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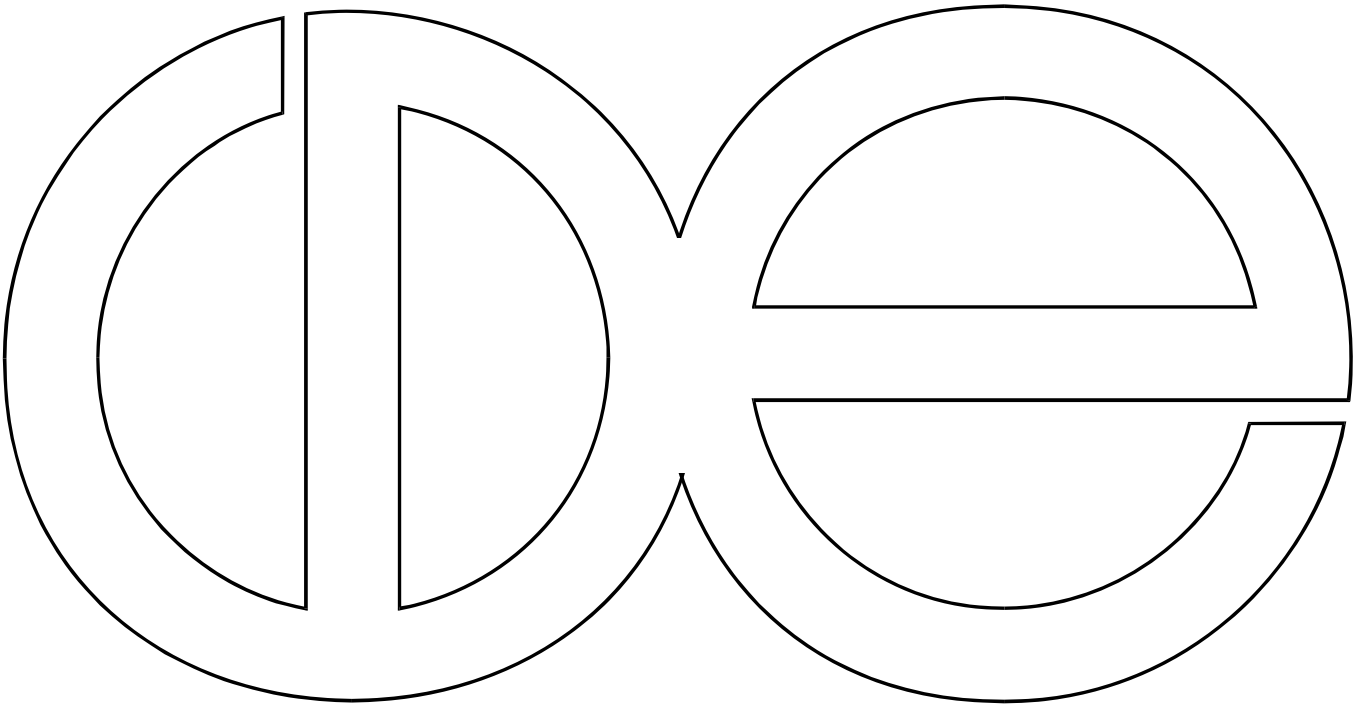

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Abstract

Infant mortality at birth may help illuminate the usefulness of season of birth as an indicator of early life exposures. We obtained data for infant mortality rates (IMR) at the county (*municipio*) level during the late 1920s-early 1940s in Puerto Rico using historical records and linked IMR with individual birth year and place using the PREHCO (Puerto Rican Elderly: Health Conditions) study. We classified PREHCO respondents into two groups according to high or low infectious disease load, corresponding to lower or higher proportion endogenous mortality in the year respondents were born. We estimated the effects of IMR (using continuous, logit, quartile, Box-Cox transformations) and season of birth on adult heart disease and diabetes for all respondents and then by subgroups, controlling for age, gender, obesity, respondent's educational level, adult behavior (smoking, exercise) and other early life exposures (childhood health, knee height, childhood SES). Findings: (1) no significant associations between IMR and heart disease or diabetes but significant associations between high IMR and low knee height, low education, older age and no rigorous exercise as an adult; (2) stronger effects of season of birth on adult health among respondents born in years with lower infectious disease loads; (3) strong effects of season of birth even after controlling for other childhood and adult factors. Conclusions: (1) effects of poor environmental conditions during the first year of life on adult health may be mediated by other life course factors; (2) season of birth is a useful indicator of early life exposures under restricted conditions; (3) in this population of older Puerto Rican adults, season of birth provides (weak) evidence in support of the importance of *in utero* (endogenous) factors affecting adult heart disease and diabetes.

Introduction

Disentangling the effects of early life conditions to better understand the conditions that precipitate the onset of adult disease continues to be of particular relevance. Infant mortality rates (IMR) and their components (neonatal and post neonatal mortality) may help provide insight.

We know that neonatal mortality rates (deaths during the first month of life) are strongly associated with non-environmental or endogenous causes.[1] Barker and Osmond's (1986) classic geographical analysis in England and Wales showed strong positive associations between neonatal mortality and adult mortality due to coronary heart disease in the 1960s. Nearly 80% of overall neonatal mortality was due to congenital causes. Endogenous mortality thus became an important clue as to the source of early life conditions which led to adult mortality. Barker later concluded that, "coronary heart disease is associated with past infant mortality because it originates in conditions *in utero*, rather than poor conditions in childhood, though these contribute" (Barker 2002). In Barker's view, living conditions may alter the risk of disease or conversely slow early growth may increase vulnerability to the effects of poor living conditions but poor living conditions are not an important confounding factor (Barker et al. 2001) but rather can add to the effects of early life conditions (Barker 1995).

The Barker hypothesis has been controversial, precisely because there are others who believe that circumstances such as poor living conditions are indeed confounding factors in explaining adult health. Important background conditions that influence early growth and development could also have independent effects on later health and mortality (Huxley, Neil, & Collins 2002;

[1] Endogenous infant mortality is defined as "cases in which the child bears within itself, from birth, the cause resulting in its death, whether that cause was inherited from its parents at conception or acquired from its mother during gestation or delivery" (Bourgeois-Pichat 1952). Exogenous infant mortality is defined as "cases in which the infant picks up the factor which causes its death in the environment in which it lives" (Bourgeois-Pichat 1952).

Joseph & Kramer 1996). Factors such as poor childhood socioeconomic conditions and poor childhood health can have substantial impacts on adult health (Davey Smith & Lynch 2004; Elo & Preston 1992; Gunnell et al. 1998; Hertzman 1994; Lundberg 1991; Wadsworth & Kuh 1997). Failing to control properly for these conditions will inflate the association between any chosen valid indicator of early growth *in utero* and adult health and mortality.

A few studies have attempted to disentangle the effects of *in utero* and early infancy on adult health by using IMR as an indicator of disease load during the first year of life and local food prices as an indicator of access to food and nutrition during pregnancy. Bengtsson and Lindstrom's study of four rural Swedish parishes from 1700-1800s showed a strong positive association between infant mortality and adult mortality but no association between food prices and adult mortality (Bengtsson & Lindstrom 2000). In the case of Sweden during the 1700-1800s, IMR were on average high (200 per 1000 live births) and airborne diseases such as smallpox and whooping cough were predominant and crossed all socioeconomic levels, thus leading them to conclude that "economic factors are of rather small importance" in terms of the causes of IMR in Sweden during this period (Bengtsson & Lindstrom 2000). Other studies from the Netherlands and Belgium which have used IMR and local food prices on populations in the 19th century also found weak evidence in support of the effects of *in utero* conditions on adult health (Svensson, Broström, & Oris 2004; Van den Berg, Lindeboom, & Portrait 2006). In these studies, IMR was also similarly high[2] and in both studies there was little evidence to suggest that *in utero* nutrition affected adult mortality. Rather it was either the economic conditions into which one was born (Van den Berg et al. 2006) or infectious diseases during the first year of life (Svensson, Broström, & Oris 2004) which were important to adult mortality. In a different type

[2] In fact, the Netherlands experienced a very high level of IMR in the early 1800s—higher than other European country and mostly due to economic conditions (Van den Berg, Lindeboom, & Portrait 2006; Bideau 1997).

of study, Doblhammer (2004) also found a positive association between IMR in the early 20th century and adult mortality in Denmark.

In previous work we used season of birth as an indicator of poor nutrition and infectious disease *in utero* to disentangle the effects of early life conditions on heart disease and diabetes among older adult Puerto Ricans (McEniry et al. 2008). We found that the odds of self-reporting heart disease and diabetes increased for those who were born at the end of the lean season (i.e. born during a period where the risk of being more fully exposed to poor nutrition and infectious disease during late gestation was highest in comparison with those born at the end of the harvest season). Furthermore, we found that these effects were independent of the effects of an indicator of self-reported childhood health. Poor childhood health was associated with childhood illnesses such as malaria, asthma, bronchitis, dengue fever, pneumonia, rheumatic fever, and smallpox and with periods of deprivations due to health but not with season of birth.

In spite of these results, it could be argued that season of birth represents exogenous (environmental) factors during the first year of life and not endogenous (*in utero*) conditions. This is because birth month is a broad indicator of early life exposures. One way to address these concerns is to use IMR and its components to help illuminate the meaning of season of birth. Endogenous mortality reflects, for the most part, conditions originating *in utero* whereas exogenous mortality reflects more environmental conditions such as infectious disease load during the first year of life (Bourgeois-Pichat 1952). If the Barker hypothesis has merit, there should be significant associations between endogenous mortality and adult morbidity (heart disease and diabetes) whereas if environmental conditions during the first month of life are important, there should be important associations between exogenous mortality and adult morbidity. Even if there are scant data on the components of IMR at a particular granular level

such as a county,[3] it may be possible to reasonably group older adults according to if they were born when endogenous mortality was more predominant.[4,5] As IMR declines, seasonality of infectious diseases may weaken whereas strong nutritional seasonality may still persist, thus providing a way to test the Barker hypothesis through examining the effects of season of birth in these subgroups.

In this paper, we examine early life exposures and older adult health using a sample of older Puerto Ricans born in the late 1920s-early 1940s, adding infant mortality as another important factor that could help disentangle the effects of early life. We expect to find two major regularities regarding the association between IMR (or their components) and adult health: **First**, (1) if early life environment (during the first year of life) is important to older adult health, then we expect to observe a significant association between IMR and adult heart disease and diabetes; (2) if season of birth reflects exogenous causes, then we expect to observe that the effects of birth month are attenuated when IMR are added to model estimation. **Second**, if we can reasonably divide the sample of Puerto Rican older adults into two groups according to the predominance of endogenous mortality when they were born and if the Barker hypothesis has merit, we expect to observe that the effects of season of birth are stronger among those respondents born when the predominance of endogenous mortality is higher (lower IMR and better environmental conditions).

[3] Such as at the county level as in the study by Barker and Osmond (1986).
[4] At higher IMR there are poorer environmental conditions and endogenous mortality accounts for a lower fraction of IMR. At lower IMR there are better environmental conditions and endogenous mortality accounts for a higher fraction of IMR. When IMR begins to decline, it is usually due to improvement in environmental conditions and thus as it continues to decline the fraction of exogenous mortality becomes smaller while the fraction of endogenous mortality becomes larger.
[5] See also Johansson and Beise (2008).

Before examining the veracity of these expectations, we first provide a brief background description on infant mortality during the 1920s-1940s in Puerto Rico and identify a critical assumption to modeling IMR and its components.

Infant Mortality in Puerto Rico during the 1920s-1940s

In the early 20th century, Puerto Rico's population was predominantly rural[6] and overall it had a high population density (Bureau of Vital Statistics 1926). Times were difficult during the late 1920s through early 1940s for many Puerto Ricans, especially those who lived in the rural countryside where wages were the main source of income, underemployment and/or unemployment were highly cyclical due to the predominant sugar cane industry and living conditions were particularly precarious (Clark 1930; Morales et al. 1937; Morales et al. 1939; Morales & Pérez 1939; Pérez 1941). Only 45% of the population was literate (Clark 1930) and increased exposure to infectious diseases during the hurricane season brought augmented exposure to infectious disease such as dysentery, diarrhea and malaria (Rigau Pérez 2000). Thus, in spite of improving health conditions during the 1930s in some areas of the island, the leading cause of death in Puerto Rico at the end of the 1930s was still diarrhea in children under the age of two (Garrido Morales 1939/40).

Infant mortality was high during this period[7] and during the late 1920s about 26-27% of all deaths were children under the age of one (Fernós Isern 1930/31; Ortiz 1929/30). During the late 1920s-early 1940s in Puerto Rico, IMR began to improve as environmental conditions improved, due to the efforts of public health interventions[8] and laws passed to provide stricter regulation

[6] About 78% in 1920 and about 72% in 1930 (US Bureau of the Census 1932).
[7] Throughout the late 1920s-early 1940s overall IMR in Puerto Rico was 129 (1927), 150 (1928), 146 (1929), 126 (1930), 122 (1931), 132 (1932), 139 (1933), 113 (1934), 115 (1935), 127 (1936), 138 (1937), 121 (1938), 112 (1939), 113 (1940), 116 (1941), 103 (1942), 95 (1943). Sources are the annual reports of the commissioner of health in Puerto Rico.
[8] See Annual Reports of the Health Commissioner in reference section.

over midwives (Belaval 1945) (see Figure 1). The overall IMR were still relatively high compared with the US mainland and with other countries such as England and Wales[9] and environmental factors were thus clearly more predominant in explaining IMR in Puerto Rico than in the US, England and Wales.

[insert Figure 1]

An examination of the trend in overall IMR from the 1930s-1980s in Puerto Rico shows the decrease in the fraction of post neonatal mortality over time as conditions improved and an increase in the fraction of neonatal mortality (Table 1). In addition, stillbirths were relatively high in the 1930s (66 per 1000 total births) and a large percentage of perinatal mortality (80-82%) was due to stillbirths indicating that conditions were difficult and many did not survive until birth.[10] Using the Bourgeois-Pichat (1952) biometric method to estimate endogenous mortality, it is not surprising to observe that endogenous mortality is more prevalent at lower levels of IMR.[11]

[insert Table 1]

[9] In 1924-25 IMR, neonatal and post neonatal mortality in Puerto Rico were 148.6, 43.3 (29%), and 105.4 (71%) respectively (Fernós Isern 1928). In 1924, for the state of New York excluding New York City: 69, 40.74 (59%), 28.29 (41%) (Fernós Isern 1928); in 1921-1925 in England and Wales: 76, 33.4 (44%), 42.6 (56%) (Barker & Osmond 1986); and in 1928-1938 in England & Wales: 61.7, 30.9 (50%), 30.8 (50%) (Woolf 1947).

[10] As a way of comparison, Corsica in France in the 1950s had an endogenous mortality of 26.4 per 1000 and a stillbirth mortality of 19.9 for a total perinatal mortality rate of 46.3 per 1000, 43% of which was stillbirths (Pressat 1961).

[11] The Bourgeois-Pichat method makes the following assumptions: (1) exogenous mortality conserves a stable age structure, (2) there is little endogeneity after the first month of life. Therefore, the role of medicine is minimized in this method although it probably was not an issue in the 1920s-1940s in Puerto Rico.

Cause of death

Puerto Rico is situated in the tropics and experienced different climate and disease environments than those of England or Wales during the 1920s. High temperatures year round nurtured infection and thus resulted in generally higher IMR than experienced in northern climates. Malaria and hookworm were common infectious diseases during the late 1920s-early 1940s and add to the complexity of understanding IMR in Puerto Rico.

There were four major reported causes of IMR during the late 1920s-1940s. Table 2 describes the situation in a typical year, 1934: (1) diseases of the digestive system (about 35% of cases), of which diarrhea and gastro-enteritis were predominant; (2) diseases of early infancy (about 24% of cases), of which congenital disability predominated; (3) diseases of the respiratory system (about 18% of cases), of which acute bronchitis predominated; and (4) infectious diseases (about 16% of cases). Factors impacting IMR differed with the age of the infant as can also be seen in Table 2. Most early infancy causes of death including congenital disability occurred during the first month of life whereas diarrhea, acute bronchitis and other infectious diseases were predominant causes of death during post neonatal (1^{st}-11^{th} month of life).

As for the first major cause of death, there was a strongly held belief by public health officials of the day that even though there were difficulties in the reliability of cause of death data, feeding habits were an important cause of diarrhea (Fernós Isern 1928). It was noted by officials that breastfeeding, which is natural and important in the first six months of life, was often impeded due to mothers (especially poor mothers) having to return to working 10-12 hours per day (Fernós Isern & Rodriguez Pastor 1930). This led to irregular hours of breastfeeding. Although cow's milk was scarce in Puerto Rico, mixed or artificial feeding (canned milk, mixtures of herb teas in dirty bottles) during the first month of feeding occurred (Fernós Isern &

Rodriguez Pastor 1930).[12] Unclean breasts may also have been a contributing factor in some cases. It is also important to note that mortality from diarrhea under the age of two was not associated with climate change or rainfall variations with the seasons of the year whereas for children over the age of two, mortality was associated with rainfall and season (Fernós Isern & Rodriguez Pastor 1930; Phelps 1928), thus leading to a stronger argument that feeding habits during the first year of life may truly have been an important cause of death whereas environmental factors became important at older ages.

[Table 3 about here]

Infections such as malaria and hookworm may be an important explanatory factor for the second leading cause of death, congenital weakness, in Puerto Rico during the 1920s-1940s. Both malaria and hookworm infection reflect the burden of disease in pregnant women which may have manifested themselves partially by congenital problems at birth. Prevalence of malaria occurred mostly in the very hot coastal regions where sugar cane was planted. An association between congenital mortality and malaria was noted by many Puerto Rican public health officials of the time[13] (Fernós Isern & Rodriguez Pastor 1930) and was thought to have more to do with the mother having the disease since the mother's weakened condition can lead to congenital weakness in the infant depending on the seriousness of the infection. The highest

[12] Wegman et al. (1942) also found that in the rural municipio of Ciales (page 237) a very high percentage of mothers who used the public health unit breastfed up to 6 months (not a representative sample). Low IMR are associated with breastfeeding and in the rural areas many mothers would be breastfeeding and "rural mothers tend to nurse their babies longer" (Wegman et al. 1942). Wegman et al. also noted two unusual facts about IMR—1) rapid decline in neonatal mortality rate but not 1st-11th month rate; 2) geographical distribution of IMR—higher IMR in western part, coastal regions and lower IMR in central and eastern regions of Puerto Rico (page 244).

[13] Example: The municipio, Santa Isabel (located in the southern coast) experienced high IMR but then a dramatic decline in IMR after a successful malaria campaign in the late 1920s. Mortality from congenital disability was also decreasing by the late 1920s (Fernós Isern & Rodriguez Pastor 1930).

IMR during the late 1920s and early 1940s tended to be in the coastal southern and western regions of Puerto Rico where the prevalence of malaria was high.[14] Prevalence of hookworm was high in rural areas and suggests that many lived with chronic infection throughout the year.[15] High loads of hookworm are associated with intrauterine growth retardation, prematurity and low birth weight among newborns born to infected mothers and can lead to anemia especially if poor nutrition (protein and iron) is involved.[16] Because it can cause protein malnutrition, anemia is a potential confounding factor for the Barker theory. A high percentage of adults did not wear shoes in rural areas and thus there was a high prevalence of hookworm disease.[17] While hookworm tends to be associated with men and certain occupations (coffee in the case of Puerto Rico and mining in the case of England and Wales),[18] in certain cases adult women in Puerto Rico may also have been more severely affected than men in the coffee regions because of soil conditions close to housing (Hill 1926).[19]

In terms of the third leading cause of death--respiratory infections--public health officials in Puerto Rico at the time generally thought that the "most important factor…is direct contagion

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[14] Is this partially explained by mothers who had malaria and thus resulted in congenital difficulties?

[15] Malaria and hookworm infections were chronic conditions in the rural areas of Puerto Rico but were not seasonal. Note: a high prevalence of hookworm infection does not mean high severity. Historical records do describe the severity (worm burden) of hookworm in certain regions and *municipios* of Puerto Rico (Hill 1926), but the severity of the infection in other regions is not clear. In the mountainous interior of the island, infestation averages were estimated at 500 parasites per person whereas in the coastal areas 200 per person (Hill 1926). In our case, we are interested in maternal infection which may have caused nutritional problems for the developing child. There is some evidence to suggest that hookworm infection by itself may not be the problem but other nutritional factors which affect disease (Gilles & Ball 1991). Nevertheless, even Gilles and Ball (1991) noted the high average infestation averages for Puerto Rico (450) suggesting that the infection was a serious health issue in Puerto Rico during the late 1920s-1930s.

[16] According to the World Health Organization, "In pregnant women, anemia resulting from hookworm disease results in several adverse outcomes for both the mother and her infant, including low birth weight, impaired milk production, and increased risk of death for both the mother and the child." See the following website: http://www.who.int/vaccine_research/diseases/soa_parasitic/en/index2.html

[17] In a typical sugar plantation, about 60-69% of those above 15 years old did not wear shoes. And many young children do not wear shoes (Morales Otero et al. 1937, p. 460).

[18] Highest rates in coffee cultivation; average infestation rate of 90% with an extremely high prevalence of hookworm disease—a disease prevalent in Puerto Rico since 1530 (Howard 1928; Daengsvang 1932; Hill 1926).

[19] It is more difficult to identify the severity of hookworm disease since the number of worms is important in terms of morbidity risks for the mother—the mother could be pregnant and be infected but not have that many worms although low worm burdens may also lead to anemia.

from older persons suffering from bronchitis and influenza…" (Fernós Isern & Rodriguez Pastor 1930). Already undernourished children exposed to adults with the disease were more susceptible to the disease. Thus, educating older persons to protect children from disease in combination with socioeconomic conditions were important underlying factors explaining the prevalence of these infections.

The fourth leading cause of death was from epidemic, endemic and other infectious diseases affecting newborn infants such as malaria and tetanus. Other diseases such as tuberculosis and measles could escape detection as causes of death and be hidden in diagnoses of either gastrointestinal or respiratory causes of death (Fernós Isern & Rodriguez Pastor 1930).

Variation among municipios

Conditions during the late 1920s-early 1940s in Puerto Rico were such that while IMR began to improve during this period, there was a still large amount of variation within and among municipalities (*municipios*) across time. Figure 3 shows IMR per year for each *municipio* but sorting *municipios* from lowest to highest IMR in 1929. The bold line in the figure shows IMR in 1929 and the bold dashed line shows IMR in 1943. Higher IMR in the late 1920s tended to be found in coastal areas where sugar cane was planted and especially in western Puerto Rico where sugar cane and coffee were planted, where there was a high prevalence of malaria and where temperatures were consistently high throughout the year.[20] Higher IMR also existed in urban centers because the population density was greater than in more rural areas due to overcrowding of families in urban areas. Lower IMR in the late 1920s were found in the highlands where

[20] It is noteworthy to observe that all *municipios* in 1929 which had an IMR >=200 came from the southern or western coastal regions of Puerto Rico which were predominantly sugar cane regions although some parts of the coffee region in the western region also experienced higher IMR. *Municipios* showing IMR >=200 in 1929 included: Aguada, Anasco, Arroyo, Camuy, Guanica, Guayama, Hormigueros, Juana Diaz, Lajas, Las Marías, Maricao, Mayaguez, Moca, Patillas, Ponce, Rincón, Sabana Grande, Salinas, Santa Isabel and Yabucoa. Although there was improvement with time in IMR, many of these same *municipios* still ranked the highest in IMR in 1943.

tobacco was grown and the lowest IMR in the island at the time were found curiously enough in the center of the island between the coffee and tobacco regions but close to the sugar cane region.[21] Within each county across the time period of the late 1920s-early 1940s, IMR were high during the late 1920s and then improved again during the 1930s as more public health units[22] were constructed and made available in *municipios* throughout the region.

[insert Figure 2 about here]

Methods

Data

The data for this paper come from two major sources: (1) yearly data from 1927-1943 on IMR by *municipio*, acreage planted and amount harvested for major crops (sugar cane, tobacco, and coffee) in 1929 found in either US Census reports (1929), Puerto Rican public health journals or annual reports on the health of the island produced by the Puerto Rican Health Commissioner during the 1920s-1930s; and (2) comprehensive data from the Puerto Rican Elderly: Health Conditions (PREHCO) project,[23] a project designed to gather quality baseline data on issues related to the health of the non-institutionalized population age 60 and over and their surviving spouses.

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[21] All *municipios* in 1929 which had an IMR <100 came from the central regions of Puerto Rico where mostly tobacco was grown. These included: Aguas Buenas, Aibonito, Barranquitas, Ciales, Cidra, Coamo, Jayuya, Naranjito, Orocovis, Vega Alta and Villalba. Some public health officials attributed lower IMR in the central region to the milder climate in the mountain ranges (Fernós Isern & Rodriguez Pastor 1930). Although population density was associated with higher IMR in Puerto Rico (Fernós Isern & Rodriguez Pastor 1930), when we did an analysis of the five highest IMR and lowest IMR *municipios* according to population density, there were equally higher and lower density areas in both groups.

[22] The first public health unit was created in 1926 and was funded by the Rockefeller Foundation and the US Public Health Service. By 1938, each municipality had its own public health unit.

[23] See http://www.ssc.wisc.edu/cdha/projects/projects.html.

Today Puerto Rico is divided into 78 *municipios* governed by an elected mayor and legislative assembly. The current location of *municipios*, for the most part, conforms to how Puerto Rico was divided in the late 1920s-early 1940s. According to the 1920 and 1930 census, most of the population of Puerto Rico was classified as rural and only a few *municipios* were predominantly urban.[24] The collection of vital statistics in Puerto Rico in the early 20th century was the responsibility of *municipio* clerks under direct supervision from the mayor of each municipality. While law required that "daily statistics shall be kept in all the registers which must contain such information as may be determined by the Director of Health" (Pérez 1926), the Department of Health did not have direct oversight into the local registers and how data were collected until the 1930s. In fact, it is rather dubious if municipal authorities inspected the civil registers in the early 1920s. However, Puerto Rican laws established during this period clearly outlined fines for not reporting deaths and required that a physician sign the death certificate and thus the Department of Health viewed the numbers of deaths collected by each *municipio* as fairly accurate and believed that almost all deaths were reported. According to the Department of Health, over 90% of births were reported (Porto Rico Health Review, October 1926, page 15) and thus the reporting of numbers of births and deaths was viewed as being fairly accurate.[25]

[24] US census defined rural as communities with less than 2500 inhabitants (U.S. Bureau of the Census, 1932). Only San Juan, Bayamon and Ponce had predominantly urban populations in 1920. By 1930, Catano and Mayaguez became predominantly urban.

[25] The 90% estimate agrees with official estimates of underreporting in later annual reports to the governor. Furthermore, the problem with underreporting births appears to be related with missing birth reports for infant deaths under one year. In the Fiscal Year (FY) 1930 report, an examination of December 1929 deaths under one year with matching birth reports found that about 45% of births associated were not reported (FY 1930 Annual Report to Governor, page 8): "According to law death reports are 100 per cent complete as no burial permits are issued without a certificate of death from the physician, but on the other hand, birth reports, taking the month of December, 1929, as a basis are only about 55 per cent complete…The infant mortality rate….would be 125 per 1,000 live births and after proper checking 118 per 1,000 live births." Thus, in this example, there was about a 6% difference between the higher calculated and lower corrected infant mortality rate for December 1929. It is not clear from the annual reports to the governor the degree to which underreporting of births may have occurred in instances which did not also involve the death of the infant under one year of age.

The important weakness of the system was the classification of births and deaths by race, domicile, age and occupation and the reporting of the cause of death and morbidity. There were no uniform standards across *municipios* for classification into groups. In the 1920s, the Department of Health actively classified deaths according to international standards of disease classification, but many Puerto Rican physicians did not conform to the international standards of disease classification or did not report communicable (transmissible) diseases as required by law. In addition, because of the scarcity of physicians,[26] especially in the rural areas, the physician who signed the death certificate often did not actually see the deceased but gave a diagnosis based more on guessing than scientific reasons.

In terms of the survey data used in this paper, the PREHCO sample is a multistage, stratified sample of older adults residing in Puerto Rico with oversamples of regions heavily populated by people of African descent and of individuals aged over 80. The data were gathered through face-to-face interviews with targets and with their surviving spouses, regardless of age. The data collected offer a substantial amount of information within the limits permitted by face-to-face interviews in a cross-section. The questionnaire included extensive modules on a variety of topics including demographic characteristics, health status and conditions, cognitive and functional performance, anthropometric measurements and physical performance. A total of 4,291 interviews with primary respondents were conducted between May 2002 and May 2003 and second wave data were collected during 2006-2007 both with very high response rates.

Measures

Infant Mortality.—Data for infant mortality for 1927-1943 were obtained by *municipio* and for Puerto Rico as a whole based on official reports compiled by the Department of Health in

[26] In some areas there was one physician per 15,000 persons (Report of the Bureau of Vital Statistics 1925-1926; Porto Rico Health Review 2 (September 1925-June 1927):15).

Puerto Rico.[27] For the years 1927-1931 data were obtained using the Porto Rico Review of Public Health and Tropical Medicine which reported monthly births and deaths for each *municipio*. Yearly infant mortality rates by *municipio* were then calculated using these numbers.[28] For 1932-1943, infant mortality rates were obtained using the Puerto Rican Commissioner of Health annual report to the governor of Puerto Rico which contained already calculated yearly infant mortality rates for each *municipio*.

Infant mortality rate for each *municipio* was modeled in several ways: (1) as a continuous variable with no transformation; (2) as a continuous variable using a suitable transformation; we tried a logit transformation and also chose a square root and cubed root based on Box-Cox power transformation; (3) as a discrete variable using deciles, quartiles and quintiles of IMR; (4) as a discrete variable identifying extreme values of IMR for each *municipio* (values greater than 1 standard deviation from *municipio*-specific trend) using the Hodrick-Prescott (1997) method for decomposing trend from cycle using a filter value of 100.[29] We also use trend data as predicted IMR at birth to address the large variation in IMR across time within some *municipios*.

In addition, our first idea was to decompose IMR into neonatal and post neonatal mortality and then endogenous and exogenous mortality by using the Bourguois-Pichat (1952) method at the *municipio* level. However, information on neonatal and post neonatal mortality was only available at the level of Puerto Rico as a whole. Thus, we modified this approach by splitting respondents into two groups: those born in years of lower IMR and those born in years of higher IMR. If we can reasonably divide PREHCO respondents into two groups according to higher or lower IMR at respondent's birth, then we can estimate separate models for different early life

[27] See the citations for Fernós Stern, A. 1930/31, 1942/43, 1944/45; Garrido Morales, E. 1934/35, 1935/36, 1937/38, 1939/40; and Ortiz, P. N. 1925/26, 1926/27, 1929/30 in the references.
[28] For 1927 only January-July were published and for 1931 only January-September were published.
[29] We followed the suggestion of Bengtsson & Lindstrom (2000).

exposures—one reflecting poorer environmental conditions when exogenous mortality was more predominant and the other reflecting better environmental conditions when endogenous mortality was more predominant.

To divide the PREHCO sample we relied on the only known data on IMR and neonatal mortality in Puerto Rico from early periods (1932-1984) (Vázquez Calzada 1988). We then used the Bourgeois-Pichat method to estimate exogenous mortality[30] and estimated endogenous mortality accordingly. We noted at this point that it is only at very low levels of IMR when endogenous mortality is more predominant, but in the PREHCO sample of respondents IMR were still relatively high in 1927-1943 and thus there were fewer cases at very low levels of IMR. We thus searched for a more reasonable cutoff point by which to divide the sample. We made an important assumption. We assumed that the relationship observed between IMR and neonatal mortality in 1932-1984 was similar to the relationship observed in earlier years (1927-1943) so that we could use the same relationship to say something about IMR during 1927-1943. We tested three different cutoff points for endogenous mortality: (1) IMR less than or equal to 100 (completely arbitrary but close to IMR of the Barker & Osmond study in 1986); (2) lowest quartile of IMR (cutoff IMR 96.7); (3) lowest tercile of IMR.[31] We created suitable dichotomous variables for each of these cases and then used the variables to estimate separate multivariate models for endogenous and exogenous mortality. The results using all of these cutoff points were very similar and thus in this paper we show only one of the cutoff points---the lowest tercile of IMR (cutoff IMR 112 for actual IMR at birth and 113 for predicted IMR at birth).

[30] According to Bourgeois-Pichat (1952), about 25% of exogenous mortality was found in neonatal deaths and thus to obtain an estimate for exogenous mortality one need multiply post neonatal mortality by 1.25.

[31] Using Table 2, linear extrapolation and the Bourgeois-Pichat method we estimate that at the cutoff point of 100, the percent of neonatal mortality is 32% (endogenous mortality 15%). Similarly for the other cutoff point of 96.7 the percent of neonatal mortality is about 33% (endogenous mortality 16%) and for the cutoff point of 105 the percent of neonatal mortality is 31% (endogenous mortality 14%).

Prenatal exposure to poor nutrition.—We defined seasonal exposure to poor nutrition and infectious diseases based on the months of the slack or lean season (July–December) in the Puerto Rican sugar cane industry (Clark, 1930; Gayer, Homan, & James, 1938). Mid to late gestation and early infancy may all be periods sensitive to poor nutrition. However, we began with the supposition that late gestation is most relevant (Barker, 1998) and that exposure to poor nutrition and infectious disease during late gestation was higher in those infants born in the lean season. We called this broad definition of exposure to poor nutrition and infectious diseases in model estimation *exposure period*, which identified whether the respondent had been born during the lean season or after the sugar cane harvest (July–December).

Adult Health.— Elderly adult health was defined by dichotomous variables using self-reported heart disease and self-reported diabetes from the PREHCO study. These variables ask the respondent if a doctor has ever diagnosed them with heart disease or diabetes.

Childhood conditions.—Early life conditions in addition to *in utero* exposures (season of birth) included lowest quartile of knee height, an indicator of nutrition during early childhood (Eveleth & Tanner 1990) and possibly earlier; a retrospective question asked of respondents regarding socioeconomic conditions during childhood and mother's exposure to malaria. This indicator was obtained by identifying *municipios* where malaria infection was highest either through published accounts[32] or through an analysis of acreages of sugar cane planted obtained through the US census (1932).

Adult conditions.—These conditions included the number of years of education, obesity (a dichotomous variable indicating if BMI>=30) and adult behavior (smoking, and exercising).

[32] These *municipios* were identified in several articles that appeared in the *Porto Rico Review of Public Health and Tropical Medicine* on malaria. *Municipios* with high malaria rates were Barceloneta, Fajardo, and Ponce (Earle 1925). In addition, hookworm was more prevalent in the coffee growing areas and so we also identified predominant coffee growing regions.

The smoking variable was defined according to non-smokers (never smoked), former smokers and current smokers. In terms of exercising, respondents were asked if during the last year they had played sports, jogged, walked, danced or did heavy work three or more times per week. Responses to this question were dichotomized with 1 reflecting an affirmative response to the question and 0 reflecting a negative response to the question.[33]

Analysis

Imputation.—We used multiple imputation procedures (Raghunathan, Reiter, & Rubin 2003; Rubin 1987; Van Buren, Boshuizen, & Knook 1999) using IVEware (Raghunathan, Solenberger, & Van Hoewyk 2007) to ensure that all cases were included. The results obtained with multiple imputation are statistically efficient and avoid systematic biases likely to arise when one deletes cases with missing observations. The validity of our results relies on a weak assumption about missingness (Rubin 1987). The number of missing responses among the subsample of those born in Puerto Rico who lived in the countryside as a child was small (less than 1 percent) in most study variables; knee height and obesity had about 3% missing. However, we were primarily interested in imputing items about living in the countryside as a child, for which about 14% of the cases were missing primarily because proxies were not asked this question.

Subsample for estimation.—We selected a subsample of older adults born in Puerto Rico who responded affirmatively to a survey question that asked them if they had lived for a prolonged period of time in the countryside prior to the age of 18. The imputation created five imputed data sets, each of which varied slightly in sample size (from 1447-1464). We only considered respondents aged 60 to 74 to generate estimates for the subpopulation that was most at risk of

[33] Weighting imputed results for 60-74 year olds who lived in the countryside as children: about 21% responded saying that they drank alcoholic beverages on a weekly basis during the last three months; about 44% responded saying that they exercised at least three times a week during the last year and about 65% responded saying that they had never smoked; 25% were former smokers and 10% current smokers.

having been affected by harsh early childhood experiences and, simultaneously, had larger probabilities of surviving due to their exposure to the massive deployment of medical technologies and public health measures during the period after 1930. Thus, this cohort may be able to provide some insights into whether early childhood experiences are indeed important in later life because it was less affected by mortality-driven selection than the group of cohorts who preceded it (those aged 75 and older).

Preparation for model estimation.— Municipality-level IMR data were linked with the *municipio* of birth of PREHCO respondents who were born between 1927 and 1943 (excluding those respondents not born in Puerto Rico). We transformed IMR using either a logit, square root or cubed root transformation based on Box-Cox power transformations. Given the large variation in IMR across time within some *municipios,* we also estimated trends for each *municipio* across the time period using the Hodrick-Prescott (1997) method for decomposing trends from cycles with a filter value of 100 and then also used predicted values in model estimation.

Estimation.—We first examined the bivariate association between predictors and health outcomes both overall and within subgroups. We estimated the effects of IMR (using continuous and different Box-Cox transformations, quantiles, lowest tercile of IMR) on adult heart disease and diabetes using logistic regression and both actual and predicted IMR at birth. Models for heart disease controlled for age, gender, education, obesity, poor childhood health, poor childhood socioeconomic status (SES), low knee height, and exposure (lean) period. Models for diabetes also included a variable indicating whether the respondent had a family member with diabetes. We also tested interaction effects of season of birth and IMR at birth and then estimated separate models for each of the two subgroups.

Results

The distribution of IMR at respondent's birth when linked with PREHCO respondents (n=1447, first imputed data set) shows that most respondents were born in years with relatively high IMR. Approximately 40% of rural-born 60-74 year old respondents were classified as being born when infectious disease loads were lower and endogenous mortality more predominant (cutoff point of 112) (Figure 4). An examination of several childhood and adult characteristics with IMR at respondent's birth showed (Table 3) that there were significant differences between IMR and age—a result to be expected since infant mortality improved between 1927 and 1943 ($F=50.55$, df 2, $p=0.0000$). There were also significant associations between higher IMR at respondent's birth and lower adult education ($F=2.99$, df 3, $p=0.0298$); low knee height ($F=18.52$, df 1, $p=0.0000$); and no exercise as an adult ($F=8.94$, df 1, $p=0.0028$). There were no significant differences between IMR at respondent's birth and other childhood and adult variables including adult heart disease and diabetes. The results remained the same with each of several different transformations of IMR (continuous, logit, square root, cubed root, deciles, quartiles, quintiles and extreme values), using multivariate models that also included a basic model with only IMR as predictors and estimating separate models for each exposure period (results not shown). There were no significant interaction effects between season of birth and IMR at birth (results not shown). An examination of associations with knee height, education and exercise and other childhood and adult characteristics suggested several possible complex pathways through which IMR could possibly negatively impact health leading to adult heart disease and diabetes (Figure 5). In particular, the importance of knee height, educational attainment and exercise as an adult stand out as possibly playing a potentially mediating role.

[insert Figures 4 & 5 and Table 3]

The strong association between IMR at respondent's birth and low knee height is further examined in Table 4. Previous studies have shown low knee height is associated with higher odds of adult diabetes (Palloni et al. 2005) and thus it may be that knee height mediates the effects of IMR on adult health. There are very strong associations between IMR at birth and knee height using either the continuous or lowest tercile measure for IMR as noted in basic models controlling for age and gender (Models 1 & 2). Controlling for other childhood conditions such as poor child health, poor child SES and lean exposure period in addition to adult obesity and education do not change the results (Models 3 & 4).

[Insert Table 4 about here]

In the full model for heart disease (Table 5, Model 1), the odds of reporting heart disease are about 40% higher for those born during the lean season than those born during the harvest season Separate estimation by infectious disease load groups showed that the effects of (1) exposure period (lean period) are significant and stronger for the **Low IMR at birth** group (Models 2a & 2b) and become insignificant for the **High IMR at birth** group (Models 3a & 3b); (2) obesity is stronger in the **Low IMR** group; (3) previous smoking is significant in the overall Model 1 but then only significant in the **Low IMR** group; (4) exercise shows a protective effect on heart disease in the overall Model 1 and then only in the **High IMR** group.

[Insert Table 5 about here]

In the full model for diabetes (Table 6, Model 1) there are strong effects of having a family member with diabetes, being born in the exposure (lean) period, having a family member with diabetes and being born in the exposure period, being obese and exercising. Exercising lessens the odds of diabetes by about 33%. When models are estimated separately, smaller sample sizes for the **Low IMR** group produced less robust effects. Tentatively then, the effects of: (1) exposure (lean) period on diabetes are stronger in the **Low IMR** group (Models 2a & 2b) than in the overall model and insignificant in the **High IMR** group (Models 3a & 3b); (2) effects of having a family member with diabetes are strong in both the **Low IMR** group and the **High IMR** group; (3) interaction effects between family member with diabetes and exposure period are significant only in the **Low IMR** group; (4) obesity is significant only in the overall model and the **High IMR** group; (5) exercise shows a protective effect on lowering the odds of diabetes by about 33-37% across all models while smoking shows a similar protective effect in the **High IMR** group.

[Insert Table 6 about here]

Discussion

In this paper we collected historical data on IMR per *municipio* in Puerto Rico from 1927-1943 and linked them with respondent's place and year of birth using a comprehensive survey of Puerto Ricans aged 60 and older (PREHCO) in order to help disentangle the effects of early exposures *in utero* and early infancy. By the late 1920s and early 1940s, IMR was declining in Puerto Rico although it was still relatively high in comparison with the US and the UK,

reflecting poorer environmental conditions. The **first** set of results concerns associations between IMR and adult health and the degree to which the effects of season of birth on adult heart disease and diabetes are attenuated when IMR is included in model estimation. We found no significant association between IMR and adult heart disease or diabetes and thus no attenuation of seasonality effects. The odds of reporting heart disease and diabetes were higher among those born in the lean period as noted in previous work (McEniry et al. 2008) even when other early life conditions and adult life course factors (smoking, drinking and exercising) were added to model estimation. However, IMR was associated with higher age, low knee height, lower education and not exercising as an adult and these variables are associated either directly or indirectly with adult heart disease and diabetes. Initial conclusions: (1) IMR, largely reflecting environmental and poor economic conditions in Puerto Rico, affects adult health indirectly through a number of complex pathways; (2) the effects of IMR may be mediated through knee height and educational attainment.[34] The **second** set of findings examined the effects of season of birth according to the predominance of endogenous mortality as proxied by low and high IMR. We found, as expected, that there were stronger effects of season of birth among PREHCO respondents who were born in years when the fraction of endogenous mortality was higher (i.e., lower IMR and better environmental conditions) as compared with PREHCO respondents who were born in years when the fraction of endogenous mortality was lower (i.e., higher IMR and poorer environmental conditions). There were strong seasonality effects even after controlling for other childhood and adult life course factors. Curiously, we also found that the effects of adult behavior were different between groups—strong positive effects of being a

[34] Educational attainment and adult exercise are also important but we examined knee height in this paper due to previous work with diabetes (Palloni et al. 2005).

former smoker on heart disease among those born when endogenous mortality was more predominant.

As to the **first set of findings**, other studies have found a strong positive association between IMR at respondent's birth and adult mortality (Bengtsson & Lindstrom 2000; Bengtsson & Lindstrom 2003). It may be that the results are partially explained because of the different reasons for the causes of infant deaths in Sweden during the 18th and 19th century and Puerto Rico during the early 20th century. Smallpox and whooping cough dominated IMR in Sweden at the time; these diseases affected everyone across social classes and thus economic factors were not the main drivers of IMR (Bengtsson & Lindstrom 2000). This was not the case in Puerto Rico. Even though there are studies which attribute poor childhood economic conditions to adult health (Hertzman 1994; Lundberg 1991; Wadsworth & Kuh 1997; Wickrama, Conger, & Abraham 2005), the exact mechanisms by which these conditions could lead to adult heart disease and diabetes are not clear. It may thus be that IMR in the case of Puerto Rico is too crude an indicator of environment and economic disturbances to be able to discern particular pathways to adult heart disease and diabetes.

Other reasons that explain differences in non-significance of results for IMR are: (1) the health outcome is morbidity whereas other studies have examined adult mortality; there may be more ambiguity in measuring morbidity. However, even though there may be an underestimation of disease from self-reported heart disease or diabetes, other studies have shown that underestimation provides more conservative estimates but not extremely so (Banks et al. 2006; Goldman et al. 2003; Riosmena & Wong 2008). Regardless, the results obtained in this paper could be considered conservative and more so since imputed results also produced more conservative results than did non-imputed results; (2) the cross sectional nature of the PREHCO

study means that we cannot observe those who have already died before the study began. However, mortality selection is less of a problem in the younger age group of older adults selected (60-74 years old) than it is with much older adults.

The observation that there is an association between childhood health, poor childhood SES, low knee height and education but not between IMR at respondent's birth and childhood health or poor childhood SES suggests that childhood health and childhood SES represent environmental conditions at different periods in childhood. Why is there no association between IMR at respondent's birth and childhood health or childhood SES? Again, it may be that each is tapping into a different dimension of the environment.

The significant association between IMR at respondent's birth and age is not surprising given that mortality and IMR began to significantly decline during this period in Puerto Rico. However, the associations between higher IMR and low educational attainment, not exercising and low knee height as an adult raise interesting issues. **First**, the negative association between IMR at respondent's birth and ***adult educational level*** suggests that poor environmental conditions during the first year of life impacts adult educational attainment, a finding pointed out by many others (Palloni 2006). The association between IMR at respondent's birth and education merits further investigation (beyond the scope of this paper) to better understand the different pathways to adult diabetes and heart disease through education and adult exercise. It may be that the curious direct association between IMR at respondent's birth and adult exercise can be explained by the association between IMR and education. It is reasonable to believe that there is a more complex pathway from IMR to adult heart disease or diabetes and that this pathway is through education--lower IMR at respondent's birth is associated with higher educational levels, higher educational levels are associated with more exercise as an adult, and

exercise is associated with lower prevalence of adult diabetes and heart disease. **Second**, the significant association between IMR at respondent's birth and low knee height suggests that poor environmental conditions during the first year of life are associated with stunting in childhood. Knee height is an important factor affecting adult diabetes in Puerto Rico (Palloni et al. 2005) although not as important (disappointingly so) to adult health in other Latin American or Caribbean countries. It may be that knee height is a more suitable indicator of poor early childhood conditions but not necessarily *in utero* conditions, or it may be that other confounding factors are present. The initial association between knee height and diabetes, disappearing after "family member with diabetes" is entered into model estimation, certainly suggests that a mixture of environmental and genetic factors affect knee height.

In terms of the **second set of findings** which divide older adults into subgroups according to the predominance of endogenous mortality, the results suggest that endogenous (*in utero*) conditions have an important effect on older adult heart disease and diabetes, thus providing (weak) evidence in support of the Barker (1998) hypothesis. These results do not mean that there are no endogenous effects on adult health at higher IMR because other studies show strong associations between neonatal and adult mortality (Barker & Osmond 1986). Rather they suggest that season of birth may be a better indicator of endogenous exposures at lower levels of IMR when there is strong marked nutritional seasonality but weaker seasonality of infectious diseases.

There were necessarily smaller sample sizes in the group reflecting high endogenous mortality due to the relatively high IMR in Puerto Rico during the 1920s-1940s and thus model convergence was a problem in the case of diabetes. The cutoff points by which we split the sample are arbitrary (cutoff points were at approximately 30% neonatal and 15% endogenous

mortality) although they correspond with other studies (Barker & Osmond 1986; Doblhammer 2004). The ideal would have been to have a lower cutoff point for IMR but there were an insufficient number of cases with respondents born with IMR lower than 60 (for example). In that sense, we may indeed be observing what some have described as the "tip of the iceberg" (Palloni et al. 2005) because the PREHCO study includes only those born up until 1943. Thus, there may be a larger group of older adults born in the late 1940s and 1950s who are also at risk due to poor early life exposures.

The results obtained showing different effects of adult behavior (smoking in particular) according to lower or high fractions of endogenous mortality are intriguing. The Barker hypothesis suggests that *in utero* exposure to poor nutrition may lead to increased vulnerability or susceptibility to environmental conditions after birth (Barker 1995; Barker et al. 2001). It may be that smoking taps into this vulnerability.

In addition to the limitations already cited we present a few more caveats to the analysis appearing in this paper. **First**, approximately 10% of infant births were not reported during the time period 1927-1943 which would tend to overestimate IMR at respondent's birth. We do not have data to show if this was the case across all *municipios* or across only a few. However, the use of subgroups of respondents and the use of predicted IMR at birth may minimize the error due to underestimation of births. **Second**, there are no reliable data on IMR by rural versus urban areas and thus the IMR we use in the models are at the entire *municipio* level. However, about 50% of the *municipios* were identified as completely rural by the US census of 1930 (US census 1932) and about 10% were completely urban with the remaining showing a high percentage of rural residents. IMR was higher in the urban areas but because of the predominant rural population the error we make when we use IMR at the *municipio* level is probably small.

Third, even public officials of the day warn against interpreting rates by municipality (Morales Otero et al. 1937) because in some cases the rates are based on small numbers and there are different factors that may influence the rates from *municipio* to *municipio*. We partially addressed this by using predicted IMR at birth. **Fourth**, we use cross sectional data which is representative of the population of Puerto Ricans aged 60 and older but which is not representative of the *municipios*. Thus, we cannot examine IMR by *municipio* and adult health as did Barker and Osmond (1986).

In conclusion, by examining the effects of IMR at respondent's birth and season of birth on adult heart disease and diabetes overall and according to subgroups of older adults exposed to different early life exposure, we have added to understanding the conditions under which season of birth is an important indicator of early life exposures. The findings provide some evidence that it can be. In that regard, there is merit to continue to dig even further in order to fully illuminate this most interesting and compelling story regarding early life conditions and adult health in Puerto Rico.

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Table 1: Infant neonatal and post-neonatal mortality and endogenous and exogenous mortality in Puerto Rico

Year	IMR	Neonatal	Post-neonatal	% neonatal	Endogenous	Exogenous	% Endogenous
1935-39	122.6	36.1	86.5	29.5	14.5	108.1	12
1940-44	105.3	32.9	72.4	31.2	14.8	90.5	14
1945-49	**78.9**	**28.8**	**50.1**	**36.5**	**16.3**	**62.6**	**21**
1950-54	64.7	26.6	38.1	41.2	17.1	47.6	26
1955-59	52.6	25.9	26.7	49.2	19.2	33.4	37
1960-64	44.6	25.1	19.5	56.3	20.2	24.4	45
1965-69	34.8	23	11.8	66.1	20.1	14.8	58
1970-74	26.1	20.2	5.9	77.3	18.7	7.4	72
1975-79	19.9	15.7	4.2	78.7	14.7	5.3	74
1980-84	17.5	14	3.5	80.0	13.1	4.4	75
1985	14.9	12.1	2.8	81.1	11.4	3.5	77

Source: First five columns are from Vázquez Calzada (1988) and are based on 5 year averages. Endogenous and exogenous infant mortality were calculated by the author using the Bourgeois-Pichat (1952) assumptions and biometric method. Bolded is a level of infant mortality which is very similar to overall infant mortality in England and Wales at the time of the Barker and Osmond study on infant mortality (1986).

Table 2: Major causes of infant mortality in Puerto Rico in 1934

Category	Predominant Diseases	All ages	Under one month	1st-11th month
Digestive system	Diarrhea & gastro-enteritis	35%	12%	48%
Early infancy	Congenital disability	24%	59%	5%
Respiratory system	Acute bronchitis	18%	9%	22%
Infectious diseases	Malaria, tetanus, TB, measles	16%	13%	17%
Total deaths		7442	2615	4827

Source: Report of the Commissioner of Health of Puerto Rico to the Governor of Puerto Rico for the fiscal year ending June 30, 1935, page 81. Note: Numbers for 1935, 1937, 1939 show similar percentages. Percentages in table do not add to 100. Not shown is other category (7-8%) comprised of unknown causes, nephritis and infantile convulsions.

Digestive system: diarrhea and gastro-enteritis, dysentery
Early infancy: congenital disability, other diseases of earl infancy, congenital malformations
Respiratory system: acute bronchitis, pneumonia, whooping cough
Infectious diseases: malaria, tetanus, TB, measles, syphilis, meningitis, diphtheria

A very high percentage of congenital disability falls within one month of life (82%) as does syphilis (64% of all cases of syphilis), infantile tetanus (99%), other diseases of early infancy (97%), and infantile convulsions (55%). All other mentioned diseases fall predominantly in 1-11 months of life.

Table 3: IMR at respondent's birth + comparison between lower/higher disease load due to infectious diseases

Variable	IMR	Lower load	Higher load	Significance
	Mean (sd)	%	%	p-value
Gender				
Female	119 (36.6)	54	50	0.239
Male	120 (35.1)	46	50	
Age group [1]				
60-64	107 (26.2)	51	31	0.000
65-69	121 (32.5)	33	38	
70-74	138 (45.3)	15	32	
Education [1]				
None	125 (38.1)	6	7	0.271
Primary	120 (35.8)	46	45	
Secondary	119 (36.7)	35	35	
Higher	116 (32.1)	12	12	
Obesity				
Obese	120 (37.1)	28	26	0.338
Not obese	119 (35.4)	72	74	
Poor childhood health				
Yes	121 (33.9)	23	26	0.298
No	119 (36.5)	77	74	
Poor childhood SES				
Yes	120 (36.6)	38	38	0.995
No	119 (35.4)	62	62	
Low knee height [1]				
Yes	128 (41.7)	19	28	0.000
No	117 (33.4)	81	72	
Family w diabetes				
Yes	120 (35.9)	60	60	0.423
No	118 (35.9)	40	40	
Smokes?				
Never	118 (35.3)	68	62	0.145
Previous	121 (37.0)	23	27	
Current	124 (36.3)	9	11	
Exercises? [1]				
Yes	117 (34.6)	47	41	0.017
No	121 (36.7)	53	59	
Broad exposure				
Harvest	119 (33.7)	48	50	0.755
Lean	120 (37.8)	52	50	
Heart Disease				
Yes	122 (38.5)	16	19	0.366
No	119 (35.3)	84	81	
Diabetes				
Yes	120 (36.9)	33	29	0.892
No	119 (35.4)	67	71	

Source: PREHCO, first wave, imputed data set, weighted; only those living in countryside as children and between 60-74 years of age (n=1447) using first imputed data set and weighted results. The overall weighted average for IMR at respondent's birth was 119 (35.7), n=1447. Group indicator: lowest tercile of IMR (lower disease load due to infectious diseases) and remaining terciles of IMR (higher disease load due to infectious diseases). Average IMR for lower disease load group 94 (21.1) and higher disease load 144 (30.5).

[1] Cases where there was a significant association with IMR at respondent's birth using age group ($p<0.001$); education categories ($p<0.05$); low knee height ($p<0.001$); and exercises ($p<0.01$). When predicted IMR at birth was used, these associations remained the same with the exception of education. When predicted IMR at birth is used to identify groups, results also remain the same as shown in table.

Table 4: Effects of IMR at respondent's birth on low knee height

	Knee Height Model 1 OR (SE)	Knee Height Model 2 OR (SE)	Knee Height Model 3 OR (SE)	Knee Height Model 4 OR (SE)
Female	1.13 (.14)	1.13 (.14)	1.09 (.14)	1.10 (.14)
Age 60-64 (ref)				
Age 65-69	1.01 (.15)	0.94 (.14)	0.96 (.15)	0.89 (.14)
Age 70-74	1.12 (.19)	0.97 (.17)	1.02 (.18)	0.88 (.16)
Education (yrs)			0.94 (.01)***	0.94 (.01)***
Obesity			0.82 (.13)	0.81 (.13)
Poor child health			0.79 (.12)	0.79 (.12)
Poor child SES			1.04 (.14)	1.04 (.14)
Lean period			0.89 (.12)	0.89 (.12)
IMR at respondent's birth (logit)		3.02 (.71)***		3.01 (.73)***
IMR at respondent's birth (lowest tercile)	0.60 (.08)***		0.59 (.08)***	
Range of n[1]	1421-1436	1421-1436	1421-1436	1421-1436
Range of LL[1]	-799, -787	-795, -783	-789, -774	-785, -770

Source: Puerto Rican Elderly: Health Conditions project (PREHCO 2007), imputed; all 60- to 74-year olds who lived in the countryside as children. Results shown are based on combining multiple imputation results.

*p<0.05, ** p<0.01, ***p<0.001

[1]The multiple imputation procedure required us to work with five alternative completed data sets. In this case it was not clear how to calculate conventional statistics, such as chi square, BIC, or Akaike's information criterion, all of which are functions of data-specific log-likelihood functions. As a partial resolution to the conundrum, we present in this table the range of values for the chosen statistics obtained after estimating models for each of the imputed data sets.

Table 5: Effects of early life conditions on heart disease by disease load due to infectious diseases

IMR at respondent's birth	Overall Model 1 OR (SE)	Lower IMR at birth[1] Model 2a Actual IMR OR (SE)	Lower IMR at birth[1] Model 2b Predicted IMR OR (SE)	Higher IMR at birth[1] Model 3a Actual IMR OR (SE)	Higher IMR at birth[1] Model 3b Predicted IMR OR (SE)
Female	1.12 (.18)	1.41 (.38)	1.05 (.27)	0.98 (.19)	1.13 (.23)
Age 60-64 (ref)					
Age 65-69	1.40 (.24)*	1.55 (.41)	1.65 (.44)	1.33 (.30)	1.33 (.30)
Age 70-74	1.62 (.29)**	1.76 (.55)	1.77 (.52)*	1.56 (.36)	1.56 (.37)
Education (yrs)	0.99 (.02)	0.98 (.03)	0.99 (.03)	1.00 (.02)	1.00 (.02)
Obesity	1.85 (.27)***	2.29 (.55)***	2.26 (.53)***	1.69 (.32)**	1.68 (.32)**
Poor child health	1.49 (.24)*	1.52 (.41)	1.31 (.35)	1.45 (.29)	1.60 (.32)*
Low knee height	1.03 (.17)	1.13 (.35)	0.93 (.30)	0.97 (.19)	1.10 (.22)
Poor child SES	1.09 (.16)	1.10 (.27)	0.97 (.24)	1.07 (.20)	1.20 (.22)
Lean period	1.42 (.20)**	2.20 (.52)***	2.86 (.68)***	1.12 (.20)	0.95 (.17)
Never smoked (ref)					
Previous smoker	1.44 (.25)*	2.27 (.63)**	1.96 (.52)*	1.11 (.24)	1.21 (.27)
Current smoker	0.77 (.22)	0.40 (.26)	0.57 (.33)	0.91 (.30)	0.83 (.28)
Exercises	0.71 (.10)*	0.96 (.23)	0.82 (.20)	0.59 (.11)**	0.65 (.12)*
Range of n[2]	1428-1443	570-577	571-580	851-860	849-857
Range of LL[2]	-688, -672	-251, -242	-254, -250	-428, -419	-426, -411

Source: Puerto Rican Elderly: Health Conditions project (PREHCO 2007), imputed; all 60- to 74-year olds who lived in the countryside as children. Results shown are based on combining multiple imputation results.
*p<0.05, ** p<0.01, ***p<0.001

[1]**Lower IMR at birth** is defined as the lowest tercile of either actual IMR at birth (cutoff 112) or predicted IMR at birth (cutoff 113) and **higher IMR at birth** is defined as the second and third tercile of either actual IMR at birth or predicted IMR at birth. Model 1: Full model; Models 2a and 3a used actual IMR at birth to separate respondents into groups whereas Models 2b and 3b used predicted IMR at birth to separate respondents into groups. Although the cutoff points are similar, actual and predicted IMR at birth do not always coincide and thus respondents were not all classified similarly when using actual and predicted values. About 85% of the cases matched; 8% changed from lowest tercile to a higher tercile and about 7% changed from highest tercile to a lower tercile when using predicted values.

[2]The multiple imputation procedure required us to work with five alternative completed data sets. In this case it was not clear how to calculate conventional statistics, such as chi square, all of which are functions of data-specific log-likelihood functions. As a partial resolution to the conundrum, we present in this table the range of values for the chosen statistics obtained after estimating models for each of the imputed data sets. Sample sizes for groups vary slightly because differences between actual and predicted IMR resulted in slightly different division into groups.

Table 6: Effects of early life conditions on diabetes by disease load due to infectious diseases

	Model 1	Lower IMR at birth [1]		Higher IMR at birth[1]	
IMR at respondent's birth		**Model 2a Actual IMR**	**Model 2b Predicted IMR**	**Model 3a Actual IMR**	**Model 3b Predicted IMR**
	OR (SE)	**OR (SE)**	**OR (SE)**	**OR (SE)**	**OR (SE)**
Female	0.94 (.13)	1.11 (.24)	1.00 (.22)	0.83 (.15)	0.89 (.15)
Age 60-64 (ref)					
Age 65-69	1.18 (.17)	1.39 (.30)	1.14 (.25)	1.05 (.20)	1.15 (.22)
Age 70-74	1.27 (.19)	1.82 (.46)*	1.41 (.37)	1.02 (.21)	1.15 (.24)
Education (yrs)	0.99 (.01)	1.01 (.02)	1.02 (.02)	0.98 (.02)	0.97 (.02)
Obesity	1.42 (.21)*	1.11 (.23)	1.27 (.27)	1.67 (.32)**	1.51 (.29)*
Poor child health	1.22 (.17)	1.33 (.30)	1.07 (.26)	1.22 (.22)	1.33 (.23)
Low knee height	1.28 (.18)	1.25 (.31)	1.55 (.40)	1.33 (.23)	1.16 (.20)
Poor child SES	1.03 (.14)	1.47 (.30)	1.16 (.24)	0.79 (.16)	0.94 (.17)
Family member with diabetes[2]	4.08 (.83)***	**4.17 (1.33)*** **	**5.26 (1.77)*** **	3.91 (1.02)***	3.39 (.85)***
Exposure period	1.86 (.42)**	2.09 (.71)*	2.09 (.77)*	1.62 (.52)	1.76 (.53)
Family member X exp. period interaction	0.54 (.15)*	0.44 (.18)*	0.41 (.18)*	0.71 (.27)	0.68 (.24)
Never smoked (ref)					
Previous smoker	0.78 (.12)	1.01 (.24)	1.15 (.28)	0.63 (.13)*	0.60 (.12)*
Current smoker	0.79 (.17)	0.44 (.19)	0.87 (.35)	0.94 (.25)	0.68 (.19)
Exercises	0.67 (.09)**	0.63 (.13)*	0.76 (.15)	0.66 (.11)*	0.63 (.11)**
Range of n[3]	1429-1444	570-577	571-580	851-860	849-861
Range of LL[3]	-832, -859	-341, -332	-332, -320	-503, -484	-516, -500

Source: Puerto Rican Elderly: Health Conditions project (PREHCO 2007), imputed; all 60- to 74-year olds who lived in the countryside as children. Results shown are based on combining multiple imputation results.
*p<0.05, ** p<0.01, ***p<0.001

[1] **Lower IMR at birth** is defined as the lowest tercile of either actual IMR at birth (cutoff 112) or predicted IMR at birth (cutoff 113) and **higher IMR at birth** is defined as the second and third tercile of either actual IMR at birth or predicted IMR at birth. Model 1: Full model; Models 2a and 3a used actual IMR at birth to separate respondents into groups whereas Models 2b and 3b used predicted IMR at birth to separate respondents into groups. Although the cutoff points are similar, actual and predicted IMR at birth do not always coincide and thus respondents were not all classified similarly when using actual and predicted values. About 85% of the cases matched; 8% changed from lowest tercile to a higher tercile and about 7% changed from highest tercile to a lower tercile when using predicted values.
[2]Smaller sample sizes for the lower disease load group produced higher standard errors for this variable and thus should be interpreted with caution.
[3]The multiple imputation procedure required us to work with five alternative completed data sets. In this case it was not clear how to calculate conventional statistics, such as chi square, all of which are functions of data-specific log-likelihood functions. As a partial resolution to the conundrum, we present in this table the range of values for the chosen statistics obtained after estimating models for each of the imputed data sets. Sample sizes for groups vary slightly because differences between actual and predicted IMR resulted in slightly different division into groups.

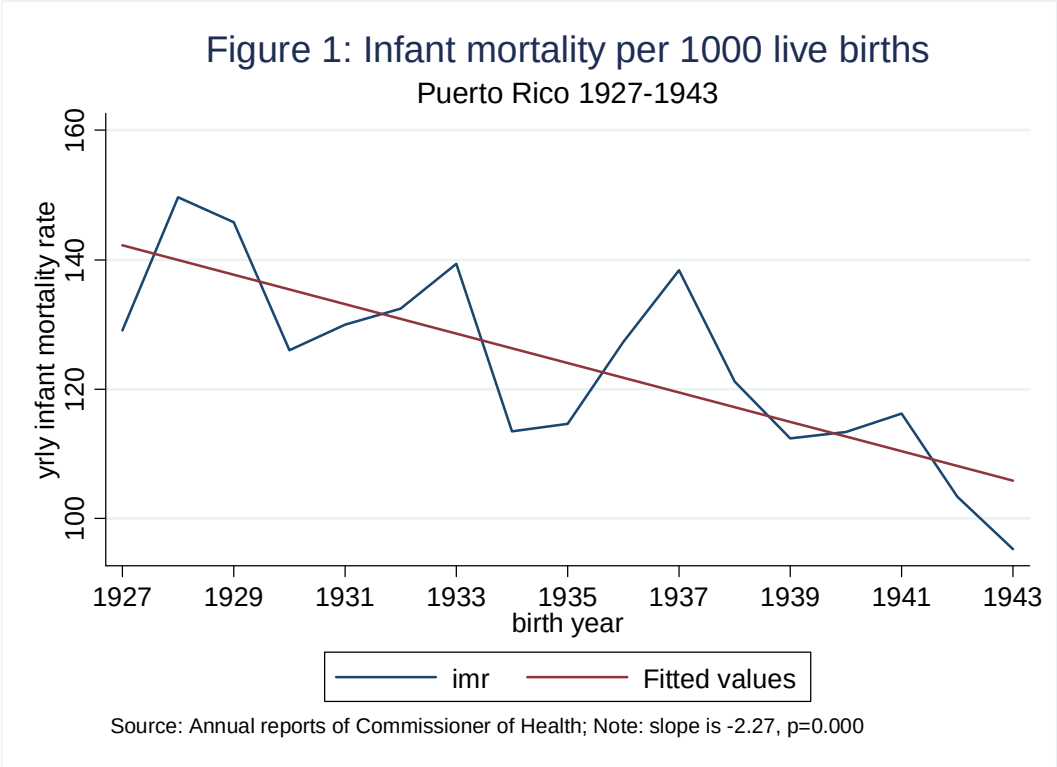


Figure 1: Infant mortality per 1000 live births
Puerto Rico 1927-1943
yrly infant mortality rate
100
120
140
160
1927
1929
1931
1933
1935
1937
1939
1941
1943
birth year
imr
Fitted values
Source: Annual reports of Commissioner of Health; Note: slope is -2.27, p=0.000

Figure 2: Mortality rates in Puerto Rico (1927-1943)

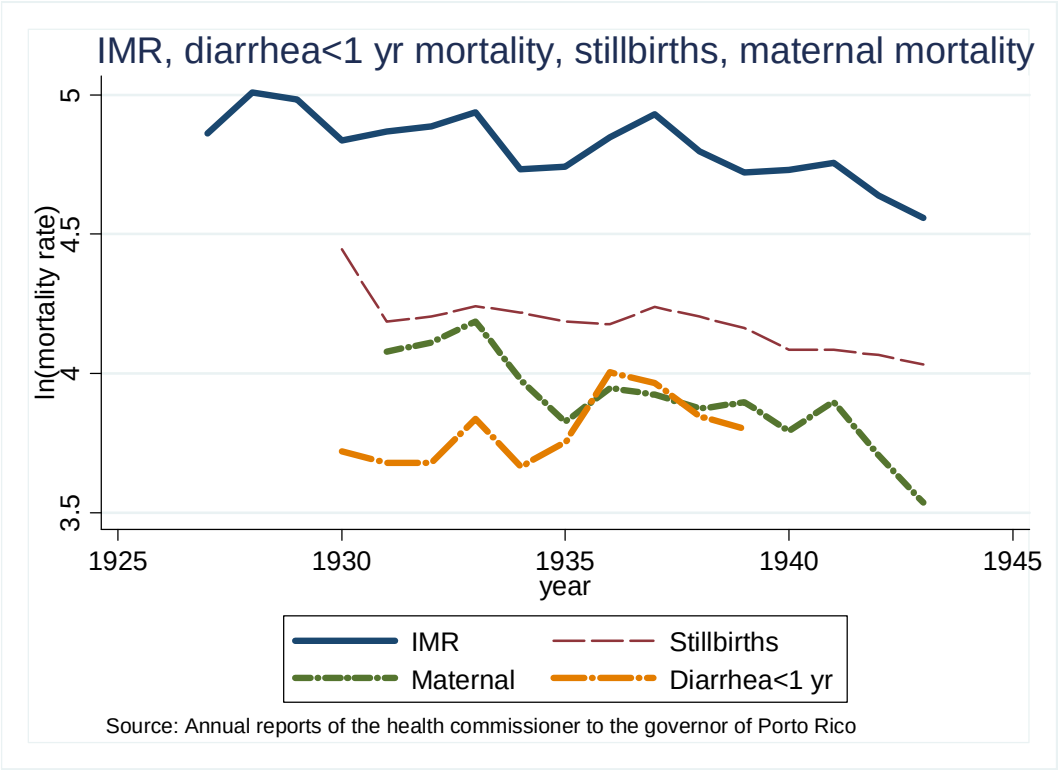


Figure 3:

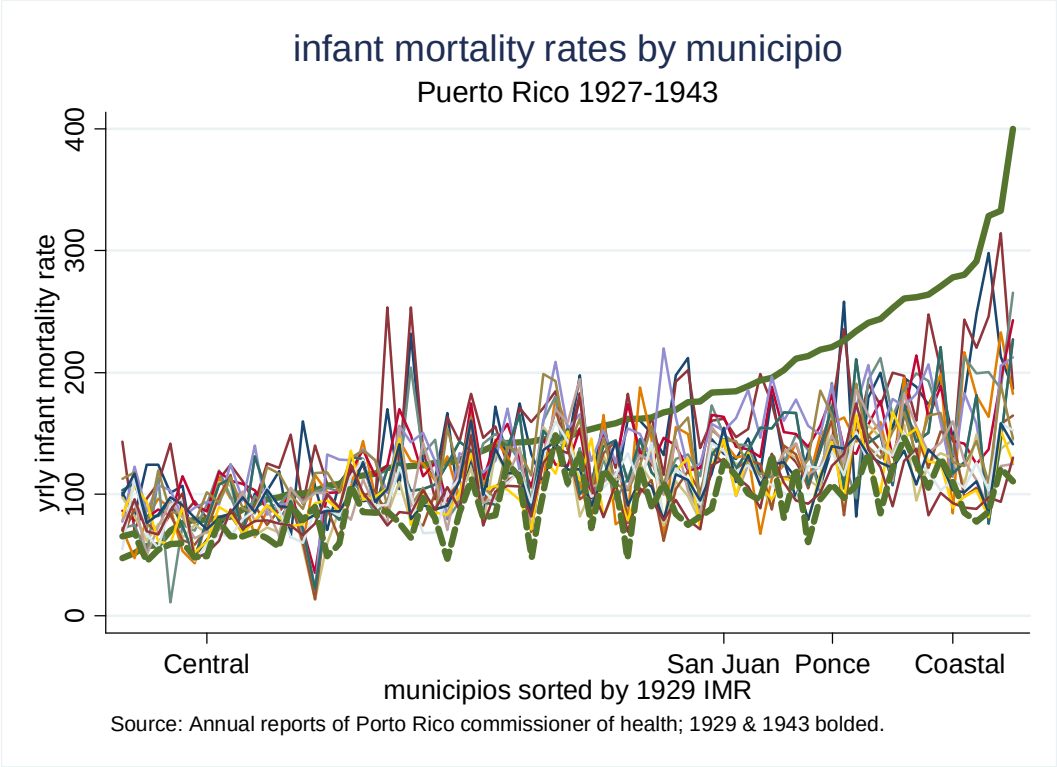
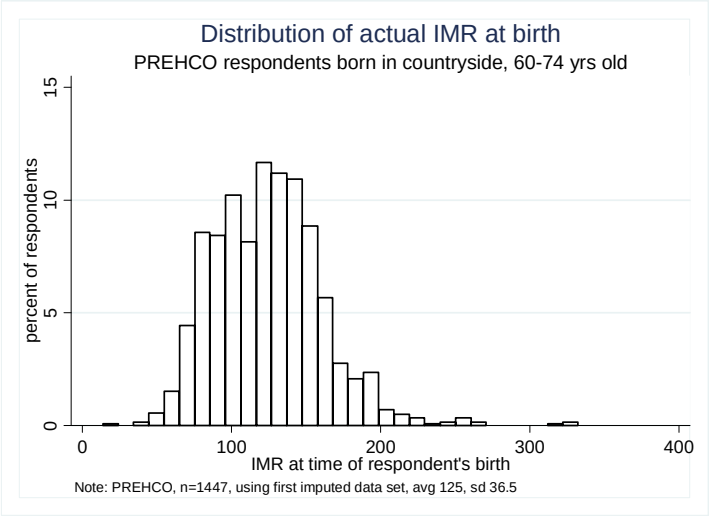

infant mortality rates by municipio
Puerto Rico 1927-1943
yrly infant mortality rate
0
100
200
300
400
Central
San Juan
Ponce
Coastal
municipios sorted by 1929 IMR
Source: Annual reports of Porto Rico commissioner of health; 1929 & 1943 bolded.

Figure 4:


Distribution of actual IMR at birth
PREHCO respondents born in countryside, 60-74 yrs old
percent of respondents
0
5
10
15
0
100
200
300
400
IMR at time of respondent's birth
Note: PREHCO, n=1447, using first imputed data set, avg 125, sd 36.5

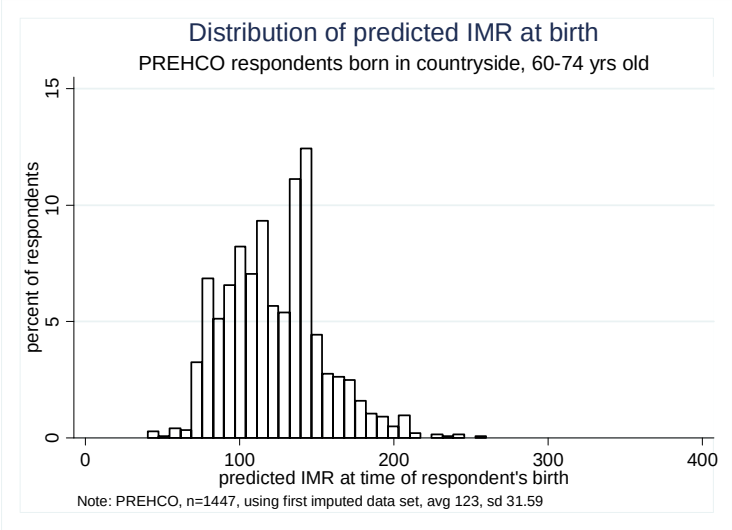
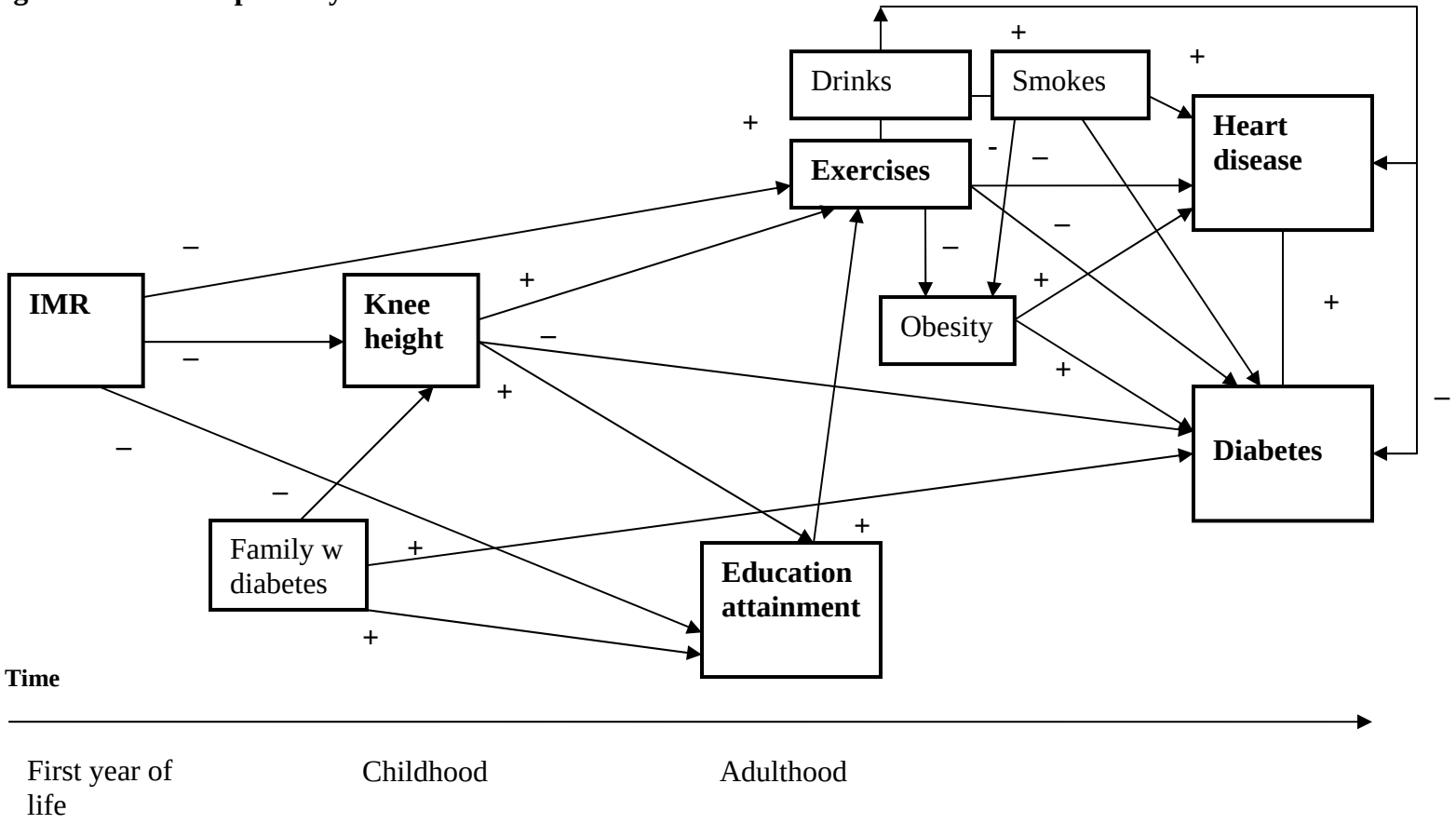

Distribution of predicted IMR at birth
PREHCO respondents born in countryside, 60-74 yrs old
percent of respondents
0
5
10
15
0
100
200
300
400
predicted IMR at time of respondent's birth
Note: PREHCO, n=1447, using first imputed data set, avg 123, sd 31.59

Figure 5: Different pathways to adult disease: Observed associations between IMR childhood and adulthood characteristics



Note: Poor childhood health and poor childhood SES were not associated with IMR but were negatively associated with educational attainment. Season of birth (exposure period) was associated only with heart disease and diabetes. Age not included in above diagram although it was positively associated with IMR (Source: PREHCO, first wave, imputed and weighted 60-74 years old living in countryside as child (n=1447).)

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