Social Class, Infant Development And Adult Disease Mortality.

Since at least the beginning of the twentieth century there has been an interest in the link between infant development and adult disease mortality. A number of clinical studies in the 1940s and 1950s indicated a relationship between infant health and mortality from a variety of adult diseases, including coronary heart disease, stroke and stomach cancer. Most of these of these studies were flawed by poor design methodologies, but in 1964 Geoffrey Rose published the results of carefully designed research which indicated that the mortality patterns amongst families of heart disease patients were significantly higher than amongst the families of control patients suffering from other diseases.[1] The most important findings were as follows:

Table 1: Mortality Amongst Parents And Siblings Of Male Heart Disease Patients And Controls[2]

	Heart Disease Patients	*Controls*
No. Of Families	65	65
No. Of Fathers Dead Before Age 45	11	3
No. Of Mothers Dead Before Age 45	8	2
No. Of Siblings Born	319	367
Percentage Dying In First Year	22%	10%
Percentage Dying After One Year	28%	21%

The families of heart disease patients suffered both from higher parental mortality and infant & child mortality, but death in the first year of life was particular high amongst siblings.

Rose's paper had little influence on subsequent research but the link between infant mortality and heart disease was confirmed by an ecological study of Norway carried out by Forsdahl.[3] He found a significant correlation between county infant mortality rates and mortality from arteriosclerotic heart disease in people aged between 40 and 69 years, and put forward the hypothesis that 'great poverty in childhood and adolescence followed by prosperity, is a risk factor for arteriosclerotic heart disease.'[4] Although the emphasis on adult prosperity has not been widely accepted,[5] the link between infant mortality and heart disease has been supported by subsequent research. For example, Williams et. al. established a correlation between infant mortality rates during 1885-1948 and adult ischaemic heart disease mortality for the counties of England & Wales in 1969-73 .[6]

The link between infant mortality and heart disease mortality was partially confirmed by Buck and Simpson in 1982. They found that in the 1917-21 birth cohorts of 17 United States Registration States, infant mortality from was significantly related to arteriosclerotic heart disease at ages 40-44 and 50-54 in both sexes, after controlling for contemporary infant mortality.[7] However, there was no association between infant mortality and heart disease for a later birth cohort 1927-31, which led them to re-analyse their data. They discovered that it was infant mortality due to diarrhoea and enteritis that was mainly responsible for the link between infant mortality and heart disease in the 1917-21 cohort. Diarrhoea and enteritis largely disappeared between 1917-21 and 1927-31, which Buck and Simpson believed was the reason for a lack of correlation between infant mortality and heart disease mortality in this later period.[8] This finding illustrates the importance of historical change in shaping patterns of infant and adult disease mortality.

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[1] Geoffrey Rose, 'Familial patterns in ischaemic heart disease', *British Journal Of Preventative Social Medicine*, 18 (1964), 75-80.

[2] *Ibid*, p.76.

[3] A. Forsdahl, 'Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease?', *British Journal Of Preventative And Social Medicine*, 31 (1977), 91-95.

[4] *Ibid*, p. 91.

[5] George Davey Smith, 'Adult factors contributing to socio-economic differentials in cardiovascular disease', in Dinah Kuh and Yoav Ben-Shlomo (Eds), *A Lifecourse Approach To Chronic Disease Epidemiology* (Oxford 1997), p. 246.

[6] D.R.R. Williams, Sarah J. Roberts, and T.W. Davies, 'Deaths from ischaemic heart disease and infant mortality in England & Wales', *Journal Of Epidemiology And Community Health*, 33 (1979), 199-202.

[7] Carol Buck and Helen Simpson, 'Infant diarrhoea and subsequent mortality from heart disease and cancer', *Journal Of Epidemiology And Community Health*, 36 (1982), 27-30.

[8] *Ibid*, p.27.

In 1986, Barker and Osmond produced evidence confirming the association between infant mortality and ischaemic heart disease in local authority areas in England & Wales.[9] They found a correlation between both neo-natal and post neo-natal mortality in 1921-25 and heart disease mortality in 1968-78, with strong associations between all infant causes of death and heart disease. They also established correlations between infant and other adult disease mortality: bronchitis, stomach cancer, stroke and lung cancer. Barker and colleagues concluded that 'poor nutrition in early life increases susceptibility to the effects of an affluent diet' for the development of heart disease.[10]

Leon and Davey Smith in a recent study confirmed the link between infant mortality in 1921-23 and adult disease mortality in 1991-93 for twenty-seven countries.[11] Controlling for current levels of infant mortality, they found significant correlations between infant mortality and mortality from respiratory tuberculosis, stomach cancer and stroke, but only weak or non-existence correlations between infant mortality and lung cancer and coronary heart disease.[12] This lack of a correlation between infant and coronary heart disease mortality may be due to the historical shift in patterns of infant mortality discussed by Buck and Simpson. Leon and Davey Smith speculate that infections associated with high infant mortality - such as *H pylori* - might be responsible for the higher rates of mortality from respiratory tuberculosis, stomach cancer and stroke.

Bengtsson and Lindstrom have recently produced evidence on the link between infant mortality and a range of adult diseases in a number of parishes in southern Sweden during the period 1760-1894. The authors found no correlation between maternal or infant nutrition and adult disease, but did find a strong association between infant and adult mortality, particularly with respect to airborne infectious diseases.[13] Infectious diseases were more important in the nineteenth than the twentieth century, and most of the work on the link between infant mortality and coronary heart disease has been for the twentieth century. It is probable that the 'epidemiological transition' led to the replacement of infectious diseases by degenerative ones in shaping patterns of mortality during the twentieth century.[14]

Forsdahl, Barker and some other epidemiologists have assumed that infant mortality, birthweight and weight at one year are primarily shaped by infant and maternal nutrition. The basic assumption is that poverty has influenced foetal and infant health and development through inadequate nutrition. Recently for example, Dorling and colleagues have argued that the pattern of poverty mapped by Booth in his classic study of London at the end of the nineteenth century was reflected in subsequent adult mortality from the middle of the twentieth century onwards.[15]

Research at the Open University however on infant mortality has found that there was little or no correlation between poverty and infant mortality in England before the twentieth century. A study of infant mortality in Fulham, one of the areas covered by Booth, found the following pattern of mortality, based on the poverty colour-coding of the streets in which children were born.

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[9]D.J.P. Barker and C. Osmond, 'Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales', *The Lancet*, (May 10, 1986), 1077-1081.

[10] *Ibid*, p. 1077.

[11] David A. Leon and George Davey Smith, 'Infant mortality, stomach cancer, stroke, and coronary heart disease: Ecological Analysis', *British Medical Journal*, Volume 320 (24 June 2000), 1705-1706.

[12] *Ibid*, p.1705.

[13] Tommy Bengtsson and Martin Lindstrom, 'Childhood misery and disease in later life: the effects on mortality in old age of hazards experienced in early life, Southern Sweden, 1760-1894.' *Population Studies*, 54 (2000), 263-277.

[14] A.S. Osram, 'The epidemiologic transition theory. A preliminary update', *Journal Of Tropical Pediatrics*, 29 (1983), pp. 305-316..

[15] Danny Dorling, Richard Mitchell, Mary Shaw, Scott Orfod and George Davey Smith, 'The ghost of Christmas past: health effects of poverty in London in 1896 and 1991', *British Medical Journal*, Volume 321 (December 2000), pp. 1547-1551. It should be pointed out however, that George Davy Smith and colleagues have emphasised a life-course approach to the development of adult disease, which looks at poverty and social class across a range of age groups.

Table 2: Infant Mortality in Fulham, 1876-1888.[16]

Colour Code of Birthplace Street	*Number of Births*	*Number of Infant Deaths*	*IMR*
Light Blue/ Dark, Blue/ Black	651	100	154
Light Blue/Purple, Purple + Purple/ Pink	865	133	154
Pink + Purple/ Pink/ Red	829	124	150
Pink/Red + Red + Red/ Orange	385	53	138

There was little or no difference in the first three colour-coded streets and a slight reduction in mortality amongst the fourth middle class colour classification. Provisional research also indicates little or no social class/ infant mortality gradient was found during the period 1871-1885 in the registration districts of Fulham, Ipswich, Bungay, Felixstowe, Loughborough and Hollingbourne.[17]

This lack of a social class gradient in infant mortality in the 1870s requires special comment. A clue was provided by Newsholme in a special report on infant mortality at the beginning of the twentieth century: "probably 80 per cent of the mothers of infants in wage-earning classes suckle their infants partially or entirely ... [whereas] the proportion of mothers in the well-to-do classes who are able or willing to continue to give their infants this immenely important start in life is believed, I think rightly to be much smaller. There must be some reasons of great potency, enabling infants of the well-to-do to survive in much higher proportions to the end of the first year of life, notwithstanding the heavy handicap against them."[18]

In a special cohort study of infant mortality in Ipswich, it was found that mortality was much higher amongst the professional classes in the first two months of life than it was amongst labourers' families - 41 per 1000 as against 24 per 1000 births respectively.[19] There was no mortality gradient by the age of four months, suggesting that any disadvantage amongst professional families due to lack of breastfeeding was compensated at a later age by superior nutrition and hygiene.

Early evidence suggests that the social class gradient for infant mortality increased sharply between the 1870s and 1920s. For example, a special study of cohort mortality in Ipswich matching labourers with professionals for the period 1871-1895 revealed the following pattern:

Table 3: Cohort Analysis Of Mortality By Age Four Months In a Matched Sample of Professionals and Labourers, Ipswich 1871-1895. (Mortality Rate Per 1000 At Risk, Numbers At Risk In Brackets)[20]

Period	*Labourers*	*Professionals*
1871-78	50.9 (179)	50.4 (177)
1884-95	100.5 (155)	41.1 (174)

16 The figures are based on births and infant deaths in the years 1876, 1877, 1881, 1882 1887 and 1888. I am grateful to Sue Smith for allowing me to quote these figures from her post-graduate research at the Open University.

[17]These studies were carried out on vaccination birth and infant death registers lodged in local record offices. These registers were copies of the civil registers made for purposes of compulsory vaccination. See Michael Drake and Peter Razzell, *The Decline Of Infant Mortality In England And Wales 1871-1948: A Medical Conundrum* (Interim Report to the Wellcome Trust, available from the Faculty of Social Science, The Open University).

[18] There is some independent evidence to confirm Newsholme's statement that the middle classes breastfed much less frequently than the working classes at the end of the nineteenth and beginning of the twentieth century. See Valerie Fildes, 'Infant feeding practices and infant mortality in England, 1900-1919', *Continuity And Change,* 13 (1998), p. 258; Valerie Fildes,'Breastfeeding in London, 1905-19', *Journal Of Biosocial Science*, 24 (1992),.p. 59.

[19] The numbers of children at risk in the two groups were 236 amongst professional families, and 240 amongst labourers' families.

[20] These figures are based on the analysis of vaccination birth registers deposited in the Ipswich Record Office which are copies of the civil birth registers, with additional information on the dates of vaccination and death before vaccination. Cohort mortality is calculated on the number of births and deaths before vaccination.

These figures indicate that infant mortality doubled amongst labourers between 1871-78 and 1884-95, a period when when early infant mortality was falling slightly amongst professional families. The social class gradient of overall infant mortality further sharpened between 1887 and 1911, as indicated by the following figures for occupational mortality in Ipswich:

Table 4: Infant Mortality Rates By Occupation Group In Ipswich, 1887-1911. (Number Of Births In brackets)[21]

Period	*Labourers*	*Carpenters*	*Clerks &Professionals*
1887-1895	175 (2404)	144 (694)	106 (743))
1896-1905	185 (3366)	148 (722)	105 (882)
1906-1911	144 (2111)	105 (343)	52 (533)

This increasing social class gradient is reflected in the national figures of infant mortality derived retrospectively from the 1911 Fertility Census and subsequent Registrar-General's figures. Haines has published this material by occupational group as follows:

Table 5: Infant Mortality Rates by Occupation England & Wales, 1896-1950.[22]

	Infant Mortality Rate							
	1896	1899	1905	1911	1921	1930-32	1939	1949-50
Father's Occupation								
Professional	101.5	100.6	76.4	55.1	38.4	32.7	26.8	18.4
Farmers	97.3	97.1	80.2	74.2	51.2	46.3	40.5	24.3
Teachers	104.7	106.0	80.0	57.5	41.6	37.1	28.9	16.6
Clerical	124.3	119.1	95.9	81.2	51.2	42.0	35.1	21.8
Miners	178.8	183.7	158.8	160.0	104.7	81.9	65.5	43.4
Farm Workers	115.5	116.9	101.4	95.9	67.6	58.2	47.2	29.9
Building Labourers	177.0	180.9	149.5	138.5	93.0	64.8	58.0	38.1

Infant mortality nearly halved amongst professionals and teachers between 1896 and 1911, whereas the fall in mortality amongst miners, farm workers and building labourers was only of the order of 10 to 20 per cent. The social class gradient sharpened significantly during this period, confirming the trend revealed by the figures for the local communities quoted above. Although infant mortality continued to fall amongst all groups during the first half of the twentieth century, there was still a two to one gradient in 1949-50. Infant mortality fell to a very low level after that date with most deaths occurring in the first month of life for mainly "endogenous" biological reasons, but in the 1970s children born to unskilled parents were four times more likely to die between one month and one year than those born to professional families.[23]

Given the link between infant and adult disease mortality, it is possible that the changes in the social class gradient of infant mortality were reflected in later mortality rates for heart and other adult disease mortality. The social class gradient in male heart disease mortality in England & Wales was as follows:

[21] Mortality rates are calculated be expressing the number of infant deaths in the first year of life as a ratio of the number of births in each occupational group. The raw data was collected from copies of the civil birth and death registers lodged in the Ipswich Record Office.

[22] Michael R. Haines, 'Socio-economic differentials in infant and child mortality during mortality decline England & Wales, 1890-1911', *Population Studies*, 49 (1995), p.313.

23 Power, Manor and Fox, *Health And Class*, p.1.

Table 7: Heart Disease Mortality Amongst Males By Social Class: Standardised Mortality Ratios[24]

Period	*Social Class I*	*Social Class V*
1921	92	105
1931	98	109
1951	123	101
1961	94	116
1971	86	115
1981	69	141

The gradient continued to widen in the 1980s, but heart disease mortality began to fall amongst Social Class V between 1979-83 and 1991-93.[25] Much of the change in the social class gradient in heart disease mortality is probably due to variations in adult life style risk factors, including diet, smoking and physical exercise. However, as the *Health of the Nation* Working Group found, risk factors such as smoking, drinking and diet only explain "around a third of the socio-economic gradient in coronary heart disease mortality and morbidity".[26]

Considering the figures in Table 7, there is also the difficulty that definitions of heart disease mortality have changed over time, and were probably associated with changing medical assumptions about the relationship between social class and heart disease.[27] Given uncertainty about the reliability of the figures, it is impossible to reach firm conclusions about the timing of changes in heart disease and other forms of adult disease mortality. In general terms, it appears that the social class gradient for heart disease mortality increased significantly from about 1951 onwards, fifty years or so after the gradient in infant mortality began to establish itself. Whether this link is a causal one must await further research, and the reasons for the association must at this stage be largely speculative.

The work of Buck and Simpson linking death from diarrhoea in infancy with later mortality from heart disease may provide a clue to explaining the link. The middle classes appear to have become aware at the end of the nineteenth century of the important role of breastfeeding and good personal and domestic hygiene in preventing infant death, particularly from diarrhoea. The fall in infant mortality amongst middle class families was particularly strong with respect to diarrhoea occurring within the first two months of life,[28] and this may be a central key to explaining the association between infant mortality and coronary heart disease. Barker and colleagues have produced evidence to show that "mortality from coronary heart disease has similar but separate trends with neonatal and postneonatal mortality."[29] Reductions in neo-natal mortality among the middle class could therefore be important in explaining subsequent declines in mortality from heart disease in this social group, but only detailed cohort studies of individual families will enable clarification of these complex issues on the relationship between social class, early infant development and subsequent adult disease mortality.

The above data indicates that infant mortality fell initially and most significantly amongst the middle classes, suggesting that the elimination of poverty and increases in per capita consumption of food were not the primary reasons for the reduction of infant mortality. There is some confirmation of this conclusion in McGonigle's classic study of poverty and mortality in Stockton-on-Tees during the late 1920s and early 1930s. This research focused on the effects of re-housing nearly a half of a local slum population, ninety per cent of which were unemployed. These families were relocated to new council housing with modern sanitary facilities, yet overall mortality increased by about fifty per cent amongst this rehoused population, whereas mortality declined slightly amongst the group left in the slum area. McGonigle attributed this paradoxical finding to the growth of poverty and a decline in the consumption of food amongst the rehoused population resulting from a large increase in rent paid for new housing. However, infant mortality fell significantly amongst the rehoused population whereas the reduction was less

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[24] George Davey Smith, 'Adult factors contributing to socio-economic differentials in cardiovascular disease', in Dinah Kuh and Yoav Ben-Shlomo (Eds), *A Lifecourse Approach To Chronic Disease Epidemiology* (Oxford 1997), p. 251.

[25] Frances Drever and Margaret Whitehead (Eds), *Health Inequalities* (HMSO, London 1997), p. 106.

[26] *Ibid*, p.199.

[27] *Ibid*, pp. 248-250.

[28] This appears to have been the case in Ipswich, but has yet to be confirmed in other studies.

[29] D.J.P. Barker, *Mothers, Babies And Disease In Later Life* (BMJ 1994),p. 8. There were probably other links between social class, infant mortality - such as that due to *Heliocabacter pylori* infection - and adult disease mortality as evidenced in the work of Davey Smith, Leon and others, but these links are likely to be highly complex, and involve a number of additional variables.

significant amongst those left in the slum area, suggesting that the fall in infant mortality was not the result of better nutrition but was due to improved domestic hygiene associated with new bathrooms, running hot water and other improved facilities.[30]

There is a range of evidence that housing conditions shape levels of infection and infant mortality. Systematic reviews of evidence in third world countries show that better water and sanitation is associated with decreased diarrheal morbidity, improved nutritional status and lower childhood mortality.[31] A study of appendicitis in Anglesey concluded that the provision of hot water systems and fixed baths were important in reducing the incidence of the disease.[32] In England, poor quality housing has been found to be linked to respiratory illnesses,[33] and damp housing to general ill-health amongst adults.[34] There is also evidence that housing conditions are associated with levels of childhood infection which influences the later development of adult disease. Infection with *Helicobacter pylori* is linked to poor housing conditions, overcrowding, and sharing a bed with parents,[35] and the lack of private indoor tapped water supply in childhood was found to be associated with increased mortality from coronary heart disease in later life.[36] An ecological study of found a correlation between overcrowding levels in 1936 and mortality from stomach cancer in 1968-78, but this finding was not confirmed in a cohort study linking housing conditions to later mortaluity levels.[37] Recent unpublished research by the MRC Environmental Epidemiology Unit at Southampton University on Finnish population and medical registers has found that failure of growth in height and weight between birth and one year, in association with domestic overcrowding, is linked with the long-term development of coronary heart disease.[38]

Barker and colleagues have produced a series of seminal papers establishing an association between foetal/ infant development, as measured by birthweight and weight at one year, and a range of adult diseases.[39] This research was based initially on the Hertfordshire Health Visitors' Register, and the pattern of correlation between birthweight/ weight at one year and a number of these adult diseases has been confirmed by a number of studies for other areas and in other countries.[40] It has been widely assumed that birthweight and weight at one year are the result of maternal and infant nutrition, both before birth and during the first year of life. The role of maternal nutrition in

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[30] G.C.M. M'Gonigle and J. Kirby, *Poverty And Public Health* (London 1936), pp. 112-115.

[31] Susan E. Burger and Steven A. Esrey, 'Water and sanitation: health and nutrition benefits to children', in Per Pinstrup-Anderson, David Pelletier and Harod Alderman (Eds), *Child Growth And Nutrition In Developing Countries* (Cornell University Press, 1995), pp. 156, 157.

[32] D.J.P. Barker, *Mothers, Babies, And Disease In Later Life* (BMJ London 1984), p. 112.

[33] P. McCarthy, D. Byrne, S. Harrison, and J. Keithley, 'Respiratory conditions: effect of housing and other factors', *Journal of Epidemiology and Community Housing*, 39 (1985), pp. 15-19.

[34] Claire N. Packer, Sarah Stewart-Brown, Sarah E. Fowle, 'Damp housing and adult health: results from a lifestyle study in Worcester, England', *Journal of Epidemiology and Community Health*, 48 (1994), pp. 555-559.

[35] David Leon and Yoav Ben-Shlomo, 'Preadult influences on cardiovascular disease and cancer', in Dinah Kuh and Yoav Ben-Shlomo (Eds), *A Lifecourse Approach To Chronic Disease Epidemiology* (Oxford 1997), p. 61.

[36] D.J. Dedman, D. Gunnell, G. Davey Smith, S. Frankel, 'Childhood housing conditions and later mortality in the Boyd Orr cohort' (Unpublished paper).

[37] D.J. P. Barker, D. Coggan, C. Osmond and C. Wickham, 'Poor housing in childhood and high rates of stomach cancer in England and Wales, *British Journal of Cancer*, 61 (1990), pp. 575-578; D. Coggan, D.J.P. Barker, H. Inskip, G. Wield, 'Housing in early life and later mortality', *Journal of Epidemiology and Community Health*, 47 (1993), pp 345-348.

[38] Personal communication from Professor David Barker.

[39] See D.J.P. Barker, *Mothers, Babies And Disease In Later Life* (BMJ 1994); D.J.P. Barker (Ed), Fetal And Infant Origins Of Adult Disease (BMJ London 1992).

[40] David Leon and Yoav Ben-Shlomo, 'Preadult influences on cardiovascular disease and cancer', in Dinah Kuh and Yoav Ben-Shlomo (Eds), *A Lifecourse Approach To Chronic Disease Epidemiology* (Oxford 1997), pp. 54, 55.

influencing birthweight is however a matter of controversy, and it is likely that nutrition is only important in determining birthweight above a certain minimal threshold of malnutrition.[41]

There are reasons to doubt the role of poverty in shaping patterns of birthweight in the Hertfordshire sample used by Barker and his colleagues. They produced the following figures on social class and birthweight/ weight at one year:

Table 8: Birthweight And Weight At One Year In Men According To Social Class At Birth[42]

Social Class At Birth (Based On Father's Occupation)	*Number*	*Birthweight (Pounds)*	*Weight At One Year (Pounds)*
I	96	7.7	22.2
II	372	7.9	22.5
IIINM	279	7.8	22.4
IIIM	920	7.9	22.2
IV	512	7.7	22.1
V	228	7.8	21.7

The was no significant relationship between social class and birthweight, but a slight correlation between social class and weight at one year in this Hertfordshire sample. The information on father's occupation was retrospectively gathered and the number of cases in Social Class I was very small, and it was possible to only trace less than half of the cases selected from the Hertfordshire Health Visitors' Register.[43] More reliable evidence on socio-economic status and birthweight/ weight at one year is available from research on the rateable values of houses in which children were born in the 1920s and 1930s. This is contemporary information and is available on about three-quarters of all cases in the Hertfordshire Health Visitors' Register.

The following tables summarises findings of research on the relationship between the rateable value of houses in which children were born and stillbirths, premaurity, birthweight, weight at one year, infant and child mortality in two Hertfordshire towns, Berhampstead and Hoddesdon, during the 1920s and 1930s.

Table 9: Stillbirths, Prematurity, Birthweight, Weight At One Year, Infant & Child Mortality, By Rateable Value In Hoddesdon, 1915-39 (Number Of Cases In Brackets)

Rateable Value	*Stillbirths (% Births)*	*Premature (% Births)*	*Birth Weight (pounds)*	*Weight 1 Year (pounds)*	*Infant Mortality (per 1000 births)*	*Child Mortality 1-4 Years (per 1000 cases))*
£4-£6	5.0% (120)	10.0% (120)	7.6 (74)	20.7 (79)	96 (114)	80 (50)
£7	1.0% (299)	5.4% (299)	7.8 (170)	21.2 (215)	54 (295)	36 (111)
£8	1.8% (226)	10.2% (226)	7.6 (156)	21.5 (157)	54 (222)	26 (117)
£9	2.9% (209)	6.7% (209)	7.6 (142)	22.0 (161)	45 (202)	16 (127)
£10	1.3% (239)	8.8% (239)	7.5 (162)	21.5 (167)	39 (236)	7 (135)
£11-£14	3.2% (436)	9.0% (436)	7.7 (289)	21.9 (296)	31 (421)	4 (247)
£15+	3.2% (338)	9.8% (338)	7.6 (184)	22.3 (185)	8 (331)	0 (168)

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[41] Ivan J. Perry, 'Fetal growth and development: the role of nutrition and other factors', in Dinah Kuh and Yoav Ben-Shlomo (Eds), *A Lifecourse Approach To Chronic Disease Epidemiology* (Oxford 1997), p.158; D.J.P. Barker, *Mothers, Babies And Disease In Later Life* (BMJ 1994), p. 124.

[42] C. Osmond, D.J.P. Barker, P.D. Winter, C.H.D. Fall, S.J. Simmonds, 'Early growth and death from cardiovascular disease in women', *British Medical Journal* (11th December 1993), Volume 307, pp. 1522.

[43] There is some evidence for a correlation between social class and birthweight In England & Wales, but this is for the period after the Second World War and the scale of the correlation is very modest. See M.E.J. Wadsworth, *The Imprint Of Time* (Oxford 1991), p.25; O.G. Brooks, 'Effects on birthweight of smoking, alcohol, caffeine, socioeconomic factors, and psychosocial stress', *British Medical Journal*, 298 (25 March 1989), pp. 795-801; T. Coms-Orme et.al., 'Predicting birth weight: relative importance of socio-demographic, medical and prenatal care variables', *Social Science Review*, 67 , 4 (December 1993), pp. 617-30.

Table 10: Stillbirths, Prematurity, Birthweight, Weight At One Year, Infant & Child Mortality, By Rateable Value In Berkhampstead, 1923-39 (Number Of Cases In Brackets)

Rateable Value	*Stillbirths (% Births)*	*Premature (% Births)*	*Birth Weight (pounds)*	*Weight 1 Year (pounds)*	*Infant Mortality (per 1000 births)*	*Child Mortality 1-4 Years (per 1000 cases)*
£3-£6	1.7% (174)	10.3% (174)	7.4 (146)	20.1 (141)	51 (156)	12 (136)
£7	1.1% (190)	8.4% (190)	7.5 (157)	20.5 (153)	66 (182)	20 (154)
£8-£9	1.9% (160)	12.5% (160)	7.5 (125)	21.6 (124)	47 (149)	31 (129)
£10-£11	1.9% (157)	12.1% (157)	7.5 (123)	21.5 (123)	48 (145)	8 (125)
£12-£15	3.1% (321)	12.5% (321)	7.6 (262)	21.4 (248)	14 (281)	16 (244)
£16+	0.7% (286)	26.0% (293)	7.4 (153)	21.7 (141)	20 (249)	0 (199)

There were significant correlations between rateable value and the post-natal variables of weight at one year, infant and child mortality in both towns, but no association between rateable value and the pre-natal variables of stillbirth, prematurity and birthweight.[44] Given that rateable value was a reflection of the market value of housing, this suggests that poverty and housing conditions had a very powerful effect on post-natal child development, but little or no impact on pre-natal development.

Rateable value can be seen as a measure of socio-economic status as well as an indication of housing conditions, and it is known that there was a close link between poverty/ wealth and the consumption of food. Michael Nelson has summarized the association between soci0-economic status and the consumption of food after 1900 as follows:

"Lower-income diets continued to be based mainly on bread and potatoes, and compared with higher-income diets contained much less meat and fish, eggs and milk, separated fats and sugar, fruit, and vegatables other than potatoes. They also provided less energy and other nutrients per family. Income rather than physiological requirements was the major determinant of food choice and quantity ... it was not until the Second World War, with the imposition of a rigorous rationing system, changes in the laws on the provision of welfare foods, and a marked reduction in income differentials, that permanet changes in the social class distribution of food took place."[45]

In 1936/37 families in the top five per cent income group spent an average of about 17 shillings per head on food, compared to the 6 shillings per head spent by the bottom fifteen per cent.[46] It was estimated that none of the top five per cent income group were below the BMA food expenditure minimum, compared to 48 per cent in the bottom fifteen per cent income group.[47] Given that rateable value is a measure of socio-economic status, the lack of a correlation between rateable value and birthweight and a strong association between rateable value and weight at one year in Hertfordshire, suggests that nutritional levels had little or no imact on birthweight, but a significant influence on weight at one year.

There were major increases in the per capita consumption of food during the first half of the twentieth century, associated with growing real incomes and improvements in the standard of living. Total expenditure on food per head rose by 47 per cent between 1900 and 1955, and increases occurred in nearly every category of food covered by the food expenditure surveys.[48] If nutrition had different impacts on birthweight and weight at one year, it is hypothesized that there would be no changes in average birthweight during the twentieth century, but significant increases in mean weight at one year due to the growth of per capita consumption of food. The following figures are based on an analysis of changes in birthweight and weight at one year over time in th two Hertfordshire towns,

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[44] Ref to pattern of correlation re data.

[45] Michael Nelson, 'Social class trends in British diet, 1860-1980', in Catherine Geisler and Derek Oddy (Eds), *Food, Diet And Economic Change Past And Present* (Leicester, ?), p.104.

[46] Richard Stone, *The Measurement Of Consumers' Expenditure And Behaviour In The United Kingdom, 1920-38*, Vol. 1 (Cambridge 1954), p.167.

[47] John Charlton and Mike Murphey (Eds), *The Health Of Adult Britain*, Vol. 1 (HMSO London, 1997), p.102.

[48] Richard Stone, *The Measurement Of Consumers' Expenditure And Behaviour In The United Kingdom, 1920-38*, Vol. 2 (Cambridge 1954), p. 130.

Hoddesdon and Berkhampstead, and in a later period average birthweights in the two Health Authority areas in which they are located:

Table 11: Changes In Mean Birthweight And Weight At One Year In Hoddesdon And Berkhampstead, 1911-39, And East & North Hertfordshire And West Hertfordshire, 1986-98. (Number Of Cases In Brackets)[49]

Period	*Hoddesdon*		*Period*	*Berkhampstead*	
	Birthweight (Pounds)	Wt At 1 Year (Pounds)		Birthweight (Pounds)	Wt At 1 Year (Pounds)
1911-19	7.7 (94)	21.2 (245)	1923-28	7.4 (471)	21.1 (447)
1920-29	7.4 (747)	21.5 (723)	1929-34	7.4 (514)	21.3 (496)
1930-39	7.4 (1116)	21.7 (1109)	1935-39	7.3 (365)	21.5 (356)
	East & North Hertfordshire			*West Hertfordshire*	
1986-98	7.4 (64932)		1995-98	7.4 (27167)	

There was no significant change in birthweight between 1911 and 1998, whereas there was a modest increase in weight at one year in both Hoddesdon and Berkhampstead in the 1920s and 1930s.[50] These figures suggest that nutrition did not play a significant role in changing birthweight, whereas it may have been partly responsible, along with improvements in housing conditions, for increasing weight at one year.

The determinants of birthweight are highly complex, and it is possible that parental characteristics play a role in shaping birthweight, both through genetic and other influences. For example, Davey Smith and others found that mothers of heavier babies were taller, had higher body mass index and died from cardiovascular disease a third less frequently than mothers of small babies,[51] suggesting that genetic or congental factors may play a role in shaping patterns of birthweight.[52] It is also possible that demographic factors - such as maternal age and parity - play a large part in determining birthweight.[53]

The evidence from the study of rateable value and birthweight and weight at one year suggests that birthweight was not influenced by socio-economic environmental variables, whereas weight at one year and other post-natal factors were strongly affected by such environmental factors. Birthweight might be a biological marker for genetic and other biological variables which are not directly influenced by the socio-econopmic or nutritional environment, and it is possible that pre-natal factors measured by birthweight affect later adult disease patterns in a different way from post-natal variables shaped by the soci-economic environment.

Infant development in the first year of life has not attracted the same attention as foetal development in epidemiological research, although historically infant growth was recognised as very important outcome of envuiromental poverty. In 1910, Dr John Robertson , Medical Officer of Health for Birminhagm, published an important paper on the effect of female employment and povery on infant development and health. He showed that family income had a strong impact on weight at one year, as demonstarted in the following table:

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[49] The figures for Hoddesdon and Berkhampstead were calculated from the Hertfordshire Health Visitors' Register, copies of which were made available in computerised form by the MRC Environmental Epidemiology Unit at Southampton; the figures for East & North Hertfordshire and West Hertfordshire were made available by the Hertfordshire Health District Authority.

[50] The figures for birthweight and weight at one year are calculated from the Hertfordshire Health Visitors' Register. There is also some evidence that birthweight did not change significantly at a national level during the second half of the twentieth century: the proportion of babies born under 2500 grams was 8.0 per cent in 1955 and only fallen slightly to 7.2 per cent by 1986. See M.E.J. Wadsworth, *The Imprint Of Time* (Oxford 1991), p.26.

[51] George Davey Smith, Carole Hart, Catherine Ferrell, Mark Upton, David Hole, Victor Hawthorne, Graham Watt, "Birthweight of offspring and mortality in the Renfrew and Paisley study: prospective and observational study", *British Medical Journal*, Vol. 315 (8 November 1997), pp. 1189-1193.

[52] See Paul McKeigue, 'Diabetes and insulin action', in Kuh and Ben-Shlomo, *op.cit.*, pp. 92, 93; Yoav Ben-Shlomo and Dinah Kuh, 'Conclusions', in Kuh and Ben-Shlomo, *op.cit.*, p. 300.

[53] See M.L. Nordstrom, 'Effects on birthweight of maternal education, socio-economic status and work related characteristics', *Scandinavian Journal Of Social Medicine*, 24, 1 (March 1996), pp 55-61.

Table 12: Family Income And Average Weight At One Year In Birmingham, 1909.

Total Income	*Number Of Babies Weighed*	*Average Weight At One Year (Pounds)*
Under 10 Shillings Per Week	52	16.8
10 - 20 Shillings	303	17.5
20 - 30 Shillings	300	18.3
Over 30 Shillings	39	18.8

Robertson concluded that "large number of infants start life at a very great disadvantage, because during intra-uterine life or during the first six months their mothers have not been able to nourish them ... what food comes into the house is given to the children or the husband, while they themselves go on from day to day in a state of semi-starvation."[54] Robertson appended to his report six charts which tracked weight in the first year, suggesting that most of the differential weight gain between those living below and above the poverty line occurred after birth, and was the result of both poverty and infection. Robertson implied that it was the lack of good-quality breastmilk along with infection that was responsible for inadequate growth in infancy

Infant development in the first year of life has also perhaps been under-estimated in its effect on later adult disease mortality. In the studies carried out by Barker and colleagues, the correlation between weight at one year and coronary heart disease mortality amongst males was significantly stronger than the correlation between birthweight and coronary heart disease mortality. [55] A special study carried out by Fall and colleagues found no correlation between birthweight and the incidence of coronary heart disease in a Hertfordshire sample of men, but a a strong and linear correlation between weight at one year and heart disease.[56]

The research reviewed in this paper suggests that infant mortality and development in the first year of life is critical for the understanding of coronary heart and other adult diseases. The Hertfordshire Health Visitors' Register provides the basic information for the further exploration of both birthweight and weight at one year, as well as breastfeeding, infection and infant mortality. These variables need to be linked to data on rateable value, father's occupation, housing conditions, overcrowding, and the family links between siblings and parents. The former will allow the exploration the role of socio-economic and other environmental factors, and the latter the genetic links with regard to birthweight and adult disease mortality, enabling greater clarification of the infant origins of coronary heart and other adult diseases.

Peter Razzell

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[54] *Ibid,* p.19.

[55] D.J.P. Barker, *Mothers, Babies, And Disease In later Life* (BMJ London 1994), p.41.

[56] C.H.D. Fall, M. Vijayakumar, D.J.P. barker, C. Osmond, S. Duggleby, 'Weight in infancy and prevalence of coronary heart disease in adult life', *British Medical Journal*, Vol. 310 7th January 1995, p. 18.