in July in Georgia in 1979 received a different exposure. We assess how that variation in exposure affects the probability of developing asthma, taking into account a possible national trend in asthma diagnoses across years with the ρ_y.

Our main results are displayed in Table 3. We report our analysis using log-transformed sunlight to express our results in terms of percentage changes, but in the Online Appendix we show similar results with a nonlinear functional form.[25]

23 For this approach to provide meaningful evidence on a possible vitamin D connection, we need to assume that this variation in sunlight correlates with vitamin D levels. Though this may seem obvious, to provide supportive evidence, we merged our data from Study II with that from the American Time Use Survey to show that the variation we exploit correlates with time spent outside. Since we view this result as supportive, we refer the reader to the Online Appendix, Section 4, for more on this point.

24 Ideally, we would have liked to control for birth weight in our regressions as well, in order to isolate the effect on asthma beyond the birth weight effect documented in Trudeau, Conway, and Menclova (2016). However, the fraction of the NHIS sample that has birth weight data is very small; thus, unfortunately we simply do not have the power to produce such an analysis.

25 See Online Appendix Table A.5. To be fair, Study II's results seem more robust to functional form than Study I's.

TABLE 3. Regression output of main results from Study I

Dependent variable	(1) Asthma	(2) Asthma	(3) Asthma
Log hours of sun 3rd trimester	0.001	0.002	0.001
	(0.007)	(0.007)	(0.007)
Log hours of sun 2nd trimester	−0.016[a]	−0.016[a]	−0.017[a]
	(0.006)	(0.006)	(0.006)
Log hours of sun 1st trimester	0.003	0.003	0.004
	(0.007)	(0.007)	(0.007)
State of birth × month of birth fixed effects	x	x	x
Year of birth fixed effects	x	x	x
Race controls		x	x
Gender			x
N	264,533	264,533	264,533
R^2	0.008	0.009	0.011

Notes: The results indicate that greater potential sunlight exposure during the second trimester reduces the probability of being diagnosed with asthma later in life. For the coefficient on the second trimester sunlight, $p = 0.0091$, 0.0095, and 0.0079 for columns 1 to 3, respectively. All regressions are done with robust standard errors clustered at the state level. [a]$p < 0.01$, [b]$p < 0.05$, [c]$p < 0.10$.

This table's striking finding is that sunlight exposure during the second trimester has a large and significant effect on the probability that a child is later diagnosed with asthma. More specifically, doubling the amount of sunshine during this period lowers the subsequent probability of asthma by 1.15 percentage points.[26] Thus, at the mean incidence rate of 11.49 percent, such an increase in sunlight would lower the probability of asthma by around one-tenth. In contrast, during neither the first nor the third trimester is sunlight significant. To put these results in context, the average number of hours of sunlight in the summer in Nevada is about twice that for New Hampshire. Similarly, a baby born in December in New Mexico has on average about three times as much sunlight during the second trimester as a baby born in May in Michigan.

Bounding the time window as mentioned in the data section, these results provide evidence for significance inside weeks 10–26 of the pregnancy. This is a close match with the window of weeks 11–26 we specified earlier.[27]

26 This number comes from multiplying the coefficient on the second trimester in column 3 by ln(2): $-0.01653 \times \ln(2) = -0.0115$.

27 Given that data from both studies are at the month level, we repeat the analysis at this finer level in the Online Appendix, Section 3, to see whether we can learn more about when during this period sunlight exposure is most important (Table A.2).

Our results pass various robustness checks detailed in the Online Appendix, Section 6. Namely, if we add an additional pregnancy's worth of sunlight data both before conception and after birth to our main regressions, none of these extra terms is significant while our main effect persists. We also reestimated our analyses with state-specific time trends and found similar results.[28] Finally, to explore whether the effect is constant across time, we also reestimated our analysis on separate time intervals. Interestingly, we find that the effect we observe is largely driven by more recent births, arguably making our findings more relevant for current policy.

B. STUDY II

The main regression used in this study is the following:

$$ASTHMA_{cmy} = \alpha + \lambda_{cm} + \rho_y + \gamma \ln(SUN_{cmy}) + \varepsilon_{cmy},$$

where $ASTHMA_{cmy}$ is the per capita number of emergency department discharges for asthma in county c for individuals born in month m and year y; λ_{cm} are county interacted with month of birth fixed effects; ρ_y are year of birth fixed effects; and $\ln(SUN_{cmy})$ is the log-transformed measurement of sunlight.

In words, what this specification does is as follows. The cohort of individuals born in Mercer County, New Jersey, in March 2006 received a certain exposure to sunlight in utero; individuals born there in the March 2007 cohort received a different exposure. We are determining how that variation affects the incidence rate of emergency department discharges for asthma for those cohorts, taking into account possible trends across birth years that would affect all counties. Our main results from this study are in Table 4. As above, we report our analysis using log-transformed sunlight, but reestimating our models with alternative functions of sunlight yields very similar outcomes.[29]

This table shows that there is a large negative and highly statistically significant coefficient on the relative amount of sunlight during the second trimester, the exact same period we documented in Study I. Other trimesters show no effect. To interpret our second trimester coefficient, consider the following. Across this data set, compared with the mean potential sunlight exposure within a specific county-month pair, the minimum and maximum levels are approximately -0.2 to 0.2 log points away. If everyone had the sunniest second trimester in the data set for their month and county of birth, this would thus yield on average approximately 20 percent more sunlight. That 20 percent increment would lower the rate of asthma emergency department discharges by $0.2 \times 0.03 = 0.006$, which at the mean rate of 0.10 observed in this data set, is about a 6 percent reduction relative to

28 These would help control for factors that might be changing differentially across time in states, such as use of sunscreen or behavioral patterns affecting time spent outside.

29 See Table A.6 in the Online Appendix.

TABLE 4. Regression output of main results from Study II

Dependent variable	(1) Asthma	(2) Asthma
Log average sunlight intensity 3rd trimester	0.011	−0.023
	(0.024)	(0.034)
Log average sunlight intensity 2nd trimester	−0.032[a]	−0.060[a]
	(0.010)	(0.021)
Log average sunlight intensity 1st trimester	0.021	−0.012
	(0.020)	(0.020)
County of birth × month of birth fixed effects	x	x
Year of birth fixed effects	x	x
Birth years	1999–2009	1999–2004
N	3,036	1,656
R^2	0.762	0.747

Notes: We estimate the model regression on the full sample and a subset of the birth years. One might be concerned that there are fewer years of discharge data available from 2005 to 2009 for cohorts born after 2004 since they were alive for a smaller portion of the five-year observation period. The birth year fixed effects in the main regression should take care of these level differences, but may be then entangled with an abnormally large or small cohort year. Column 2 therefore omits the birth cohorts born after 2004, and reestimates the main regression on the 1999–2004 cohorts. Each of these six cohorts has a full five years of discharge data. The second trimester coefficient in column 2 is actually larger than that of the main result in column 1, strongly suggesting that the main result is robust to the inclusion of younger cohorts. The results indicate that greater potential sunlight exposure during the second trimester reduces that cohort's per capita rate of emergency department discharges for asthma later in life. For the coefficient on the second trimester sunlight, $p = 0.004$ and 0.009 for columns 1 and 2, respectively. All regressions are done with robust standard errors clustered at the county level. [a]$p < 0.01$, [b]$p < 0.05$, [c]$p < 0.10$.

the average.[30] These results pass robustness checks similar to those described for Study I (see the Online Appendix, Section 6, Table A.8).[31]

C. RACIAL DIFFERENCES

Blacks suffer from vitamin D deficiency at much higher rates than do whites. Two factors explain this disparity: blacks have a greater incidence of lactose intolerance (which limits

30 If we repeat this analysis on a similar sample from Study I (babies born in December in New Jersey), we see a 4.8 percent decrease in asthma diagnoses at the mean. While these values are of similar magnitude, the two are in fact significantly different. This is not surprising though, as given the range of symptoms present in asthmatics, it seems likely that the effect of sunlight may differ for those who receive borderline diagnoses versus those with sufficiently extreme cases to entail emergency department visits.

31 One additional robustness check we conducted added county-specific annual time trends. This addition negligibly affected our results. See Tables A.9 and A.10 in the Online Appendix.

TABLE 5. Regression output estimating our models for blacks and whites separately for Studies I and II

Dependent variable Study Racial group	(1) Asthma I Whites	(2) Asthma I Blacks	(3) Asthma II Whites	(4) Asthma II Blacks
Log sunlight 3rd trimester	−0.001 (0.008)	0.007 (0.015)	−0.004 (0.014)	0.161 (0.146)
Log sunlight 2nd trimester	−0.013[c] (0.007)	−0.035[c] (0.021)	−0.053[a] (0.009)	−0.064 (0.059)
Log sunlight 1st trimester	0.006 (0.008)	−0.003 (0.016)	−0.025[c] (0.012)	0.039 (0.102)
Region of birth × month of birth fixed effects	x	x	x	x
Year of birth fixed effects	x	x	x	x
Race controls	x	x	x	x
States (counties)	All	All	NJ (18)	NJ (18)
N	214,688	41,553	2,376	2,376
R^2	0.010	0.025	0.658	0.334

Notes: For the coefficient on the second trimester sunlight, $p = 0.0585$, 0.0963, 0.00002, and 0.295 for columns 1–4, respectively. All regressions are done with robust standard errors clustered at the state level. [a] $p < 0.01$, [b] $p < 0.05$, [c] $p < 0.10$.

their ability to consume vitamin D–fortified dairy products), and their greater skin pigmentation inhibits the production of vitamin D from sunshine. The benefit of additional sunlight exposure for blacks may be greater or less than it is for whites. Sunlight is converted to vitamin D at a lower rate for blacks, but likely provides a larger marginal benefit given their lower base level. We thus sought to explore how our observed effects differ by race.

We see in Table 5 that the second trimester coefficients are actually larger for blacks, though within each study the differences are not statistically significant. In terms of each individual regression, the evidence is less statistically significant than in the pooled regressions, but we do still see evidence of an effect within each racial group. If blacks have lower baseline levels of vitamin D, then perhaps the qualitatively larger second trimester coefficients derive from a diminishing marginal benefit of vitamin D during the second trimester. We leave further analysis of this hypothesis to future work.

D. ALTERNATIVE HYPOTHESES

We provide evidence that within-location variation in sunlight across birth years affects the probability of asthma pathogenesis. To conclude that our results imply a causal role for vitamin D, we must assume that our variation in sunlight correlates with maternal vitamin D levels and that it is uncorrelated with other factors that might actually determine asthma.

While we cannot rule out every possible alternative story, we addressed as many plausible ones as we could, given our data. From the fetal development literature, specific factors that have received detailed study are smoking while pregnant, pollution, and temperature.[32] Namely, there have been associational studies that document correlations between in utero exposure to each of maternal smoking and pollutants such as carbon monoxide and asthma later in life (e.g., Stick et al. 1996; Gilliland, Li, and Peters 2001; Mortimer et al. 2008). We note that apart from only reporting correlations, the exact biological pathways in utero connecting these contaminants to asthma remain unclear. In comparison, the connection between vitamin D and sunlight is extremely well documented, and the preexisting evidence on in utero vitamin D and asthma has a clear, well-researched biological pathway. To consider these further, we explore below each of these hypotheses in greater detail.

E. SMOKING

One could imagine various stories by which the variation in sunlight we are exploiting correlates with smoking rates. For example, since a sunny day can reduce stress and give many individuals more options for leisure and amusement, one might expect it to reduce smoking rates. Alternatively, given regulatory restrictions and social sanctions, individuals now mostly smoke outdoors (as opposed to in their workplaces or homes), and so a less sunny day could result in reduced smoking. If we believe that smoking affects fetal lung development and therefore asthma rates, then our observed sunlight coefficient could conceivably be picking up an effect from smoking behavior.

The American Time User Survey (ATUS) asks respondents about the number of minutes spent smoking per day.[33] This variable can be used to test the relative impact of sunlight on smoking, using the same econometric framework as above. Table 6 shows the results of this analysis.

The coefficients are extremely small in magnitude and are far from statistically significant. A one log-point increase would increase the mean amount of time spent smoking by only a few seconds, which is even less than one cigarette. With the standard errors in column 3, we can rule out at the 5 percent level any effect bigger than 9 fewer seconds $((0.036 - 0.09 \times 2) \times 60)$ or 13 more seconds spent smoking on an unusually sunny day. Finally, because of some data concerns with the ATUS, in Section 5 of the Online Appendix we do a separate analysis using the CPS Tobacco Use Supplements.

F. POLLUTION

One may also be concerned about a potential correlation between sunlight and pollution levels. The study cited above specifically refers to carbon monoxide (CO), which

32 The negative effects of cigarette smoke on current asthmatics is very well documented; note here we are referring to the smaller literature on in utero exposure to smoke.

33 The ATUS has a single code for "Tobacco and drug use." We are assuming here that since tobacco use rates are much higher than drug use rates, and that smoking is still the primary method of tobacco use, this variable can safely be used to represent time spent smoking.

TABLE 6. Smoking results, women 15–44, using ATUS

Dependent variable	(1) Time spent on smoking	(2) Time spent on smoking	(3) Time spent on smoking
Log average sunlight intensity for reference day	0.046	0.040	0.036
	(0.095)	(0.093)	(0.090)
State × month fixed effects	x	x	x
Day of week and year fixed effects		x	x
Demographic controls			x
N	27,340	27,340	27,340
R^2	0.114	0.116	0.125

Notes: Regression output of time spent on smoking and drug use on average sunlight for the population-weighted state centroid on the reference day. The results indicate no relationship between greater sunlight and time spent on smoking / drug use ($p > 0.1$ for all). All regressions are done with robust standard errors clustered at the state level.

a separate literature has consistently shown to be a pollutant with adverse effects on child health (Neidell 2004; Currie, Neidell, and Schmieder 2009; Schlenker and Walker 2011).

To investigate whether our results are robust to this potential issue, we apply the regression framework of Study II to pollution data,[34] and also to a combined regression that includes both pollution and sunlight data. The analysis below uses measured CO level (in parts per million), averaged over each calendar month and year. As with the satellite sunlight data, each county is matched with the pollution monitor that is the closest to its population-weighted centroid.

The log of a three-month moving average level of CO is modestly negatively correlated with sunlight (−0.2). This adds plausibility to the concern described above, since higher in utero sunlight levels, which should reduce asthma rates, would coincide with lower in utero CO levels, which could also reduce asthma rates.

In Table 7, column 1 repeats the main results for Study II from above for comparison. Column 2 replaces the log of average sunlight in each trimester with the log of the average CO level in each trimester. Finally, column 3 includes both sets of independent variables to see how greatly CO levels reduce the magnitude and significance of this paper's main finding.

Column 3 shows the results of including both the in utero CO level and sunlight level. The second trimester sunlight result remains extremely statistically significant, but its magnitude is reduced about 10 percent. While this is consistent with a hypothesis that the in utero CO level does have an effect on asthma, and that this effect is somewhat

34 Daily pollution data from the Environmental Protection Agency is publicly available at http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html (see Currie, Neidell, and Schmieder 2009; Sanders 2012).

TABLE 7. Study II, carbon monoxide results

Dependent variable	(1) Asthma	(2) Asthma	(3) Asthma
Log average sunlight intensity 3rd trimester	0.011		0.012
	(0.024)		(0.024)
Log average sunlight intensity 2nd trimester	−0.032[a]		−0.030[a]
	(0.010)		(0.009)
Log average sunlight intensity 1st trimester	0.021		0.025
	(0.020)		(0.017)
Log average CO level 3rd trimester		0.009	0.009
		(0.009)	(0.009)
Log average CO level 2nd trimester		−0.001	−0.001
		(0.007)	(0.007)
Log average CO level 1st trimester		0.006	0.005
		(0.007)	(0.007)
County of birth × month of birth fixed effects	x	x	x
Year of birth fixed effects	x	x	x
Birth years	1999–2009	1999–2009	1999–2009
N	3,036	3,036	3,036
R^2	0.762	0.763	0.763

Notes: Here we repeat the analysis done earlier for Study II. Column 1 contains the same main result as in Table 4. Column 2 repeats the analysis using instead the log of the average CO level. Column 3 estimates a "horse race" including both sets of independent variables. Robust standard errors clustered at the county level. The p-value for the second trimester sunlight coefficient in column 3 is 0.005, versus 0.004 in column 1. [a]$p < 0.01$; [b]$p < 0.05$; [c]$p < 0.10$.

correlated with the effect of sunlight, it does not diminish the robustness of the main result of this paper. The effect of sunlight is not simply proxying for the effects of pollution, but rather represents its own substantial result.

G. TEMPERATURE

Finally, average monthly sunlight levels are highly correlated with average monthly temperatures (correlation of 0.82). A broad literature documents the connection between temperature and health (as reviewed in Deschenes 2013), though that relationship has diminished over time (Barreca et al. forthcoming). While there is no literature specifically linking temperature in utero to asthma, there is work linking temperature to birth outcomes (Barreca, Deschenes, and Guldi 2015).

We repeat the above analysis using the log of the average monthly temperatures over the three months of each trimester. Data are from US Historical Climatology Network Monthly (USHCN), which consists of 1,218 weather stations spread throughout the

United States.[35] For the Arizona and New Jersey counties in Study II the population-weighted county centroid is matched to the nearest weather station.

Table 8 shows the results of estimating an analogous regression of relative temperatures on asthma rates, as well as a "horse race" with both sunlight and temperature as possible explanatory variables. So while contemporaneous temperature may affect mortality, fertility, and birth outcomes, temperature in utero appears to have minimal future effect on asthma.

Here, the results are even stronger than above, with temperature in utero having a minimal effect on asthma rates, and when included in a "horse race" regression having no impact on this paper's main results.

One additional temperature robustness check in columns 4–6 instead adds the average number of days per month that the temperature was "extreme," that is, under 15°F, over 80°F, and over 95°F (following Barreca, Deschenes, and Guldi 2015). While the magnitude of the main second trimester effect of sunlight is somewhat decreased, the results remain robust overall.[36]

Finally, while our data availability did not enable us to repeat this analysis with Study I, it is worth highlighting how the temperature extremes may have played a differential role across time in that study, because of the advent of indoor climate control (as pointed out by a referee). Hence, omitting temperature in Study I's regressions could diminish the coefficients on sunlight in earlier decades, which is consistent with what we see in Table A.11 in the Online Appendix.

V. Conclusion

Our results from two independent studies provide evidence that greater sunlight during the second trimester of pregnancy reduces the probability the unborn child will later develop asthma. Further, we find evidence that sunlight has an effect for both blacks and whites, which is important given the increased prevalence of asthma within the African American community.[37] The presumed causal channel operates through the mother's conveyance of vitamin D to the fetus, the vast majority of which comes from her sunlight exposure. Similarly, we have provided evidence that sunlight exposure is correlated with the variation in weather that we exploit. A qualification to our identification strategy is that there might be unobservable factors other than vitamin D that are correlated with sunlight levels and are the true causal determinants of asthma. Given our data, we cannot rule out all such possible unobservables, but given the strength of the preexisting hypothesis surrounding in utero vitamin D and asthma, and our results from several external data sets detailed in the Online Appendix, we believe the results to be strongly suggestive. To judge the overall significance of the results from our two studies, we note that if the true effect of second trimester sunlight levels on asthma incidence were zero, the probability

35 The data from NOAA are available at ftp://ftp.ncdc.noaa.gov/pub/data/ushcn/v2.5/readme.txt.

36 The daily data necessary for this piece are available from NOAA's Global Historical Climatology Network. See ftp://ftp.ncdc.noaa.gov/pub/data/ghcn/daily/readme.txt.

37 We thank a reviewer for encouraging us to explore this direction.

TABLE 8. Study II, temperature results

Dependent variable	(1) Asthma	(2) Asthma	(3) Asthma	(4) Asthma	(5) Asthma	(6) Asthma
Log average sunlight intensity 3rd trimester	0.011		0.010	0.017	0.016	0.013
	(0.024)		(0.026)	(0.027)	(0.026)	(0.024)
Log average sunlight intensity 2nd trimester	−0.032[a]		−0.033[a]	−0.028[b]	−0.026[c]	−0.030[b]
	(0.010)		(0.010)	(0.010)	(0.012)	(0.011)
Log average sunlight intensity 1st trimester	0.021		0.019	0.023	0.021	0.025
	(0.020)		(0.210)	(0.023)	(0.024)	(0.021)
Log average temperature 3rd trimester		0.007	0.009			
		(0.017)	(0.019)			
Log average temperature 2nd trimester		0.010	0.008			
		(0.015)	(0.019)			
Log average temperature 1st trimester		0.004	0.001			
		(0.017)	(0.016)			
Average days/month temperature <15°F 3rd trimester				0.002		
				(0.001)		
Average days/month temperature <15°F 2nd trimester				0.002		
				(0.002)		
Average days/month temperature <15°F 1st trimester				0.002[c]		
				(0.001)		
					>80°F	>95°F
Average days/month temperature hot 3rd trimester					0.000	0.000
					(0.001)	(0.001)
Average days/month temperature hot 2nd trimester					0.000	−0.001
					(0.001)	(0.001)
Average days/month temperature hot 1st trimester					0.000	−0.001
					(0.001)	(0.001)
County of birth × month of birth fixed effects	x	x	x	x	x	x
Year of birth fixed effects	x	x	x	x	x	x
Birth years	1999–2009	1999–2009	1999–2009	1999–2009	1999–2009	1999–2009
N	3,036	3,036	3,036	3,036	3,036	3,036
R^2	0.762	0.762	0.762	0.763	0.762	0.763

Notes: Here we repeat the analysis done earlier for Study II. Column 1 contains the same main result as in Table 4. Column 2 repeats the analysis using instead the log of the average temperature level. Column 3 estimates a "horse race" including both sets of independent variables. Columns 4–6 instead add the average number of days per month that a temperature was "extreme." Robust standard errors clustered at the county level. The p-value for the second trimester sunlight coefficient in columns 3–6 are 0.003, 0.012, 0.041, and 0.013 versus 0.004 in column 1. [a]$p < 0.01$, [b]$p < 0.05$, [c]$p < 0.10$.

of finding effects at less than the 1 percent significance level in two independent studies would be less than one in 10,000.

Our results suggest low levels of maternal vitamin D during pregnancy may be more harmful than previously thought. As mentioned earlier, vitamin D deficiency and insufficiency are extremely widespread, with multiple studies of pregnant women documenting overall rates in excess of 80 percent (Bodnar et al. 2007; Holmes et al. 2009; Johnson et al. 2011). Hence, our results suggest that if we could raise vitamin D levels in these women, the savings in terms of both future quality of life and health-care costs could be substantial.

The two most practical and cost-effective methods for augmenting vitamin D levels would be supplements and increased sunlight exposure. Both may be helpful, but when regularly available, we prefer the latter for two reasons. First, many women already take supplements during pregnancy that include vitamin D, but some studies have found that their insufficiency rates are similar to those of women who do not take supplements (e.g., Holmes et al. (2009), or see Vieth et al. (2001)). This may be due to inadequate dosages, absorption problems, or compliance issues, but it suggests that supplementation is not completely straightforward. Second, sunlight exposure is a very efficient means of vitamin D production. A rule of thumb in the medical literature is that most people may obtain a sufficient daily dose of vitamin D from 10 minutes of direct sunlight exposure (Holick 2007).[38] Further, unlike supplements, which can only deliver vitamin D in fixed quantities, production of vitamin D from the skin automatically shuts off when levels are sufficiently high (Holick and Garabedian 2006). The main cost of additional sunlight exposure would be a slightly higher risk of sunburn and melanoma, but given how little sun exposure most people need to obtain sufficient vitamin D compared with the amount necessary for sunburn, much less melanoma, this risk seems modest. To be fair, Trudeau, Conway, and Menclova (2016) do find evidence that more sunlight may slightly increase the probability that a white mother has a lower birth weight child. However, a very rough back of the envelope calculation of the magnitude of the broad economic surplus from widespread vitamin D supplementation is $5 billion per year.[39]

Additionally, while this study focused on asthma because of data limitations, other diseases (such as multiple sclerosis, schizophrenia, and cardiovascular disease) have been linked to low levels of vitamin D. Future work could consider whether increased sunlight

38 The specific amount required varies with ethnicity, altitude, latitude, etc. There are several calculators available online, such as http://nadir.nilu.no/~olaeng/fastrt/VitD-ez_quartMEDandMED_v2.html. To be fair, in some geographies, it is simply impossible to get enough vitamin D from sunlight exposure alone. Even when the location and timing make it feasible to obtain the necessary level of vitamin D from the sun, other factors may make this difficult in practice, such as sunlight obstruction from tall urban buildings.

39 This estimate is calculated as follows. From Study I, the reasonable relative range of sunlight hours is double. Multiplying ln(2) by the regression coefficient (−0.0165) and dividing by the mean incidence (11.5 percent) yields a 10 percent reduction. With the annual cost of asthma at $56 billion (Barnett and Nurmagambetov 2011), this would be a surplus of $5.6 billion. The costs of supplements is minimal compared with these surpluses, not to mention the even smaller cost of more time outdoors. This analysis also ignores the risk of increased low birth weight children for white mothers suggested by Trudeau, Conway, and Menclova (2016).

reduces the incidence of those diseases as well, perhaps using this paper's empirical framework.

Prior research has shown that the mechanisms that underlie asthma are numerous and complex. We find evidence suggesting that sunlight during the second trimester (which we posit contributes to and proxies for maternal vitamin D levels) is a significant factor. Given the extent of the asthma epidemic afflicting the world today, securing a better understanding of this complex disease and its numerous contributing factors is extremely important. On a broad note, our paper contributes to the growing economics literature that uses applied econometrics to shed light on medical questions. We believe that such econometric analyses have the ability to complement (not supplant) the standard randomized controlled trials of medicine. They can provide substantially cheaper, larger-scale analyses of preexisting data that can also help to guide future randomized controlled trials. Econometric studies and randomized controlled trials are not rivals in research; they are partners.

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