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ABSTRACT

Fertility Decline and the Heights of Children in Britain, 1886-1938^{*}

In this paper we argue that the fertility decline that began around 1880 had substantial positive effects on the health of children, as the quality-quantity trade-off would suggest. We use microdata from a unique survey from 1930s Britain to analyze the relationship between the standardized heights of children and the number of children in the family. Our results suggest that heights are influenced positively by family income per capita and negatively by the number of children or the degree of crowding in the household. The evidence suggests that family size affected the health of children through its influence on both nutrition and disease. Applying our results to long-term trends, we find that rising household income and falling family size contributed significantly to improving child health between 1886 and 1938. Between 1906 and 1938 these variables account for nearly half of the increase in heights, and much of this effect is due to falling family size. We conclude that the fertility decline is a neglected source of the rapid improvement in health in the first half of the twentieth century.

JEL Classification: I32, J13, N33, N34

Keywords: fertility decline, heights of children, health in Britain

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Introduction

It now well understood that growth during childhood is conditioned by nutrition and disease. A host of studies in a variety of historical and development settings have explored the link between nutrition and growth, so much so that height has become widely accepted as a key indicator of physiological well-being. It is also widely acknowledged that the disease environment matters: children subject to frequent bouts of disease such as diarrhoea and respiratory infections are less able to convert the available food supply into the growth of bone and sinew. While it is possible to regain lost ground through lengthening the growth years, catching up may be partial and so deficits in nutrition and health can leave a permanent mark on final height. Research by epidemiologists and others has also shown that poor health during childhood and diminished height is correlated with adult health conditions such as ischaemic heart disease and with increased risk of mortality from all causes.¹

In the past 25 years economic historians have unearthed a vast amount of data on heights that has painted a much clearer picture of long-run trends in the biological standard of living (Steckel, 1995; 2009). For the United States and a number of European countries the results typically show a steady increase in average heights that broadly correlates with the pace of economic development, although with some temporary reverses such as those observed in the United States, Britain, the Netherlands, Sweden and Germany around the middle of the nineteenth century.² The steady growth trend in the late nineteenth century was followed by a much steeper upward trend that started in most countries around the end of the nineteenth century and extended well beyond the Second World War.³ The remarkable pace of improvement in the health of children in the first half of the twentieth century has been widely attributed to better nutrition as a direct result of the growth in per capita income as well as to public heath improvements, the availability of antibiotics and vaccination, and medical progress more generally.

¹ For useful surveys, see Elo and Preston (1992) and Galobardes et al. (2004).

² See the country studies in Floud and Steckel (1997) and the overview by Haines (2004),

³ The trends for a number of countries are compared in Floud (1994, pp. 16-19) and presented graphically by Weir (1993). More recently, Baten (2007, p. 11) shows that the sharp discontinuity in the trajectory of heights in western Europe and North America around the turn of the 20th century is not paralleled in the populations of other continents.

quickening in the growth of income per capita and they also largely pre-date the expansion of heath services and public welfare programs (McKeown and Record and Turner, 1975).

One factor that that has been neglected in much of the debate is the diminishing size of the family resulting from the decline in fertility that began in the late nineteenth century and continued until the 1930s.⁴ This trend has been interpreted as the progressive substitution away from large numbers of children per family towards higher average child 'quality'. The preference for quality in dimensions such as health and education over quantity has been linked to trends such as lower infant mortality, restrictions on child labour, the rise of education (especially among parents) and a host of other factors associated with economic development. Clearly, if smaller families are causally linked to improved outcomes for children, then the fertility decline should have been a powerful force for improvement. It is worth stressing that the focus here is exclusively on the effects of changing family circumstances rather than on their causes.⁵

The quality-quantity trade-off has been subject of a lively empirical debate among economists since the pioneering analyses of Becker and Lewis (1973) and Becker and Tomes (1976). A host of studies have explored the links between childhood conditions and health and educational outcomes using modern microdata, which include, among other explanatory variables, the number of children in the household. Although there are issues of causal inference, the weight of evidence favours a negative relationship between the number of siblings and the average child outcome.⁶ In this light it is surprising that so little attention has been paid to this key relationship in explaining the dramatic improvements in health from the end of the nineteenth century.

Empirical analysis of country-level data has firmly established the historical link between income per capita and the heights of young adults (Floud, 1994; Steckel, 1995). Other variables matter too, including inequality and, interestingly, infant mortality both of which have negative effects. The latter finding points to the importance of the disease

⁴ One important exception is Reves (1985) who argued that declining fertility played a significant role in reducing infectious disease among children in England and Wales from the late nineteenth century.

⁵ There is an extensive literature on the factors influencing the choice of smaller families, which include expanding consumption possibilities, increasing education, and changing social norms. Recent important contributions on Britain include Garrett et al. (2001), Humphries (2007) and Scott (2008).

⁶ For the effects on education see Hanushek (1992), Booth and Kee (2009).

environment in conditioning growth, and the decline in infant mortality has sometimes been related to the decline in fertility. One of the few studies to make the direct link between fertility and height is Weir (1993) who analysed regional data on the heights of French conscripts from 1840 to 1911. Using this panel he found that, in the presence of other effects, falling marital fertility contributed half to three-quarters of a centimetre to the increase in heights across the nineteenth century. This accounts for about a quarter of the total gain in height between 1790 and 1911. Although contributions such as these that rely on aggregate data have added much to our understanding, by their very nature, they are unable to identify the mechanisms at work at the household level.

Some of the literature also studies heights at the individual level.⁷ Broadly speaking, the results indicate that height is positively associated with income or occupational status and with rural residence. Apart from the well-known selection issues that confront the use of data on military recruits, these studies typically suffer from a number of other shortcomings. The most important is that the information available for use as explanatory variables is often severely limited. Apart from age, the explanatory variables used often relate to occupation or father's occupation, literacy, place of birth and a few other individual characteristics. These variables stand as proxies for variables, such as family income, that are not directly observed. A second issue is that individuals are often observed as adults and so assumptions have to be made about the conditions in which they grew up. Given these limitations it is perhaps not surprising that household characteristics such as the number of siblings have been neglected and that, as a result, the potential impact of changing family size has been largely obscured.

This paper offers new results using household data on the heights of children in British families during the 1930s. This allows us to pursue the type of microeconomic analysis that is used by economists on modern datasets but for a period much closer to the fertility decline. We find that the heights of children are positively related to family income per capita and negatively to the number of children in the household. The key channels of influence appear to be nutrition, as represented by food expenditure, and the disease environment, which is related to overcrowding and housing quality. Using these

⁷ Studies that present of individual-level econometric analysis of heights include Johnson and Nicholas (1997), Whitwell et al. (1997), Riggs (1994).

results we can assess the contribution of falling family sizes to the trend in the heights of children from the 1880s to the 1930s. The bottom line is that falling family size accounts for more than a third of the increase in heights over this period.

Trends in health, height and socioeconomic conditions

Height data for Britain come from a variety of sources and those most relevant to our argument are listed in Table 1. They are reported by date of observation rather than by date of birth. Those for army recruits (adjusted for truncation) show that, after some decline, average heights increase by about 1cm per decade from 1885-9 to 1910-13. This would be equivalent to an increase of about 0.8cm per decade for 10 year-old children. The upper right hand panel of Table 1 illustrates the trend in heights among boys at Christ's Hospital School, which shows that from the 1870s to the 1910s heights increase at about 0.6cm per decade for 10 year olds and 0.9cm per decade for 14 year olds. It also illustrates the distinct acceleration in heights referred to earlier. From 1906-18 to 1930-33 heights increase by 1.6cm per decade for 10 year olds and by as much as 2.2cm for 12 year olds and 2.8 cm for 14 year-olds.⁸

For the period from 1908 we are fortunate to have more representative data on the heights of children that have been carefully assembled by Bernard Harris from the reports of school medical officers (see Harris, 1994, 1995). The practice of measuring children dates from the establishment of the school medical service in 1908 and was continued into the 1950s. As the results were not reported for all ages in all towns in all years, we have used fixed effects regressions with year dummies to establish the trends in heights for boys and girls at age 5, 8 and 12. The four-year averages presented in the lower panel of Table 1 also show remarkably strong growth in average heights over three decades up to 1939. Linear trends fitted to the underlying annual data give the average annual increase in height for boys of 1.5cm per decade for five year olds, 2.5cm per decade for eight year olds and 1.9cm per decade for 12 year olds. For girls, the average increases per decade at the three ages are, respectively, 1.6cm, 2.5 cm and 2.3cm. These figures

⁸ Christ's Hospital is a charitable school for poor and fatherless children that received a royal charter from Edward VI shortly after its foundation in 1552. The basic criteria for admission remained largely unchanged over the period examined here and so the trend in heights should not be influenced by changing socioeconomic mix among the pupils.

reinforce the impression gained from more fragmentary data that the heights of children increased particularly rapidly from around the turn of the century.⁹

An alternative indicator that is often seen as a general measure of the health status of populations is infant and child mortality. While infant mortality indirectly reflects nutritional status, it is generally taken as more closely reflecting the disease environment. As Figure 1 shows, infant mortality in England and Wales averaged about 150 per thousand in the late nineteenth century after which it went into a sharp secular decline in the first half of the twentieth century, reaching about a third of its former level by the late 1940s. Among the proximate causes of the steep fall in infant mortality were diseases associated with prematurity, diarrhoea and dysentery as well as respiratory diseases such as bronchitis, pneumonia and influenza. Woods et al. (1988, p. 362) argue that the upturn around the turn of the century was largely due to unusual climatic conditions, which particularly affected deaths from diarrhoea and that the underlying trend decline dates from the early 1890s.

Mortality rates for children aged 1-4 and 5-9 declined from the middle of the nineteenth century, largely as a result of the declining incidence of diseases such as scarlet fever, diphtheria, measles and whooping cough. Floud et al. (1990, p. 301) comment on the relationship between cohort mortality and the trends in heights in the half century to 1914 as follows: "The match is amazingly close. In other words, the fall in mortality in late nineteenth century England and Wales follows almost exactly the pattern that we would expect from the evidence of nutritional status. The height data make the link between nutrition (although in a wider sense) and mortality which McKeown [1976] could only infer." They go on to comment that the decline in mortality and the increase in the heights of successive cohorts "could best be explained by improvements in the health of children" (1990, p. 314). Thus it appears that while improved nutrition underpinned the trend increase in height and the trend fall in child and adult mortality from the third quarter of the nineteenth century, significant improvements in the disease environment affecting children (as reflected in infant mortality) date largely from the 1890s.

⁹ This is consistent with other data presented by Rosenbaum (1988) and earlier studies by Karn (1937), Clements (1953) and Boyne et al. (1957). For a longer-term perspective see Floud et al. (1990, p. 166).

The argument put forward here is that these striking improvements in the health of children owe much to the fall in the number of children per family. Although the fall in infant and child mortality increased the survival rate, this effect on family size was overwhelmed by the falling birth rate. Figure 2 shows that births per woman aged 15-44 fell from a peak of 154 per thousand in the 1870s to a low of 62 per thousand in the 1930s. Over the same interval the total period fertility rate fell from 4.8 to 1.7 while the gross reproduction rate fell from 2.3 to 0.9. The clear implication of these trends is smaller families, particularly among the working class where the fertility decline was later and steeper.

Much of the literature on health and height—most notably that inspired by the pioneering work of McKeown (1976) and Fogel (2004)—has stressed the importance of the link with real wages and per capita income. And most of the focus has been on the nineteenth century, with an emphasis on nutrition and only indirectly on the disease environment. Such a focus is difficult to square with the British experience between 1850 and 1950. Feinstein's (1995) figures show that real earnings increased by 1.2 percent per annum in 1875-1900, 0.8 percent in 1900-1925 and 1.6 percent in 1925-1950. Overall the average rate of wage increase was the same in the half centuries before and after 1900. However, for families with children, household income per capita grew more strongly than real wage rates between the 1880s and the 1930s because of the fall in family size.

These trends, particularly after the turn of the century, are consistent with the sharp decline in poverty among working class families that was noted in a variety of poverty surveys. In his investigation of York in 1899, Rowntree (1901, p. 37) found that large families with young children were particularly likely to be in poverty. In his later survey he found that the fall in primary poverty between 1899 and 1936 was principally due to the increase in wages for the low paid and the decrease in large families (meaning those with more than three children). Rowntree found that the primary poverty rate for children under 14 in working class families fell from about 30 percent in 1899 to 13 percent in 1936, while their share of those in poverty fell from 58 percent to 47 percent.

Using more generous poverty standards, the child poverty rate fell from 55 percent to 43 percent while their share of total poverty fell from 38 percent to 34 percent.¹⁰

A number of studies have examined the environmental conditions associated with disease and mortality. These conditions include the well-known differences between urban and rural localities, population density, industrial or occupational structure and the sanitary infrastructure of towns (Woods et al. 1988; Lee, 1991; Millward and Bell, 1998; Williamson, 1990, Ch. 9). Such variables are often interpreted as proxies for sanitary conditions generally and clean water supply in particular.¹¹ The evidence for British towns suggests that the sanitary infrastructure improved particularly rapidly from the end of the nineteenth century (Bell and Millward, 1998). This positive effect on the health environment eventually outweighed the negative effect of continuing urbanisation.

The degree of overcrowding also appears to have a strong negative effect on health as measured by infant mortality, suggesting that conditions within the household were important. Overall, housing conditions improved, especially after the turn of the century, both in terms of quality and overcrowding. In York, the proportion of the working class population living in overcrowded conditions (defined as more than two persons per room) fell from 10.1 percent in 1899 to 3.6 percent in 1936 (Rowntree, 1941, p. 269). Examining trends in Glasgow and Edinburgh, Cage and Foster (2002) found a positive effect of overcrowding on infant mortality, an effect that diminished sharply between 1911 and 1931.¹² Below we offer further evidence that, through its effect on overcrowding, declining family size significantly improved health outcomes for children.

One factor that cannot account for much of the improvement in the disease environment is the discovery and dissemination of new medical treatments. As McKeown (1976) has shown, many of the landmark medical discoveries of the twentieth century did not become available until around the Second World War. Streptomycin (effective against tuberculosis) was introduced in 1947, sulphonamides and sulphapyridine (effective against bronchitis, pneumonia and influenza and whooping cough) not before

¹⁰ The primary poverty line was defined by Rowntree (1901, p. 86) as a living standard sufficient merely to maintain physical efficiency. The figures in the text for primary poverty are calculated from the comparative data given by Rowntree (1941, p. 110). The higher poverty lines are, for 1899, primary plus secondary poverty and, for 1936, the 'human needs' standard. These are not strictly comparable.

¹¹ Cutler and Miller (2005) show that the introduction of clean water supplies account for up to half of the mortality decline in major US cities between 1900 and 1940. See also Preston and Haines (1991).

¹² For a comparison with Sydney, Australia, see Jackson and Thomas (1995).

1938 and antibiotics still later. Similarly, treatments for other childhood diseases such as measles and scarlet fever were developed in the 1930s, long after the steep decline in these diseases. Only the timing of the reduction in diphtheria mortality from around the turn of the century seems be consistent with the advent of treatment by antitoxin.

A more recent line of research suggests that advances in health technology at the domestic level were far more important. Mokyr (2000) argues that improved knowledge of nutrition and the channels through which disease is transmitted led mothers to devote more time and effort to child nurturing and housework than they otherwise would have. Specific improvements identified in the literature include better quality milk supplies, better knowledge of hygiene and feeding methods (particularly bottle feeding) as well as the growing importance of local health services in the form of midwives and health visitors, and not least of all the health of the mothers themselves (Dyehouse, 1978; Fildes 1998; Millward and Bell, 2001). These advances are likely to have been all the more effective when taken in conjunction with smaller families. Indeed, the quality-quantity tradeoff suggests that smaller family size was a key factor that made possible such improvements in child-nurturing practices.

The Boyd Orr Cohort.

In order to shed more light on the relationships between health, income and family size, we draw on the records from the Boyd Orr survey. This inquiry was undertaken in 1937-9 by the Rowett Research Institute at Aberdeen, led by Sir John Boyd Orr.¹³ It represents the culmination of a line of social investigation originating in the late nineteenth century that focused on the relationship between poverty and life chances. It is one of the few interwar surveys for which the original records have survived and the only one that contains information on the heights and health of children. The original survey covered a total of 1343 households containing 4999 children under the age of 19. It was confined to families with children and it was targeted to over-represent poor families. 16

¹³ The work on the survey was interrupted by the War and the report summarizing the results was not published until 1955 (Rowett Institute, 1955). The original survey records were recovered and have been augmented with follow-up information on later life outcomes by epidemiologists in the Department of Social Medicine, University of Bristol.

different towns and villages where chosen and these were intended to be representative of urban and rural locations.¹⁴

The original survey recorded the number of children and total household members with details of income, food expenditure and housing conditions. The clinical part of the survey collected a variety of indicators of the anthropometric and health status of the children including height, leg length, weight, incidence of medical conditions and dental decay. The clinical survey did not include all the households nor did it cover all children in households that are represented.¹⁵ As compared with the original sample the clinical survey under-represents infants, those over age 14 and, most important, those in certain locations. Here we focus on children aged 2 to 14 for whom height and weight measurements are available for 2946 children in 1131 households in 14 of the 16 localities.¹⁶

The heights by age of boys and girls are plotted in Figure 3. They increase fairly linearly from around 85 cm at age 2 to 152 cm at age 14, or about 5.6 cm per year of age. Some of the other characteristics of those who were measured appear in Table 2, which lists the means across individuals and the means across households. The average age of the children is 7 years and 11 months and a little over half are female. The average child comes from a family with 4.56 children and 6.75 people in total, whereas the average family consists of 3.74 children and 5.79 persons. As we show below, these families are much larger than the typical working class family with children in 1938. In this respect

¹⁴ The 16 locations are, in Scotland: Aberdeen, Kintore, Hopeman, Barthol Chapel, Methlick, Tarves, West Wemyss, Coaltown of Wemyss, Dundee and Edinburgh; and in England: Barrow-in-Furness, Liverpool, Yorkshire, Wisbech, Fulham and Bethnal Green. These locations are identified on a map in Martin et al. (2005), p. 743.

¹⁵ The survey report is somewhat vague on this point, commenting that "For various reasons all children in all surveyed families could not be examined although the attempt was made to include them all" (Rowett Institute, 1955, p. 50). One difficulty seems to have been arranging attendance at a school or clinic where measuring instruments could be used. But another seems to have been simply a matter of logistics: two of the original locations (Edinburgh and Kintore) are not represented at all in the clinical survey. These two locations account for about 40 percent of the missing observations for children aged 2 -14.

¹⁶ According to the original survey report 3762 children were examined, and the records for most of these have been found. We exclude those under age 2 and over 14 because they are underrepresented and may be subject to selection bias, and also because the height measurements for the very young children are thought to be less reliable.

the households in the Boyd Orr cohort have a demographic structure that is more representative of about 30 years earlier.¹⁷

Weekly per capita income of each household was recorded by the survey only as a categorical variable, with four categories: less than 10 shillings, 10-15 shillings, 15-20 shillings and greater than 20 shillings. Similarly per capita food expenditure is grouped into four categories: less than 5 shillings, 5-7 shillings, 7-9 shillings and greater than 9 shillings. An income of less than ten shillings per family member was considered as living in poverty by the standards of the time. Boyd Orr (1936, p. 49) found that children below this poverty line had a food intake that was deficient in almost every constituent while those in the next income bracket suffered deficiencies mainly in certain minerals and vitamins.

As Table 2 shows 71.6 percent of children and 59.8 percent of families were below this poverty line and 54.8 percent of children and 43.6 percent of families subsisted on a food intake costing less than 5 shillings per week. Applying a similar poverty line Rowntree (1941, pp. 42, 144-149) found that, in York (a fairly typical town) in 1936, 37 percent of working class households and 43 percent of children under 14 were in poverty. In this respect too, those families observed in the Boyd Orr survey are more typical of working class households 40 years earlier. Thus, while the population in the Boyd Orr survey is somewhat unrepresentative of the later 1930s, this is a strength for the purposes of comparison with earlier decades.

The survey also collected a limited amount of information on living conditions in the household, including the number of persons per room. 42 percent of these households were living in overcrowded conditions, on the widely used criterion of more than two persons per room. The investigators also gave an assessment of the general level of cleanliness of the dwelling, ranking a third of them as good or excellent and the remainder moderate or poor. Only 37 percent of the dwellings possessed a flush toilet inside with the remainder being non-flush and/or outside. Finally, nearly three-quarters of dwellings were assessed as having good or excellent ventilation, with the rest designated as moderate or poor.

¹⁷ One reason for this is that almost all the families have at least one child of school age. We suspect that this is because families were contacted by first approaching the schools.

Family-level determinants of heights

For the purposes of analysis we standardise the heights of children in the Boyd Orr cohort by age and sex by converting them to z-scores. The z-score converts heights into units of standard deviation by taking the deviation of the individual's height from the median and dividing by the standard deviation for that age/sex category. Thus the mean z-score is approximately zero and the standard deviation is approximately one. In order to measure the family-level determinants of heights we estimate a cross sectional model in which the dependent variable is the average z-score of height for the children who were measured in each household.

The recent debate in the economic literature noted above has been much concerned with avoiding biases that might arise from unobserved factors that determine both the health, as represented by height, and the number of children in the family. One solution is to use instrumental variables in order to correct any bias that arises from correlation between one or more of the explanatory variables and the error term. Here we present both ordinary least squares and instrumental variables estimates. The issue of endogeneity is explored in more detail in another paper (Hatton and Martin, 2008).¹⁸ Later we shall use these estimates to construct counterfactual trends and our purpose here is to insure against relying on coefficients that are biased in favour of our hypothesis.

The instruments used are of three types. The first is a dummy variable for families that contain twins at last birth. The incidence of twins has often been used as an instrument for family size, on the argument that the birth of twins is an exogenous influence on the number of children. However families that choose more births are more likely to have a twin birth, so the incidence of any twin birth is naturally correlated with family size. It is therefore necessary to create the dummy for a given parity (in this case last birth) in order to avoid reverse causality. In the regression analysis that follows the

¹⁸ The basic model is written as $\bar{q}_j = \beta(\bar{z}_j + \alpha N_j) + u_j$, where \bar{q}_j is the average 'quality' of children in household *j*, in this case the average *z*-score for height. Averaging by family abstracts from systematic birth order effects and random individual-specific effects which are interpreted as variations around the family mean. Thus we do not conflate birth order (which across individuals is correlated with family size) and family size. \bar{z}_j is average resources per child in family *j*, and *N* is the number of children. β is expected to be positive and α will be positive if there are economies of scale in the production of child quality or negative if there are diseconomies of scale. The endogeneity issue arises if there is an unobserved component of the family-specific disturbance u_i that is also correlated with \bar{z}_j or N_j .

twins themselves (who are significantly shorter) are dropped from the analysis, so that the instrument relates to other children in families that have experienced a shock to family size.

The second instrument is the average weekly income for the occupational class of the head of household (6 classes) constructed using national estimates reported by Routh (1965, p. 104). This is strongly correlated with the household's income but excludes heterogeneity at the household level. Other instruments are based on the household's location and include the share of the labour force in agriculture and the share of population urban, based on county level data from the census. These variables capture well-known income differences between rural and urban locations as well as reflecting different local health environments. In order to capture current economic conditions we also include the local unemployment rate for 1937-9 as reported by the Ministry of Labour. The first stage estimates for income per capita and the number of children are reported in the Appendix. Twins at last birth is highly significant in both equations, despite the fact that there are only 21 such families in the data, and the other variables give the expected signs. Both equations pass the conventional F-test for the relevance of the instruments (F > 10).

Table 3 reports a set of regressions of the average height z-score across families in the Boyd Orr cohort. The first two columns show the effect of per capita income alone. The OLS estimate indicates that moving up one income class increases the height of children in the family by a third of a standard deviation or about two centimetres for an 8year old. When instrumental variables are used, the coefficient increases slightly in size and remains highly significant. It is worth noting that the test for endogeneity suggests that income per capita is endogenous but the overidentification test is right on the margin of 5 percent significance, suggesting that some of the instruments may have a direct effect, rather than working only through their effect on income per capita.

The third and fourth columns test the hypothesis that the number of children in the family has an effect on health in addition to its effect on income per capita. Since per capita income is not adjusted by an equivalence scale this might induce a positive bias in the coefficient on the number of children. The coefficients in columns (3) and (4) are negative, suggesting that there is some crowding effect on height over and above that

which results from the dilution of income per capita. Note also that the IV coefficient is larger than the OLS coefficient, suggesting that the negative coefficient is not simply the result of some unobserved behavioural factor which leads both to smaller families and to healthier children. Although statistical significance is somewhat weaker in the IV estimate, the important point is that the OLS coefficients do not seem to be biased towards zero. Note also that the test for endogeneity is now only marginal but that, with the additional variable, the regression now passes the overidentification test.

It is possible that the effects of family size are driven predominantly by large families. Since the number of children in the households under study range from one to eleven it is possible to test this hypothesis. Adding the squared number of children to the regression in column 3 of Table 3 produced a coefficient (not shown) of 0.004 ('t' = 0.88), which rejects the hypothesis of a non-linear effect. An alternative variant of this test is to see if the family size effect differs depending on the birth order of the child. Table 4 provides OLS estimates of height z-scores by parity, so that the dependent variable is now an individual child rather than the average for the family. The results show that per capita income effects are remarkably stable across different birth orders. It also shows that the family size effect is always negative. Although the size and significance of the coefficient varies across parities, there is no evidence of a trend.

A number of other robustness checks were made on the regression in column (3) of Table (3). It is possible that the effect of per capita income diminishes as income increases. But adding the square of income per capita produces a positive but insignificant coefficient of 0.03 ('t' = 0.18), which does not support the hypothesis of a nonlinear effect. But since income per capita is a categorical variable this is not a very strong test. Another possibility is that the effect of poverty bites harder the more children there are. However, there is no support for this hypothesis as the interaction of income per capita and the number of children gives a negative coefficient ('t' =1.1). Finally, similar results to those in Table 3 were obtained when the dependent variable is the z-score of leg length rather than total height.¹⁹

¹⁹ If anything, the effect of family size on leg length is even greater than on total height. Previous analyses of leg length in the Boyd Orr cohort, including a link to longevity, can be found in Gunnell et al. (1998a; 1998b).

We can probe a little deeper into the proximate determinants of height by replacing income and the number of children by household food expenditure per capita and the degree of crowding in the household as measured by persons per room. Food expenditure and crowding are chosen subject to the household's overall budget constraint, which is determined by income and family size. For this reason we exclude income and the number of children when examining the effects of these more proximate determinants. It is interesting to note how closely the key elements of the budget constraint are related to the more proximate determinants of health. Regression analysis shows that food expenditure is positively related to per capita income ('t' = 31.4) and negatively related to per capita income ('t' = 6.9) and positively related to the number of children ('t' = 15.3).

The OLS result in the first column of Table 5 shows that per capita food expenditure has a positive effect on height while overcrowding has a negative effect. The latter is somewhat parallel with the results on the number of children in Table 3, and it suggests that overcrowding is one channel through which the number of children in the family negatively affects the height of the average child. Thus it is consistent with the literature that suggests that overcrowding worsens the disease environment, which in turn leads to poorer health outcomes.²⁰

The second column of Table 5 adds the index for the investigators' assessment of the cleanliness of the household. This has a positive effect as might be expected, but with only marginal effects on the other coefficients. Consistent with the disease interpretation, the result indicates that negative health effects are associated both with the degree of overcrowding and with the general lack of cleanliness of the household environment. When two dimensions of housing quality are added they add little to the explanatory power and the cleanliness index also becomes insignificant. However, when good ventilation is the only variable representing the household environment, it takes a significant positive coefficient. It seems likely that good ventilation reflects the overall quality of the house by capturing the distinction between dark, dingy tenements and more modern housing. Together the results in Table 5 suggest that the fabric of the building

²⁰ IV estimates (not reported) support the same conclusions.

may have been one of the main factors contributing to household squalor while the particular facilities that it contained played a secondary role.

Disease and socioeconomic conditions

The effects of overcrowding and cleanliness on heights provide strong circumstantial evidence that disease was one of the important mechanisms through which low income, and especially large families, negatively affected the health of children. Studies of the links between height and diseases point to the importance repeated respiratory and gastrointestinal infections. Rona and Florey (1980) found that British children with respiratory infections in the preceding year were around 0.4 standard deviations shorter. In their survey of the effects of microbial infection on human height Beard and Blaser (2002, p. 486) give the example of the bacterium *Heliobacter pylori* the transmission of which is related to the number of children living in the household.²¹ Here we provide further corroborative evidence by focusing directly on the results of clinical examinations that were reported in the Boyd Orr survey. These clinical examinations included taking a blood sample, assessing various skeletal abnormalities, as well as identifying various types of infection. All the clinical observations were made by two doctors using a common standard to minimise the degree of subjectivity involved in making comparisons across individuals.²²

It is worth stressing, however, that results are limited in two important respects. First, they do not include some of the most important illnesses that would have attenuated growth, either by reducing food intake or by limiting the ability to convert nutrients into building bone and tissue. These include recurrent conditions such as diarrhoea and dysentery, as well as major illnesses such as tuberculosis, whooping cough, scarlet fever, chickenpox and measles. Thus, while we can explore links between household conditions and disease, we do not necessarily capture those conditions that are most likely to have arrested growth. Second, the clinical observations relate largely to current conditions and

²¹ In an important historical study Burstrom et al. (1999) find that, when controlling for socioeconomic status, the number of children in Swedish families in 1885-1910 was a significant determinant of death from measles but not from bronchitis or pneumonia. It seems likely that the latter would affect growth but would not necessarily result in death.

²² For the analysis of disease incidence we take children aged from 1 to 14. Unfortunately we lose another of the locations, Aberdeen, for which the results of clinical examinations are incomplete.

abnormalities; they do not record medical histories. Since height reflects the cumulative effects of nutrition and disease, currently observed medical conditions are likely to be an imprecise indicator of the underlying disease burden.

The only measure that was recorded from the blood sample was the haemoglobin count. This is converted to a dummy equal to one if the haemoglobin count was less than 75 percent of the normal level, which was taken to be 14.8 grams per 100ml of blood. Less than 11.1 grams per 100ml is consistent with the World Health Organisation definition of anemia. This condition is associated with dietary deficiencies and in more severe cases it presents symptoms such as weakness, fatigue and shortness of breath, Using this definition, of the 2839 children examined 5.0 percent were anemic. The clinical observations also included a variety of skeletal disorders and abnormalities. Here we use the observations on frontal bossing, Harrison's sulcus and pigeon chest, which are markers of rickets, and we construct a dummy for the presence of any of these conditions. Of 3294 children examined 15.9 percent of the sample has at least one of these defects. ²³

The most important infections are respiratory infections of one kind or another. The clinical observations identified chronic upper respiratory catarrh, bronchitis and coryza, which is common cold or cold-like symptoms. Our measure of respiratory disease is the presence of any one of these conditions and it affected 19.1 percent of the 3294 children examined. Finally, the clinical examination also looked for eye and ear infections. The two eye conditions recorded are blepharitis, which is inflammation around the eye (particularly the eyelids) and conjunctivitis. The only ear condition that was recorded is otitis media, which is inflammation of the middle ear. We combine these observations into one dummy for any occurrence of eye or ear infection, which was present in 7.4 percent of the 3292 cases examined.

To what degree are these medical conditions related to the heights that were previously analysed? Table 6 shows the results of a regression of height z-scores on the four different groups of medical conditions recorded in the clinical examinations, estimated across the individuals for which both measures are available. The coefficients are all negative as would be expected but they should not be interpreted as causal. The

²³ Some clinical observations were also made of knock knees and flat feet, but these are not included here, as they are not necessarily markers of disease.

first two columns show that height is negatively associated with anemia and with skeletal defects but in neither case is the relationship statistically significant. Perhaps the latter is not surprising since these conditions do not refer to the long bones such as the leg bones which are key determinant of height. The third and fourth columns reveal significant correlations between height and the incidence of respiratory disease or eye and ear infections. While these results are consistent with a link between infection and growth, the strength of these associations is relatively low. In all likelihood this is because we do not have evidence on some of the main conditions that limit growth, and we lack systematically obtained information on the full history of infections.²⁴

In order to explore the link between household circumstances and medical conditions we present regressions relating each condition to the same explanatory variables that were used in the earlier analysis of heights. As before, in order to focus on the effects at the family level, for each disease outcome, we take the average across all the children in the household who were subject to examination. The resulting variable for the average disease or infection rate ranges between zero and one, and so we perform tobit regression with censoring at the upper and lower bounds. Because some medical conditions are age-related we also include the average age of the children in the household. A variable for the sex composition was also introduced but was never significant and hence it is excluded from the reported regressions.²⁵

Table 7 shows that for all the conditions analysed the sign of the coefficient on income per capita is negative while that on the number of children is positive. It also shows that the incidence of disease was generally negatively related to age. For anemia, the association with income per capita is weak while the association with the number of children is stronger. The reverse is true for skeletal abnormalities. Respiratory infections are more strongly related both to per capita income and to the number of children. Eye and ear infections are strongly associated with family size, but not with income per capita. These results suggest that family size was particularly important for infectious

²⁴ However, the results are consistent with other evidence—see for instance Rona and Florey (1980) who examined the correlations between respiratory symptoms over the past year (as reported by parents) and heights of British children in the early 1970s.

²⁵ These variables were not included in the earlier regressions because the z-scores for height adjust for sex and year of age.

diseases, consistent with the idea that larger families created adverse disease environments by increasing the rate of infection.

Table 8 probes a little deeper into the channels of influence by testing the associations with food expenditure, overcrowding and cleanliness. The first column indicates that anemia is strongly associated with per capita food expenditure but not with overcrowding. This makes sense as anemia is chiefly associated with dietary deficiency. Skeletal abnormalities are also inversely associated with per capita food expenditure but there is only a weak positive association with overcrowding. By contrast respiratory infections are strongly positively associated with overcrowding as well as inversely associated with food expenditure per capita. This result parallels that for the number of children in Table 7. It suggests that the spread of infection within the household was greater the more crowded the house. That finding is replicated for ear or eye infections which are also likely to have been passed between individuals in the household. The effect of cleanliness, while always giving the expected sign is only significant at the 5 percent level for anemia and for ear and eye infections.

The results reported in this section offer some support for the argument that the variables that determine heights are also associated with the disease environment in the household. Although the inferences are necessarily limited, they provide some corroborative evidence for an association between the degree of crowding and the spread of infectious disease. This in turn suggests that the negative effect of the number of children in the household on their average height that was reported in Tables 3 and 5 operates partly through the channel of recurrent infections arresting growth.

Explaining Heights 1886-1938

As mentioned earlier, the evidence indicates a steep increase in heights from the turn of the century onwards. Here we use our results on heights to 'explain' that improvement. As noted earlier, the households in the Boyd Orr cohort are more representative of living standards a generation earlier, which can be considered a strength for the purposes of backcasting. Of course we must be careful in inferring the causes of change over time based on coefficients estimated from a cross-section. If the effects of household income and family size become smaller as knowledge of nutrition and hygiene disseminates, and as the general disease environment improves, then our estimates for the late 1930s will understate the average effects for the previous half century.²⁶ Similarly our projections will be too modest if changes in family income have decreasing effects as households become richer (Steckel, 2009, p. 142), although we find no evidence of this. Nevertheless, in order not to overstate the effects over time we use the relevant OLS coefficients, which are more precisely estimated and which, if anything, are biased towards zero.

In order to trace the likely effects of changing income and family composition over time and to be consistent with the families in the Boyd Orr Cohort we focus on a standard family type—working class male-headed households with a non-working wife and at least one child present. Table 9 offers some stylised facts for this classic family type and the basis for these calculations is given in the notes to the table. The dates chosen are those of the Board of Trade or Ministry of Labour wage surveys of 1886, 1906 and 1938. The latter two dates are also close to the surveys of working class budgets undertaken in 1904 and 1938. The calculations in the fourth row of Table 9 show dramatic increases in family income per capita, which was due to the combination of increasing real earnings and decreasing family size.

Calculations of the possible effects of these changes appear in Table 10. Taking an increase of one income category in the Boyd Orr survey to be worth five shillings, the increase in per capita income between 1906 and 1938 would be equivalent to 6.7/5 =1.34. Using the coefficients in the third column of Table 3, the effect of rising income per capita increased height by $0.258 \times 1.34 = 0.35$ standard deviations. The direct effect of falling family size increased heights by (-) $0.073 \times$ (-) 1.7 = 0.12 standard deviations. The total effect of 0.35 + 0.12 = 0.47 standard deviations equates to an increase in height of 2.9cm for eight year olds and it accounts for nearly half of the increase in heights between 1906 and 1938. The effect of falling family size alone (holding wage rates constant at the 1906 level but allowing supplementary earnings to decline as family size decreases) increases height by 1.8cm. Thus about three fifths of the explained part of the

²⁶ We find no evidence of non-linearity in the effects of per capita income in our data. Adding the square of income per capita in column 3 of Table 3 gives a coefficient of 0.03 ('t' = 0.82). But since income per capita is a categorical variable this is not a very strong test.

increase in heights is due to the direct and indirect effects of the fall in the number of children, which therefore accounts for around 30 percent of the total observed increase.

Similar calculations for the period from 1886 to 1906 indicate that growth in per capita income added around 0.9 cm to the height of the average eight year-old while the separate effect of falling family size contributed 0.5 cm. The total increase of 1.4 cm would probably account for about three quarters of the increase in heights across these two decades and more than half of that effect is due to falling family size (holding wage rates constant). Although we lack precise estimates of the increase in children's heights over this period it seems likely that these variables would account for a very substantial share of it, perhaps as much as 90 percent.

Two observations flow from these calculations. First, over the half century from 1886 changes in per capita income and family size increased the height of eight-year olds by 4.2cm, with about 2.6cm, or around three fifths of the total observed increase, due to falling family size. The second point is that falling family size also contributed to the acceleration in heights, starting at zero before the 1880s, adding 0.4cm per decade from then up to 1906 and 0.6 cm per decade from 1906 to 1938. But after the turn of the century declining family size and increasing real income are only part of the story and these effects leave room for other factors such as improved knowledge of child health and nutrition as well as improvements in sanitation and housing infrastructure.²⁷

It is interesting also to consider predictions over time from the estimates that include per capita food expenditure and persons per room. These are more speculative because there is a greater element of choice over expenditure on food, housing and other goods. Here we apply the coefficients in the first column of Table 5 to the estimated changes reported in Table 9. For the classic family with children we estimate that between 1906 and 1938 the growth in food expenditure per capita added about 1.8 cm

²⁷ Although not the focus of this paper, it is worth considering what happened in the decades after 1938 when heights continued to increase despite the rebound in family size associated with the postwar baby boom. While it is tempting to suggest that the family size effect faded out, studies of the 1958 birth cohort indicate otherwise (Goldstein, 1971; Fogelman, 1975). Between 1938 and 1958 real wages grew by 49 percent, and the number of children increased by about 1.2. That would imply an increase of 1.4 cm due to per capita family income and a reduction of 0.5cm due to the separate effect of the number of children. Thus the income effect dominates but the combined effects are smaller than during the interwar period. However, the health of children and are likely to have been enhanced by a variety of welfare and medical improvements that came into effect in the early postwar period.

while the fall in persons per room contributed just 0.1 cm. About two thirds of the total effect is accounted for by the falling family size.

The predictions based on food expenditure and persons per room 'explain' rather less of the increase in heights than those based on income per capita and the number of children. This is probably because the former fails to reflect the improvement in the quality of housing, some of which is captured in the effects of cleanliness and housing conditions that feature in Table 5. How such qualitative variables might have evolved is difficult to judge. In his surveys of York Rowntree (1941, p. 225-6) found that between 1899 and 1936 the share of working class families living in slums fell from 25.5 percent to 11.3 percent, while those living in the best quality housing increased from 12.7 percent to 34.1 percent. Much of the progress in housing quality was due to the postwar building programme of York Corporation and it seems likely that such developments contributed to the remarkable improvement in the health of children in the decades following the First World War.²⁸

As noted earlier, improvement in the disease environment is reflected in the dramatic falling infant mortality. Analysing time series data for average heights of schoolchildren in British towns Hatton (2009) finds that the local infant mortality rate had a negative effect on subsequent heights of children. On these results, the fall in infant mortality between 1901-5 and 1941-5 accounts for an increase in heights of about 0.6cm per decade, or a little over a quarter of the total increase in the heights of 6-9 year-olds. These effects cannot simply be added to those reported in Table 10, as some of the decline in infant mortality would ultimately be due to increasing family incomes and falling family size. Nevertheless the results do underline the fact that improvements both in nutrition and in the disease environment contributed to the trend increase in the heights of children.

Conclusion

Using a unique survey of working class families for the late 1930s we have found that the heights of children are positively related to income per capita and negatively

²⁸ According to the coefficients in the last column of Table 5, increasing housing quality, as measured by ventilation, by one class, e.g. from 'moderate' to 'good' would increase average height by about 1 cm. This is equivalent to the effect of increasing food expenditure per capita by about 1s 2d a week.

related to the number of children in the family. These effects can be further decomposed into those factors that are associated with nutrition and those that are associated with disease, both of which affect growth during childhood. While nutrition is mainly associated with per capita expenditure on food, the spread of infectious disease within the household was exacerbated by overcrowding and poor quality housing. Our examination of medical conditions suggests that infectious diseases were closely associated with the degree of crowding within the household. Thus the number of siblings in the family negatively affected the health of children, both through reducing per capita food expenditure and by worsening the disease environment.

These findings suggest an answer to the puzzle of why the health of children (and their subsequent outcomes as adults) improved so dramatically in the first half of the twentieth century. All the evidence points to a distinct acceleration in the rate of increase in the heights of children around age 8-10, from less than 1cm per decade in the late nineteenth century to more than 2cm per decade in the first half of the twentieth century. In the late nineteenth century rising incomes brought better nutrition, with consequent improvements in health and height. But the external disease environment improved only slowly as the effect of public health improvements were offset by growing urbanization and increasing population density. This is reflected in the fact that while child mortality rates fell as a result of improved nutrition, the infant mortality rate remained obstinately high.

In the first half of the twentieth century real wages increased no faster on average than they had in the previous half century. Yet the dramatic fall in fertility meant that, for households with children, income per capita increased more strongly and poverty rates declined more rapidly than they would have otherwise. It also meant an improvement in the disease environment within the household as the degree of crowding decreased. More rapid improvements in urban infrastructure, in housing quality, and in practical knowledge of hygiene probably explain most of the improvement in child health that is not accounted for by rising family income and falling family size. But improvements in medical technology contributed little, because the discovery or diffusion of most of the new medical treatments came too late to confer much benefit on working class families until after the Second World War. Taken together, these trends suggest that unless we recognise the effect of falling family size it is difficult to fully account for the sharp improvements in child health in the half century before the advent of the National Health Service.

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1	Army Recruit	S	Christ's Hospital Boys			
	Age 18	Age 20		Age 10	Age 12	Age 14
1860-64	166.1	167.1	1870s	130.1	137.8	146.4
1880-84	164.3	166.1	1906-18	132.7	140.4	149.9
1885-89	162.3	165.9	1919-22	132.4	142.4	152.0
1890-94	162.8	166.9	1923-26	135.4	143.5	154.0
1895-99	163.1	167.1	1927-30	135.7	143.6	154.7
1900-04	163.8	166.9	1930-33	136.0	145.3	155.6
1905-09	164.3	168.7				
1910-13	164.8	168.1				
		School Ch	ildren in Brit	ish Towns		
	Boys 5	Boys 8	Boys 12	Girls 5	Girls 8	Girls 12
1908-11	101.4	117.4	135.0	101.0	114.9	135.2
1912-15	101.9	118.3	134.9	101.8	117.2	135.5
1916-19	102.2	119.2	135.6	102.2	117.3	135.4
1920-23	103.1	119.7	135.3	102.5	118.8	135.9
1924-27	104.0	121.0	136.4	103.4	119.6	137.3
1928-31	104.1	121.8	137.9	104.2	120.5	138.6
1932-35	104.8	123.1	139.0	104.7	121.3	139.8
1936-39	105.7	123.7	139.6	105.5	122.4	141.1

Table 1Heights of Army Recruits and School Children, 1860-1939

Notes: Heights of Army Recruits (adjusted for truncation) from Rosenbaum (1988) Table 1 p. 282; heights of boys at Christ's Hospital from Rosenbaum (1988), Table 8 p. 288. Heights of school children in British Towns calculated from data collected by Bernard Harris and supplied by the AHDS at the University of Essex. The underlying data is an unbalanced panel of average heights by age and by town. The figure calculated for each year, is based on the coefficients on the year dummies in regressions for each age and sex, which include town fixed effects. The regressions also include an adjustment for the difference between the year of age and the estimated average age of the children in each observation (i.e. if the average age of eight year olds in a given observation is 8.5 then the variable takes the value 0.5). The omitted category (the intercept) is Aberdeen in 1908.

Table 2	
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	By individual	By household
No of cases	2946	1131
Individual characteristics		
Average age	7.92	7.85
Percent female	52.7	53.9
Household characteristics		
Number of children in family	4.56	3.74
Family size	6.75	5.96
Family income per capita < 10s	71.56	59.8
Food expenditure per capita < 5s	54.75	43.50
Persons per room > 2	43.76	42.01
Clean house	32.26	33.11
Flush toilet inside	35.40	37.00
Good ventilation	70.56	73.00

Characteristics of Children who were Measured in the Boyd Orr Survey

Note: due to missing data some of the means are calculated on a smaller number of observations as follows (individuals, households): income per capita (2911, 1112), persons per room (2605, 984), clean house (2734, 1045), flush toilet (2099, 786) good ventilation (2707, 1037).

Table 3

Effect of Per-capita Income and Number of Children on Height

	OLS	IV	OLS	IV
Constant	-0.41**	-0.56**	-0.03	0.16
	(8.1)	(6.3)	(0.3)	(0.4)
Income per capita	0.32**	0.42**	0.26**	0.29**
	(11.7)	(8.2)	(8.5)	(3.8)
No. of children in			-0.07**	-0.14*
family			(5.0)	(2.0)
\mathbb{R}^2	0.13	0.13	0.14	0.13
F	135.8	66.4	80.4	34.8
Endogeneity		0.03		0.05
Overidentification		0.05		0.11
No. Families	1102	1102	1102	1102

Note: 't' statistics in parentheses computed from robust standard errors. ** = significant at 1%; * = significant at 5%%; $\dagger =$ significant at 10%.. The number of observations is reduced from that in Table 1 because of the exclusion of twins and missing data for income class. Endogeneity and overidentification are the p-values for the Hausman and Hanson J tests respectively.

Table 4

	Birth order 1	Birth order 2	Birth order 3	Birth order 4
Constant	-0.11	0.05	-0.26	0.06
	(0.2)	(0.4)	(1.5)	(0.3)
Income per capita	0.26**	0.28**	0.29**	0.26**
	(6.0)	(6.7)	(5.3)	(3.0)
No. of children in	-0.05*	-0.10**	-0.05†	-0.08**
family	(2.0)	(4.3)	(1.8)	(2.7)
\mathbb{R}^2	0.09	0.13	0.07	0.05
F	36.2	52.0	20.2	8.8
No. Children	738	729	577	378

Effect of Per-capita Income and Number of Children on Height by Birth Order

Note: 't' statistics in parentheses. ** = significant at 1%; * = significant at 5%; $\dagger =$ significant at 10%. Twins are excluded.

Table 5

Effect of Per-capita Food Expenditure and Housing Conditions on Height

	OLS	OLS	OLS	OLS
Constant	-0.24**	-0.55**	0.63**	-0.68**
	(2.6)	(3.9)	(2.9)	(4.1)
Per capita food	0.27**	0.27**	0.27**	0.26**
expenditure	(8.9)	(2.9)	(7.8)	(8.6)
Overcrowding	-0.09**	-0.07**	-0.08**	-0.06*
(persons per room)	(3.2)	(2.9)	(2.6)	(2.2)
Cleanliness index		0.13**	0.04	
		(2.8)	(0.7)	
Flush toilet inside			0.003	
			(0.1)	
Good ventilation			0.10	0.16**
			(1.6)	(3.1)
R^2	0.14	0.14	0.16	0.14
F	64.1	45.7	24.8	45.2
No. Families	978	953	737	946

Note: 't' statistics in parentheses computed from robust standard errors. ** = significant at 1%; * = significant at 5%; $\dagger =$ significant at 10%. The number of observations is reduced from that in Table 1 because of the exclusion of twins and missing data for expenditure class and persons per room.

Table 6

Relationship between Medical Conditions and Height

	Anemia	Skeletal	Respiratory	Ear or eye
		abnormality	infection	infection
Constant	-0.001	0.004	0.020	0.012
	(0.04)	(0.20)	(1.01)	(0.66)
Medical condition	-0.188†	-0.037	-0.119	-0.199
	(1.79)	(0.74)	(2.55)	(2.86)
\mathbb{R}^2	0.001	0.000	0.002	0.003
No of obs.	2600	2912	2912	2910

Note: Regressions on individuals aged 2-14. 't' statistics in parentheses. ** = significant at 1%; * = significant at 5%; $\dagger =$ significant at 10%.

Table 7

Effect of Per-capita Income and Number of Children on Disease Incidence

	Anemia	Skeletal	Respiratory	Ear or eye
		abnormality	infection	infection
Constant	-0.15	-0.18	0.20†	-1.03**
	(0.9)	(1.5)	(1.7)	(7.0)
Income per capita	-0.10†	-0.08*	-0.23**	-0.03
	(1.7)	(2.2)	(5.6)	(0.7)
Number of children	0.07**	0.03†	0.05**	0.10**
	(2.8)	(1.9)	(3.3)	(5.5)
Average age	-0.14**	-0.02*	-0.05**	-0.002
	(6.6)	(2.1)	(4.6)	(0.2)
Pseudo-R ²	0.10	0.01	0.06	0.05
Likelihood ratio $\chi^2_{(3)}$	81.9	21.7	106.2	52.5
No. Families	1058	1134	1134	1134
Uncensored cases	112	310	323	183

Note: Tobit regressions of family average disease incidence, for children aged 1-14. 't' statistics in parentheses. ** = significant at 1%; * = significant at 5%; $\dagger =$ significant at 10%.

Table 8

	Anemia	Skeletal	Respiratory	Ear or eye
		abnormality	infection	infection
Constant	0.39	-0.04	0.04	-0.45*
	(1.6)	(0.19)	(0.2)	(2.5)
Per capita food	-0.15**	-0.10**	-0.08*	-0.11**
expenditure	(2.7)	(2.8)	(2.1)	(2.9)
Overcrowding	0.02	0.05	0.17**	0.09**
(persons per room)	(0.6)	(1.6)	(5.4)	(2.8)
Cleanliness index	-0.16*	-0.06	-0.10**	-0.14*
	(2.1)	(1.1)	(1.8)	(2.4)
Average age	-0.11**	-0.01	-0.033	0.02
	(5.9)	(1.0)	(2.92)	(1.6)
Pseudo-R ²	0.10	0.02	0.048	0.04
Likelihood ratio χ^2 (3)	69.1	23.8	75.91	41.8
No. Families	923	990	990	990
Uncensored cases	100	269	283	165

Effect of Per-capita Food Expenditure, Overcrowding and Cleanliness on Disease Incidence

Note: Tobit regressions of family average disease incidence, for children aged 1-14. 't' statistics in parentheses. ** = significant at 1%; * = significant at 5%; $\dagger =$ significant at 10%. The number of observations is reduced from that in Table 7 because of missing data on expenditure class and persons per room.

	1886	1906	1938	Change	Change
				1886-1906	1906-38
No of children	4.6	3.5	1.8	-1.1	-1.7
No in Family	6.6	5.5	3.8	-1.1	-1.7
Family income (1938 shillings)	56.0	62.5	68.6	6.5	6.1
Per capita income (shillings)	8.5	11.4	18.1	2.9	6.7
Food expenditure (shillings)		37.5	34.3		-3.2
Per capita food expenditure		6.8	9.0		2.2
Overcrowding: persons per room		1.1	0.9		-0.2

Table 9Trends in Working Class Families, 1886-1938

Notes: The standard working class family is taken to be a male breadwinner household with a non-working wife and children under age 18. The data in Table 8 were derived for this standard family in England and Wales as follows:

Number of children per family. The 1904 survey of working class budgets, which covered 'normal' families with a male breadwinner and at least one child, records an average of 3.6 children of all ages (Bowley, 1941, p. 129); an estimate from the 1901 census suggests a figure of 3.4 children (Gazeley and Newell, 2007, p. 12). We take a figure of 3.5 children for 1906. From the 1938 budget survey a figure of 1.8 children can be inferred (Rowett Institute, 1955, p. 24; Prais and Houthakker, 1955, p. 187-8). The figures for 1906 and 1938 are fairly close to the trend in the total period fertility rate: 4.58 in 1881-5, 3.46 in 1901-5 and 1.78 in 1931-5 (Figure 3). We therefore adopt a figure for 1886 of 4.6 children. It should be noted that completed family size for working class families with children would be higher than these figures.

Earners and earnings per family. Weekly earnings for adult males, based on the periodic wage enquiries, were taken from Bowley (1937, p. 49): 24s in 1886, 29s in 1906 and 60s in 1938. The 1904 survey of working class budgets recorded average weekly family income of 36s 10d, or 27 percent more than the adult male wage. Assuming that 20 percent of children present were aged 14 -17, the average number of supplementary wage earners in 1906 would be 3.5*0.2 = 0.7, this implies that one supplementary earner would have earned about 0.27/0.7 of adult male earnings or about 40 percent. For 1938 the 1.8*0.2 = 0.36 supplementary earners would have contributed 14.4 percent, a figure that is consistent with Rowntree's (1941, p. 129) finding that supplementary earners added 14.3 percent. For 1886 the same calculation gives 4.6*0.2 = 0.92 supplementary earners contributing 37 percent over adult male earnings. These estimated family incomes were converted to 1938 prices using the cost of living index (Feinstein, 1995, p. 264-5). *Expenditure on food*. The working class families surveyed in 1904 spent 60 percent of their income on food (Bowley 1941, p. 131). In the 1938 survey only 40 percent was spent on food but this included a wider range of household types (in particular households without children). The figure would have been higher for the standard family and so we estimate a food share for 1938 at 50 percent.

Persons per room. In 1931 the average number of persons per room was 0.85 for a family of 4, 1.03 for a family of 5, and 1.20 for a family of 6. Since families with children experienced somewhat greater crowding we assume a figure of 1.0 for 1938. Unfortunately we do not have comparable figures for 30 years earlier. However, the average number of persons per room in four-roomed dwellings across all family types fell from 1.12 per room in 1901 to 0.95 in 1931. Allowing for changes in family size it seems reasonable to assume a decrease in crowding of about 0.2 persons per room over the 30 year period.

Table 10Effects of Family Income and Family Size on Heights, 1886-1938

	Change 1	Change 1886-1906		Change 1906-38	
Change in:	SD	cm	SD	cm	
Family income per capita	0.15	0.9	0.35	2.1	
No in Family	0.08	0.5	0.12	0.7	
Total	0.23	1.4	0.47	2.9	
Of which due to family size	0.17	1.1	0.30	1.8	
Change in:			SD	cm	
Per capita food expenditure			0.30	1.8	
Overcrowding: persons per room			0.02	0.1	
Total			0.32	1.9	

Source: Based on data in Table 5 and coefficients in col. (3) of Table 3 and col. (1) in Table 5. The counterfactuals are calculated by holding family size constant at the beginning period value and adjusting the change in income to allow for the contribution of older children.

APPENDIX TABLE

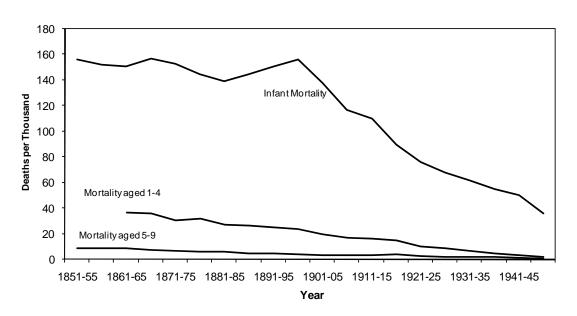
	Income per	No. of
	capita	children
Constant	0.75	4.03**
	(1.2)	(11.4)
Twins last birth	-0.42**	1.40**
	(3.4)	(4.3)
Occupational income	0.02**	-0.01**
	(15.7)	(5.9)
Share of labor force in	-0.70**	-0.99*
agriculture	(2.9)	(2.0)
Share of population	-0.26†	0.49
urban	(1.8)	(1.6)
Local unemployment	-0.01	0.04**
rate	(1.2)	(2.9)
\mathbb{R}^2	0.27	0.08
F	59.0	22.5
No. of observations	1102	1102

First Stage Estimates for Table 3 Col. 4

Note: t- statistics in parentheses. As none of the instruments appear in the second stage, the R^2 reported here is the partial R^2 for excluded instruments and the F-statistic is the test for excluded instruments.

Sources for instruments: Occupational income is constructed by allocating a value to each of the six social classes recorded for the households in the Boyd Orr survey on the basis of the occupational class of the head of household. A weekly income for each class was calculated from the data reported by Routh (1965) p. 104. This in turn was derived from the Ministry of Labour's earnings survey for 1935/6. Other variables are based on the locality. The share of the labour force in agriculture and the share of the population urban are measured at the county level, taken from the 1931 census. The unemployment rate is the average unemployment rate of adult males for 1937-9, based on the Ministry of Labour's local unemployment index. These are for employment exchange areas and are reported in the *Ministry of Labour Gazette*. Infant mortality rates (per thousand live births) for 1937-9 are taken from the *Statistical Reviews* of the Registrar General for England and Wales and the Registrar General for Scotland. These are measured either for the borough or for the relevant rural district.

Figure 1: Infant and Child Mortality Rates: England and Wales, 1851-1950



Source: Registrar General (2004), Table 4, p. 5.

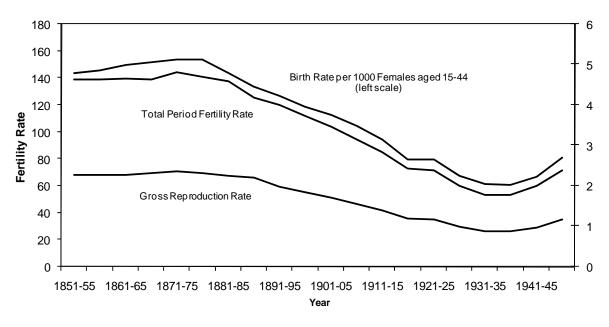


Figure 2 Fertility Rates, England and Wales 1851-1950

Source: OPCS (1987), Table 1.4, p. 26.

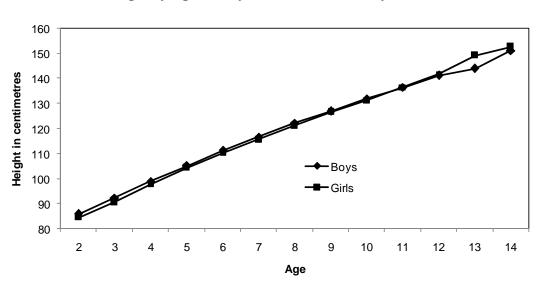


Figure 3 Height by Age of Boys and Girls in the Boyd Orr Cohort