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

**Dr Liam Delaney,**

Lecturer UCD School of Economics & UCD School of Public Health & Population Science, UCD Geary Institute, University College Dublin, Belfield, Dublin 4, Ireland. Tel: + 353-1-7164631, Fax: + 353-1-7161108, Liam.Delaney@ucd.ie

**Mark McGovern, M.A,** and

Researcher, UCD Geary Institute, University College Dublin, Belfield, Dublin 4, Ireland. Tel: + 353-1-7164650, Fax: + 353-1-7161108, mark.mcgovern@ucd.ie

**James P. Smith[Professor]**

Senior Economist, RAND Corporation, 1776 Main Street, P.O. Box 2138, Santa Monica, CA 90401-3208, U.S.A. Tel: + 1-310-451-6925, Fax: + 1-310-451-6935, smith@rand.org

### 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 II. 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 12% to 18%.

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There has been remarkable growth in life expectancy across the developed world in the past century. Advances in longevity in the 20th 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 has 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 led to a convergence of urban and rural infant mortality rates, essentially eliminating the urban mortality penalty. We argue that one of the reasons for the sharp fall in infant mortality

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during this period was due in part to legislative changes that improved sanitation and water, including food safety and rubbish disposal.<sup>1</sup> With appropriate time lags, these changes improved contemporaneous health of infants and contributed 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 database 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 later 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 socioeconomic distribution.

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 0.3 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.<sup>2</sup>

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 United States were concentrated before age 14. In the second half of the century, two-thirds of gains were felt by those over 45, 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 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 account for 70% of declines in mortality since 1960, are leading economists to focus on the importance of initial health endowments.<sup>3</sup> Similarly, Ireland's poor performance in life expectancy (compared to other developed nations) over the recent

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<sup>1</sup>Details are discussed in section II.A.

<sup>2</sup>Life expectancy at age 65 stagnated until the mid-1980s; there followed an increase of remaining life expectancy from 16 to over 19 years by 2006.

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 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 argue that a series of interventions succeeded (albeit belatedly) in helping to eliminate 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 about 13 to 17%.

Early life conditions may also be important for explaining variation across social class. Although several studies have shown separately that Irish people from lower socioeconomic 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 groups with lower socioeconomic status (SES) 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 behavior and circumstance will not enable us to accurately characterize 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 that 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.<sup>4 5</sup>

<sup>3</sup>Black et al. (2007) shows 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 I.B discusses previous studies in more depth.

<sup>4</sup>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 fetus 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).

<sup>5</sup>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).

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) demonstrates 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 that a 10% increase in birth weight implies a 0.57 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 United States to provide exogenous variation in early life conditions. Data from the U.S. Census (1960, 1970, 1980) suggest that the 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) argues 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 that 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 industrialization and urbanization. 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 United States.<sup>6</sup> For U.S. 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 sewage disposal, leading to a consequently higher degree of contaminated water and food; and garbage and carrion in the streets (Haines, 2001).<sup>7</sup>

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 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 systems in Dublin and then Cork (1929) proved effective in reducing local refuse.

#### A. The 1947 Health Act

Following World War II, 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 ten years.<sup>8</sup> 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.<sup>9</sup> 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 show the age distribution of child mortality declines, demonstrates that by far the main mortality improvement is focused on infants, i.e., those

<sup>6</sup>See, for example, Williams and Galley (1995) for a discussion of the experience in England.

<sup>7</sup>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.

<sup>8</sup>The Irish Times, October 3, 1947.

<sup>9</sup>These measures were particularly aimed at combating problems associated with tuberculosis.

who were less than one year 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 declines of both diarrhea 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 and 1961 allow us to examine county-level correlates of infant mortality.<sup>10</sup> 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 a county, and both are associated with higher levels of infant mortality. 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.

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 percentage 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 in Dublin had halved by this time.

Our claim that the 1947 Health Act was an important contributor to the elimination of the urban mortality penalty still has to deal with the plausibility of alternative explanations. Our claim is not solely based 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—in particular, but 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 were most correlated with the changes in county-level infant mortality. In addition, the association of infant mortality with the percentage of families using shared sanitation and infant mortality is eliminated 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 a role for other possible factors. Three plausible alternative possibilities are the 1932–1938 trade war

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<sup>10</sup>Relevant county-level data is only available for these two years.

with Britain, the Second World War (WWII), and the introduction of penicillin and sulfa drugs. 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 including the period from 1940 to 1955 after the trade war was initiated.

As our mortality figure by cause showed in Figure 4, WWII 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 and other drugs such as sulfa drugs. For example, penicillin was first introduced into Ireland in 1945, so the timing is certainly coincident with the fall in urban mortality. However, a thorough search by us found 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 rollout 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 predated the discovery of penicillin and has been largely attributed to improved hygienic measures, such as improved sewage, water purification, removal of refuse, and improved milk supplies (McKeown et al., 1975; Williams and Galley, 1995; and Cutler and Meara, 2003).

In particular, Cutler and Meara (2003) shows that 83% of the decline in infant mortality in the United States 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 would be in common. The later timing of the elimination of the urban mortality penalty in the 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 and other drugs in the Irish case (the timing, extent, and geography of the penicillin rollout) is simply not available at present. Until we have that evidence, we should remain cautious in our summary conclusions and allow for a contributory role for introduction of new drugs to the market.

### III. Data

#### A. Census Data on Disability and Health

This research on relating infant mortality declines to later life health relies on successive waves of Irish census data from 2002 and 2006.<sup>11</sup> There are several advantages to these 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.

<sup>11</sup>Irish census data is freely available from the Irish Social Science Data Archive ([www.ucd.ie/issda](http://www.ucd.ie/issda)).

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 is quite high, as Table 2 demonstrates. Among those with a disability, only 14% 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 that their health is fair or bad, compared to 6% for the non-disabled.

The question on employment status identifies those out of work due to illness, but there are small numbers in this category. We combine these measures into a single disability variable.<sup>12</sup> Table 3, which shows types of disability broken down by 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.

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 the disability rate for people born in urban areas (Dublin, Cork, Limerick, Waterford, and Galway county boroughs) and rural areas for five-year birth cohorts.<sup>13</sup> 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 in section V.

## B. Infant Mortality Data

For our analysis, we use information gathered on annual infant mortality by county of birth.<sup>14</sup> Due to restrictions mentioned above on knowing a person's exact age, we average infant mortality in a county by five-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, the 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.

<sup>12</sup>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 is included. A concern is that disabilities 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).

<sup>13</sup>Due to the process of anonymization employed by the Irish Central Statistics Office, we only know a person's five-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 over 75. We also limit the sample to those over 25, as many under this age may be still in school.

<sup>14</sup>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 an electronic format.



## IV. Results

### A. Model Specification

We examine effects 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.<sup>15</sup> We did not control for adult education, since reductions in infant mortality could have led to increases in adult education (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.

These models also control for county of birth fixed effects, county of current residence fixed effects, county-specific age quadratics, 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 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 each census separately reaches similar conclusions.<sup>16</sup> The following equation summarizes the model.  $\gamma$  is the vector of control variables outlined above.

$$\begin{aligned} \text{Disability}_{ijt} = & \alpha + \beta_1 \text{Age}_{it} + \beta_2 \text{AgeSquared}_{it} + \beta_3 \text{Sex}_i \\ & + \beta_4 \text{InfantMortality}_{jt_0} \\ & + \gamma_{ijt} \end{aligned}$$

We first present marginal effects at the mean of the independent variables. However, to examine whether early life conditions have the same effect across socioeconomic 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 that does not change over time. Our inclusion of county-specific trends has important consequences for the analysis, as identification of the effect of infant mortality is reduced to using sharp nonlinear breaks, such as the dramatic declines in the urban areas in the 1940s.<sup>17</sup> 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 nonlinear 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.

<sup>15</sup>We did not also control for cohort effects, since the well-known identity between cohort, time, and age effects precludes that.

<sup>16</sup>Some individuals are in merged 2002 and 2006 census twice. The Irish government only releases a 5% sample of the census, so about 0.25% of the sample is in common. Thus, the effect on standard errors is quite small. We cannot identify these people in the publicly released data, but this effect of this degree of clustering must be very minor.

<sup>17</sup>In fact, omitting county trends has little effect on either the magnitude or significance of these results.

## 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 1,000 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 of disability, as is being male. Most central to our hypothesis, a unit decrease in the infant mortality rate (deaths per 1,000 live births) is associated with a 0.506 percentage-point decrease in the probability of currently suffering from a disability. This result is strongly statistically significant.

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 a slightly smaller 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 later 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. 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 with a fall of around 35 points), was associated with an approximate 12% to 18% 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.

We also estimated separate models as in Table 4 separately for men and women. The estimated effects on adult disability were concentrated on men. For example, the male coefficient in the first column of Table 2 was 0.918 which was statistically significant at the one percent level while the female coefficient was 0.141 which was not statistically significant. Given this result, we examined this issue further. First, the decline in infant mortality rates since the late 1940s in Ireland was much higher among boys than among girls- a 33 percent larger decline in infant mortality. In retrospect, this may not be surprising since boy babies are known to be more fragile than girl babies. (Drevenstedt et al, 2008; Wells, 2000). In addition, an important form of disability is that associated with work. Using our data in the age span 40–60 years old, 81% of Irish men are working compared to only 55% of Irish women.

Table 5 presents the marginal effects from our main specification evaluated at different levels of education, again scaled by 1,000.<sup>18</sup> Taking schooling as a proxy, there is a clear indication that those from the lowest socioeconomic groups are much more vulnerable to the adverse effects of poor early life conditions. The marginal effect of infant mortality on the

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<sup>18</sup>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. When we estimated a model of adult education, county-level infant mortality explained very little of the education increase.

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.

**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 produced increases in health disability at older ages due solely to composition effects. 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. The 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-emigrating 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 1930s 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, 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 13 to 17%. There is also evidence that individuals from lower socioeconomic 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. Finally, these effects are concentrated on men which may not be surprising in light of the more rapid decline in male infant mortality rates over time and the higher male labor force participation rates in light of the concentration of disability in the work force.

We present evidence that an important 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. 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 contributing to the elimination of 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 at least in part due to 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 distinguish the different facets of sanitation that may have been responsible with the existing data. The fact that major urban clear-outs were under way from the early 1930s onward without impacting on infant mortality suggests that crowding itself may have been less of a problem than actual treatment of water and rubbish.

Some other competing explanations are unlikely to be the real mechanism behind improvements in Irish infant health in the 1940s. A time series analysis suggests that there is no evidence of a relationship between either national gross domestic product per capita or real wages and infant mortality. At a regional level, Table 2 demonstrates that 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 WWII, in part due to changes in registration legislation. However, this occurred too early to explain the fall in infant mortality after 1947.

The most difficult explanation to assess relates to introduction of new effective drugs, in particular penicillin and sulfa drugs. The existing literature is inconsistent on this issue. Some studies have shown an important role for drug innovations in life expectancy improvements around the world (Acemoglu and Johnson, 2007) while Cutler and Meara (2003) and McKeown et al., (1975) demonstrated that improvements in urban infant mortality in the United States and England predated the introductions of drugs such as penicillin. Based on timing alone, it is certainly possible that these drug innovations contributed to reductions in urban infant mortality in Ireland even if they did not initiate the trend. But a more compelling case requires careful documentation of the speed and intensity of the actual roll-out of these drugs in urban and rural areas of Ireland. Without that detailed documentation, we should not rule out a contributory role for such drug innovations.

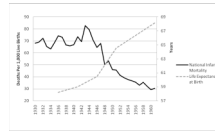
Overall, the evidence presented in this paper suggests that the changes in health policy in the 1940s in Ireland both improved the contemporaneous health of infants and equipped the cohorts who benefited with more robust health as adults.

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**Figure 1. Irish Infant Mortality and Life Expectancy 1930–1961**

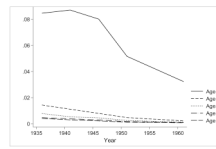
Source: Irish Life Tables and Central Statistics Office. Note: The figures for life expectancy are from the years 1936, 1941, 1946, 1951, and 1961 and refer to expected years of life remaining at birth.



**Figure 2. Urban and Rural Infant Mortality 1930–1961**

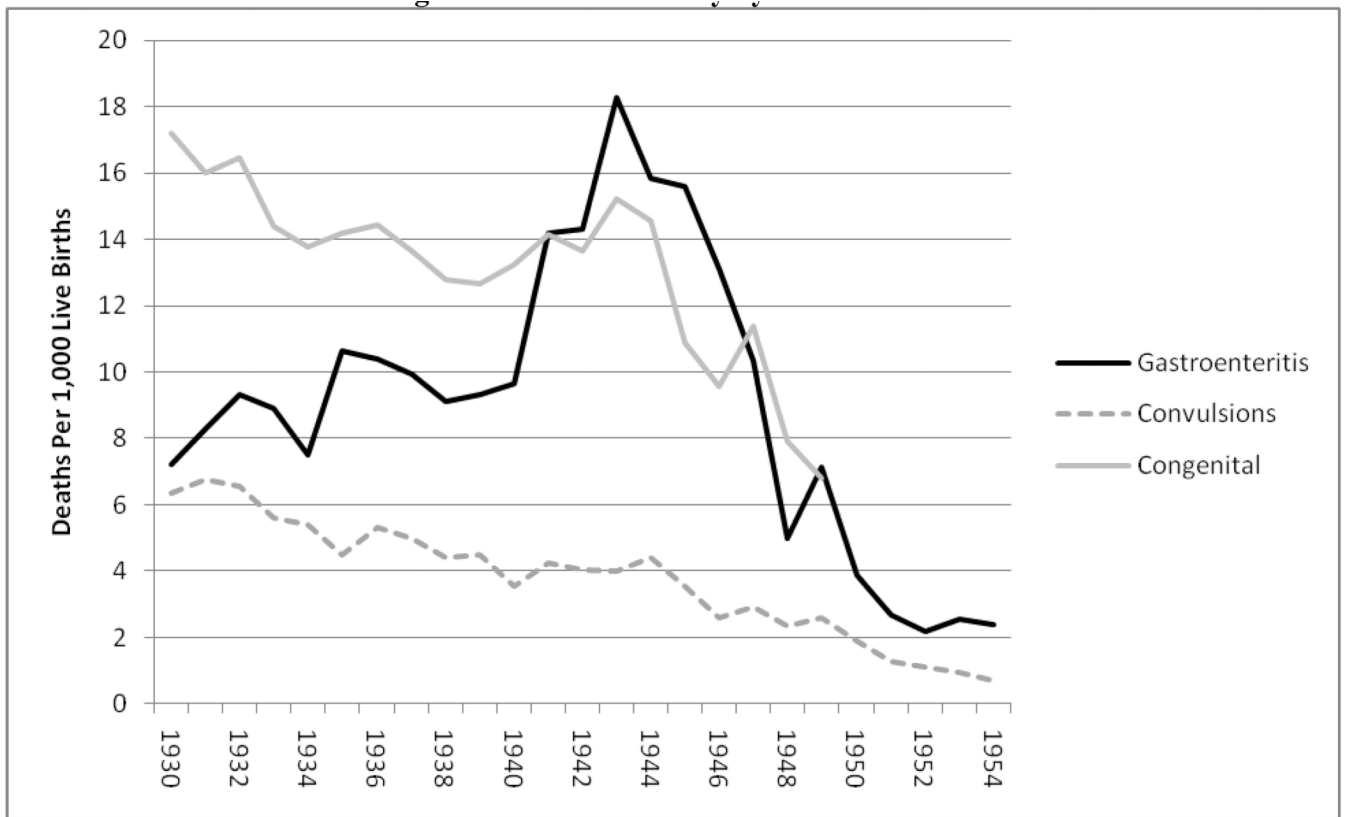
Source: Irish Infant Mortality Database. Note: The urban areas include Dublin, Cork, Limerick, Waterford and Galway county boroughs, with the rural areas comprising the other county areas.





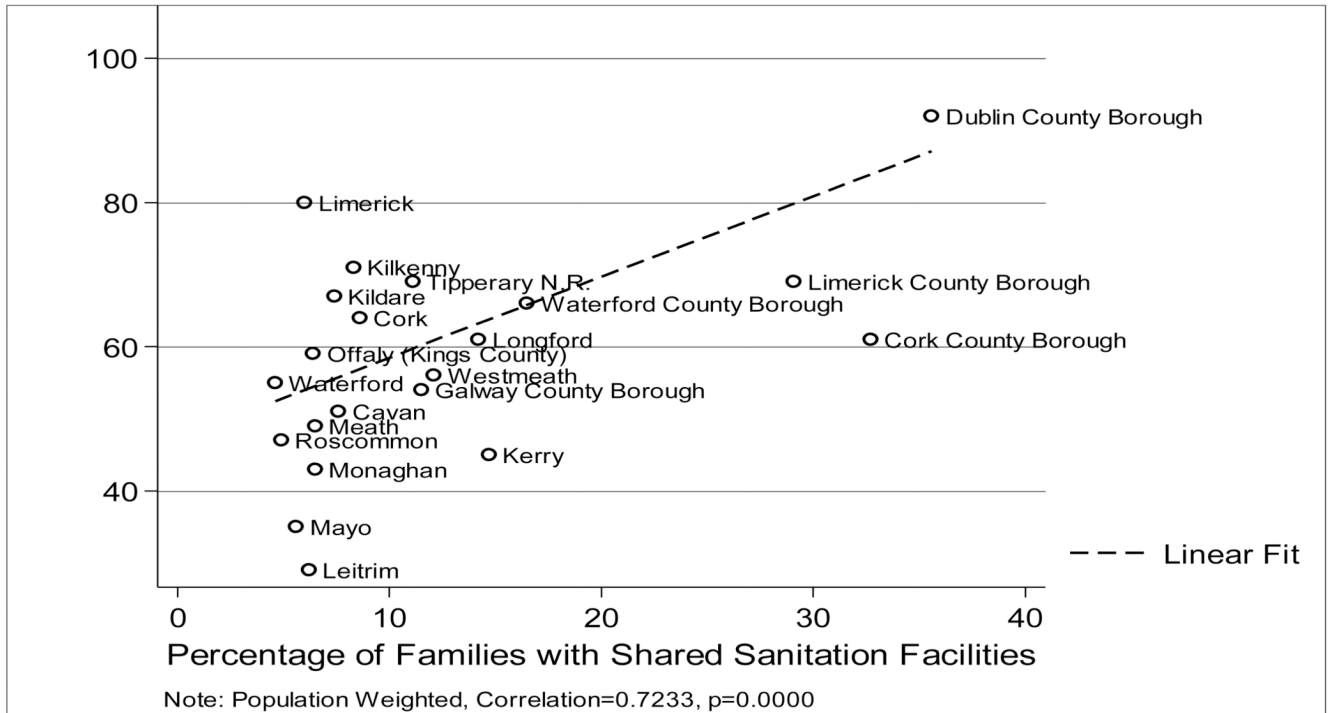
**Figure 3. Early Age Mortality Rates 1936–1961**

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



**Figure 4. Infant Mortality by Cause 1930–1954**

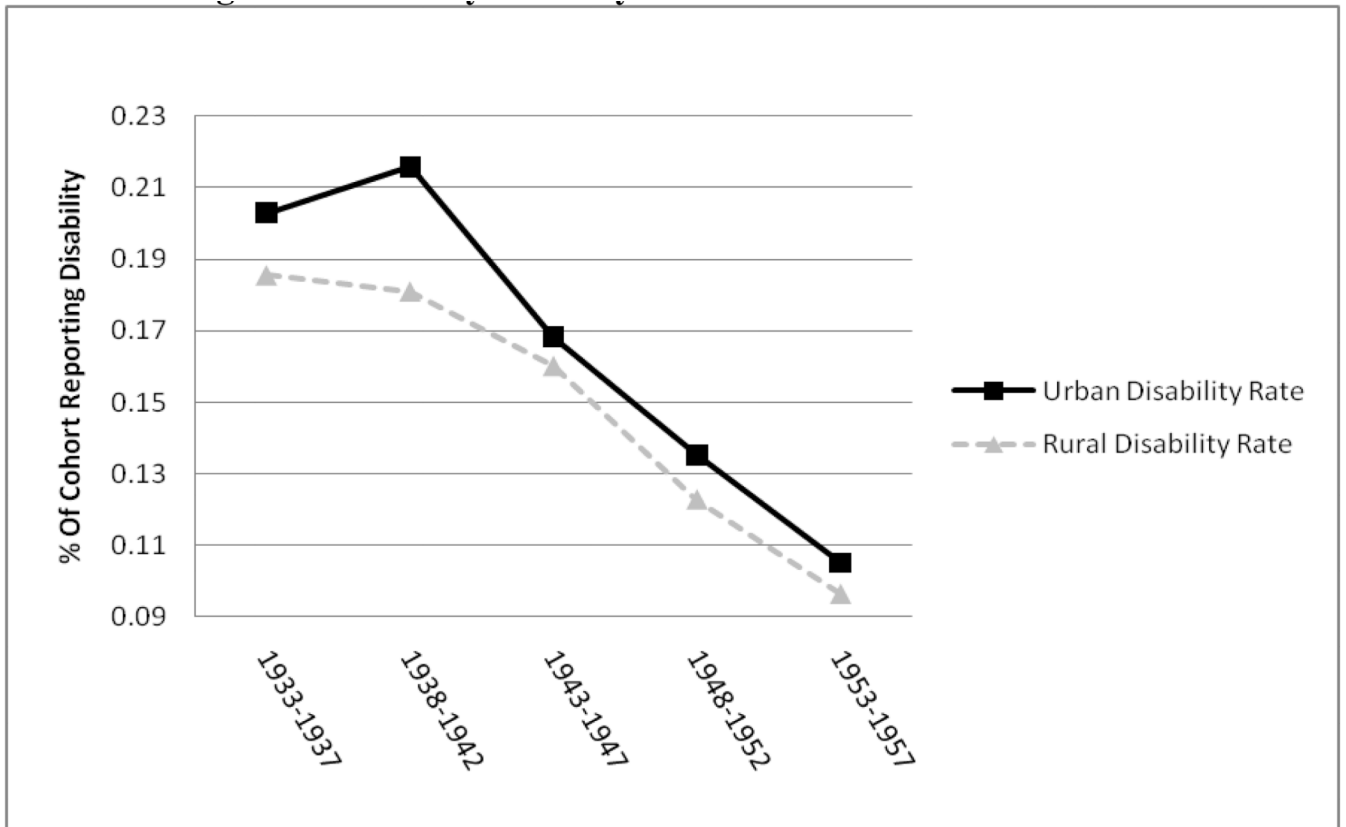
Source: Central Statistics Office. Data exist only for the years 1930–1954. Note: Due to changes in the coding for the congenital series, this cause can only be shown up to 1950.



**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.



**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.

**Table 1**

## County-Level Determinants of Infant Mortality in 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 Counties	30	

Standard errors are 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 censuses in 1946 and 1961.

**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; or 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](http://www.cso.ie/releasespublications/documents/labour_market/current/healthstatus.pdf)

**Table 3**

Cause of Disability by Type

Cause	Type of Disability									
	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	
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	100

Source: National Disability Survey, 2006. N=14,518. Note: The table shows the causes of each type of disability (as a percentage of that particular disability). For example, of people who have a seeing 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>



**Table 4**

## Marginal Effects for Disability

Variables	Disability	Disability (>40)	Disability (Lags and Leads)
Age	3.22 (3.09)	5.38 (9.02)	3.54 (3.41)
Age Squared	-0.0001 (.033)	0.0789 (.084)	0.0076 0.0359
County Infant Mortality (Five-Year Average)	0.506*** (0.144)	0.478*** (0.151)	0.307** (0.146)
Female	-0.9798 (2.2149)	-4.7859 (4.0381)	-0.9369 (2.2143)
County Infant Mortality +5 Years			-0.0783 (.237)
County Infant Mortality +10 Years			-0.4714* (.2821)
County Infant Mortality -5 Years			0.0921 (.1563)
County Infant Mortality -10 Years			-.1407 (0.1013)
Wald Heteroskedasticity Test	22.13***	5.00**	13.43***
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 the 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.