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Article

Season of birth and depression in adulthood: Revisiting historical forerunner evidence for in-utero effects

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ABSTRACT

Evidence showing a relationship between season of birth and adult well-being is long-standing, but is now largely overlooked or dismissed. In light of increasingly compelling evidence for the effects of in-utero conditions on adult health, however, it is instructive to revisit the relationship, with an eye toward resolving the reasons for skepticism. This study uses data from the first National Health and Nutritional Examination Survey to examine the effects of month of birth on adult depression. The data correspond to an important time in history and the analysis points to one reason why enthusiasm for birth seasonality in depression has faded: although there was a strong relationship between month of birth and depression in the early 20th century, with spring and summer month births corresponding to significantly more depression, the relationship was largely eliminated by the 1940 birth cohort. Few adults alive today would be subject to this effect, but when it was apparent it was enormously consequential. Population attributable risk scenarios indicate that among those born between 1900 and 1920 the prevalence of major depression would have been reduced by approximately 22% if all births had been confined to November through March. The percent rises to 26% among those born between 1900 and 1910, and was likely even higher in earlier cohorts. Additional analyses point to the importance of nutritional deficits in explaining these effects. In the early 20th century, the relationship between month of birth and depression was weaker in circumstances where the food supply was less seasonally sensitive. For this reason, the turn-of-the-century relationship between month of birth and depression was much weaker among the well-educated, in Southern states, and in urban areas. Although birth seasonality in depression can be regarded as a historical artefact of diet and nutrition, evidence for its prior existence nonetheless speaks to the significance of other in-utero effects, both past and present.

The idea that season of birth affects health and well-being is ancient, but has mostly faded from the imagination of contemporary scientists. In the US, scientific interest in birth season effects grew following the publication of several provocative studies in the 1930s. These studies found birth seasonality in a host of psychiatric disorders and dimensions of personality (Huntington, 1938). Other studies quickly followed (e.g., Knobloch & Pasamanick, 1958). By the 1960s and 70s, however, enthusiasm for birth season effects had waned as the evidence grew more mixed and inconclusive. Although some studies continued to find a relationship (e.g., Bailar & Gurian, 1965; see Torrey, Miller, Rawlings, & Yolken, 1997 for a review), other studies failed to find a significant effect, prompting skeptics to dismiss the entire idea of birth season effects as an esoteric form of speculation, aligned more or less with astrology (Woodruff, Guze, & Clayton, 1974, p. 926). Pointed skepticism of this sort was perhaps rare but it found a receptive audience, even among scientists with more moderate inclinations. Many scientists, it would seem, were eager to cast the idea of birth seasonality in health to the dustbin of history, focusing their efforts instead on unraveling the many contemporaneous conditions relevant to adult health. The idea of fundamental causes is consistent with this turn (Link & Phelan, 2010).

Yet in the 21st century the credibility of a relationship between season of birth and adult health has grown in light of the growing acceptance of in-utero effects. Although in-utero effects are rarely cast in terms of seasonality *per se*, they do point to the enduring importance of the conditions surrounding gestation and birth, much like the original birth season research did. Perhaps the most influential evidence of this sort stems from the so-called Barker hypothesis, positing a relationship between in-utero conditions, indicated by birth weight, and adult health, especially cardiovascular disease (Barker, 1992; Barker, 1998). Since the initial statement of the idea, the basic parameters of the Barker hypothesis have expanded, encompassing a variety of in-utero insults and an assortment of adult health outcomes, including mental health (Langley-Evans & McMullen, 2010). It requires little stretch to

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see parallels between this contemporary literature and the seemingly more archaic insights of the birth season literature. There is, in fact, already evidence linking month of birth to adult mortality, channeled through some of the same metabolic mechanisms behind the Barker hypothesis (e.g., Doblhammer & Vaupel, 2001).

The present study attempts a rapprochement between earlier and recent evidence. It does so using the lens of history, by exploring the relationship between season of birth and adult depression using survey data drawn from cohorts born in the early 20th century, between 1900 and 1950, and collected when respondents were adults, in the mid-1970s. In particular, this study explores how the relationship between season of birth and depression changed during a particularly important time period. A focus on between-cohort change provides a framework for thinking about the evidence for birth-season effects, while also pointing to the importance of nutritional deficiencies in shaping them. Perhaps one reason contemporary scholars have often failed to find a relationship between season of birth and adult well-being is that the relationship between the two has, in fact, faded over time, especially as the conditions that made it possible have evolved. Evidence for a declining relationship with season of birth does not, however, obviate the significance of in-utero effects altogether, and the conclusion discusses the implications of birth season effects for contemporary research on other in-utero determinants of adult depression.

Background

Although research on birth season effects is often overlooked, much of the early evidence is quite persuasive. Entertaining the idea of birth season effects depends only on recognizing two things: that in-utero conditions can exert a lasting impact on health and that such conditions can be influenced by seasonal factors. Much of the early evidence regarding season of birth and mental illness focused on severe and persistent disorders, especially schizophrenia. In general, this literature finds an elevated risk associated with winter and spring, corresponding more precisely to births between December and March (in the Northern hemisphere) (see Torrey et al., 1997). Although there is less evidence pertaining major depression—and less still with respect to subclinical disorders—what studies exist point to a somewhat later risk period, corresponding to births between March and May. Contemporary reviews are circumspect: they generally regard the birth season effect as small but significant. One systematic review of schizophrenia studies found a population attributable risk for being born during winter and spring months of only 3.3% (Davies, Welham, Chant, Torrey, & McGrath, 2003). Reviews of Southern hemisphere data also conclude the effects of season of birth are significant but, if anything, they demonstrate even smaller effects than are apparent in Northern hemisphere data (McGrath & Welham, 1999).

In addition to seemingly small effects, one reason contemporary audiences are skeptical of the literature is its general inability to identify a precise mechanism. In absence of a mechanism, the literature appears overly speculative. To date, most of the literature has focused on ruling out explanations rather than presenting evidence favoring one mechanism over another. Much of the initial effort, for instance, focused on testing artifactual explanations and, therefore, did not address mechanisms at all. For instance, there are natural patterns in birth seasonality that are apparent in the general population (Martinez-Bakker, Bakker, King, & Rohani, 2014). To the extent that studies employ samples of psychiatric patients without appropriate comparisons or statistical corrections, they risk incorrectly concluding that one season is risker than another when in fact the distribution of birth seasons among patients merely reflects normal seasonal variation in births. Other artefactual explanations focus on the behavior of parents. Some, for instance, have posited that those with certain psychiatric disorders have procreation habits that are seasonal, creating birth patterns but ones that likely reflect genetic influences rather than seasons *per se* (see Bleuler, 1991 with respect to depression). Studies examining the siblings of those with psychiatric disorders generally find no evidence for this interpretation and, in general, explanations focused on assorted behavioral correlates of birth seasonality have not been successful (Pulver et al., 1992).

Subsequent to ruling out artefactual explanations, studies have settled on two types of mechanisms: either seasonal variation in nutrient supply or seasonal variation in infectious disease exposure (Disanto et al., 2012). These explanations differ in the proximate mechanisms they emphasize, whether with respect to the specific infection or the specific nutrient, but are nonetheless united around the idea that some in-utero exposure or deficiency alters developing fetal tissues. One set of hypotheses focuses on seasonal variation in exposure to infections, especially the flu. The regular timing of flu season lends this idea its plausibility. Despite year-to-year variation in the specific strains of influenza that appears there are remarkable regularities in the timing of flu pandemics (Lofgren, Fefferman, Naumov, Gorski, & Naumova, 2007). Flu season generally peaks in February, corresponding, then, to late-term in-utero exposure and, in turn, to additional risk associated with November to March births. Some evidence for the relevance of in-utero influenza exposure is more direct. Some studies, for instance, have linked in-utero exposure to especially significant flu pandemics to the development of schizophrenia in adulthood (e.g., Adams, Kendell, Hare, & Munk-Jørgensen, 1993). To be sure, these pandemics tend to be exceptionally virulent and, therefore, represent an especially powerful dose, but they nonetheless add to the general case that in-utero influenza exposure affects fetal development.

Other hypotheses focus on nutritional deficiencies. Nutritional intake can vary over seasons and studies have posited a variety of relevant influences, including vitamin C, vitamin K, and protein (Susser, Hoek, & Brown, 1998; Thane et al. 2002; Tochigi, Okazaki, Kato, & Sasaki, 2004). The idea that maternal sunlight exposure can also affect fetal development is a different version of the same idea. Seasonal changes in sunlight exposure might affect the availability of vitamin D in pregnant women and, in turn, adversely affect the development of the fetus (McGrath, Burne, Féron, Mackay-Sim, & Eyles, 2010). Beyond seasonal variation, this idea also implies an interaction between season of birth and latitude, as higher latitudes will correspond to even less sun exposure during the winter and early spring. Although the interpretation that birth seasonality in schizophrenia reflects nutritional deficiencies is speculative, there is at least high-quality evidence linking maternal nutritional adequacy to the mental health of offspring (Jacka et al., 2013).

When explaining birth season effects, it is difficult to evaluate the role of nutritional deficiencies directly, but the idea has a number of testable implications. For one, to the extent that birth season effects reflect nutrition, it is likely that the effects have shifted considerably over the course of the 20th century, especially in the early part. In particular, seasons likely played less of a role in nutritional adequacy in the middle of the century than they did earlier and, furthermore, the overall nutritional content of food has improved. These improvements were broad and multifactorial. Food supply, preservation methods, and nutritional content each improved over the early part of the century (Bennett & Peirce, 1961). Refrigerators with freezers, for instance, became available for households during the 1920s, abruptly changing the supply of food families were able to have on hand (Centers for Disease Control, 1999). At the same time, the scientific understanding of nutrition was improving rapidly during this period (see Preston & Haines, 1991 for a review of science and nutrition at the turn of the century). In the early 20th century, little was known about the nutritional content of food or its consequences, apart from a rudimentary understanding of the effects of severe inadequacy. In this context even a well-resourced family was unlikely to maintain perfect nutrition year-round, even if most families were able to maintain adequate caloric intake (Wait, 1909). Among high-income families, the demand for nutritional content *per se* only emerged in the middle of the century, when the benefits of nutrition were more credibly established (Beatty & LaFrance, 2005).

Other important changes pertained to broader public health initiatives, which likely reduced the relationship between season and nutritional intake. Beginning in the 1920s, for instance, public health officials, armed with a better understanding of disease, focused on alleviating some specific deficiencies in the American diet (Martini & Phillips, 2009). Reflecting the same interest, nutrient supplementation to staples such as wheat flour began in the 1940s. Other improvements occurred at approximately the same time, including in other aspects of food supply. The per capita supply of citrus fruit, for instance, increased through most of the 20th century, improving the intake of vitamin C (Gerrior, Bente, & Hiza, 2004). Indeed, the supply of most vitamins in food likely increased over the 20th century, and some improvements were especially pronounced in the early part of the century (Gerrior et al., 2004; Gortner, 1975). The discovery of specific nutrients related to disease led to a rapid decline in, for instance, pellagra (caused by a lack of niacin) and rickets (caused by a lack of vitamin D), much of it occurring in the 1930s and 40s (Centers for Disease Control, 1999).

If nutrition played a role in shaping birth season effects in depression, there are likely to have been other social influences that were relevant as well. In the early 20th century, dietary nutrition was perhaps especially sensitive to seasonal fluctuation, but it was almost certainly not equally sensitive over all regions and people. In general, urban areas provided more variety in the food supply than did rural ones (Bryant, 1898). In addition, some entire regions experienced less variation in the supply. Evidence from surveys conducted in the early 1900s, for instance, found great seasonal consistency in the diet of Southerners, in part because of the difficulty of preserving food in a warm climate combined with regional patterns in agriculture and food supply (Martini & Phillips, 2009; Wait, 1909). A typical meal often involved little more variety than could be gleaned from corn meal, cured or salted pork, and items baked with wheat flour (Wait, 1909). Fresh fruits were rarely consumed, even in the summer. Cross-cutting these regional differences was the likely significance of social class. At the turn of the century, food was a significant part of the household budget (U.S. Bureau of Labor, 1906), with some poor families spending nearly 70% of their income on food (Forman, 1906). With less strain on their household budgets, upper-class families are likely to have enjoyed more consistent nutrition over the seasons than did lower-class families. Some evidence for this is already apparent in research on birth seasonality in schizophrenia, especially research conducted in the context of psychiatric hospitals. There is more evidence for seasonality apparent in public psychiatric hospitals than private hospitals (Barry & Barry, 1964).

Summary

Altogether the historical literature points to the possibility both of birth season effects on adult depression and for a host of moderators of these effects. To the extent that nutrition is important, the relationship between month of birth and adult depression is likely to have attenuated over consecutive birth cohorts in the early 20th century. In addition, the relationship between month of birth and depression is likely to have been weaker among those of higher social classes, among those born in the south, and among those born in urban areas. Investigating moderators of this sort provides a window not only on the role of nutrition in birth season effects, but also the conditions that can both produce and extinguish in-utero effects. Investigating moderators also situates the history of depression in a larger setting of socioeconomic development. The data used in this study allow for the exploration of these influences over an especially significant window of historical time.

Data and research methods

Data for this study are drawn from the first National Health and Nutrition Examination Survey (NHANES), the initial entry in the ongoing series and referred to as NHANES-I (Centers for Disease Control, 1975). The survey can be regarded as historical in the sense that it is not the latest in the series, but it was collected over an especially significant time period that allows the analyst to shed light on contemporary debates. It was conducted between April 1971 and October 1975, encompassing births from 1900 to 1950. Although the entire NHANES-I sample is representative of those age 1 to 74, a subsample of adults age 25 to 74 were administered a more detailed battery of questions regarding emotional well-being. This battery contains questions on depression, including the well-known Center for Epidemiological Studies Depression scale, which serves as the foundation for this analysis. The NHANES-I is a three-stage stratified probability sample of non-institutionalized persons. Because of the complex survey design, all the analyses use survey weights. These weights serve two purposes: they account for survey nonresponse and the unequal probability of selection given the sampling design (Landis, Lepkowski, Eklund, & Stehouwer, 1982).

The NHANES-I is valuable for several reasons. For one, the NHANES-I is a large study, even when restricted to the subsample of respondents who received the supplementary questions on emotional well-being. In total, 3048 respondents were asked the relevant questions. A very small number did not report values sufficient to code the dependent variable, leaving a total available sample of 3,033. This represents a large sample relative to previous studies. In previous studies, a not insignificant problem has been relatively small sample sizes involved (Hare, 1975). Assuming a small true effect of season of birth, many previous studies might simply have not had the power to detect a statistically significant relationship. Another valuable aspect of NHANES-I is its representative nature. As noted, many prior studies have relied on clinical samples, including samples of residents in psychiatric hospitals. From these data, researchers have looked for excess births in a given month relative to normal expectations. The use of clinical samples can be illustrative, to be sure, and, in fact, sparked the entire birth season literature, but a general reliance on such samples has been one reason some scientists are skeptical of birth season effects. NHANES-I does not, of course, suffer from this problem, and the dependent variable, discussed more shortly, reflects the full range of distress, even as it allows for identifying an analogue of major depression.

Dependent variable

The primary dependent variable is the Center for Epidemiological Studies Depression scale (CES-D), among the most popular dimensional measures of distress in epidemiology and a cornerstone of the sociology of mental health (Radloff, 1977). All 20 items of the CES-D were included in NHANES-I, allowing for a full summative range of 0 to 60, with higher values reflecting more depression. As in other surveys, in NHANES-I the CES-D shows very high coefficient reliability ($\alpha = .851$). The full CES-D serves as the dependent variable for most of the models, in large part because it provides strong statistical sensitivity in the context of well-established empirical parameters. Some parts of the analysis consider major depression, conventionally assigned to those scoring a 16 or higher, and corresponding to about 17% of the NHANES-I sample. Designating a respondent as suffering from major depression does not indicate he/she has been formally diagnosed with the disorder, though this represents a strength of the research design, as it does not require such a designation by a clinician. When using the CES-D as the dependent variable, the models are linear regression. When using major depression, the models are logit regression.

Independent variables

There are multiple ways to specify birth seasonality effects, though the underlying process described in most of the interpretations discussed above implies gradual change. So-described, the process almost

Table 1
Summary of variables used in regression models, NHANES-I.

		Cohort				
Variable	Overall Mean	1900–1910	1911–1920	1921–1930	1931–1940	1941–1950
CES-D	8.949	8.929	9.254	8.956	9.045	8.656
Major Depression (CES-D > = 16)	.174	.153	.176	.175	.179	.177
Month of Birth	6.517	6.679	6.562	6.369	6.500	6.536
Age	46.046	68.778	68.432	48.541	38.553	28.767
Female	.526	.571	.524	.511	.514	.524
Black	.096	.083	.096	.112	.091	.095
Northern State	.238	.194	.233	.250	.239	.253
Rural	.301	.308	.324	.286	.308	.286
Southern State	.191	.202	.196	.198	.176	.189
State Centroid Latitude	38.767	38.401	38.943	38.689	38.712	38.949
High School +	.353	.207	.335	.359	.417	.388

Note: Sample size is 3033, except for state centroid latitude, which is restricted to respondents born in US states (N = 2820).

always involves a single peak in risk, rather than a more discontinuous function consisting of multiple peaks and valleys. With this as background, most of the regression models specify seasonality using a continuous month-of-birth function. Month of birth is scored from 1 to 12, corresponding to January to December, and all the models include both month and month-squared terms. This specification permits curvilinearity with a single inflection point, as is implied in the idea that there is a single period of especially high risk (though perhaps one spanning a couple months). Some models also estimate coefficients for especially risky months (e.g., birth from April to October versus all other months), though this specification is used only for some specific calculations, including the population attributable risk scenarios, for which a categorical designation is required. A continuous-month specification is also preferred on empirical grounds. Exploratory tests of model fit revealed that a continuous month and month-squared specification fit better than a fully relaxed dummy variable specification, with 11 dummy variables, one for each month minus a reference category. A single risk period, specifically April to October births, also fit better than a model with 11 dummy variables.

A continuous month and month-squared specification is useful in another way. This specification allows for more statistically powerful tests of moderators, based on simple multiplicative interactions. If the relationship between month of birth and adult depression has diminished over time, interactions between month of birth and year of birth should be significant (as well as between month of birth-squared and year of birth). The coefficients for the interactions should be signed in the opposite direction as the coefficients for the main effects, implying, then, a graduate reduction in a once strong relationship. Because in NHANES-I birth year cohorts begin in 1900—and, thus, at zero because birth year is coded year minus 1900—the main effect of birth month (and birth month-squared) has a simple interpretation: it represents the effect of birth month among those born in 1900.

Although the focus of this study is obviously on month of birth, there are a number of confounding influences to consider. Births are naturally distributed over all months of the year. There are, however, some social and geographic correlates of seasonality. Many of these correlations are small, but the presence of such correlations nonetheless necessitates the use of robust geographic controls. In general, the peak month for births occurs earlier in the year the further one is from the equator. In the early 20th century, for instance, Northern states showed a peak in June, whereas Southern states showed a peak in November (see maps presented in Martinez-Bakker et al. (2014), especially Fig. 2). In addition, seasonal variation between states was greater in the early 20th century than it is now (though variation still exists). To the extent that place of birth also affects depression, it is important to control for regional effects. To this end, the regression models capture geographic differences in a variety of ways. Some models control for Northern states, based on maps presented by Martinez-Bakker et al. (2014). Some models control for Southern states, using the South Central designation of the US Census Bureau. In addition, some models explore the latitude of the state of birth, based on a state centroid (with data also available from the US Census Bureau, https://www.census.gov/geo/reference/state-area.html). This measure only applies to those born in US states, as the birth latitude of the remainder of the sample cannot be coded with the same precision. A series of additional control variables are included in all or some of the models. These include race (black versus all other race/ethnicities), sex (female versus male), rural (rural versus other), and education (coded as having at least a high school degree versus less than a high school degree, which is a parsimonious split chosen in light of the typical levels of education among those born in the first half of the 20th century). Not all of these covariates pertain to birth conditions, although they still speak to them. Rural residence, for instance, pertains to current residence, though, at the level of urban versus rural, current residence is positively correlated with residence at birth. Similarly, education pertains to the respondent's education, though in some models it is used, in part, as a proxy for maternal education. These two variables, too, are likely correlated. Table 1 presents descriptive statistics. Values are consistent with expectations, with a sample that has slightly more women than men, where the prevalence of major depression is under 20%, and the average age is in the mid-40s. About a third of the sample has at least a high school diploma.

Results

Table 2 begins with a basic regression model of the relationship between month of birth and depression. Two models are presented, one for the CES-D and the other for major depression. The models are estimated over the entire sample and include basic demographic controls. The coefficients from the first model indicate a significant non-linear relationship between month of birth and depressive symptoms, with depression rising and then falling over the approximate midpoint of the year. The second model, for major depression, shows the same non-linear pattern, though the effect is not statistically significant and is substantively small. To illustrate this, Fig. 1 presents expected values from both models, arrayed over months, with two y-axes, one for each dependent variable. Depression rises to a peak in June and July before declining again. In total, though, the effect is very small. The span of the relationship is a little less than one unit (January = 8.55 and July = 9.21) for the CES-D and half a percentage point (January = .1719 and July = .1747) for major depression.

Table 3 presents models that introduce a more rigorous set of controls, beginning with geography. The table focuses on the CES-D (the only outcome for which the initial differences were significant) and presents four models. Model 1 controls for Northern states. Model 2 controls for the centroid latitude of the state of birth. And Model 3 uses

Table 2
Regression models of depression on month of birth and control variables, NHANES-I.

	CES-D	Major Depression
Age	-.010	.003
	(.045)	(.016)
Age^2	.0001	-.0001
	(.0005)	(.0002)
Female	2.892***	.911***
	(.162)	(.065)
Black	2.918***	.800***
	(.382)	(.134)
Birth Month	.286**	.009
	(.110)	(.037)
$Birth\ Month^2$	-.022*	-.0007
	(.009)	(.0028)
Constant	6.660***	-2.140

Note: Model for CES-D is linear regression, whereas model for major depression is logit regression.
* $p < .05$,
** $p < .01$,
*** $p < .001$ (two-tailed test, standard errors in parentheses). N = 3033

Table 3
Regression of CES-D on birth month and assorted geographic and socio-demographic control variables, NHANES-I.

	Model 1	Model 2	Model 3	Model 4
Age	-.001	.027	-.006	-.003
	(.044)	(.045)	(.046)	(.046)
Age^2	.0000	-.0003	.0000	-.0001
	(.0004)	(.0005)	(.0005)	(.0005)
Female	2.968**	2.944***	2.946****	2.978***
	(.162)	(.173)	(.159)	(.160)
Black	2.614***	2.547***	2.696***	2.671***
	(.384)	(.374)	(.435)	(.433)
Birth Month	.263*	.321**	.254*	.250*
	(.109)	(.107)	(.109)	(.107)
$Birth\ Month^2$	-.020*	-.025***	-.019*	-.019*
	(.009)	(.007)	(.008)	(.008)
Northern State	-1.487***			
	(.310)			
State Centroid Latitude		-.126***		
		(.020)		
Rural	-.322	-.091	-.323	-.310
	(.164)	(.189)	(.171)	(.170)
High School +				-.557**
				(.183)
Constant	7.001***	10.669***	8.096***	8.100***
State Fixed Effects			√	√
N	3033	2820	3033	3033

Note: Model 2 restricted to respondents born in US states.
* $p < .05$,
** $p < .01$,
*** $p < .001$ (two-tailed test, standard errors in parentheses).

state fixed-effects, effectively a dummy variable for each state (minus one). Altogether these specifications capture geographic differences in an especially fine-grained fashion, but none alters the relationship between birth month and the CES-D in a meaningful way. Although it would also be useful to control for socioeconomic conditions at birth, the data do not contain such indicators. Model 4, however, controls for a reasonable proxy: the education of the respondent. This control variable, too, does not significantly alter the relationship between birth month and the CES-D.

Together Tables 2 and 3 demonstrate a relationship between birth month and depression, not entirely dissimilar to what has been shown (in some studies) for schizophrenia. The specification of the models, however, implies an average effect, realized over multiple birth years and different social/geographic conditions. If, as anticipated, the relationship between birth month and depression has attenuated over the course of the 20th century, the average relationship between birth month and depression will reflect a blend of stronger effects from births earlier in the century with much weaker effects from births later on. Table 4 tests this possibility directly by introducing multiplicative interactions between birth month and year of birth. It returns to the specifications presented in Table 2 and presents models for both the CES-D and major depression. Multiplicative interactions are included for both components of the birth-month specification: month of birth and month of birth-squared. Because age is determined by birth year, the age covariates are not included in these models (though, of note, the

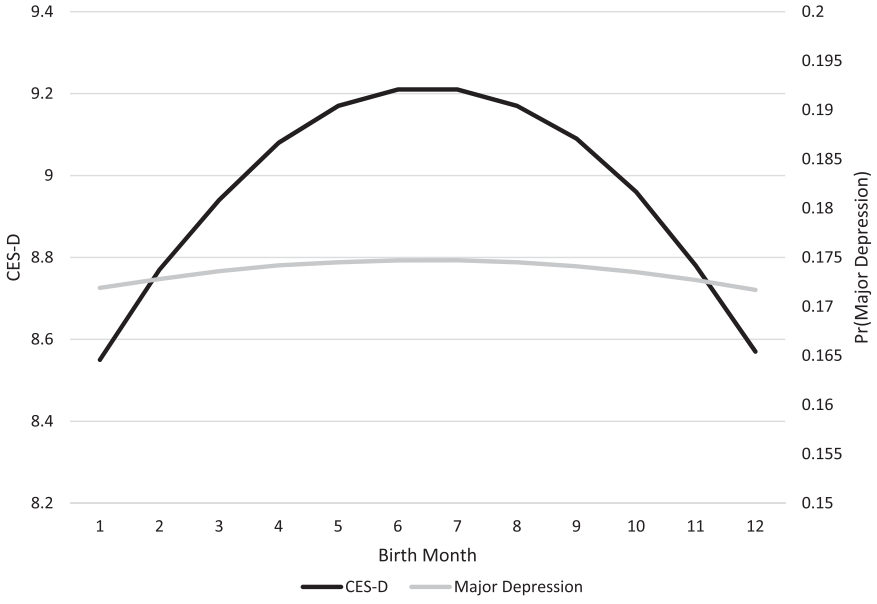

Fig. 1. Relationship between month of birth and depression, NHANES-I. *Note*: Predicted values from models presented in Table 2.

Table 4
Coefficients from regression of CES-D and major depression on birth month and interactions with birth year, NHANES-I.

	CES-D	Major Depression
Birth Year	.062**	.035**
	(.021)	(.011)
Birth Month	1.159***	.400***
	(.239)	(.081)
Birth Month^2	-.092***	-.032***
	(.019)	(.005)
Birth Month × Birth Year	-.031***	-.013***
	(.008)	(.003)
Birth Month^2 × Birth Year	.002***	.001***
	(.001)	(.000)

Note: Models also include controls for sex and race (coefficients not shown).
*$p < .05$,
** $p < .01$,
*** $p < .001$ (two-tailed test, standard errors in parentheses). N = 3033

age coefficients were not statistically significant in the models presented in Table 2). The models yield highly significant interactions. Because year of birth begins at zero, the main effects of birth month can be interpreted as the relationship between birth month and depression among those born in 1900. For both the CES-D and major depression, the main effect is significant and large, though the interactions indicate a graduate weakening of the relationship over time.

Fig. 2 illustrates this change. It presents predicted values from the model, based on five different decennial birth years. For clarity, the figure only presents values for the CES-D, although the results for major depression are similar. The relationship between birth month and depression was very strong in 1900, but was eliminated sometime between 1930 and 1940. Although the results also indicate that those born in the winter months show some elevation in depression over time—and thus an apparent loss of a protective effect—this elevation is a result of a significant positive relationship between year of birth and depression in the fully interacted model. In deriving expected values, the value of year of birth is allowed to change. If the same value for year is assigned for both cohorts (even as the values for the interaction are allowed to differ), those born in January or December of 1940 report lower depression than those born in the same months in 1900 (6.448 and 6.722 relative to 7.584 and 7.184 respectively).

Table 5
Coefficients from regression of CES-D on interactions between birth month and select moderators among those born 1900 to 1920, NHANES-I.

	Model 1	Model 2	Model 3	Model 4
Main Effect of Moderator	1.727	4.367***	1.213	-.273**
	(1.071)	(1.251)	(1.438)	(.086)
Birth Month				
Birth Month	1.027***	1.051***	1.185***	-.048
	(.301)	(.319)	(.285)	(1.425)
Birth Month^2	-.092***	-.095***	-.109***	-.031
	(.021)	(.023)	(.020)	(.100)
Interaction with Moderator				
Birth Month	-.754*	-1.378**	-1.308**	.020
	(.310)	(.513)	(.469)	(.038)
Birth Month^2	.052*	.109**	.113***	-.001
	(.022)	(.036)	(.033)	(.003)
Moderator	Rural	Southern State	High School +	State Centroid Latitude

Note: Models also include controls for sex, age, age-squared, and race (coefficients not shown).
* $p < .05$,
** $p < .01$,
*** $p < .001$ (two-tailed test, standard errors in parentheses). N = 966 (Models 1 through 3) and 892 (Model 4).

A plausible interpretation of the elimination of the birth month effect pertains to seasonal deficiencies in nutrition. If this were the case, there would be other interactions with month of birth, corresponding to different patterns in the food supply by season. In the early 20th century food supply was generally more constrained by the seasons than it was by mid-century, but not all regions experienced equally strong seasonal variation. In general, urban areas had a greater variety of foods available year round. In addition, the South experienced relative consistency in diet over the seasons. Finally, seasonal fluctuation was less significant for those with sufficient financial resources, here indicated by respondent education. Table 5 presents interactions between all three of these characteristics and birth month and birth month-squared. The sample is limited to those born between 1900 and 1920.

The interactions are all significant, indicating that the relationship between birth month and depression is shallower for three critical

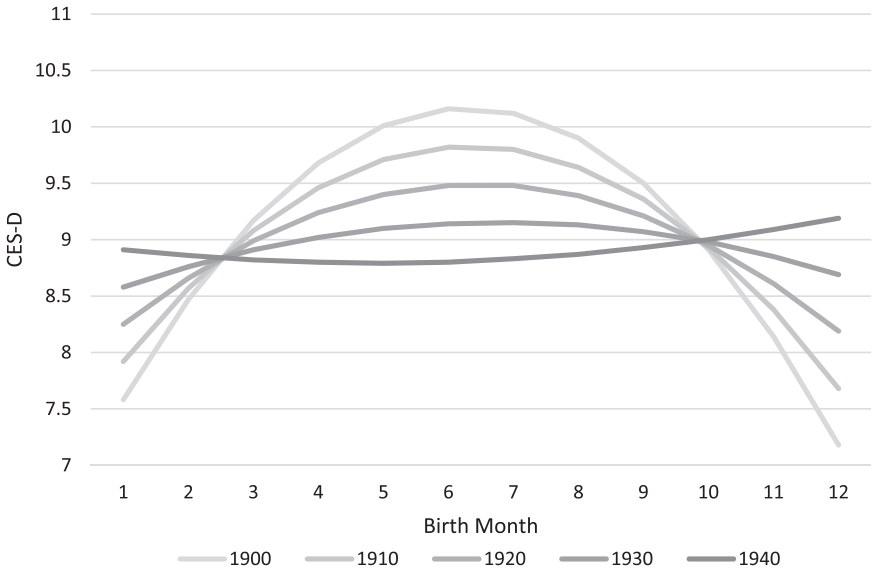

Fig. 2. Changing relationship between month of birth and CES-D by birth cohort, NHANES-I. *Note*: Predicted values from model presented in Table 4.

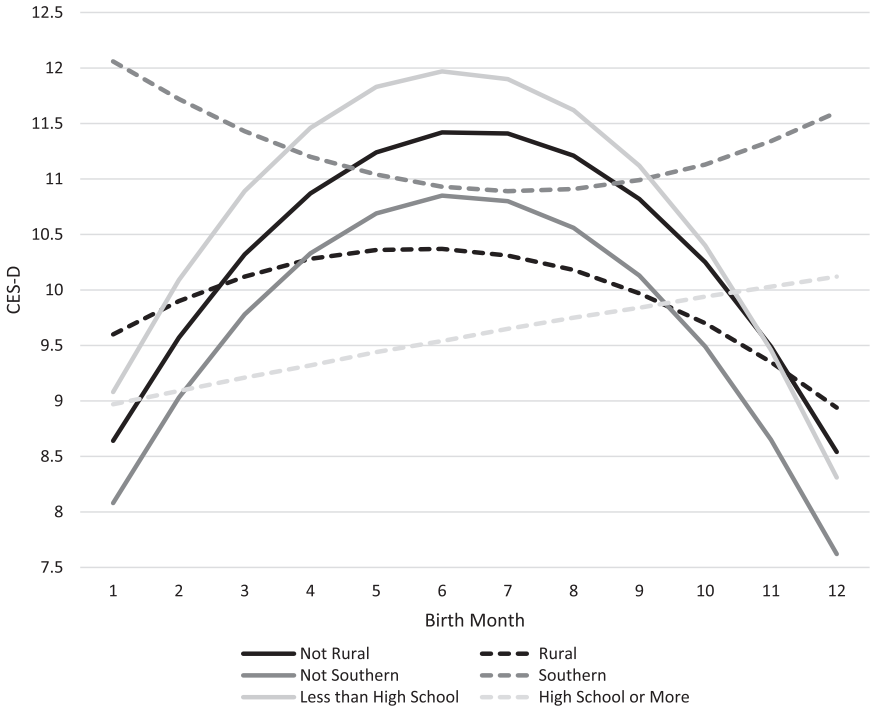

Fig. 3. Relationship between month of birth and CES-D among select subgroups, 1900 to 1920 birth cohort, NHANES-I. *Note*: Predicted values from Models 1 through 3 of Table 5.

groups: those living in rural areas, those born in the South, and those with at least a high school degree. In all three groups, the interpretation is likely premised on the same thing: for these groups, the diet of pregnant women is expected to be less sensitive to seasonal variation (though not always "better" than the diet for the reference category). To clarify this interpretation, Fig. 3 presents expected values. One line is presented for each of the six groups (the group represented by the dummy-variable, as well as the group represented by its reference category). Relative to those with less than a high school degree, those with at least a high school degree report lower levels of depression for virtually every birth month. Conversely, those born in the South generally report more depression than those not born in the South, even though depression in the South is much less sensitive to birth month effects. Table 5 presents one additional interaction. If the effects of birth month were premised on maternal sunlight exposure, the most powerful interaction would be between month of birth and latitude, with a sharper effect of birth month in states at higher latitudes. The final model, however, reveals no such interaction.

Recall that an additional reason for skepticism surrounding the effect of birth season pertains to the magnitude of the effect rather than its statistical significance. Table 6 explores the issue of magnitude, using attributable risk comparisons (see Newson 2013 for exposition, formulas, and statistical programs). In these calculations, the dependent variable is major depression, though the models are estimated in a somewhat different fashion from what has been presented thus far. To allow for population attributable risk calculations, the birth month effect is estimated using a single dummy variable, corresponding to a birth between April and October. Using a model so-specified as the baseline, the table presents a variety of alternative scenarios. It presents three percentages for each of four cohorts: the first corresponding to the expected prevalence of major depression for that cohort, based on predicted values from the model; the second corresponding to the expected prevalence of major depression if all births in that cohort were confined to November through March (and, thus, no high-risk April to October births); and the third corresponding to the percent reduction in the burden of major depression under this alternative scenario relative to the actual one. The table reveals the progressive elimination of the month of birth effect over time, but also the enormous role that month

Table 6
Population attributable risk percentages over alternate birth cohort and birth month scenarios, NHANES-I.

	Odds Ratio for April to October Birth [95% CI]	Expected Prevalence of Major Depression	Expected Prevalence of Major Depression with No April to October Births	Population Attributable Percentage [95% CI]
Birth Cohorts				
1900 – 1910	1.823 [1.448 - 2.295]	15.2	11.3	26.0 [16.2 - 34.6]
1900 – 1920	1.615 [1.373 - 1.899]	16.6	13.0	21.5 [14.3 – 28.1]
1900 – 1930	1.218 [1.049 - 1.414]	17.0	15.5	8.9 [2.0 – 15.4]
1900 – 1940	1.264 [1.078 - 1.482]	17.2	15.4	10.5 [3.1 - 17.3]
1900 – 1950	1.150 [1.021 - 1.295]	17.4	16.3	6.3 [.9 – 11.4]

of birth played in the epidemiology of major depression in the early and mid-20th century. Among those born between 1900 and 1910, for instance, the burden of major depression in adulthood would have been reduced by 26% if all births had been confined to November through March. This impact was nearly as large for births up to 1920, though it diminished quickly thereafter.

In evaluating the magnitude of the month of birth effect, another population-level comparison sheds additional light. Education is an especially powerful predictor of depression. It remains among the most important fundamental causes of mental health in social epidemiology. Yet the population-level impact of education is smaller than that of season of birth. If everyone in the NHANES-I sample had attained at least a high school degree—at a time when only about 35% of adults did—the prevalence of major depression would be reduced by about 10% (models not shown). In other words, the population attributable risk associated with education is less than half the risk associated with month of birth.

Discussion

Although the study of birth seasonality in psychiatric disorders is no longer prominent, the study of the in-utero determinants of health is thriving. This study provides an empirical explanation for this discontinuity, and it does so without discrediting the idea of seasonality altogether. Although season of birth effects in depression were very strong in the early 20th century, they faded in relevance as seasonal deficits in nutrition were eliminated. This finding can be interpreted in terms of its implications for understanding the history of depression in the US and in terms of its implications for 21st century social epidemiology. On the one hand, the results point to a parallel between depression and other developmental problems in the early 20th century. At the time, the impact of nutritional deficits were certainly not limited to depression, nor, for that matter, to adults. They were also apparent in high levels of morbidity from pellagra, goiter, and rickets, which were especially common among children (Centers for Disease Control, 1999). Public health initiatives eventually reduced or eliminated such diseases, and it is possible that, in so doing, these efforts also reduced depression as these children became adults, even if this reduction was not anticipated by medical professionals. In this way, the results speak to ongoing debates on the role of science in understanding modern health improvements. Psychiatric disorders are usually not part of that discussion, given how the scientific understanding of psychiatric disorders has generally lagged behind the understanding of many other forms of morbidity. Nonetheless, the two are related and deserve parallel attention.

On the other hand, the findings point to the significance of in-utero determinants well into the contemporary era. Seasonality and nutrition are, of course, not the sum total of potential in-utero patterns and influences, and simply because some influences have faded from relevance does not mean that others have not taken their place. The results do not mean that maternal nutrition is now unrelated to depression in offspring. They only reveal the fading relevance of seasonality. In demonstrating the relevance of one kind of in-utero influence, this study further solidifies the case for considering others. The results also encourage more exploration of the population-level implications of such determinants, which should not be neglected. Scientists have been keen to identify designs appropriate for testing in-utero effects, recognizing the relevance of confounding and how may common in-utero insults are often correlated with a host of other risk factors. In this light, scientists have been drawn to large-scale natural experiments, such as famines and pandemics, which allow for a strong empirical test. These events, however, affect a relatively small segment of the population and only at a certain time. Other in-utero risk factors are more difficult to study, though will have a larger effect at the level of the population. A focus on historically unusual influences should not obscure an interest in more typical contemporary influences or attempts to uncover their relevance empirically.

Limitations

This study has several imitations. First, it was only able to make the case for the influence of diet in a conjectural fashion. It included no direct measures of diet and, rather, inferred it from the intersection of information on year of birth, month of birth, and other birth characteristics. With some of the same data points, another possible interpretation is exposure to seasonal infections. Influenza has well-established seasonal patterns, as does typhoid. Furthermore, at least for typhoid, the incidence of the disease decreased precipitously over the early 20th century, overlapping, then, with the between-cohort patterns found here (Harmon 1930). The relevance of infections is tempered, however, by at least two things. For one, if flu infection were the primary mechanism, the interactions with birth cohort would likely be insignificant. The flu season is a highly regular occurrence, and apart from occasional pandemics, including the 1918 pandemic, there was not a sharp decline in flu infections over the early 20th century. Typhoid shows seasonal patterns, too, and did, in fact, decline early in the century, but it affected far fewer people than influenza. At its height, in 1921, the incidence of typhoid was only about 44 per 100,000 people (Centers for Disease Control, 1999, Fig. 1). In order to account for the birth month effects found here, typhoid's in-utero effects would need to be implausibly large. Furthermore, in the case of both influenza and typhoid, we would not expect the other moderators to be significant or in the direction found here. The flu tends to spread rapidly over space, usually in just over 5 weeks (Viboud et al., 2006), and, if anything, the infection rate is higher in urban areas than rural ones (Paynter, Ware, & Shanks, 2011). This situation would lead to interactions in the opposite direction from those found here, indicating more of an effect of birth month in urban areas. Of course, direct measures of in-utero conditions and exposures would alleviate these concerns and the need to rely on conjecture.

In addition, this study inferred the significance of in utero effects, though it is possible they reflect neonatal effects. Much of the evidence has focused on in utero effects and, during that period, there are a number of plausible pathways to link nutritional deficiencies to depression. Nonetheless, even if we accept that the sensitive period is in utero, this study is imprecise about the most sensitive gestational period. The findings presented here differ from previous studies in at least one important respect. This study found a peak in the risk for depression occurring in spring-summer, which is later than what is usually uncovered for other disorders (Torrey et al., 1997). In research on birth seasonality in schizophrenia, for instance, the peak is usually from December to March. To be sure, some evidence points to a later risk period for depression in particular, but usually this period does not extend into late summer. One study of suicide, for instance, found an excess risk occurring over a somewhat later window, births from April to May (Salib & Cortina-Borja, 2006). One possible explanation for these differences centers on differences between disorders in the most developmentally sensitive trimester: in-utero factors might matter more in the third trimester for schizophrenia (corresponding to deficits in the winter and births in the late winter and early spring) and more in the first trimester for depression (corresponding to deficits in the winter and births in the summer and early fall). Another explanation for these differences, though, is embedded in a different aspect of the findings presented here: even if all studies of birth season effects are capturing nutritional deficiencies, the peak of such deficiencies is likely to vary over the many axes adumbrated in this study, including time, geography, and sociodemographic position. This variation ultimately leads to a good deal of imprecision in timing, and little can be firmly concluded regarding the precise trimester of exposure if the exposure itself is a moving target.

The use of more direct and precise measures would improve confidence in other ways, too. For instance, more precise indicators of

location of birth would improve the precision surrounding the effects of, for instance, latitude. In addition, family background measures would alleviate the need to use current respondent characteristics as proxies for past experiences. All this points to some trade-offs in the study of in-utero determinants, at least when using existing surveillance or observational data. In particular, there are trade-offs in the adequacy of control variables for evaluating in-utero conditions relative to evaluating contemporaneous adult conditions. One reason the study of birth seasons is attractive is because many popular datasets contain information on month of birth, even if they contain few other measures of birth conditions. This is not an uncommon situation in the in-utero determinants literature more generally. One reason Barker initially focused on birthweight is because the data were easy to collect. The same sort of necessity applies to studies exploring the lingering consequences of in-utero exposure to flu pandemics (e.g., Almond, 2006). These studies often contain rich information regarding the adult social environment, appended to more limited information regarding in-utero exposures. Improving the study of in-utero determinants will require data explicitly designed to do so, rather than relying on data that are coincidentally useful for the purpose.

Finally, this study is based on cross-sectional data. The interpretation of the interaction between age and month of birth rests on a cohort interpretation. The justification for this is based on previous research and from some empirical considerations. In the models (see Table 3), age and age-squared do not themselves have a significant relationship with depressive symptoms. Although this insignificance lends credence to a cohort interpretation with respect to the interactions, the insignificant main effects are inconsistent with previous research. Previous research ordinarily finds a u-shaped relationship between age and depressive symptoms (Mirowsky & Ross, 1992). It is possible that the effects of month of birth are masked between cohorts: they only emerge later in life and, therefore, are not yet apparent in the youngest members of the NHANES-I sample. Longitudinal data could shed light on this issue, though such a pattern would be unusual with respect to the descriptive epidemiology of depression. The median age of onset for major depression is usually under the age of 30, suggesting most people in the NHANES-I sample who will ever develop depression have developed it already (Kessler et al., 2005).

Conclusion

This study demonstrates both the value of thinking about in-utero conditions as determinants of adult depression and the value of thinking about social epidemiology in a historical framework. One key and often underappreciated aspect of the fundamental cause approach is that it alerts scholars to the value of thinking about history. In making the case for social conditions as fundamental causes, Link and Phelan (1995) emphasize the persistence of social determinants over time, even as the underlying mechanisms that explain those determinants change. In this sense, their approach is premised on an appreciation of the passage of time, though this idea is worth expanding more than it usually is. It is worthwhile, for instance, to also consider the ebb and flow of the kinds of early-life determinants that might shape both adult health and adult social conditions. If scientists appreciate that in-utero causes are real, they might also appreciate that such causes are as contingent in their effects as other more well-established contemporaneous causes. It is also worthwhile to think about the political and economic conditions that set the stage for in-utero effects. To the extent that birth seasonality in depression reflects nutritional deficiencies, for instance, it is important to not regard such deficiencies as strictly proximate or biological determinants. At the time they were apparent, birth season effects were almost certainly supported by a host of more upstream influences, including a weak scientific understanding of nutrition, poverty that prevented a more varied and nutritionally compete diet, regional differences in food supply, and access to technology that could aid food preservation. Given all these upstream influences, the study of in-utero determinants—even when construed in terms of deficiencies in specific vitamins and nutrients—is entirely compatible with a widescreen fundamental cause approach.

It is also important to consider how historical influences can shape research agendas, including what topics attract scientific attention and what questions scientists ask. It is perhaps no coincidence that a more environmental approach to the study of psychiatric disorders emerged at approximately the same time as when the influence of one particularly important in-utero influence was fading. At the moment when psychiatric patients appeared less likely to have been born with their disorders, scientists might have gravitated toward considering other causes, shifting their focus from endowments to attainments. Being attentive to historical change in the environment could prevent scientists from developing epidemiological models that are cast in too transhistorical a fashion. The past can shape the future in more ways than one.

Ethical statement

I have abided by the ethical guidelines for publication in SSM – Population Health. I have no interest or conflict that might bias this work. And I did not receive any outside funding for the research.

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