"From Angela's Ashes to the Celtic Tiger:
Early Life Conditions and Adult Health in Ireland"

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Abstract

We use data from the Irish census and exploit regional and temporal variation in infant mortality rates over the 20th century to examine effects of early life conditions on later life health. The urban mortality penalty collapsed in Ireland in the years right after World War 2. Our main identification is public health interventions centered on improved sanitation and food safety which we believed played a leading role in eliminating the Irish urban infant mortality penalty. Our estimates suggest that a unit decrease in mortality rates at time of birth reduces the probability of being disabled as an adult by about thirteen to seventeen percent.

JEL Classification: I19, N34

There has been remarkable growth in life expectancy across the developed world in the past century. Advances in longevity in the twentieth century were of the order of three years per decade, with little sign of this trend falling off. The West also experienced declining morbidity, particularly among the elderly, for whom rates of disability were reduced by 50% between 1984 and 2000 (Fogel, 2005). The age of onset of chronic disease is also occurring at later ages. Interest in the origins of these gains have led economists to focus on isolating causal mechanisms involved (Smith, 1999). This paper examines one possible contributory factor; the hypothesis that improving early life conditions equipped those who benefited with more robust health as adults, enabling part of this increased longevity. The key point is that quality of initial inputs into an individual's health production function may influence risk of disease in later life.

In Ireland, there were sharp declines in infant mortality rates beginning in the mid-1940s, with most of the gains occurring in urban areas over the next 20 years. This lead to a convergence of urban and rural infant mortality rates, essentially eliminating the urban mortality penalty. We argue that reasons for the sharp fall in infant mortality during this period were due in part to legislative changes that improved sanitation and water, including food safety, and rubbish disposal.[1] With appropriate time lags, these changes improved contemporaneous health of infants, and lead to a significant improvement in the health of affected cohorts at older ages.

Our analysis uses Irish micro-census data on the current health of affected cohorts. These data are linked to a unique data base characterizing Irish mortality conditions by county across the 20th century, enabling us to control for county level fixed effects and time trends. Strong links are found between these early life declines in infant mortality and latter life health outcomes. We also find evidence that those who benefited the most from the public health investments in the 1940s were from the lower end of the socio economic distribution.

[1] Details are discussed in section 3.2.

The rest of this paper is structured as follows. Section I provides background material on mortality trends in Ireland and reviews the relevant literature. Section II outlines regional variation in Irish infant mortality rates, and discusses interventions, including the 1947 Health Act, which aimed to eliminate the urban infant mortality penalty. Section III describes the data, and section IV contains our principal results. Section V highlights our main conclusions.

I. Background

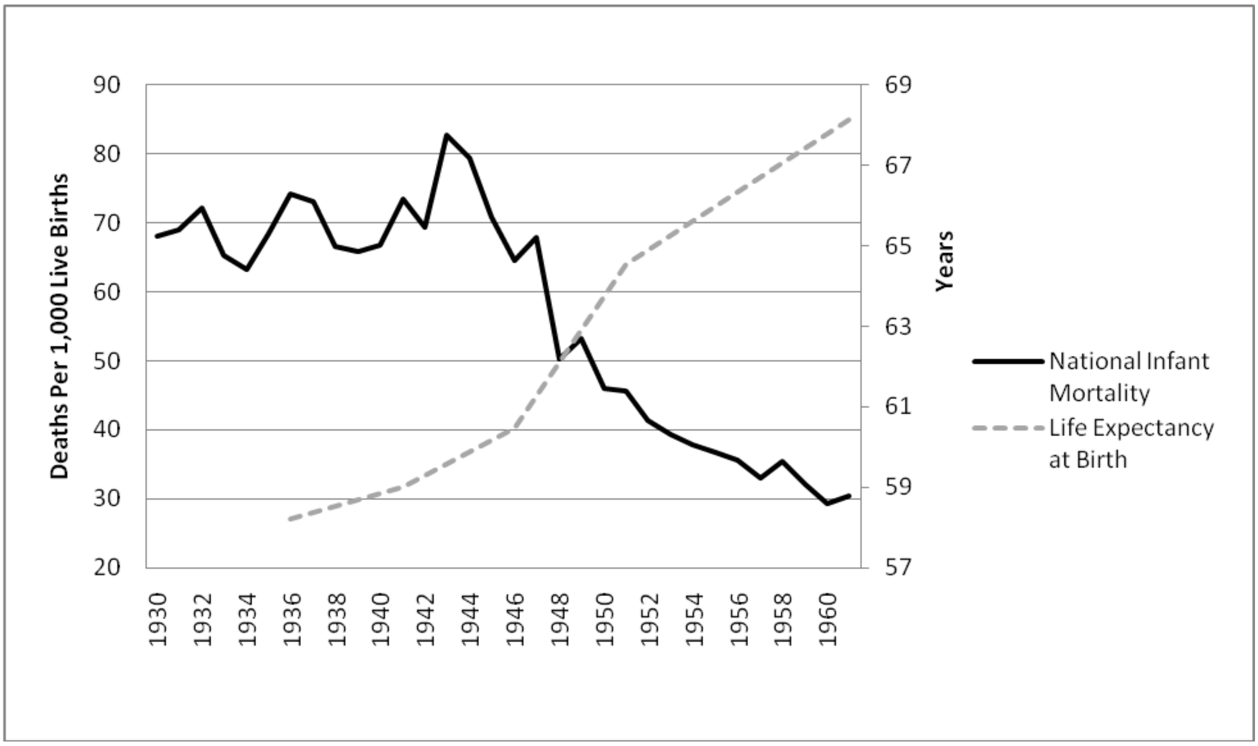
A. Trends in Mortality

Until relatively recently, Ireland lagged far behind the rest of Europe and the developed world in terms of reductions in mortality. Life expectancy at birth was 57.4 years in 1926, but rose to 79.9 by 2006 (Figure 1). On average, male life spans increased by a quarter of a year per annum since the start of the last decade, and .3 of a year per decade for women. It is projected that Irish children born today may live well into their 90s (Whelan, 2008). Mortality rates dropped significantly between 1940 and 1960, with particularly dramatic improvements in infant mortality (Figure 1). The rate of progress in Irish life expectancy slowed from 1960-1980, and then picked up from the mid 1980s on. While declining child mortality drove life expectancy gains up to the 1980s, improvements at later ages have driven recent improvements.[2]

This is the same pattern observed in other countries. According to Cutler and Meara (2003), over half of gains in life expectancy in the first half of 20th century in the US were concentrated before age 14. In the second half, two thirds of gains were felt by over 45s, mainly due to reduced deaths from heart conditions. This pattern is being repeated half a century later in developing countries. Fogel (2005) and Cutler and Miller (2005) argued that there are clear stages in the battle against mortality. The first era in the 18th century involved Fogel's famous

[2] Life expectancy at age 65 stagnated until the mid 1980s; there followed an increase from 16 to over 19 by 2006.

Figure 1- Irish Infant Mortality and Life Expectancy 1930-1961



Source: Irish Life Tables and Central Statistics Office. Note: The figures for life expectancy are from the years 1936, 1941, 1946, 1951 and 1961, and refer to expected years of life remaining at birth.

escape from hunger, the second in the 19th and early 20th century was mainly waged against infectious disease, and the modern period is characterized by falling morbidity and mortality rates among the elderly, with the main enemy being chronic conditions such as heart disease.

The cohorts in the west living longer today enjoyed improvements in early life conditions in the first part of the last century. This, and the fact that reductions in deaths from cardiovascular illness for 70% of declines in mortality since 1960 are leading economists to focus on the importance of initial health endowments.[3] Similarly, Ireland's poor performance in life expectancy (compared to other developed nations) over the recent past mirrors its lag in mortality rates at the beginning of the 20th century. The urban-rural mortality penalty is an important facet of this. What sets Ireland apart is the delay in entering the final phase; the urban

[3] Black et al. (2007) show that low birth weight has an independent negative effect on various outcomes in later life. The medical literature outlined a potential causal pathway for linking early life conditions to cardiovascular illness in later life via the Fetal Origins Hypothesis. Section 2.2 discusses previous studies in more depth.

penalty was not eliminated until the 1960s. In this paper we exploit this delayed transition to identify the effect of early life conditions on adult health.

In Ireland, we argued that a series of interventions succeeded (albeit belatedly) in eliminating the urban mortality penalty following the 1947 Health Act. This is the variation in early life conditions we examine in this paper. We use current Irish census data to tie individuals back to the historical conditions into which they were born. This enables us to exploit both regional and temporal variation in these conditions, which we proxy with county level infant mortality data. The elimination of the urban mortality penalty provides the main source of identification when we include trends in the model. Our estimates suggest that a unit decrease in infant mortality rates reduces the probability of being disabled as an adult by between about thirteen to seventeen percent.

Early life conditions may also be important for explaining variation across social class. Although several studies have shown separately that Irish people from lower socio economic groups are more at risk of poor health as infants, and more likely to suffer from chronic conditions later in life, few papers have linked these two findings. In Ireland in 1999 the prenatal mortality rate was three times higher for children of unskilled manual workers than for those born in the higher professional category (Cullen, 2002). In this paper we find that individuals from lower SES groups are more at risk from poor early life conditions, with marginal effects much larger for those at the bottom of the education distribution compared to the top.

B. Literature

This paper is part of an emerging literature which examines the role of early life conditions in determining adult outcomes. With stocks such as human capital and health, there can be significant path dependence, and initial endowments and shocks in early periods can have significant long term consequences. Given that health is a function of past inputs, simply considering current behaviour and circumstance will not enable us to accurately characterise distribution of health, nor to correctly identify various causal factors for different health states.

The literature on the role of early life determinants of current health began with ecological studies which examined correlations between infant mortality rates and subsequent death rates across regions. Two studies by Forsdahl (1977, 1978) showed correlations between infant mortality rates in Norwegian districts and subsequent arteriosclerotic heart disease. Cohorts facing high infant death rates were also cohorts with high rates of heart disease in later life. While Forsdahl focused on stress during early childhood and adolescence, Barker (1997, 2001) argued that such chronic conditions have their origins even earlier in life.[4] [5]

An ecological study based on Irish data (Pringle, 1998) examined correlations between infant mortality rates in Irish counties between 1916 and 1935, and deaths from heart disease between 1981 and 1990. Support for the early life conditions hypothesis was ambiguous. However, selective internal migration is a serious confounding factor here; according to the 2006 census around 65% of current county residents were born the same county. Our data allow us to take account of this problem since we know both county of birth and county of residence.

In addition to the evidence on infant health, childhood health may have important lasting consequences. Case, Lubotsky and Paxson (2002) demonstrate that poor health as a child is a significant predictor of ill health as an adult. Black, Devereux and Salvanes (2007) use a Norwegian administrative dataset on twins, which implicitly controls for genetic and socioeconomic endowments and eliminates the influence of length of gestation. The authors find

[4] The Fetal Origins Hypothesis provides a pathway for linking early life conditions to adult outcomes. There are periods of sensitive development for both children and infants, and any interruption to adequate nutritional intake at these particular stages would result in long term damage to the system or organ concerned. This is principally due to the foetus diverting scarce resources to the brain. The main adaptive behaviours involved include the slowing of the rate of cell division, particularly in organs in critical stages of development, changes in the distribution of cell types, hormonal feedback, metabolic activity and organ structure (Barker, 1997).

[5] Barker (2001) discusses 80 published studies exhibiting a correlation between low birth weight and raised blood pressure as adults. A recent meta analysis of studies involving over 150,000 individuals found evidence of a significant correlation between low birth weight and future risk of diabetes (Peter Whincup, Samantha Kaye, Christopher Owen, Rachel Huxley et al., 2008).

that 10% increase in birth weight implies a .57 cm increase in height, a 1% increase in income and a 1% increase in the probability of high school completion. Another approach is to use data from natural experiments. Almond (2006) uses the 1918 influenza outbreak in the U.S. to provide exogenous variation in early life conditions. Data from the US Census (1960, 1970, 1980), suggest that cohort in utero during the influenza pandemic received 1.5 less months of schooling, and were up to 5% less likely to graduate from high school than expected.

Finch and Crimmins (2004) argue that there is a strong association between early life experiences of a cohort and its subsequent death rates. They present results from an analysis of mortality rates for Swedish birth cohorts since 1751 across the entire life course, and note that mortality declines among both the young and elderly generally begin in the same cohort. Individuals experiencing improving early life conditions were also individuals who experienced declining rates of mortality at later ages. The authors argue that reduced exposure to infectious disease (particularly tuberculosis and gastroenteritis) and malnutrition in childhood reduced levels of chronic inflammation (known to be linked to vascular disease and other diseases associated with the aging process). These are precisely the disease areas which exhibited dramatic improvements following the 1947 Health Act in Ireland. These cohort effects, which are a function of the shared environment affecting these individuals in early life, lasted throughout the lifespan. Public health and medical interventions, such as the 1947 Health Act can have additional effects through enduring effects of early life conditions, a hypothesis we test in this paper.

This paper contributes to the literature by examining evidence for Ireland regarding the elimination of the urban mortality penalty. This study has a number of benefits relative to those mentioned above. First, we proxy early life conditions directly with infant mortality rates with clear variation across regions, particularly for urban versus rural counties. Second, we are not reliant on extreme events such as famine, reducing the danger that results are reflecting a highly localized effect. The next section outlines regional variation in Irish infant health in more detail.

II. The Irish Urban Infant Mortality Penalty

Following Ireland's independence in 1922, the new government faced the difficult task of organizing and financing a new state. Quite apart from economic development, Ireland lagged behind in terms of other indicators, such as industrialisation and urbanisation. Ireland lingered in the second phase of Fogel and Omran's epidemiological transition, and infectious disease and poor public health infrastructure remained to be tackled up to the 1940s. According to Garvin (2004), Irish towns had some of the worst slums in Europe at the time. The urban infant mortality penalty and its eventual elimination is one manifestation of this.

Substantial regional variation in mortality rates also existed prior to the 1940s. The urban penalty in Irish infant mortality rates remained high at around 50% throughout this period (Figure 2). It was not until the mid 1950s that this penalty was eliminated when urban and rural rates converged. Haines (2001) documents the experience in the US.[6] For states with reliable data, the penalty for infant mortality was also around 50% in 1900. By the 1930s full convergence had been achieved, and in fact cities were marginally better for infants. Excess mortality of cities was explained by greater density and crowding, leading to more rapid spread of infection; lack of adequate clean fresh water and sewerage disposal, a consequently higher degree of contaminated water and food; and garbage and carrion in streets. (Haines, 2001). [7]

Another important factor in improving public health relates to the overcrowding that existed in Ireland's major cities. From the early 1930s, increased public awareness called for

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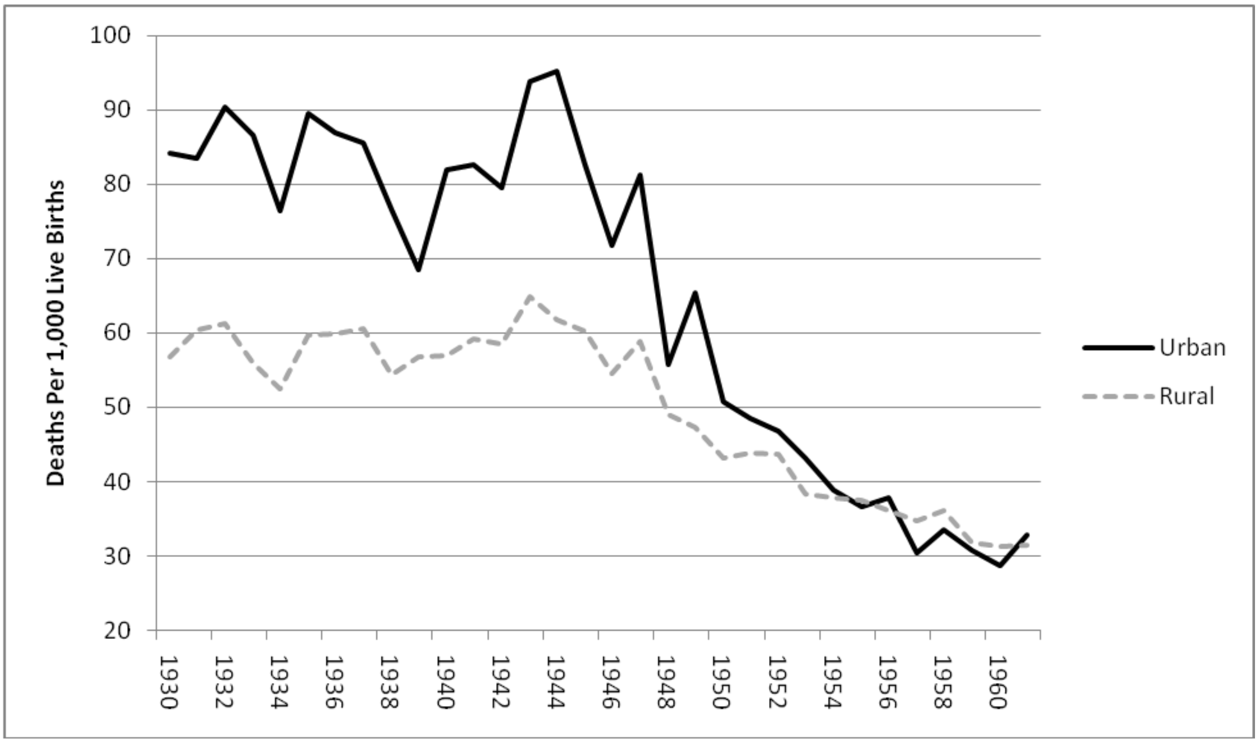
[6] See, for example, Williams and Galley (1995) for a discussion of the experience in England.

[7] Costa and Kahn (2003) argue that state expenditures on public health lowered mortality rates from typhoid, dysentery, and diphtheria between 1910 and 1940 and that city public health expenditures circa 1910, particularly on sewage and water filtration, were effective in reducing childhood and infant mortality.

slum clearance, and between 1932 and 1939 there was great investment in housing by the government. A 1944 Report of the Dublin Housing Inquiry highlighted issues of persistent tenements in Dublin County Borough, and it became policy to give tax relief on developments which involved slum clearance. Finally, experimentation with privatization of street cleaning system in Dublin and then Cork (1929) proved effective in reducing local refuse.

Figure 2- Urban and Rural Infant Mortality 1930-1961



Source: Irish Infant Mortality Database. Note: the urban areas include Dublin, Cork, Limerick, Waterford and Galway county boroughs, with the rural areas comprising the other county areas.

A. The 1947 Health Act

Following World War 2, there was renewed pressure on the Irish government to tackle public health problems, particularly evident in urban areas. In fact there was not even a dedicated department of health until 1947. The 1947 Act signified a landmark in Irish policy representing a firm commitment to improve the health of the population. The act envisaged a substantial investment in public health infrastructure, estimated to cost up to £30 million over 10 years.[8] There was new legislation in a range of areas, from water supply to sewage disposal, devolution of power to local authorities, a focus on tackling infectious diseases, and a proposed provision of free medical care to mothers, children, and the poor (Whyte, 1980).

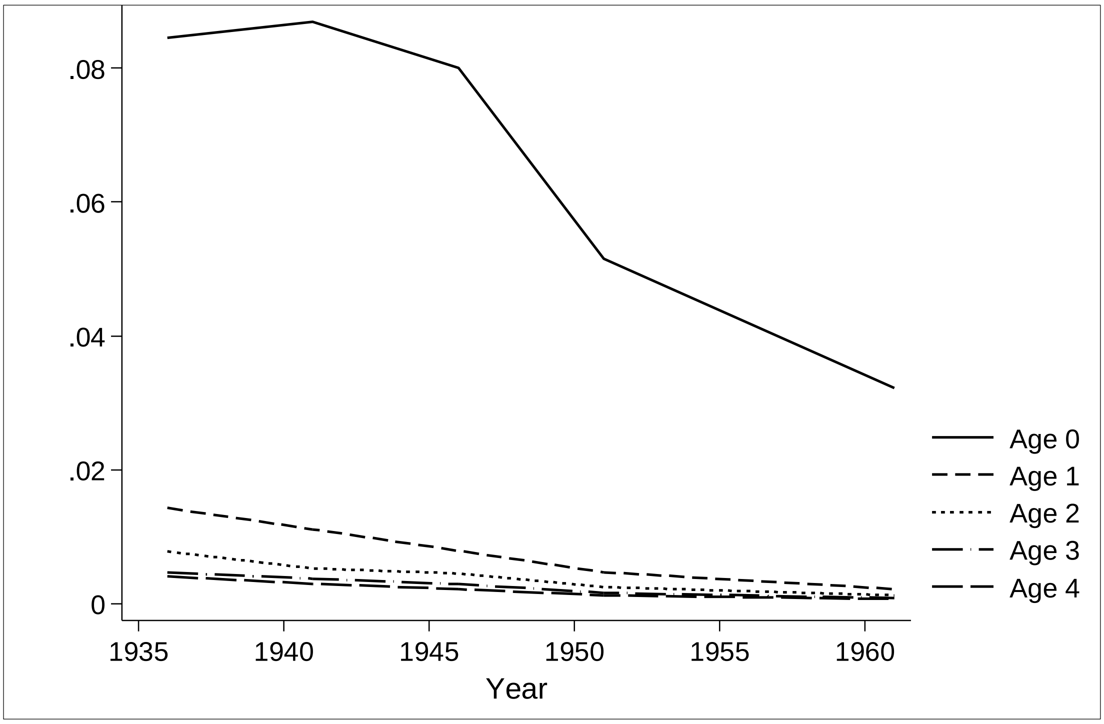
The transfer of responsibility for health to county councils involved substantial increases in funding from central government, and local authorities were given the power and obligation to monitor the well being of those in public institutions such as schools. Ministers were given sweeping powers to combat infectious disease, including the ability to impose compulsory detention for those afflicted.[9] The final set of measures related to the Mother and Child Scheme, a series of initiatives designed to provide free information, education and medical care to young mothers and babies. According to Garvin (2004), by the mid 1950s Ireland had one of the most modern health services in the world. What is clear is that a combination of these policies succeeded in eliminating the Irish urban infant mortality penalty by 1960.

The data in Figure 3, which shows the age-distribution of child mortality declines, demonstrates that by far the main mortality improvement is focused on infants, i.e., those who were less than one years of age. Although there were some improvements at ages 1 and 2, these were from a relatively low base compared to rates of over 8% for infant mortality. Mortality rates among the elderly were very stable during this time.

Figure 4 displays the main declines in infant mortality by cause. In particular, the decline of both diarrhoea and deaths due to congenital abnormalities at birth are striking. Both diseases are linked to sanitation and nutrition, and both decline very rapidly after 1947 having increased during the war. The available county level data also supports the hypothesis that sanitation and overcrowded living conditions were driving the urban mortality penalty. The censuses of 1946

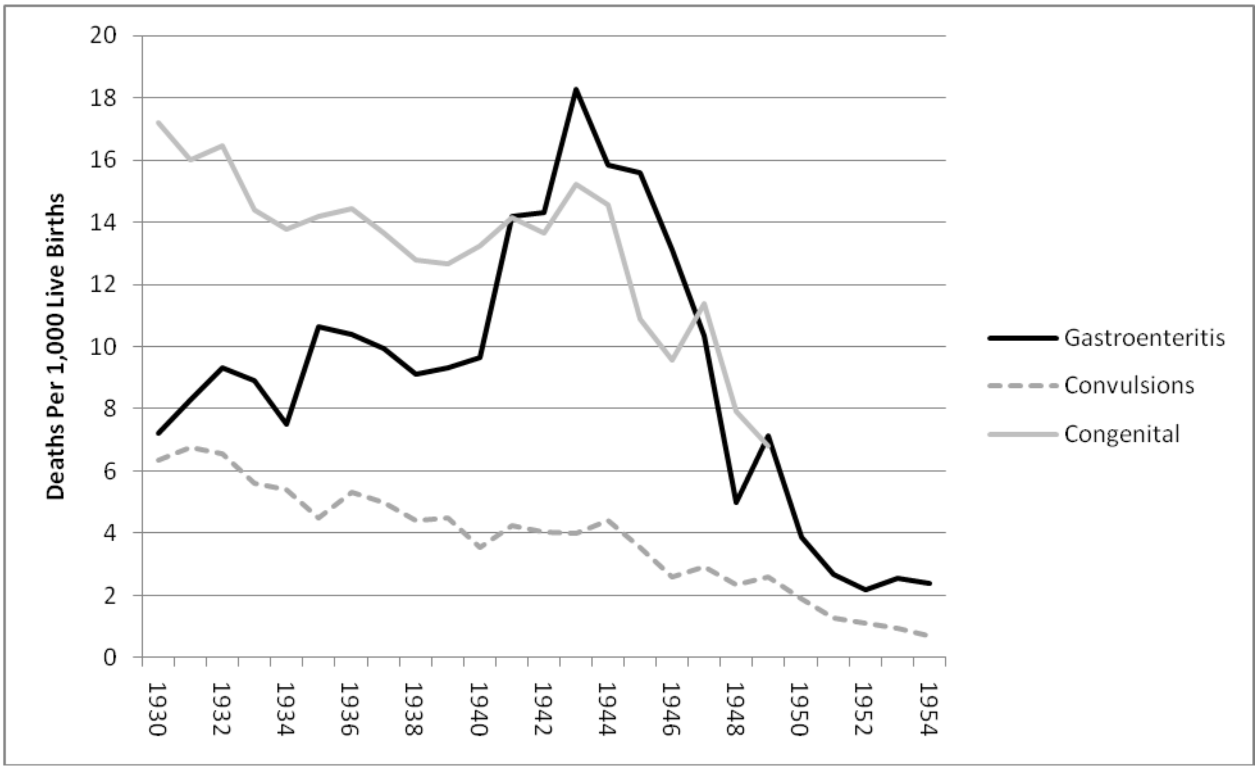
[8] The Irish Times, October 3rd 1947.
[9] These measures were particularly aimed at combating problems associated with tuberculosis.

Figure 3- Early Age Mortality Rates 1936-1961



Source: Irish Life Tables. Note: figures are for the years 1936, 1941, 1946, 1951 and 1961.

Figure 4- Infant Mortality by Cause 1930-1954



Source: Central Statistics Office. Data exists only for the years 1930-1954. Note: due to changes in the coding for the congenital series, this can only be shown up to 1950.

and 1961 allow us to examine county level correlates of infant mortality.[10] Table 1 presents a regression analysis of the determinants of changes in county level infant mortality between the 1946 and 1961 Irish Censuses. This model includes the following public health, demographic, and economic variables plausibly related to changes in county level infant mortality that are measured in both Censuses- the proportion of households with shared sanitation, the male unemployment rate, the proportion of people who were born in the county of their current residence, and the average number of people per room in the dwelling. The two variables that have statistically significant effects are the higher proportion of families living with shared sanitation facilities, and a higher proportion of migrants living in that county and both are associated with higher levels of infant mortality.[11] Importantly, the fact that male unemployment has no independent effect confirms the hypothesis that differences in income were not driving the observed cross county differentials.

Table 1: County Level Determinants of Infant Mortality 1946 and 1961

Variables	First Difference Model Change In County Infant Mortality
Proportion With Shared Sanitation	2.823***
	(0.661)
Male Unemployment Rate	-1.250
	(0.867)
Proportion Born In County Of Residence	0.005***
	(0.001)
Average Number of Persons Per Room	-65.304
	(96.916))
Constant	71.966
	(84.169)
Observations	60
R-squared	0.895
Number of county	30

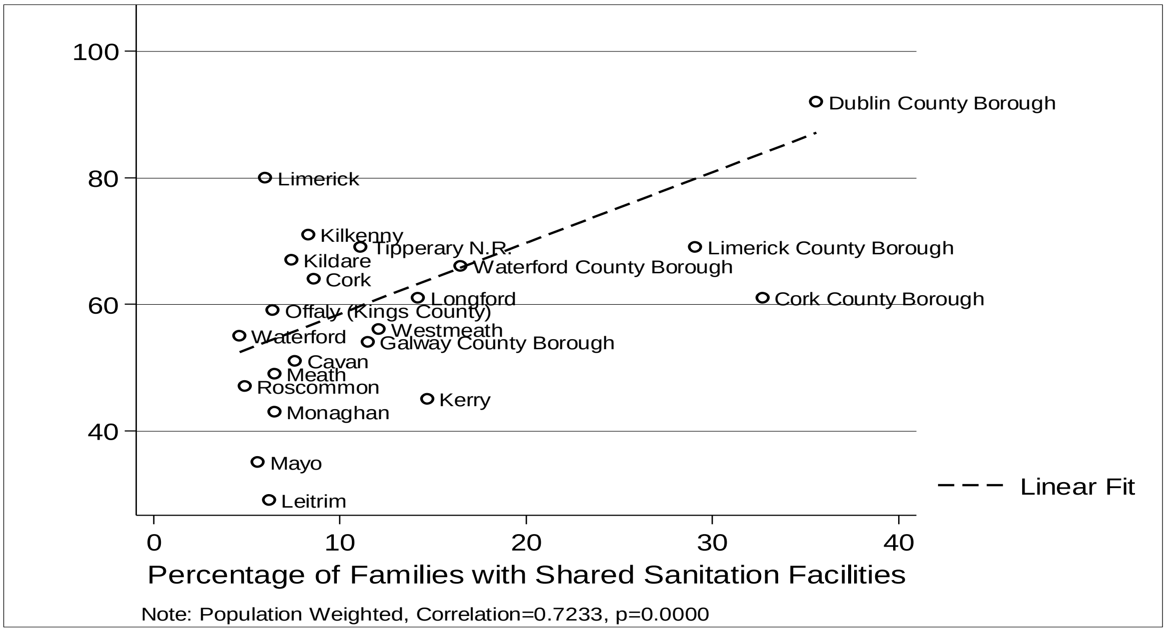
Standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.
Note: population weighted. Source: Irish infant mortality database and county level data from Irish census 1946 and 1961.

[10] Relevant county level data is only available for these two years.
[11] Other factors, in particular the proportion of families living in overcrowded accommodation, are also correlated with infant mortality, however these are collinear with other variables.

If we use the estimates contained in Table 1, the fall in the fraction with shared sanitation between 1946 and 1961 would explain 45% of the decline in national level infant mortality over that period. Since the drop in shared sanitation was much higher in the urban counties, the decline in shared sanitation 'explains' more than 80% of the fall in the infant mortality rate in the urban counties and hence most of the decline in the urban-rural mortality penalty.

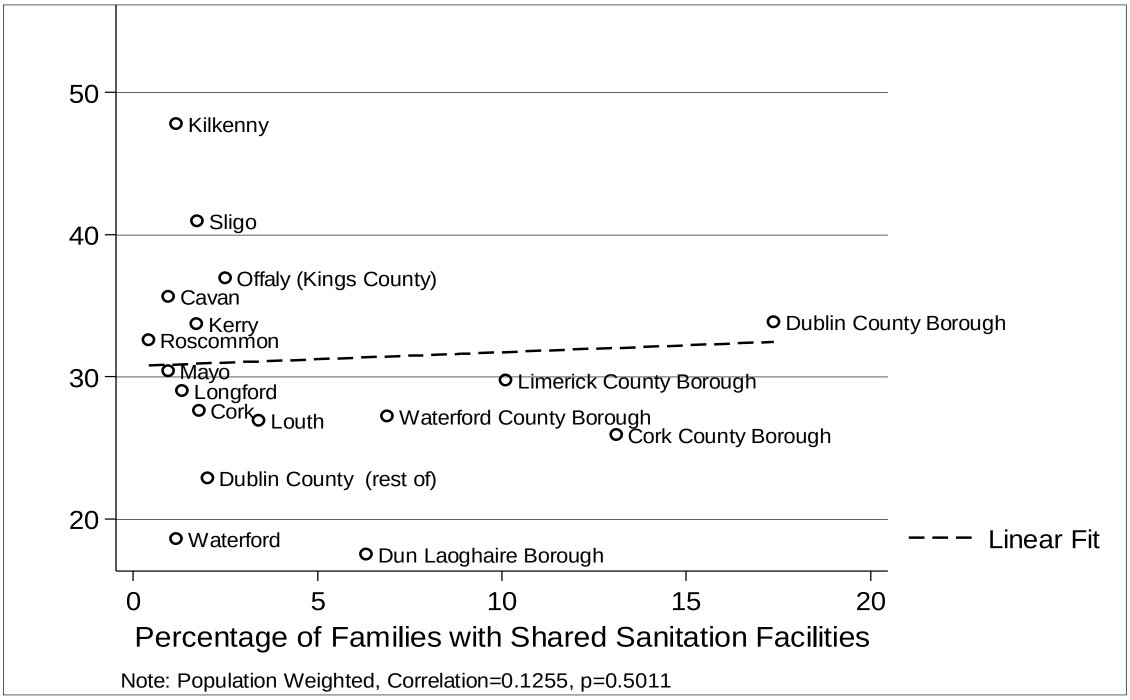
Figure 5 shows the relationship between county level infant mortality in 1946 and the percent of families with shared sanitation facilities. This figure shows that the strongly positive shared sanitation—infant mortality relationship was mainly driven by the county boroughs (i.e., urban council areas), particularly Dublin city, where 40% of households shared sanitation facilities. By the 1961 census, a similarly estimated model indicates that the relation basically disappeared (Figure 6). The numbers living in these conditions halved in Dublin by this time.

Figure 5 Infant Mortality and Percentage of Families with Shared Sanitation Facilities



Source: Irish Census 1946 and Irish Infant Mortality Database.

Figure 6 Infant Mortality and Percentage of Families with Shared Sanitation Facilities



Source: Irish Census 1961 and Irish Infant Mortality Database.

Our claim that the 1947 Health Act was mostly responsible for the elimination of the urban mortality penalty still has to deal with the plausibility of alternative explanations. Our claim is based both not only on the precise coincident timing of the decline in urban infant mortality and the fact that the components of the 1947 health act were mostly implemented initially in cities and in particular bur not exclusively Dublin. We also based our conclusion on the fact that sanitation improvement was the main focus of the 1947 Health Act. Our results in Table 1 show that falling levels of shared sanitation was most correlated with the changes in county level infant mortality. In addition, the elimination of the association of infant mortality with the percent of families using shared sanitation and infant mortality between the 1946 and 1961 Irish Censuses (Figures 5 and 6).

This consistency of time, geography, and cause of death does not by itself dismiss other possible factors. Three plausible alternative possibilities are the 1932-1938 trade war with

Britain, the Second World War (WW2), and the introduction of penicillin. While the trade war with Britain certainly influenced the Irish economy during this period, the Irish infant mortality rate is actually static throughout the 1920s and 1930s when the trade war was in effect with little convergence of the urban rate to the rural infant mortality rate. Thus, the trade war does not seem likely to be a major explanation of our results. Added to this, we have verified that our results are robust to simply including the period from 1940 to 1955 after the trade war was initiated.

As our mortality figure by cause showed in Figure 4, WW2 was associated with an increase in gastroenteritis related deaths during the war period but it is only after 1947 that infant deaths due to gastroenteritis clearly begin to fall below earlier trend levels. Very high and relatively stagnant rates of infant mortality in Ireland due to gastroenteritis preceded the war. The health act was initiated partly in response to this increased incidence of gastroenteritis during the war so the initiatives contained in the 1947 Health act may well be an indirect effect of the high levels of gastroenteritis deaths during the Second World War.

The most complex issue regards penicillin. Penicillin was first introduced into Ireland in 1945 so the timing is certainly coincident with the fall in urban mortality. However, after a thorough search by us there is little in the existing information in Irish medical journals or the yearly public health reports at the time citing any important role of penicillin for reducing infant mortality. The discovery and perhaps roll out into the population of penicillin has a common date in England, the United States, and Ireland. However, the rapid decline in infant mortality and the elimination of the infant mortality penalty in the United States and England clearly pre-dated the discovery of penicillin and has been largely attributed to improved hygienic measures such as improved sewage, water purification, removal of refuge, improved milk supplies (McKeown et al., 1975, Williams and Galley, 1995, and Cutler and Meara, 2003).

In particular, Cutler and Meara (2003) show that eighty-three percent of the decline in infant mortality in the US predated the discovery of penicillin and the elimination of the urban mortality differential predated it as well. It seems more consistent with the international evidence that the causes of the elimination of the urban infant mortality penalty be in common. The latter timing of the elimination of the urban mortality penalty in Irish case is explained by the fact that sanitation and other public health improvements occurred later in that country. But it remains true that the type of data needed to more directly test the contributory role of penicillin in the Irish case (the timing, extent, and geography of the penicillin roll out) is simply not available at present. Until we have that evidence, we should remain cautious in our summary conclusions.

IV. Data

A. Census data on Disability and Health

This research on relating infant mortality declines to latter life health relies on successive waves of the Irish census data in 2002 and 2006.[12] There are several advantages to this data. This is a representative micro sample of the entire Irish population, which allows us to examine individuals from all counties in the Republic of Ireland in all age groups. The large sample size of about 200,000 in each Census is important given our reliance on the geographic distribution of the population. Finally, the census is almost unique among Irish surveys in that it asks respondents for their place of birth so we can link to earlier data on infant mortality by county.

In this paper we examine the determinants of suffering from a disability as this is the only health related variable available in the census micro files. A legitimate question is how well disability correlates with other measures of health status. Using the main Irish Labor Force survey where both measures are present, the correlation as Table 2 demonstrates is quite high.

[12] Irish Census data is freely available from the Irish Social Science Data Archive (www.ucd.ie/issda).

Among those with a disability, only fourteen percent say that their health is very good compared to 53% claiming very good health among those with no disability. Similarly, half of the disabled state their health is fair or bad compared to 6% for the non-disabled.

Table 2 Self Reported Health Status by Disability Status

Disability Status	Self Reported Health (% in Each Category)			
	Very Good	Good	Fair	Bad/Very Bad
No Disability	14	36	39	11
Disability	53	41	6	0

Source: Quarterly National Household Survey 2007. N=21,252. Note: Figures refer to self reported health status (the question was "how is your health in general?") by self reported disability status. Disability refers to self reporting of one of the following conditions: Blindness, deafness or a severe vision or hearing impairment, a condition that substantially limits one or more basic physical activities, a learning or intellectual disability, a psychological or emotional condition, other, including any chronic illness. Further details are available from the Central Statistics Office website: http://www.cso.ie/releasespublications/documents/labour_market/current/healthstatus.pdf

The question on employment status also identifies those who are out of work due to illness, but there are small numbers in this category. We combine these measures into a single disability variable.[13] Table 3, which shows the types of disability broken down by their causes, demonstrates that random events such as accidents are typically not major contributors to overall disability rates, accounting for only around 5% for most disabilities. Such random events would be difficult to trace to childhood illnesses. On the other hand, illness is the most important cause of disability for all types of disabilities.

Table 3 Cause of Disability by Type

	Type Of Disability								
Cause	Seeing	Hearing	Speech	Mobility	Memory	Intellectual	Emotional	Pain	Breathing
Hereditary	17.6	15.3	19.5	10.0	11.0	24.7	10.4	8.5	13.0
Accident	6.2	4.8	4.2	16.1	5.0	3.4	6.6	18.3	1.7
Illness	38.7	18.7	35.6	45.5	32.7	33.1	39.6	48.9	59.1
Work	0.9	12.1	0.5	3.3	0.6	0.0	1.8	4.6	2.3

[13] The disability variable is generated from a number of questions in the census, and includes almost all dimensions. A detailed breakdown is unavailable in the census micro file we use in this paper, only the aggregated disability variable. A concern is that disability caused by events such as accidents which are obviously unrelated to early life conditions are included. However, as long as these factors do not vary systematically with the other covariates in the analysis, this is measurement error and will bias downwards the estimates of the effect of infant mortality. This does not appear to be an issue, for example other data show that urban areas are not over-represented on disabilities such as accidents (National Disability Survey, 2008).

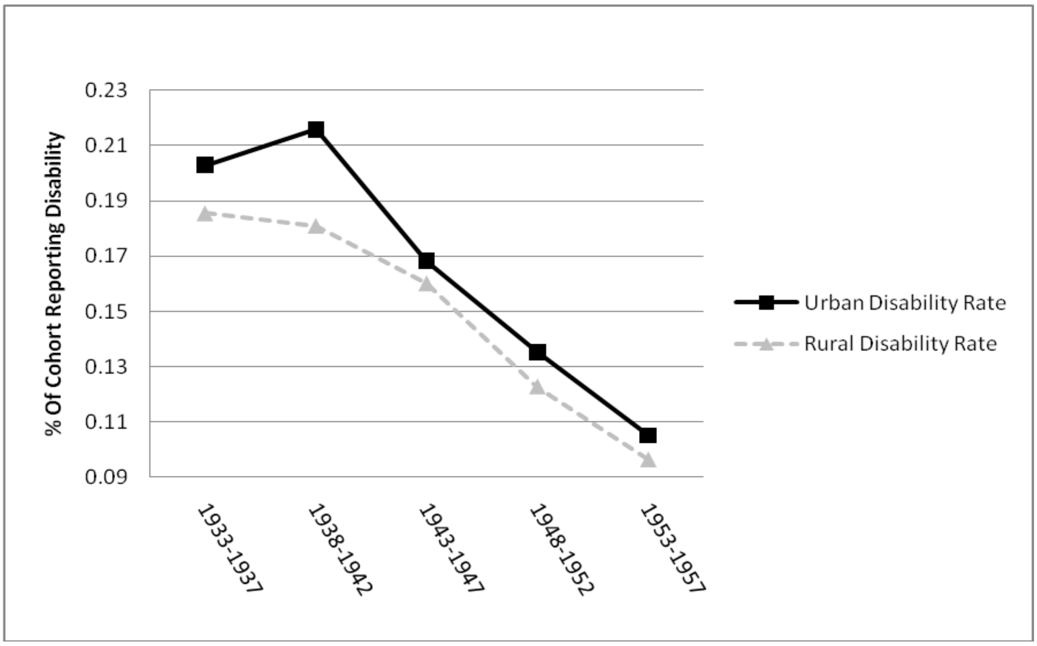
Stress	1.3	1.3	1.5	1.8	8.7	1.3	17.5	2.3	1.6
Other	15.1	21.7	18.0	13.4	18.0	11.9	12.5	9.2	10.4
No Specific Cause	9.8	12.3	8.2	4.6	10.1	8.7	4.5	3.4	3.3
Don't Know	10.7	14.1	12.8	5.9	14.4	17.5	8.3	5.5	6.4
Total	100	100	100	100	100	100	100	100	100

Source: National Disability Survey 2006, N=14,518. Note: table shows the causes of each type of disability (as a percentage of that particular disability). For example, of people who have a sight disability, 17.6% report the cause as hereditary. Disability status was classified according to the level of difficulty experienced by respondents in each domain. Further details are available from the Central Statistics Office website: http://www.cso.ie/releasespublications/nationaldisabilitysurvey06first.htm

Given the pattern in infant mortality rates across counties, we make two predictions. An individual born in the 1930s or 1940s in an urban area should entail a higher risk of adult disability, as they faced a higher risk of being in poor health as infants. However, by the 1950s birth cohorts, there should be no difference between those born into urban or rural areas. Figure 7 plots disability rate for people born in urban areas (Dublin, Cork, Limerick, Waterford and Galway county boroughs) and rural areas for 5 year birth cohorts.[14] Current adult disability rates for those born in Dublin and rural areas follow the same pattern as infant mortality—high in Dublin followed by convergence in the 1950s. We investigate the importance of early life conditions by statistical analysis discussed in Section 5.

[14] Due to the process of anonymization employed by the Irish Central Statistics Office, we only know a person's 5 year age group, as opposed to their actual year of birth. Since we only know whether a person is over 75, the analysis is restricted to those under 75. We also limit the sample to those over 25 as many under this age may be still in school.

Figure 7 Disability Rates by Urban and Rural Birth Cohorts



Source: Irish Census 2002. Note: the urban areas include Dublin, Cork, Limerick, Waterford and Galway county boroughs, with the rural areas comprising the other county areas.

B. Infant Mortality Data

For our analysis, we use information gathered on annual infant mortality by county of birth.[15] Due to restrictions on knowing a person's exact age mentioned above, we average infant mortality in a county by 5 year age bands recorded in the census and then merge this information with the census files, allowing us to proxy for early life conditions and initial health endowments. While the census only contains county level information, infant mortality database contains separate information on urban areas. Therefore, where the census lists a birth county dominated by an urban area (e.g., Dublin or Cork), the urban rate was used. Since county of birth is only listed for those born in the Republic of Ireland, we restrict our sample to those born in those 26 counties. These restrictions leave us with a sample size of around 200,000 individuals.

IV. Results

[15] There were numerous sources for this data, including the Central Statistics Office, Registrar General Reports, and the Department of Health. For a significant proportion of the information, building up this database involved obtaining the data in hard copy and inputting it into electronic format.

A. Model Specification

We examine the effect of early life conditions on adult health by regressing whether a person reported a disability on age, age squared, and infant mortality rates in that person's age group in their county of birth.[16] We did not control for adult education since reductions in infant mortality could have led to an increase in education as an adult (Case et al., 2005). However, our estimated effects of infant mortality on adult disability were not very sensitive to the inclusions of education as a variable.

[16] We did not also control for cohort effects since the well-known identity between cohort, time, and age effects precludes that.

These models also control for county of birth fixed effects, county of current residence fixed effects, county specific trends, and survey year. To check robustness of results, we also extended the model to include lags and leads of infant mortality. As the outcome variable is binary, we estimate this model using a probit. Two issues relating to standard errors arise—serial correlation and heteroskedasticity. In our case, we expect observations to be correlated within counties and age groups, as individuals were most likely exposed to similar environmental factors. We therefore cluster standard errors by county of birth X age group. To deal with the second issue, a model is estimated allowing the variance to depend on age (heteroskedastic probit). In each case, we fail to reject the null hypothesis (that variance depends on age), and conclude that this specification is correct. We present results from a combined dataset of the 2002 and 2006 census, although the analysis of the individual census separately reach similar conclusions.[17] The following equation summarizes the model. γ is the vector of control variables outlined above.

$$\begin{aligned} Disability_{ijt} &= \alpha + \beta_1 Age_{it} + \beta_2 AgeSquared_{it} + \beta_3 Sex_i \\ &+ \beta_4 InfantMortality_{jt_0} \\ &+ \gamma_{ijt} \end{aligned}$$

We first present marginal effects at the mean of the independent variables. However, to examine whether early life conditions have the same effect across socio economic groups marginal effects are also evaluated at different levels of education.

A legitimate concern is that infant mortality is correlated with some other unobserved variable. Our county level fixed effects would capture any confounding influence which does not change over time. Our inclusion of county specific time trends has important consequences for

[17] Some individuals are in the merged 2002 and 2006 Census twice. However, the Irish government only releases a five percent sample of the Census so about a quarter of one percent of the sample is in common. Thus, the effect on standard errors is quite small. We cannot identify these people in the public release tapes but this effect of this degree of clustering must be very minor.

the analysis as identification of the effect of infant mortality is reduced to using sharp non-linear breaks such as the dramatic declines in the urban areas in the 1940s.[18] Our specification has the benefit of reducing the scope for some omitted variable to bias the results. In order for some omitted factor to be driving these results, it would have to vary in exactly the same non-linear pattern as county infant mortality, i.e., differentially affect those born in urban areas up to the 1950s, and not thereafter. To further address this issue, we included lags and leads of infant mortality. This is an additional way of addressing concerns that infant mortality may be correlated with some other unobserved variable that affected cohorts at a later stage.

B. Regression Analysis

Table 4 presents marginal effects from the heteroskedastic probit regression outlined above. The control variables mentioned above are omitted from the Table, and all coefficients are multiplied by 1000 to aid ease of interpretation. The first column refers to the model outlined above. Several variables in this model are statistically significant. Not surprisingly, age is associated with an increased risk at an increasing rate of disability, as is being female. Most central to our hypothesis, a unit decrease in the infant mortality rate (deaths per 1,000 live births) is associated with a .04907 percentage point decrease in the probability of currently suffering from a disability. This result is strongly statistically significant.

Table 4 Marginal Effects for Disability

Variables	Disability	Disability (>40)	Disability (Lags and Leads)
Age	1.056	3.749	0.378
	(1.1108)	(3.1484)	(1.1036)
Age Squared	.0241**	-0.00578	0.0365***
	(0.0114)	(0.0287)	(.0124)
County Infant Mortality (5 Year Average)	0.4907***	0.455***	0.379***
	(0.1375)	(0.1548)	(0.1361)
Female	-0.964	-4.8501	-1.073
	(2.1693)	(3.522)	(2.2053)
County Infant Mortality +5 Years			.0174

[18] In fact omitting county trends has little effect on either the magnitude or significance of these results.

			(.191)
County Infant Mortality +10 Years			-0.326
			(.2201)
County Infant Mortality -5 Years			.131
			(.1589)
County Infant Mortality -10 Years			-.0898
			(.0846)
Wald Heteroskedasticity Test	26.24***	12.44***	19.97***
Observations	201,649	125,877	201,649

*** p<0.01, ** p<0.05, * p<0.1.

Standard errors in parentheses, clustered by county age group.
Note: Marginal effects at the mean of the independent variables from a heteroskedastic probit model. The standard probit model is rejected on the basis of a Wald test. The county infant mortality variable is the average mortality rate (in deaths per 1,000 live births) for individual's five year birth cohort in county of birth. The first and third columns restrict analysis to those aged over 25 and under 75. The second column restricts analysis to individuals aged over 40 and under 75. Source: Irish infant mortality database and Irish census 2002 and 2006. All coefficients are scaled by 1,000. Other variables are county of residence and county of birth fixed effects, county of birth trends, and controls for survey year.

The second column restricts the sample to those over 40, the central focus of our hypothesis. Infant mortality in the year and county of birth is still significant at the 1% level, with about the same coefficient. The final regression in the third column includes lags and leads (infant mortality 5 and 10 years before and after birth). Infant mortality at the time of birth is still significant at the 1% level, again with only a slightly smaller sized coefficient. The leads and lags of infant mortality in county of birth are not jointly statistically significant indicating that it is what happens around the time of birth that matters for latter life disability.

Results in all specifications suggest that a decline in the infant mortality rate at birth reduces the probability of being disabled. These conclusions are robust to using standard probit, logit and linear probability model approaches, with estimated coefficients ranging from .038 to .049 percentage points. A natural question is whether results of this magnitude are economically significant. Given that the average disability rate in Ireland according to the 2006 census is around 10% these are large effects. Using the estimate from this analysis, the improvement in national early life conditions in the 1940s (proxied by the infant mortality rate), a fall of around 35 ?? points, was associated with approximately a 13-17percent decrease in the probability of disability for those cohorts who benefited. Improvements over this period were even more dramatic in some counties, particularly the urban areas. For example, in Dublin the infant mortality rate fell from an average of 103 in the period 1938-1942 to an average of 43 in the period 1948-1952.

Table 5 presents the marginal effects from our main specification evaluated at different levels of education, again scaled by 1000.[19] Taking schooling as a proxy, there is a clear indication that those from lower socio economic groups are much more vulnerable to the adverse effects of poor early life conditions. The marginal effect of infant mortality on the probability of disability is much larger for those with primary education compared to those with a postgraduate qualification where the effect is not statistically different from zero..

Table 5 Marginal Effects of Infant Mortality for Each Education Group

Education Level	Marginal Effect	Standard Error	P
Primary	0.7182	0.2232	0.001
Lower Secondary	0.456	0.2687	0.09
Upper Secondary and Tertiary	-0.1155	0.972	0.235

Note: Marginal effects of infant mortality on disability status from the heteroskedastic probit model in column 1 of table 4 evaluated at different levels of education. All coefficients are scaled by 1,000.

Possible Selection Biases

There are several possible types of selection induced by the sharp decline in infant mortality that may affect our results. Two well-known forms are related to mortality selection (Deaton, 2007) (Bozzoli et al., 2009). In our case, a reduction in infant mortality could have led to less healthy babies surviving and thus produced an increase in health disability at older ages

[19] As pointed out above, higher education could be a consequence of the reduction in infant mortality. As before, we do not include education as a right-hand side variable in this model. We only use education as a way of stratifying our sample by SES to see if effects differ between high and low SES groups. With Irish Census data, there is no more direct SES measure available. The validity of this stratification depends on how much of own adult education is explained by the mechanism of improvements in childhood mortality to adult education. It turns out when we estimated a model of adult education that county level infant mortality explained very little of the education increase.

due solely to a composition effect. Similarly, a reduction in adult mortality would also lead to an increase in adult disability due to the less healthy adults now being able to survive. It is important to recognize that if present both these forms of selectivity imply that our empirical estimates are an understatement of the consequences of the reduction of infant mortality on adult health since we estimate a positive effect in reducing adult disability and this selectivity imparts a negative correlation. These types of selectivity induced by mortality cannot produce our results, but only lessen their quantitative importance.

The third type of selectivity is more unique to the Irish case—selectivity induced by out-migration from Ireland. During these periods of time, Irish out-migration is certainly important. Between 1951 and 1961, net out-migration from Ireland was 40, 877, or 14.1 per 1,000 average annual population (NESC, 1991). In recent work, we have studied out-migration from Ireland and the health of migrants over this period (Delaney et al, 2009). During these years, by far the main destination for Irish migrants was England. As have others before us (Marmot et al, 1984), we find that in contrast to the normal 'healthy' migrant effect (Rumbaut and Weeks, 1996) found in many countries for immigrants, Irish migrants to England were actually less healthy and shorter in stature than those who remained in Ireland. However, the critical issue for bias for our results is whether the remaining healthier non-immigrating adult Irish in Ireland induced by the sharp drop in infant mortality after 1946 was different than for migrants born in the years before 1946 and after 1946. The answer is no. Differences in adult health of those in Ireland and the Irish who went to England have consistently narrowed from the birth cohorts of the 1930's onwards. There is no evidence that there was a sharp break in this pattern associated with the mid-1940s birth cohort who would have been the affected by the sharp drop in infant mortality.

V. Conclusions

This paper has outlined an analysis of effects of early life conditions on later outcomes in Ireland, using a dramatic shift that occurred in initial health endowments around the middle of the 20th century. A database on county infant mortality was combined with census files, and the determinants of disability were analysed with particular focus on the role of infant health in a person's county of birth, at their time of birth. County infant mortality is significant in all specifications, with a remarkably robust coefficient, even when county trends and lags and leads were included in the model. Results suggest that the fall in infant mortality in the 1940s reduced the risk of suffering from a disability by around thirteen to seventeen percent. There is also evidence that individuals from lower socio economic groups are more at risk from poor early life conditions, with marginal effects twice as large for those at the bottom of the education distribution compared to the top.

We present evidence that the main mechanism for Ireland was the 1947 Health Act, which introduced a raft of reforms aimed at dramatically improving sanitation conditions, particularly in urban areas. The act included provision for improving food safety, refuse collection, infection control and other features of sanitation. These policies focused explicitly on urban areas, including hiring specific officers to enforce these policies and they are also more likely to have affected city areas as the more crowded urban conditions would make poorer families more vulnerable to collective sanitation hazards. We have shown that these policies were successful in eliminating the relationship between sanitation facilities and infant mortality by the 1960s. Indeed the bulk of the improvement in the infant mortality rate was generated in urban areas with the initially wide disparity between urban and rural rates disappearing by the mid 1950s. Dublin, Cork and Limerick (the main cities in Ireland) saw particularly dramatic declines.

The argument that these improvements were driven by the 1947 Health Act is given further credence by looking at the causes of death that generated the decline. In section 3 we showed that deaths from gastroenteritis and congenital deformity experienced the steepest declines. Both of these causes of death are particularly linked to poor sanitation conditions. It is difficult to separate between the different facets of sanitation that may have been responsible with the existing data. The fact that major urban clear-outs were underway from the early 1930s without impacting on infant mortality suggests that crowding itself may have been less of a problem than actual treatment of water and rubbish.

Other competing explanations are unlikely to be the real mechanism behind the improvements in Irish infant health in the 1940s. A time series analysis suggests that there is no evidence of a relationship between either national GDP per capita or real wages and infant mortality. At a regional level, Table 2 demonstrates there was no relationship between county unemployment rates and infant mortality in 1946. Furthermore, environmental factors such as rainfall did not exhibit any permanent change and do not match well the regional-temporal features of the mortality reductions. Birth rates did increase slightly during the war, in part due to changes in registration legislation, however this occurred too early to explain the fall in infant mortality after 1947.

Overall, the evidence presented in this paper suggests that the changes in health policy in the 1940's in Ireland both improved the contemporaneous health of infants, and equipped the cohorts who benefited with more robust health as adults.

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