Economics and Human Biology xxx (2012) xxx–xxx

Contents lists available at SciVerse ScienceDirect

Economics and Human Biology

journal homepage: http://www.elsevier.com/locate/ehb

Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes

Orlando Sotomayor *

University of Puerto Rico at Mayagüez, PO Box 9000, Mayagüez 00681, Puerto Rico

ARTICLE INFO

Article history:
Received 9 May 2011
Received in revised form 29 February 2012
Accepted 29 February 2012
Available online xxx

JEL classification:
I0
I12

Keywords:
Fetal origins
Hurricanes
Puerto Rico
Barker hypothesis

ABSTRACT

A natural experiment is employed to analyze the relationship between living standards, diabetes, and cardiovascular disease. Results show that shocks generated by two powerful tropical storms striking Puerto Rico during the late 1920s and early 1930s had long-term consequences consistent with the fetal origins hypothesis. Individuals in the womb or early infancy in the aftermath of the storms are more likely to report a diagnosis of hypertension, high cholesterol, diabetes, and are considerably more likely to have no formal schooling.

1. Introduction

Although some time has passed since the fetal origins hypothesis was first put forth (Barker, 1992), only recently has the field of economics contributed to the assessment of the relationship between uterine environment and later life outcomes (Almond, 2006). Adult health, disability, mortality, cognitive ability, or socio-economic status have been linked to in utero or early infancy exposure to famine, influenza, malaria, recessions, pollutants, dietary deficiencies, and Ramadan observance.[1] However, the pathways from factors affecting early life environment to outcomes in adult life are far from clear. The fetal origins hypothesis provides a potential link through biological reactions to early life deprivation that are posited to predispose individuals to diabetes, hypertension, high cholesterol, and cardiovascular disease, conditions that are leading causes of morbidity and mortality in the developed world (WHO, 2002).

Work on the biological mechanisms underpinning the predictions of the hypothesis is owed to Barker (1992). Nutrition deficiencies were posited to result in increased blood pressure required for maintaining transfer capacity across the placenta (Barker et al., 1989). High cholesterol could develop from changes in lipid metabolism in response to the same circumstances, with both conditions persisting after birth (Barker et al., 1993). Changes in fetal programming giving priority to certain organs at the expense of others like the liver were suspected to have permanent consequences on glucose and insulin metabolism (Hales et al., 1991). Research was of particular interest as it could provide an explanation for syndrome X – the association between non-insulin dependent diabetes, hypertension, and high blood cholesterol – in the form of a common origin of all three conditions. Empirical support for the proposal would also have important public health implications. Prevention of development of diabetes and cardiovascular disease would not necessarily center on moderating risk behaviors in adult life.

* Tel.: +787 649 1360.
E-mail address: orlando.sotomayor@upr.edu.

[1] See Almond and Currie (2011) for a survey.

1570-677X/$ – see front matter © 2012 Elsevier B.V. All rights reserved.
doi:10.1016/j.ehb.2012.02.009

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

Initial empirical findings pointed to an association between markers of early life nutrition, impaired glucose tolerance, increased serum cholesterol, and hypertension in adult life (Barker et al., 1989, 1993; Hales et al., 1991), but causal inference was questioned since results could have been generated by third factors (Rasmussen, 2001). In search of less problematic data settings, research turned to natural experiments for additional supportive evidence, with mixed results. While individuals exposed in utero to the 1944–1945 Dutch famine develop diabetes and coronary heart disease with greater frequency (Ravelli et al., 1998; Roseboom et al., 2000), survivors of the nineteenth century Finnish famine did not suffer from additional mortality risk (Kannisto et al., 1997). Moreover, a large medical study evaluating an extensive set of health outcomes did not establish long-term consequences related to the 1941–1944 Leningrad siege (Stanner et al., 1997), and systematic literature reviews were unable to establish material effects of fetal conditions on adult blood pressure or cholesterol levels (Huxley et al., 2002, 2004).

On September 13, 1928, hurricane San Felipe struck Puerto Rico bringing 10 inches of rain in the lowlands and up to 30 inches in the mountains over a period of two days. The San Juan weather station recorded an extreme wind velocity of 160 miles per hour and speeds over the island's higher elevations could have reached 200 miles per hour (Fassig, 1929). Damage to the Island's dominant agricultural sector was widespread, but the fact that it was concentrated in the 1928–1929 agricultural season provides the opportunity for defining exposed versus control groups in a natural experiment setting. Moreover, surveys providing information on a variety of health outcomes allow for direct tests of relationships between living standards, diabetes, hypertension, high cholesterol, and cardiovascular disease.

2. Survey of current research

In economics, research has produced more consistently supportive evidence of a relationship between early life conditions and a range of long-term outcomes. The 1918 Influenza Pandemic has been linked to reduced schooling, income, and increased rates of disease and disability (Almond and Mazumder, 2005; Almond, 2006). Exposure to the 1957 Asian influenza pandemic led to reduced cognitive ability and impaired growth among mothers who smoked (Kelly, 2011). The 1959–1961 Chinese famine has been tied to worsened literacy, labor market and marriage outcomes, and the nineteenth century Dutch potato famine to reduced life expectancy (Almond et al., 2007; Chen and Zhou, 2007; Lindeboom et al., 2010). Exposure to low levels of radiation from the Chernobyl reactor disaster translated to impaired neural development in Swedish children, iodine deficiency in Tanzania has led to reduced schooling, and exposure to malaria to lower income and higher poverty in the US (Almond et al., 2009; Field et al., 2009; Barreca, 2010). Even fasting during Ramadan has been related to poorer general health, disability, and symptoms indicative of diabetes and cardiovascular disease (Almond and Mazumder, 2011; van Ewijk, 2011). The latter study is a rare example of economic research addressing the direct implications of the Barker proposal.

Consequences of economic crises or income shocks have also been evaluated. Long-term health effects have not been associated with the severe drought and soil erosion striking the American Midwest during the 1930s, or with the phylloxera plague that decimated French vineyard production late in the nineteenth century (Cutler et al., 2007; Banerjee et al., 2010). However, weather shocks and recessions occurring during a person's infancy have been tied to height, health, and mortality (Block et al., 2004; del Ninno and Lundberg, 2005; van den Berg et al., 2006; Maccini and Yang, 2009; Skoufias and Vinha, 2011).

Exposure periods can be extended as in the case of studies examining later life effects of childhood asthma or the long-term consequences of the Head Start program (Currie and Thomas, 1995; Garces et al., 2002; Ludwig and Miller, 2007; Fletcher et al., 2010). Outcomes can also range from cognitive abilities, labor market outcomes, to welfare use (Currie and Hyson, 1999; Black et al., 2007; Case and Paxson, 2008a,b; Smith, 2009; Currie et al., 2010).

Even adult behaviors can be conditioned by childhood experiences such as disruptive family life (Belsky et al., 1991). In an evolutionary sense, early onset of menarche, an increased reproductive rate, and low offspring investment can obtain as adaptations to unstable or hazardous environments. Life history theory posits that when reproductive potential appears threatened, individuals may respond by adopting a fast life course emphasizing offspring quantity versus a slow one that would have emphasized quality. Empirical evidence establishes that across countries fertility is associated with indicators of mortality risk (Guégan et al., 2001), and that within societies parental investment in children is lower in more deprived neighborhoods (Nettle, 2010). Interpretation and processing of environmental cues is influenced by circumstances experienced during critical periods in childhood (Belsky, 2007). Reactions to stress and attitudes towards risk, for example, have been shown to depend on childhood rather than current socioeconomic status (Miller et al., 2009; Griskevicius et al., 2011).

3. The Puerto Rican economy and hurricane San Felipe

In the 1920s Puerto Rico was an agrarian economy based largely on sugar production. *Per capita* income was about a fifth of that of the US but higher than that of the neighboring island of Cuba, and several times that of Haiti or the Dominican Republic (Perloff, 1950:3). According to the US Census, agriculture accounted for 60% of employment in 1920, sugar cane alone accounting for 20% of all employment and 50% of crop value. Coffee was a secondary and declining economic activity relegated to the mountainous interior, unsuitable to large scale cane cultivation. Its contribution to crop value in 1920 was equivalent to that of fruits and starchy vegetables (18%).

The San Felipe hurricane caused damages equivalent to about a third of national income (Pérez, 1971). Loss of life was low but damages to agriculture were vast. The coffee

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

and citrus crops were almost entirely wiped out although most of the citrus trees survived (Fassig, 1929). Cane exports declined by 36% in 1929, but in being less susceptible to long-term damage, rebounded in 1930 to almost pre-hurricane levels (Dietz, 1986: Table 2.4). More than three quarters of agricultural sector employment consisted of farm laborers in seasonal employment and some of the displacement in coffee employment could have been absorbed by other agricultural activities with shorter recovery periods and by migration to urban areas.[2] US aid of $4 million in hurricane relief and $6 million in agricultural loans (Perloff, 1950:31) provided some counterweight to the income shock and helped avoid coincidental repercussions such as major outbreaks of disease. The American Red Cross supplied food, shelter, vaccines, medical personnel, emergency hospitals, job search help, and seeds for small farmers.[3]

In 1932 a second storm struck the island in late September with winds of 120 mph and causing damages of $30 million (Pérez, 1971). Although economic losses equivalent to 20% of national income were not as great as those caused by San Felipe, hurricane San Ciprián coincided with the trough of the Great Depression. During that time net income declined gradually from 1930 to 1933 and recovered during the remainder of the decade, regaining 1930 levels by 1936 and *per capita* national income by 1940 (Perloff, 1950:159). Compared to a peak to trough decline of 46% in the US, national income fell by 24% in Puerto Rico (Dietz, 1986:137). Since then, severe hurricanes have included Santa Clara in 1956 and Hugo in 1989.

4. Data

Data are derived from 1996 to 2008 Center for Disease Control's Behavioral Risk Factor Surveillance System surveys. Since collection began in 1996, they constitute the Island's main source of information on health conditions and risk behaviors of the adult non-institutionalized population. Analysis is based on individuals born between 1920 and 1940 in order to allow for a range of potential control groups to be used in estimating effects related to the 1928 San Felipe and the 1932 San Ciprián storms. As the hurricanes decimated the 1929 and 1933 crops, exposure is defined as being born in either year. The original fetal origins literature placed particular emphasis on shocks in late gestation and early infancy (Hales et al., 1991; Barker, 1992; Barker et al., 1993), but more recent research suggests mechanisms precipitating changes in the early embryo (Gluckman and Hanson, 2005). Month or quarter of birth variables would have allowed this study to evaluate timing effects, but such information is unavailable.

The sample totals 11,990 observations out of which 1197 consist of affected individuals. Cell sizes range from 237 records for individuals born in 1920 to 789 for those born in 1940 (Appendix A). Almost all observations contain demographic and outcome variables such as schooling, height, and diabetes diagnosis. Information regarding outcomes such as hypertension and high cholesterol is associated with questions surveyed every other year and cardiovascular disease diagnoses with questions included in the 1997, 2003, 2005, 2006, and 2008 surveys. Specifically, 99.7% of observations contain information on diabetes, 45% on blood pressure, 42% on cholesterol levels, and 41% on cardiovascular disease. Summary statistics presented in Table 1 provide preliminary support for the predictions of the fetal origins hypothesis. Birth in 1929 or 1933 is associated with increased prevalence of diabetes, high blood cholesterol, and hypertension. Mean differences are precisely estimated and substantial, especially in the case of diabetes, whose incidence is 22% higher among the affected population.[4] Gender-specific effects point in the same direction, although in some cases they cannot be estimated with great precision. Among the outcomes with the largest number of observations, exposure effects on diabetes, schooling, and height appear more pronounced among women.

5. Method

The study employs a natural experiment setting and follows the regression discontinuity framework in order to estimate causal effects. Exposure is related to exogenous events generating shocks in living conditions and causal inference depends on the crucial assumption of continuity. Other factors affecting health should not change abruptly around the shock, taken in this study as being in the womb or early infancy in the aftermath of a severe hurricane. If that is the case, the only average difference between individuals born in that that year and those born in a sufficiently close range around the year will be exposure to the conditions brought about by the hurricane.

The exposure effect is then the difference in outcome values between affected and unaffected individuals constructed through extrapolation of a conditional expectation function. For example, Fig. 1 displays a profile of observed incidence of diabetes as a function of year of birth as well as a conditional expectation function estimated through use of a fifth order polynomial in year of birth with indicator variables for 1929 and 1933. Diabetes prevalence among individuals unexposed to hurricanes in early life is unobserved for cohorts born in these two years, but counterfactual values can be generated by extrapolating from the conditional expectation function.[5]

$$y = \alpha + X\delta + D\beta + Z\gamma + \varepsilon \quad (1)$$

[2] Between 1920 and 1930 population growth in the three largest cane producing areas was on pace with that of the Island at 19%, while that on the three largest coffee producing areas amounted to 6%.

[3] Aid was also forthcoming from neighboring countries, US businesses, and local sources such as churches. See *El Mundo* newspaper, September 17–29, 1928.

[4] Individuals are much shorter relative to their US counterparts. Although average height on the Island increased by 1.2 centimeters between cohorts born in 1886–1890 and 1926–1930, height increased to a greater extent in the US. During the same time span the height gap rose from 6 to 10 cm (Godoy et al., 2007).

[5] The need for extrapolation is the reason behind the importance of the continuity assumption.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

Table 1
Summary statistics.

Variable	Born in 1929 or 1933		Not born in 1929 or 1933	
	Mean/share	Std. error	Mean/share	Std. error
Age	70.5	0.121	69.7	0.064
Height (in.)	64.3	0.106	64.3	0.037
Short stature	0.183	0.012	0.144	0.004
Female	0.553	0.014	0.561	0.005
Schooling				
None	0.055	0.007	0.033	0.002
1–11 years	0.490	0.014	0.518	0.005
12+ years	0.454	0.014	0.448	0.005
Diagnosis of				
Diabetes	0.320	0.013	0.263	0.004
Hypertension	0.600	0.021	0.539	0.007
High cholesterol	0.504	0.022	0.443	0.007
AMI	0.147	0.016	0.118	0.005
Coronary/angina	0.173	0.017	0.161	0.006
Stroke	0.041	0.009	0.039	0.003
Women				
Age	70.4	0.160	69.9	0.081
Height (in.)	62.2	0.104	62.3	0.035
Short stature	0.325	0.018	0.256	0.006
Schooling				
None	0.071	0.009	0.041	0.002
1–11 years	0.543	0.018	0.555	0.006
12+ years	0.385	0.018	0.402	0.006
Diagnosis of				
Diabetes	0.333	0.017	0.264	0.005
Hypertension	0.557	0.027	0.567	0.009
High cholesterol	0.526	0.028	0.498	0.009
AMI	0.115	0.018	0.094	0.005
Coronary/angina	0.169	0.021	0.157	0.007
Stroke	0.051	0.012	0.041	0.004
Men				
Age	70.6	0.188	69.5	0.107
Height (in.)	66.5	0.139	66.7	0.051
Short stature	0.026	0.008	0.014	0.002
Schooling				
None	0.035	0.009	0.022	0.002
1–11 years	0.426	0.023	0.471	0.008
12+ years	0.537	0.024	0.504	0.008
Diagnosis of				
Diabetes	0.305	0.022	0.262	0.007
Hypertension	0.653	0.033	0.503	0.012
High cholesterol	0.476	0.037	0.369	0.012
AMI	0.190	0.030	0.150	0.009
Coronary/angina	0.180	0.029	0.166	0.010
Stroke	0.028	0.012	0.037	0.005

Note: The sample is composed of individuals born between 1920 and 1940.

Fig. 1. Observed incidence of diabetes by year of birth and estimated conditional expectation function.

Expectation functions can be modeled in many ways and in order to evaluate robustness of results, parametric and non-parametric approaches are employed in this analysis.[6] In the case of the former, the empirical model is specified in Eq. (1), where y represents outcomes such as diabetic status, X is a vector of polynomial terms in year of birth, D indicator variables for birth in 1929 and 1933, β is the parameter of interest, and Z is a set of controls that

[6] Parametric approaches can involve use of global or piecewise polynomials, but unbiased estimation hinges on the use of the correct functional form. Nonparametric approaches such as kernel regressions or mean comparisons in small neighborhoods around the cutoff reduce the importance of the correct form requirement but introduce another type of bias related to the fact that they do not take into account the slope in the forcing variable. As a solution to the problem Hahn et al. (2001) suggest the use of local linear regressions.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

Fig. 2. Average height in inches by year of birth (top) and share of individuals with heights under one standard deviation from cohort mean (bottom). *Note*: Bands display 95% confidence intervals of cohort means.

Table 2
Parametric estimates of hurricane effects.

Outcome	San Felipe		San Ciprián	
	Parameter × 100	Std. error × 100	Parameter × 100	Std. error × 100
Height (in.)	−2.73	9.65	−2.53	9.65
Short stature	5.07	1.88	0.14	1.88
Diabetes	6.77	0.98	4.22	0.98
Hypertension	6.08	1.07	5.63	1.07
High cholesterol	10.06	1.21	4.67	1.21
CVD	−1.84	2.02	2.23	2.02
AMI	1.08	1.25	2.95	1.25
Coronary/angina	0.13	2.45	0.60	2.45
Stroke	0	0.83	0.96	0.83
No schooling	3.38	0.20	2.45	0.20
Low schooling	1.40	1.15	−1.85	1.15
High schooling	−2.91	1.47	2.56	1.47

Note: Effects are estimated through a fifth-order polynomial in year of birth with binary variables for birth in 1929, 1933, and controls for gender, marital status, survey year, and survey year interactions with birth year. Standard errors are clustered by year of birth. Of the 11,990 observations in the sample 11,953 contain information on education, 10,873 on height, 11,950 on diabetes, 5438 on hypertension, 5041 on high cholesterol, and close to 4900 on cardiovascular disease diagnoses.

include gender, marital status, survey year, and survey year and birth year interactions.[7] Non-parametric estimators require choices of kernel and bandwidth and following Imbens and Lemieux (2008), a rectangular kernel is selected and a variety of windows are used to see how results change with width around the cutoff points.[8] The approach is both transparent and intuitive since widening width around the cutoff is equivalent to evaluating how results change as individuals farther from the cutoff year are deemed as comparable in all ways but treatment and year of birth. In all cases, statistical inference is based on errors clustered by year of birth (Lee and Card, 2008). Analysis relies on inferences about population characteristics, particularly on differential rates of disease across birth cohorts, and sampling weights are therefore employed (Deaton, 1997).

6. Results

A profile of adult height as a function of year of birth is presented in the top panel of Fig. 2. Mean height increases over time with a small discontinuity in 1929 when the cohort value appears off trend. Table 2 establishes that the

[7] Schooling is not included as a control variable since it is a potential outcome with strong empirical support, but its use generates almost identical results.

[8] The forcing variable year of birth is discrete, non-integer width choices make little sense, and therefore widths are chosen in discrete steps.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

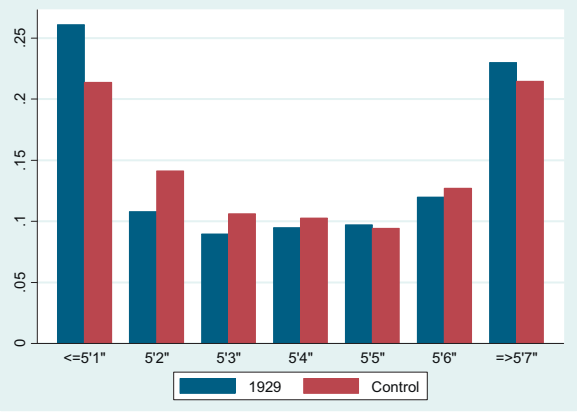

Fig. 3. Height distribution (%) of individuals born in 1929 versus born in 1927–1928 and 1930–1931.

discontinuity is not statistically significant but an alternative outcome definition focused on the incidence of low statures is significant in both parametric and non-parametric specifications. Prevalence of heights under one standard deviation from the cohort mean increases by 5.1% points among those born in 1929 (bottom panel of Fig. 2).[9] In order to gain a better understanding of exposure effects, Fig. 3 displays a comparison of height frequencies standardized by gender of individuals born in 1929, and those born two years prior and two years after that date, a frame corresponding to the narrowest year of birth window. A distinct pattern is discerned with greatest differences occurring at the bottom of the distribution where the lowest category aggregates individuals up to 5′1″ tall, the bottom quartile in the 1929 distribution. Twenty-six percent of individuals born in 1929 report a height in that range versus 21% for the control group. Changes in other height frequencies do not reflect a generalized fall in height across the remaining groups, but a reduction in the next two or three categories – those between 5′2″ and 5′4″ tall. That is, probability mass shifts from the bottom to the very bottom of the distribution, a change consistent with a pattern of effects concentrated on a population already at risk.

Profiles of hypertension and high cholesterol diagnoses rates by year of birth are shown in the top panels of Fig. 4. Visual evidence suggests clear and pronounced discontinuities both in 1929 and in 1933 that are established as statistically significant in Table 2. High blood pressure diagnoses increase by 6.1% points in 1929 and by 5.6% points in 1933. In a similar manner, being born in 1929 is associated with a 10.1% point increase in the incidence of high cholesterol and being born in the aftermath of the San Ciprián hurricane with a 4.7% point increase. Non-parametric exposure effect estimations are all in line with parametric ones and are robust to control group selection. Parametric results are similarly robust to choice of polynomial term order in year of birth (Table 4).

Visual evidence also points to causal effects on diabetes, with prevalence of the condition falling somewhat with year of birth in a fairly smooth pattern but jumping abruptly and markedly in the aftermath of the two hurricanes (bottom left panel of Fig. 4). Table 2 establishes that the sharp discontinuities in 1929 and 1933 are statistically significant and equivalent to a 6.8% point increase in diabetes incidence among individuals born in 1929 and a 4.2% point jump among those born in 1933. Non-parametric estimations are within a percentage point of parametric ones (Table 3). To date, natural experiment-based evidence linking early life conditions to diabetes had been restricted to the 1944–1945 Dutch famine. Material effects on hypertension and high cholesterol based on a natural experiment are also established for the first time, as well as support for all three predictions of the Barker proposal in a single setting.

Ultimately, concern for all three syndrome X conditions has to do with the fact that they are known risk factors of cardiovascular disease. However, evidence in Tables 2 and 3 does not suggest a link. The coefficients of diagnosis of cardiovascular disease are not statistically significant in any of the two hurricane years or model specifications. In the case of disaggregated conditions there is some evidence of effects on acute myocardial infarction but only in the second hurricane. Furthermore, visual evidence (Fig. 5) is not supportive of a relationship. The finding could have a number of explanations, the first being that individuals have not yet developed cardiovascular disease due perhaps to a late onset of risk factors. A second is that they do but have died from it, and unfortunately, lack of data on mortality precludes a test of that possibility. Thirdly, absence of a relationship may be an issue of statistical power associated with the relatively low number of observations with cardiovascular disease diagnoses.

Did the hurricanes also result in cognition shocks? Evidence in the lower right panel of Fig. 4 and in Tables 2 and 3 supports an affirmative answer. The share of individuals without any formal schooling jumps abruptly to a considerable extent in both 1929 and 1933, when the rate increases by 3.4% points with respect to the first date and by 2.5% points with respect to the second one. Relative to education levels in the two years preceding and following the hurricanes, changes are substantial and equivalent to close to 100% increases in the incidence of no formal schooling for those born in 1929 and 1933. However, results in Tables 2 and 3 establish that the abrupt change in educational achievement only occurs at the lowest level. There are no statistically significant changes in the share of individuals with higher levels of schooling. As in the case of height, effects on cognitive ability appear to be concentrated at the very bottom of the distribution.[10]

[9] Relative to pre-hurricane levels, the effect is substantial and could have affected reproductive success of impacted individuals, as established in some studies (Almond et al., 2007; Chen and Zhou, 2007), but the data do not support a relationship, at least as measured by marriage rates.

[10] As robustness checks, treatment was defined as birth in each of the three years preceding and following 1929 and 1933. Joint tests of significance do not reject the null of no increased incidence of diabetes, hypertension, high cholesterol, and zero schooling.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

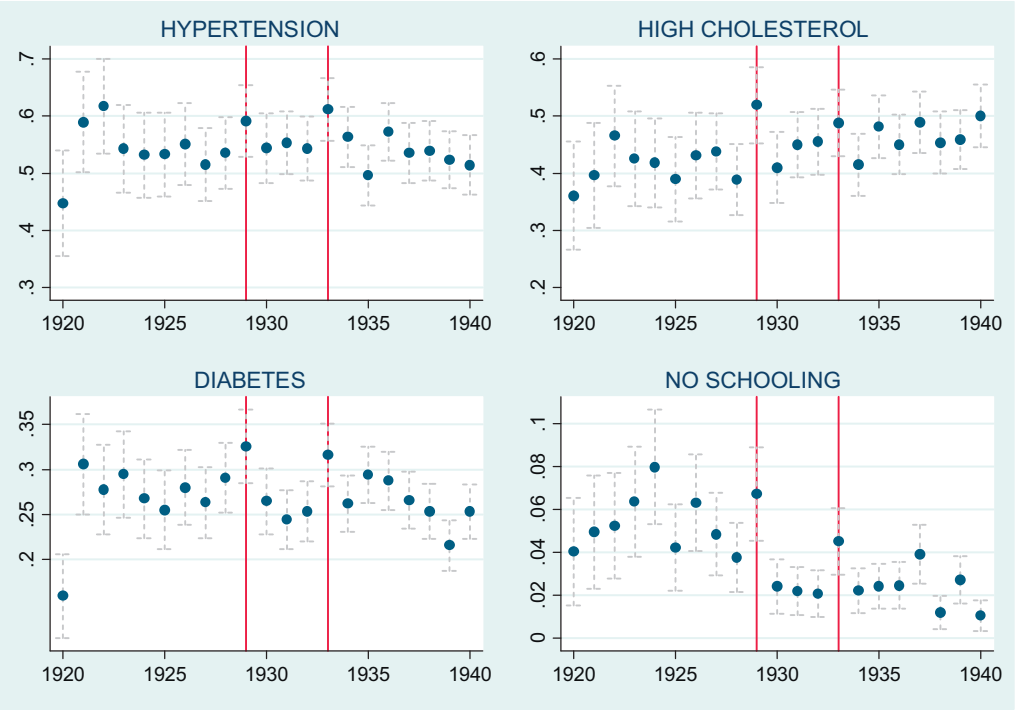

Fig. 4. Incidence of diabetes, hypertension, high cholesterol, and no schooling by year of birth. *Note*: Bands display 95% confidence intervals of cohort means.

Table 3
Non-parametric estimates of exposure effects by year of birth interval.

Outcome	San Felipe			
	1920–1932	1925–1932	1926–1932	1927–1931
Height	−11.20	−8.84	−10.11	−6.01
Short stature	4.21**	5.12**	4.77*	2.85*
Diabetes	5.94**	5.95**	5.70**	5.64**
Hypertension	4.73**	4.86**	4.89**	6.52**
High cholesterol	8.85**	8.97**	8.91**	10.41**
CVD	−1.48	−1.54	−3.21*	−3.56
AMI	0.81	0.92	0.25	−0.75
Coronary/angina	0.40	0.28	−0.80	−0.10
Stroke	−0.25	−0.06	0.10	−0.22
No schooling	3.22**	3.28**	3.09**	3.36**
Low schooling	0.64	0.54	1.06	2.41**
High schooling	−2.43	−2.12	−2.15	−3.76**
Outcome	San Ciprián			
	1930–1940	1930–1937	1930–1936	1931–1935
Height	1.49	1.27	2.03	−4.43
Short stature	1.89*	1.82*	1.70*	1.66*
Diabetes	5.43**	5.16**	4.86**	5.23**
Hypertension	6.39**	6.53**	6.37**	6.85**
High cholesterol	5.28**	5.14**	5.28**	5.17**
CVD	1.33	1.55	1.84	3.16*
AMI	3.26**	3.33**	3.49**	3.91**
Coronary/angina	−0.60	−0.42	−0.17	1.33*
Stroke	0.58	0.62	0.66	1.25**
No schooling	2.10**	2.01**	2.15**	2.24**
Low schooling	−2.35	−2.22	−2.73	−2.40
High schooling	1.86	1.97	2.60*	3.17

Note: Effects are estimated through local linear regressions employing a rectangular kernel and a variety of kernel window widths around the exogenous shocks of 1929 and 1933. Controls are included for gender, marital status, survey year, and survey year interactions with birth year. Coefficients are multiplied by 100 and standard errors are clustered by year of birth.

* Coefficients statistically distinguishable from zero with probabilities of rejecting true nulls of 5%.

** Coefficients statistically distinguishable from zero with probabilities of rejecting true nulls of 1%.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

Table 4
Sensitivity of parametric estimates of exposure effects to choice of polynomial order in year of birth.

Outcome	San Felipe		San Ciprián	
	Parameter × 100	Std error × 100	Parameter × 100	Std error × 100
First order				
Diabetes	5.93	0.65	5.70	0.65
Hypertension	4.84	0.86	7.67	0.86
High cholesterol	8.61	0.67	4.86	0.67
No schooling	2.76	0.29	1.50	0.29
Second order				
Diabetes	4.93	0.68	4.63	0.68
Hypertension	4.27	1.07	6.70	1.07
High cholesterol	8.99	0.96	5.53	0.96
No schooling	2.96	0.34	1.69	0.34
Third order				
Diabetes	4.76	0.81	4.69	0.81
Hypertension	4.69	1.03	6.61	1.03
High cholesterol	8.98	0.99	5.86	0.99
No schooling	2.82	0.36	1.91	0.36
Fourth order				
Diabetes	5.93	0.66	5.31	0.66
Hypertension	5.16	1.03	6.75	1.03
High cholesterol	9.33	1.15	5.94	1.15
No schooling	3.46	0.22	2.29	0.22
Fifth order				
Diabetes	6.77	0.97	4.18	0.97
Hypertension	6.08	1.07	5.63	1.07
High cholesterol	10.06	1.21	4.67	1.21
No schooling	3.38	0.20	2.45	0.20

7. Discussion of continuity assumption and possible biases

Whereas causal inference in regression, matching models, or similar approaches relies on appropriate control of factors relevant to the outcome generating process, in a regression discontinuity setting control is achieved by data design. If other variables affecting health outcomes evolve smoothly across cohorts, the only average difference between individuals born in (say) 1929 and those born

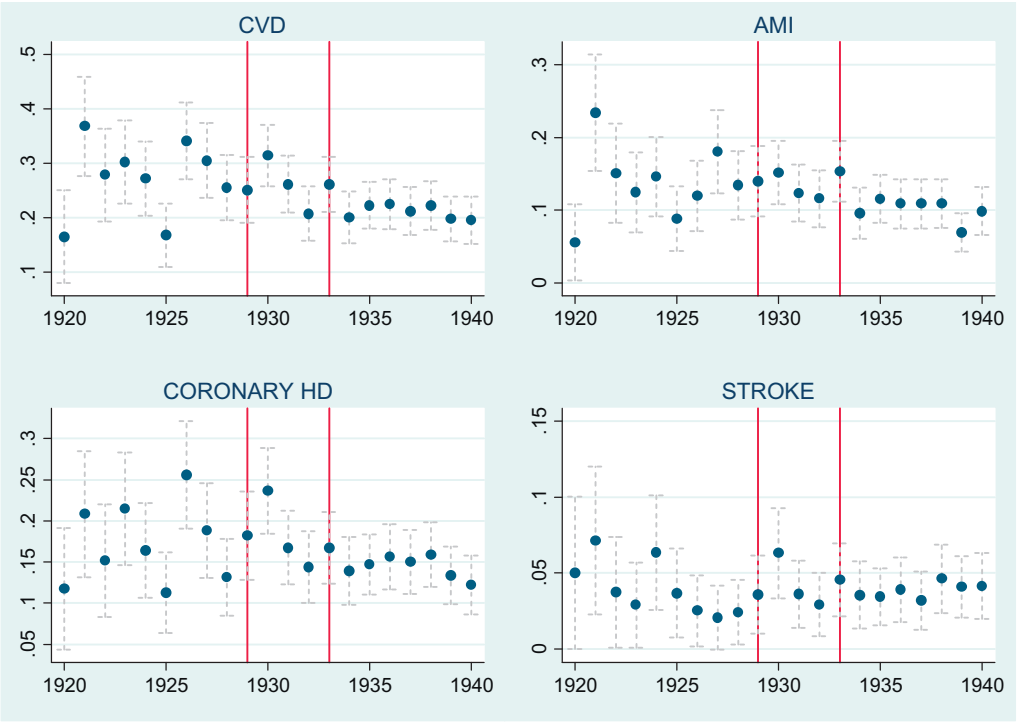

Fig. 5. Incidence of CVD, AMI, coronary heart disease, and stroke by year of birth. *Note*: Bands display 95% confidence intervals of cohort means.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

in a sufficiently close range (say) 1928 or 1930 will be exposure to the hurricane. The assumption is crucial and is required for continuity to hold (Hahn et al., 2001; Imbens and Lemieux, 2008; Lee, 2008).

In settings where data on other factors affecting outcomes are available, statistical tests could be carried out to examine the assumption (McCrary, 2008). Unfortunately, information on variables like technology, public health, or education policies is sparse although available information does not suggest discontinuities across cohorts born in the neighborhood of the occurrence of the natural phenomena. Public health initiatives regarding anemia, smallpox, and malaria had a substantial impact on health but were concentrated around the turn of the twentieth century (Dietz, 1986), too early to have had a differential impact across the cohorts examined in this study. Advances in schooling were gradual, with levels rising over the first half of the past century as a result of US government policies generating gains concentrated at the bottom of the distribution. Periodic changes in instruction language policies affected some cohorts, the largest impacting individuals born after 1933, but they had little effect on English language proficiency (Angrist et al., 2008). The equivalent of 3% of the population served in the armed forces during the Second World War (compared to 12% in the US) and potential draftees included individuals born no later than 1927. A veteran status variable is available in less than half the observations, but inclusion as a control results in almost identical estimates. Finally, obvious changes in technology pertain to advances in treatment of vector borne diseases in the early part of the century and to chemotherapy in the 1940s (Palloni et al., 2005).[11]

Still, other sources of change could have accompanied and therefore coincided with the storms. These include changes in pathogen environment, diet, housing conditions, and stress. Qualitative evidence is not supportive of substantial changes in incidence of disease. A review of the newspaper of record finds only two reports of isolated outbreaks of disease between September and December of 1928, and an extensive study being carried out at the time (Clark et al., 1930) only remarks on uncommon health conditions related to a severe hurricane striking in 1899. The low toll on lives (312) for a storm of its magnitude is perhaps reflective of the substantial preventive measures undertaken before and after the storm. However, housing conditions were undoubtedly changed. Construction was precarious and especially in rural areas building materials frequently consisted of discarded materials, thatch, bark, and structures often lacked cooking or sanitary facilities (Mountin et al., 1937:7; Perloff, 1950:3).[12] Although diet was poor and monotonous, consisting mainly of coffee, beans, rice, and tubers (Mountin et al., 1937:6; Perloff, 1950:3), shortages could have exacerbated specific dietary deficiencies independent of income. Lastly, stress related to the economic and housing circumstances could also have risen abruptly. Therefore, in absence of quantitative evidence allowing for separate tests of income, housing, nutrition, disease, and stress effects, conclusions obtained in the last section cannot strictly be tied to any one particular factor, but rather to shocks in income, diet, disease, housing conditions, and stress brought forth by the aftermath of storms. All these factors often go together and could be referred to as shocks to living standards.

Once estimated, relationships could be subject to a number of biases and it is important to at least establish their direction. First, the hurricanes could have resulted in negative selection into child mortality causing disproportionate deaths of unhealthier babies in 1929 and 1933 relative to other years. The phenomenon would have generated downward bias on estimates. Second, the hurricanes could have resulted in increased cohort migration probabilities to the extent that shocks resulted in compromised cognitive abilities and flows in following decades consisted of migrants with below-average schooling. In light of the higher levels of inequality of the Island's wage distribution, the Borjas (1987) migration model would predict outflows of negatively selected migrants. If so, individuals most affected by the storms would be less likely to be in the sample due to migration to the US. Third, the storm could have affected fertility decisions. If individuals who chose to conceive after the hurricane were a negatively (positively) selected group, estimates in Section 6 would be biased upward (downward). Fourth, selection to mortality as adults could also be playing a role.

Evidence on the existence of the first two potential sources of bias is not compelling. Historical trends on child mortality are unavailable but overall mortality rates show uninterrupted progress during the first half of the twentieth century (Perloff, 1950:200). Evidence of selective migration is only available for the decades of the 1970s, 1980s, and 1990s, but these support the case of flows composed of individuals with average rather than below-average schooling (Sotomayor, 2009). To the extent that education is correlated with health, migration did not result in disproportionate outflows of unhealthy individuals.

Ruling out selective fertility as an explanation could be achieved by exploiting month or quarter of birth information, since individuals born early in 1929 and 1933 would have been conceived before the storms struck. Such information is unavailable, but 1930 Census tables can help assess whether the hurricane caused a change in the racial profile of individuals conceived before and after the first storm. Population tabulations by age and race show that on April 1, 1930, black children accounted for a constant 27% of the population across each of the five age categories ranging from less than a year old to four years of age. The share of white children held at 73% across the same age groups (US Census Bureau, 1932:138).[13] As a measure of racial impact on relative social standing, Census tables show that 45% of black women in gainful employment worked in domestic services versus 23% of white ones. White men were 60% more likely to work in the trade sector, twice as likely to be found in clerical or public sector occupations, and

[11] The authors find correlations between markers early life conditions, obesity, and diabetes in a cross-section sample of elderly Puerto Ricans.

[12] The literature emphasizes that disasters are processes combining natural forces and social vulnerabilities (Sen, 1981; Oliver-Smith and Hoffman, 1999).

[13] Population numbers of white and black children also remain constant across birth cohorts.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

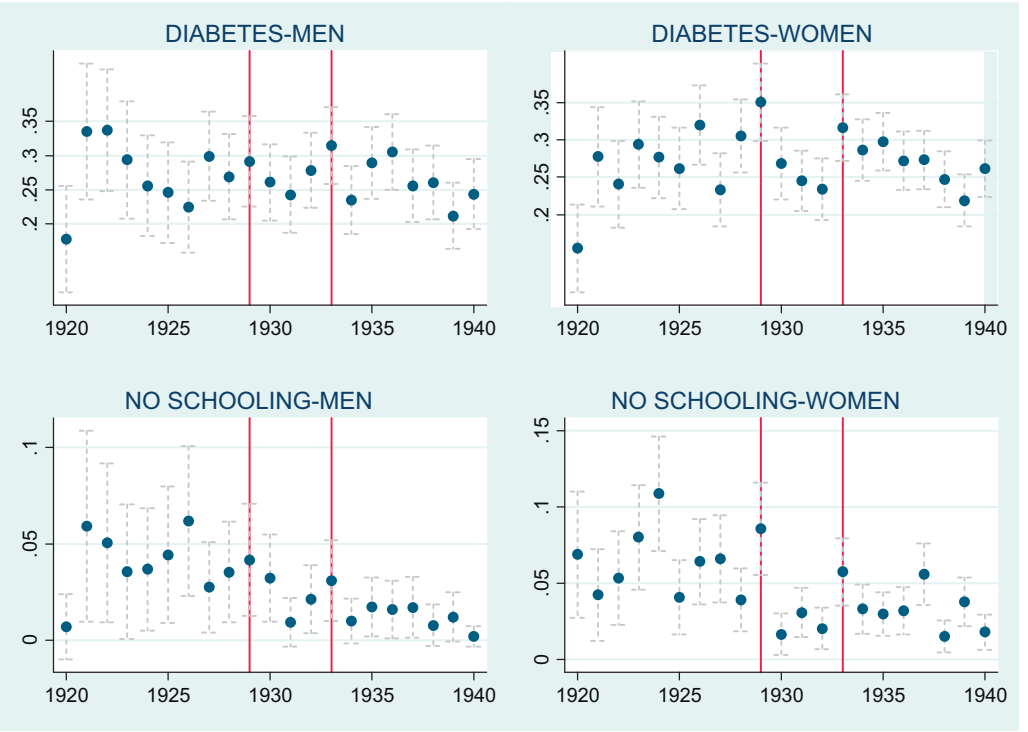

Fig. 6. Incidence of diabetes and no formal schooling by gender and year of birth. *Note*: Bands display 95% confidence intervals of cohort means.

five times as likely to be doctors, lawyers, or engineers (US Census Bureau, 1932:193–95). If race can be taken as an important indicator of socioeconomic status, evidence does not suggest that the hurricane shifted the composition of births from relatively advantaged groups to less advantaged ones, or the reverse. However, selection could still have occurred along other dimensions.[14]

The fourth source of potential bias could be playing a more important role since the sample median age is 70 and cardiovascular disease is a major cause of death and institutionalization among the elderly. In order to gain a measure of the magnitude of the bias, treatment effects are re-calculated using only data from the 1996 to 2000 surveys and outcomes available in all of them: height, diabetes diagnosis, and schooling. Coefficient estimates along with variable averages employed for estimating relative effects are presented in Table 5. The sample median age is 66 and results show that whereas in the full sample diabetes diagnoses increase by 26% and 16% among those born after the San Felipe and San Ciprián hurricanes, respectively, the numbers rise to 53% and 20% when using the sample composed of relatively younger individuals. Schooling effect differences are also substantial and in the order of increases of 105% and 110% using the full sample as compared to increases of 230% and 235% in the younger sample. Therefore, evidence points to the fact that results presented in Tables 2 and 3 and Figs. 3 and 4 represent a conservative assessment of the relationship between living standards and late-life health, especially those based on the San Felipe hurricane. Relative to the sample of younger individuals, full sample exposure effect estimates are in absolute terms about 45% lower in the case of the first storm and 15% lower in case of the second one (Fig. 6).

Table 5
Parametric estimates of exposure effects and average outcome values using the full sample and a sample restricted to 1996–2000 BRFSS surveys.

Outcome	Parameter × 100	SE × 100	Mean × 100	*N*
San Felipe – sample using 1996–2008 surveys				
Short stature	5.07	1.88	15.83	10,873
Diabetes	6.77	0.98	26.49	11,950
No education	3.38	0.20	3.22	11,953
San Felipe – sample using 1996–2000 surveys				
Short stature	7.96	1.80	15.34	3319
Diabetes	11.84	1.94	22.36	3560
No education	6.44	0.61	2.79	3563
San Ciprián – sample using 1996–2008 surveys				
Short stature	0.14	1.88	16.67	10,873
Diabetes	4.22	0.98	26.46	11,950
No education	2.45	0.20	2.23	11,953
San Ciprián – sample using 1996–2000 surveys				
Short stature	1.8	0.77	15.31	3319
Diabetes	4.71	1.29	24.06	3560
No education	3.01	0.32	1.28	3563

Note: Effects are estimated through use of a fifth-order polynomial in year of birth with binary variables for birth in 1929, 1933, and controls for gender, marital status, survey year, and survey year interactions with birth year. 'Mean' represents outcome values averaged over the two years preceding and following the hurricanes. Standard errors are clustered by year of birth. The number of observations per regression is included in the last column.

[14] The San Ciprián hurricane also does not appear to have had an effect on racial birth profile. 1940 Census tables establish that the racial profile of cohorts born in the decade of the 1930s remained unchanged at the same ratios (US Census Bureau, 1942:21).

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

Table 6
Parametric estimates of marginal exposure effects on women using the full sample and a sample restricted to 1996–2000 BRFSS surveys.

Outcome	Parameter × 100	SE × 100	Mean × 100	*N*
San Felipe – sample using 1996–2008 surveys				
Short stature	9.09	3.27	28.28	6735
Diabetes	5.77	1.97	26.34	7712
No education	3.64	0.94	3.73	7713
San Felipe – sample using 1996–2000 surveys				
Short stature	8.93	3.34	28.31	2022
Diabetes	10.22	2.77	23.61	2239
No education	4.89	0.86	4.09	2240
San Ciprián – sample using 1996–2008 surveys				
Short stature	−1.83	3.27	29.69	6735
Diabetes	−0.03	1.97	26.73	7712
No education	1.92	0.94	2.87	7713
San Ciprián – sample using 1996–2000 surveys				
Short stature	−6.14	3.34	28.15	2022
Diabetes	3.26	2.77	24.83	2239
No education	1.12	0.86	2.03	2240

Note: Marginal exposure effects refer to the additional impact of the hurricanes on women relative to men. Effects are estimated through a model fully interacted with a female indicator variable and 'Mean' represents outcomes values for women averaged over the two years preceding and following the hurricanes. Standard errors are clustered by year of birth. The number of observations per regression is included in the last column.

Sample attrition due to death or institutionalization is also lower among women, who make up 56% of the sample and 58% of individuals born in the decade of the 1920s. In order to estimate gender-specific effects, models are fully interacted with a female indicator variable and estimated using the complete sample and one constructed using only the first five surveys. Results show that exposure effects are more pronounced among women (Table 6). The San Felipe hurricane is associated with an increase in short-statured individuals that is 9.1% points larger among women. Relative to men, exposure to the hurricane resulted in an additional 5.8% point increase in diabetes, and an additional 3.6% point increase in the incidence of no formal schooling. Even greater consequences are estimated when the sample is restricted to observations from the earlier surveys. However, with the exception of no formal schooling, San Ciprián effects do not appear to vary by gender or increase using earlier surveys. At least in the case of San Felipe, evidence therefore supports the premise that attrition is introducing downward bias on estimates, although an alternative possibility of gender-specific hurricane effects could also be generating results.

8. Conclusion

A number of important contributions in economics have served to advance our understanding of the possible long-term consequences of poor early life environments. These have included the role of maternal exposure to famine, viral infection, vector-borne disease, pollutants, dietary deficiencies, recession, weather shocks, and Ramadan observance. While evidence suggests that prenatal and early infancy insults can have deleterious effects on health and other outcomes in later life, the pathways from circumstances to morbidity and mortality remain important areas of research.

This study finds that shocks caused by the tropical storms that struck Puerto Rico in 1928 and 1932 had effects that are consistent with the predictions of the fetal origins hypothesis. Diabetes and other cardiovascular disease risk factors such as hypertension and high cholesterol are markedly higher among those in the womb or early infancy in the aftermath of both storms. Moreover, the hurricanes led to pronounced increases in the share of individuals without formal schooling, a phenomenon that could have fed back to health in view of its long-established relationship with socioeconomic status. The degree of impact was substantial, with hurricane San Felipe resulting in a 26% increase in the incidence of diabetes, hurricane San Ciprián in a rise of 16%, and both hurricanes close to doubling the share of individuals in the stated schooling category. The single inconsistency with the predictions of the hypothesis is the absence of a link between the hurricane shocks and the development of cardiovascular disease, possibly resulting from late onset of risk factors, selective attrition from the sample due to death from the condition, or a true absence of relationship.

Results are noteworthy as they establish a causal link between living conditions, diabetes, and other health risk factors, and a possible pathway from poor early life environments to health and mortality later in life. Effects estimated in this paper are not only large, but also result from events of less extreme proportions than, say the Dutch famine of 1944–1945. Evidence therefore suggests that in absence of preventive measures, effects of events like Bay of Bengal cyclones, Caribbean Sea storms like hurricane Mitch, or the great Haitian earthquake may not be over for a long time.

Appendix A. Number of observations by outcome and year of birth

Birth year	Observations with information on				Retention
	Diabetes	Hyper tension	High cholesterol	CVD	
1920	237	112	100	74	0.19
1921	262	120	110	108	0.20
1922	315	134	125	106	0.24
1923	347	162	137	140	0.28
1924	396	173	156	162	0.29
1925	382	178	166	160	0.31
1926	446	184	166	174	0.34
1927	481	233	212	175	0.41
1928	530	246	233	205	0.41
1929	508	235	218	199	0.41
1930	564	257	238	260	0.44
1931	659	314	292	272	0.40
1932	666	305	286	255	0.39
1933	687	307	280	288	0.42
1934	751	337	317	271	0.46
1935	828	344	325	362	0.45
1936	761	374	351	320	0.42
1937	758	342	331	323	0.42
1938	774	353	326	331	0.43
1939	824	385	356	367	0.42
1940	789	349	321	320	0.43

Note: Total observations and observations containing information on schooling are within 5 observations of the values in the diabetes column. Retention is defined as the ratio of weighted number of observations and registered births. Attrition can result both from death as well as migration to the US.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

References

Almond, D., Mazumder, B., 2005. The 1918 Influenza Pandemic and subsequent health outcomes: An analysis of SIPP data. American Economic Review 95 (2), 258–262.
Almond, D., 2006. Is the 1918 Influenza Pandemic really over? Long-term effects of in utero influenza exposure in the post-1940 U.S. population. Journal of Political Economy 114 (4), 672–712.
Almond, D., Edlund, L., Li, H., et al., 2007. Long-term effects of the 1959–1961 China famine: Mainland China and Hong Kong. In: Ito, T., Rose, A. (Eds.), The economic consequences of demographic change in East Asia, NBER-East Asia Seminar on Economics, 19. University of Chicago Press, Chicago.
Almond, D., Edlund, L., Palme, M., 2009. Chernobyl's subclinical legacy: prenatal exposure to radioactive fallout and school outcomes in Sweden. Quarterly Journal of Economics 124 (4), 1729–1772.
Almond, D., Mazumder, B., 2011. Heath capital and the prenatal environment: the effect of Ramadan observance during pregnancy. American Economic Journal: Applied Economics 3 (4), 56–85.
Almond, D., Currie, J., 2011. Human capital development before age five. In: Ashenfelter, O., Card, D. (Eds.), Handbook of Labor Economics, vol. 4b. Amsterdam, North Holland, pp. 1315–1486.
Angrist, J., Chin, A., Godoy, R., 2008. Is Spanish-only schooling responsible for the Puerto Rican language gap? Journal of Development Economics 85 (1–2), 105–128.
Banerjee, A., Duflo, E., Postel-Vinay, G., et al., 2010. Long-run health impacts of income shocks: wine and phylloxera in nineteenth century France. Review of Economics and Statistics 92 (4), 714–728.
Barker, D., Osmond, C., Golding, J., et al., 1989. Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. British Medical Journal 298 (6673), 564–567.
Barker, D., 1992. Fetal and infant origins of later life disease. British Medical Journal Books, London.
Barker, D., Martyn, C., Osmond, C., et al., 1993. Growth in utero and serum cholesterol concentrations in adult life. British Medical Journal 307 (6918), 1524–1527.
Barreca, A., 2010. The long-term economic impact of in utero and postnatal exposure to malaria. Journal of Human Resources 45 (4), 865–892.
Belsky, J., Steinberg, L., Draper, P., 1991. Childhood experience, interpersonal development and reproductive strategies: an evolutionary theory of socialization. Child Development 62 (4), 647–670.
Belsky, J., 2007. Childhood experiences and reproductive strategies. In: Dunbar, R., Barrett, L. (Eds.), Oxford Handbook of Evolutionary Psychology. Oxford University Press, Oxford, pp. 237–254.
Black, S., Devereux, P., Salvanes, K., 2007. From the cradle to the labor market? The effect of birth weight on adult outcomes. Quarterly Journal of Economics 122 (1), 409–439.
Block, S., Kiess, L., Webb, P., et al., 2004. Macro shocks and micro outcomes: child nutrition during Indonesia's crisis. Economics and Human Biology 2 (1), 21–44.
Borjas, G., 1987. Self-selection and the earnings of migrants. American Economic Review 77 (4), 531–553.
Case, A., Paxson, C., 2008a. Stature and status: height, ability, and labor market outcomes. Journal of Political Economy 116 (3), 499–532.
Case, A., Paxson, C., 2008b. Height, health and cognitive function at older ages. American Economic Review 98 (2), 463–467.
Chen, Y., Zhou, L., 2007. The long-term health and economic consequences of the 1959–1961 famine in China. Journal of Health Economics 26 (4), 659–681.
Clark, V., et al., 1930. Porto Rico and Its Problems. Brookings Institution, Washington, DC.
Currie, J., Thomas, D., 1995. Does head start make a difference? American Economic Review 85 (3), 341–364.
Currie, J., Hyson, R., 1999. Is the impact of health shocks cushioned by socioeconomic status? The case of low birth weight. American Economic Review 89 (2), 245–250.
Currie, J., Stabile, M., Manivong, P., et al., 2010. Child health and young adult outcomes. Journal of Human Resources 45 (3), 517–548.
Cutler, D., Miller, G., Norton, D., 2007. Evidence on early-life income and late-life health from America's dustbowl era. Proceedings of National Academy of Science 104 (33), 13244–13249.
Deaton, A., 1997. The analysis of household surveys: a microeconomic approach to development policy. Johns Hopkins University Press for the World Bank, Baltimore.
del Ninno, C., Lundberg, M., 2005. Treading water: the long-term impact of the 1998 flood on nutrition in Bangladesh. Economics and Human Biology 3 (1), 67–96.
Dietz, J., 1986. Economic History of Puerto Rico. Princeton University Press, Princeton.
Fassig, O., 1929. San Felipe—the hurricane of September 13, 1928. Monthly Weather Review 56 (9), 350–352.
Field, E., Robles, O., Torero, M., 2009. Iodine deficiency and schooling attainment in Tanzania. American Economic Journal: Applied Economics 1 (4), 140–169.
Fletcher, J., Green, J., Neidell, M., 2010. Long term effects of childhood asthma on adult health. Journal of Health Economics 29 (3), 377–387.
Garces, E., Thomas, D., Currie, J., 2002. Longer-term effects of Head Start. American Economic Review 92 (4), 999–1012.
Gluckman, P., Hanson, M., 2005. The fetal matrix: evolution, development and disease. Cambridge University Press, Cambridge.
Godoy, R., Goodman, E., Levins, R., et al., 2007. Adult male height in an American colony: Puerto Rico and the USA mainland compared. Economics and Human Biology 5 (1), 255–279.
Griskevicius, V., Tybur, J., Delton, A., et al., 2011. The influence of mortality and socioeconomic status on risk and delayed rewards: a life history theory approach. Journal of Personality and Social Psychology 100 (6), 1015–1026.
Guégan, J-F., Thomas, F., Hochberg, M., et al., 2001. Disease diversity and human fertility. Evolution 55 (7), 1308–1314.
Hahn, J., Todd, P., van der Klaauw, W., 2001. Identification and estimation of treatment effects with a regression-discontinuity design. Econometrica 69 (1), 201–209.
Hales, C., Barker, D., Clark, P., et al., 1991. Fetal and infant growth and impaired glucose intolerance at age 64. British Medical Journal 303 (6809), 1019–1022.
Huxley, R., Neil, A., Collins, R., 2002. Unraveling the fetal origins hypothesis: Is there really an inverse association between birthweight and subsequent blood pressure. The Lancet 360 (9334), 659–665.
Huxley, R., Owen, C., Whincup, P., et al., 2004. Birth weight and subsequent cholesterol levels: Exploration of the 'fetal origins' hypothesis. Journal of the American Medical Association 292 (22), 2755–2764.
Imbens, G., Lemieux, T., 2008. Regression discontinuity designs: a guide to practice. Journal of Econometrics 142 (2), 615–635.
Kannisto, V., Christensen, K., Vaupel, J., 1997. No increased mortality in later life for cohorts born during the famine. American Journal of Epidemiology 145 (11), 987–994.
Kelly, E., 2011. The scourge of Asian flu: in utero exposure to pandemic influenza and the development of a cohort of British children. Journal of Human Resources 46 (4), 669–694.
Lee, D., 2008. Randomized experiments from non-random selection in U.S. House elections. Journal of Econometrics 142 (2), 675–697.
Lee, D., Card, D., 2008. Regression discontinuity inference with specification error. Journal of Econometrics 142 (2), 655–674.
Lindeboom, M., Portrait, F., van den Berg, G., 2010. Long-run longevity effects of a nutritional shock early in life: the Dutch potato famine of 1846–47. Journal of Health Economics 29 (5), 617–629.
Ludwig, J., Miller, D., 2007. Does Head Start improve children's life chances? Evidence from a regression discontinuity design. Quarterly Journal of Economics 122 (1), 159–208.
Maccini, S., Yang, D., 2009. Under the weather: Health, schooling, and economic consequences of early life rainfall. American Economic Review 99 (3), 1006–1026.
McCrary, J., 2008. Manipulation of the running variable in the regression discontinuity design: a density test. Journal of Econometrics 142 (2), 698–714.
Miller, G., Chen, E., Fok, A., et al., 2009. Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. Proceedings of the National Academy of Sciences 106, 14716–14721.
Mountin, J., Pennell, E., Flook, E., 1937. Illness and Medical Care in Puerto Rico. Public Health Bulletin 237. US Treasury Department, Public Health Service, Government Printing Office, Washington, DC.
Nettle, D., 2010. Dying young and living fast: variations in life history across English neighborhoods. Behavioral Ecology 21 (2), 387–395.
Oliver-Smith, A., Hoffman, S., 1999. The Angry Earth: Disaster in Anthropological Perspective. Routledge.
Palloni, A., McEniry, M., Dávila, A.L., et al., 2005. The influence of early conditions on health status among elderly Puerto Ricans. Social Biology 52 (3/4), 132–164.
Pérez, O., 1971. Notes on the tropical cyclones of Puerto Rico, 1508–1970. Unpublished report, National Weather Forecast Service, San Juan, Puerto Rico.
Perloff, H., 1950. Puerto Rico's Economic Future. University of Chicago Press, Chicago.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009

Rasmussen, K., 2001. The 'fetal origins' hypothesis: challenges and opportunities for maternal and child nutrition. Annual Review of Nutrition 21, 73–95.
Ravelli, A., van der Meulen, J., Michels, R., et al., 1998. Glucose tolerance in adults after prenatal exposure to famine. The Lancet 351 (9097), 173–177.
Roseboom, T., van der Meulen, J., Osmond, C., et al., 2000. Coronary heart disease after prenatal exposure to the Dutch famine, 1944–45. Heart 84 (6), 595–598.
Sen, A., 1981. Poverty and Famines: An Essay on Entitlement and Deprivation. Clarendon Press, Oxford.
Skoufias, E., Vinha, K., 2011. Climate variability and child height in rural Mexico. Economics and Human Biology 10 (1), 54–73.
Smith, J., 2009. The impact of childhood health on adult labor market outcomes. Review of Economics and Statistics 91 (3), 478–489.
Sotomayor, O., 2009. Puerto Rican migration flows and the theory of migrant self-selection. World Development 37 (3), 726–738.
Stanner, S., Bulmer, K., Andrès, C., et al., 1997. Does malnutrition in utero determine diabetes and coronary heart disease in adulthood: results from the Leningrad siege study, a cross sectional study. British Medical Journal 315 (7119), 1342–1348.
US Census Bureau, 1932. Fifteenth Census of the US: Outlying Territories and Possessions. Government Printing Office, Washington, DC.
US Census Bureau, 1942. Sixteenth Census of the US: Population, Puerto Rico. Government Printing Office, Washington, DC.
van den Berg, G., Lindeboom, M., Portrait, F., 2006. Economic conditions early in life and individual mortality. American Economic Review 96 (1), 290–302.
van Ewijk, R., 2011. Long-term health effects on the next generation of Ramadan fasting during pregnancy. Journal of Health Economics 30 (6), 1246–1260.
World Health Organization, 2002. The World Health Report 2002: Reducing Risk, Promoting Healthy Life. World Health Organization, Geneva.

Please cite this article in press as: Sotomayor, O., Fetal and infant origins of diabetes and ill health: Evidence from Puerto Rico's 1928 and 1932 hurricanes. Econ. Hum. Biol. (2012), doi:10.1016/j.ehb.2012.02.009