Papers

Size at birth and resilience to effects of poor living conditions in adult life: longitudinal study

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Abstract

Objective To determine whether men who grew slowly in utero or during infancy are more vulnerable to the later effects of poor living conditions on coronary heart disease.

Design Follow up study of men for whom there were data on body size at birth and growth and social class during childhood, educational level, and social class and income in adult life.

Setting Helsinki, Finland.

Participants 3676 men who were born during 1934-44, attended child welfare clinics in Helsinki, were still resident in Finland in 1971, and for whom data from the 1980 census were available.

Main outcome measures Hospital admission for or death from coronary heart disease.

Results Men who had low social class or low household income in adult life had increased rates of coronary heart disease. The hazard ratio among men with the lowest annual income (< £8400) was 1.71 (95% confidence interval 1.18 to 2.48) compared with 1.00 in men with incomes above £15 700. These effects were stronger in men who were thin at birth (ponderal index < 26 kg/m^3): hazard ratio 2.58 (1.45 to 4.60) for men with lowest annual income. Among the men who were thin at birth the effects of low social class were greater in those who had accelerated weight gain between ages 1 and 12 years. Low social class in childhood further increased risk of disease, partly because it was associated with poor growth during infancy. Low educational attainment was associated with increased risk, and low income had no effect once this was taken into account.

Conclusion Men who grow slowly in utero remain biologically different to other men. They are more vulnerable to the effects of low socioeconomic status and low income on coronary heart disease.

Introduction

The fetal, infant, and childhood growth of boys who later develop coronary heart disease differs from that of other boys.[1–6] They tend to have a period of retarded growth in utero and during infancy but accelerated weight gain thereafter. Altered growth may lead to disease because it is associated with permanent changes in body composition and in the structure and function of tissues such as skeletal muscle and the liver.[6] Poor living standards in adult life are known to increase the risk of coronary heart disease,[7] but whether their effects interact with those of fetal and childhood growth has not been studied.

We have previously reported on a cohort of 4630 men who were born in Helsinki, Finland, and whose growth was measured serially from birth to 12 years.[6] The study confirmed that coronary heart disease is associated with low birth weight and poor infant growth.[3] It also showed that the increased risk of disease associated with rapid weight gain after the age of 1 year occurred only among men who were thin at birth, as defined by a ponderal index (birth weight (kg)/length (m)^3) below 26. We report here on the interplay between early growth and social circumstances in childhood and adult life in determining the risk of coronary heart disease.

Methods

The original cohort comprised 4630 men who were born in Helsinki University Central Hospital during 1934-44 and who attended child welfare clinics in Helsinki and were still living in Finland in 1971.[6] Of the initial cohort, 77% also went to schools in the city and had school health records. Details of the birth, child welfare, and school health records, which included serial measures of height and weight, have been published, together with an account of the methods used to trace the men and identify all hospital admissions and deaths from coronary heart disease during 1971-97.[5 6]

Through Statistics Finland we obtained data on occupation, recorded in the 1980 census and grouped into four categories. Data on taxable household income in marks per year were obtained from the national taxation register; we converted Finnish marks to pounds sterling using the exchange rate in mid-1980. Our analyses were carried out on the 3676 (79%) men for whom these data were available. Their mean birth weight, ponderal index at birth, and weight at 1 year were similar to those of men not included in this study. For 3416 (93%) men the occupation of the father was recorded on the records of the child welfare clinic, and we grouped them according to a classification used by the central statistical office. For all except 93 men the level of education they achieved was recorded in the 1970 census. Under a classification used by the central statistical office and on the basis of

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the International Standard Classification of Education[8] we grouped the men into three levels of education: high (upper secondary and tertiary), middle (lower secondary), and low (primary).

We obtained approval for the study from the ethical committee of the National Public Health Institute, Helsinki.

Statistical analyses

We examined trends in hazard ratios with socioeconomic indices using Cox's proportional hazards model.[9] As in previous analyses we converted each measurement of height, weight, and body mass index (weight (kg)/height (m)^2) for each boy to a standard deviation score at each birthday.[5]

Results

We determined the number of men with coronary heart disease. In total 234 men had been admitted to hospital, of whom 22 died from the disease. Fifty one other men had died without admission to hospital. We therefore analysed data on 285 men with coronary heart disease.

Infant growth and social class

Previous analyses showed that hazard ratios for coronary heart disease were higher in men who at any gestational age had low birth weight and low ponderal index at birth and in those who had low weight at 1 year.[6] In a simultaneous regression both low birth weight and low weight gain between birth and 1 year increased the risk of disease, the hazard ratios being 1.31 (95% confidence interval 1.15 to 1.49, P = 0.0001) per SD fall in birth weight and 1.20 (1.05 to 1.36, P = 0.006) per SD fall in infant weight gain. Table 1 shows that hazard ratios for coronary heart disease were higher among men whose fathers were in the lower social classes. Father's social class was unrelated to size at birth but among the sons of labourers the mean weight gain between birth and 1 year was 131 g less than that of other boys (P = 0.006) and mean height gain was 0.4 cm less (P < 0.001). The effects of low social class of father on coronary heart disease remained significant after adjustment for weight at 1 year (P = 0.0005). Conversely the effects of low weight at 1 year remained significant (P = 0.001) after adjustment for father's social class.

Table 1 Hazard ratios for coronary heart disease among men according to father's social class, level of education, and social class and household income in adult life

	No of cases/ No of men	Hazard ratios (95% CI)	P value for trend
Infancy			
Fathers' social class:			
Upper middle	20/449	1.00	0.0006
Lower middle	48/709	1.44 (0.86 to 2.43)	
Labourer	202/2258	2.01 (1.27 to 3.18)	
Education			
Level of education:			
High	32/721	1.00	0.0001
Middle	111/1489	1.87 (1.26 to 2.77)	
Low	133/1373	2.25 (1.53 to 3.30)	
Adult life (1980)			
Social class:			
Higher official	62/1247	1.00	<0.0001
Lower official	69/888	1.62 (1.15 to 2.28)	
Self employed	22/240	1.83 (1.12 to 2.97)	
Labourer	132/1301	2.15 (1.59 to 2.91)	
Household income 1000 marks/year (£):			
>140 (15 700)	45/734	1.00	0.002
111-140 (15 700)	61/776	1.34 (0.91 to 1.96)	
96-110 (12 400)	41/595	1.26 (0.82 to 1.92)	
75-95 (10 700)	64/747	1.52 (1.03 to 2.22)	
≤75 (8 400)	74/824	1.71 (1.18 to 2.48)	

Table 2 Hazard ratios (95% confidence intervals) for coronary heart disease according to level of education and household income

	Level of education		
Income*	**High**	**Middle**	**Low**
>118 (13 300)	1.00	1.74 (1.03 to 2.94)	2.05 (1.20 to 3.50)
87-118 (13 300)	0.92 (0.39 to 2.14)	1.79 (1.07 to 2.99)	1.63 (0.97 to 2.74)
≤86 (9 700)	0.53 (0.12 to 2.25)	1.70 (1.00 to 2.86)	2.55 (1.58 to 4.11)

*Household income 1000 marks/year (£)

Education

Hazard ratios for coronary heart disease were higher in men who achieved only a low level of education (table 1). Education was strongly related to father's social class. Only 12% of boys in families in which the fathers were labourers reached the highest educational level compared with 37% of other boys. Men who achieved only low levels of education, though similar to others in body size at birth, had 220 g less weight gain between birth and 1 year (P < 0.001). Education had significant effects on coronary heart disease after adjustment for father's social class (P = 0.003) or weight at 1 year (P = 0.0002).

Living standards in adult life

Table 1 shows that men who as adults were in the lower social classes or had low household incomes were at increased risk of coronary heart disease. These trends remained significant after adjustment for father's social class (P = 0.0002 for social class and 0.02 for income). Conversely the trends with father's social class remained significant after adjustment for adult social class (P = 0.01). The trends with weight at 1 year remained significant after adjustment for adult social class (P = 0.002). Adult social class and income were strongly related to educational level. In simultaneous analyses with education the effect of adult social class on coronary heart disease remained significant (P = 0.003), whereas the effect of income became non-significant (P = 0.10) Table 2, in which the men's household incomes are divided into thirds, shows how, within each level of education, income had no influence on the risk of coronary heart disease.

Interactions between body size and living standards

We examined the simultaneous effects of early body size and the socioeconomic indices. There were interactions with ponderal index at birth but not with any other measure of body size at birth or at 1 year. In table 3 we have divided the cohort around a ponderal index of 26 kg/m^3, as in our previous analyses.[6] Although father's social class had a stronger effect in the high ponderal index group (table 3), the interaction was not significant (P = 0.6). There was no interaction between ponderal index and education, but there were interactions with adult social class and income. Among men in the low ponderal index group low adult social class and income were associated with increased hazard ratios for coronary heart disease.

Table 3 Hazard ratios for coronary heart disease according to ponderal index (kg/m³) at birth, father's social class, level of education, and adult social class and income

	Ponderal index ≤26.0 (n=1475)		Ponderal index >26.0 (n=2154)	
	Hazard ratio (95% CI)	Unadjusted (adjusted) P value for trend	Hazard ratio (95% CI)	Unadjusted (adjusted) P value for trend
Infancy				
Father's social class:				
Upper middle	1.00	0.06 (0.41*)	0.76 (0.32 to 1.84)	0.006 (0.02*)
Lower middle	1.52 (0.71 to 3.27)		1.05 (0.49 to 2.27)	
Labourer	1.86 (0.94 to 3.68)		1.60 (0.81 to 3.16)	
Education				
Level of education:				
High	1.00	0.004	0.60 (0.30 to 1.20)	0.007
Middle	1.29 (0.73 to 2.27)		1.50 (0.88 to 2.55)	
Low	2.00 (1.17 to 3.42)		1.47 (0.86 to 2.51)	
Adult life (1980)				
Social class:				
Higher official	1.00	<0.0001 (<0.0001†)	1.00 (0.61 to 1.66)	0.01 (0.21†)
Lower official	1.59 (0.93 to 2.72)		1.66 (1.01 to 2.71)	
Self employed	1.87 (0.88 to 3.99)		1.81 (0.93 to 3.53)	
Labourer	2.69 (1.71 to 4.25)		1.76 (1.11 to 2.77)	
Household income 1000 marks/year (£):				
>140 (15 700)	1.00	<0.0001 (0.0001†)	1.19 (0.65 to 2.19)	0.75 (0.84†)
111-140 (15 700)	1.54 (0.83 to 2.87)		1.42 (0.78 to 2.57)	
96-110 (12 400)	1.07 (0.51 to 2.22)		1.66 (0.90 to 3.07)	
76-95 (10 700)	2.07 (1.13 to 3.79)		1.44 (0.79 to 2.62)	
≤75 (8 400)	2.58 (1.45 to 4.60)		1.37 (0.75 to 2.51)	

*Adjusted for adult social class. †Adjusted for father's social class.

Among men in the higher ponderal index group, however, there was a weaker trend in hazard ratios with social class, which became non-significant after adjustments for father's social class, and no trend with income (P for interaction 0.05 for social class, 0.005 for income).

We examined whether accelerated weight gain during childhood further increased the risk associated with low adult social class and low income among men with a low ponderal index at birth. In table 4 we have divided the men with a low ponderal index into two groups according to whether their SD scores for body mass index increased or decreased between the ages of 1 to 12 years, the period used in previous analyses.[6] In those in whom the scores increased, hazard ratios for coronary heart disease rose as adult social class fell. There was no similar trend among those in whom the scores decreased. The interaction between change in childhood body mass and adult social class was significant (P = 0.02); there was no significant interaction with income.

Combined effects

In a simultaneous analysis coronary heart disease was associated with low weight at 1 year, low education, and low adult social class. The hazard ratios were 1.19 (1.06 to 1.33, P = 0.003) for each SD decrease in weight at 1 year, 1.22 (1.01 to 1.48, P = 0.04) for each decrease in level of education, and 1.17 (1.05 to 1.30, P = 0.005) for each decrease in adult social class. Addition of father's social class and income did not increase the explanatory power of the model.

Discussion

In this large follow up study the risk of coronary heart disease associated with low social class and income was greater among men who were thin at birth. Recent studies of newborn babies in Southampton (unpublished) suggest that the definition of thinness that we used would include one quarter of all boys born in Britain today. The highest risk was in men who were thin at birth but had accelerated weight gain after the age of 1 year followed by poor living standards in adult life. Men who were not thin at birth were largely resilient to the later effects of poor living standards. Low rates of fetal and infant growth, poor educational attainment, and low adult social class each had independent effects on coronary heart disease. Low social class in childhood and low adult income did not give any additional prediction of disease.

Limitations of the study

We studied men who were born in Helsinki University Central Hospital, who were taken to child welfare clinics in the city, and for whom information was recorded in the 1980 census. We have previously shown that the distribution of social class in these men was representative of the city as a whole.[5] Our information on living standards in adult life, however, did not include aspects of personal behaviour such as diet and smoking.

Table 4 Hazard ratios for coronary heart disease in men with ponderal index at birth <26 kg/m³, according to adult social class and changes in body mass index (BMI) between ages 1 and 12 years

	Change in SD score for BMI	
Social class	Decrease*	Increase†
Higher official	1.38 (0.55 to 3.52)	1.00
Lower official	1.14 (0.38 to 3.40)	1.84 (0.70 to 4.85)
Self employed	0.69 (0.09 to 5.62)	2.93 (0.86 to 10.01)
Labourer	2.18 (0.88 to 5.39)	3.78 (1.68 to 8.53)

*P=0.24 for trend. †P<0.0001 for trend.

Infant growth and social class

As has previously been shown, we found that having a father with low social class increased the risk of coronary heart disease independently of a man's own social class.[10–13] Growth during infancy was reduced in families of low social class, and poor infant growth is associated with increased risk of disease, possibly through altered liver growth and consequent re-programming of lipid metabolism and blood coagulation.[14 15] The effects of low social class on coronary heart disease, however, were only partly explained by their effect on infant growth. Its other effects could be linked to its strong association with poor educational attainment.

Education

We found that poor educational attainment was associated with later coronary heart disease. Men who achieved only low levels of education had reduced infant growth, but this explained only a small part of the association. One possible explanation has been suggested by a follow up study of children born in Aberdeen, Scotland.[16] Those who had lower scores in a mental ability test at 12 years of age died younger. Whalley and Dreary proposed that performance in intelligence tests might reflect general aspects of childhood fitness related to long term health. Greater intelligence and higher levels of education could also be associated with healthier behaviour in adult life.

Living standards in adult life

As expected, low social class and low income were associated with increased rates of coronary heart disease.[7 17 18] We found, however, that these effects were stronger in men who were thin at birth, especially if they had had accelerated weight gain in childhood. Taxable household income was strongly related to educational attainment. We found that better educational attainment protected men from the harmful effects of low income. The way in which low income may increase cardiovascular risk is controversial. Lynch et al have emphasised the material consequences of low income on living conditions.[19] While better education may enable people to reduce the effects of low income on their living standards, it is surprising that we found no effect of income once education was taken into account.

Marmot and Wilkinson have emphasised the psychosocial consequences of a low position in the social hierarchy, as indicated by low income, and suggested that perceptions of low social status and lack of success lead to changes in neuroendocrine pathways and hence to disease.[20] Our findings seem consistent with this hypothesis, although they point to the importance of a hierarchy defined by education rather than income. The hypothesis could also explain why, in our study, low income was associated with coronary heart disease only in a vulnerable group defined by low rates of intrauterine growth. People who are small at birth are known to have persisting alterations in responses to stress, including raised serum cortisol concentrations.[21] Rapid childhood weight gain may exacerbate the adverse effects of small size at birth.[22]

Conclusion

Our results show that studies of the developmental origins of coronary heart disease may reveal new biological pathways through which social influences affect the risk of disease.[23] People whose rate of growth in fetal life, infancy, and childhood differs from the norm remain biologically different and are at increased risk of coronary heart disease. Social influences that alter growth may therefore alter the risk of disease. Conversely, altered early growth may increase vulnerability to the effects of poor living standards in adult life. Therefore, improvements in fetal, infant, and child growth may prevent coronary heart disease in the next generation by improving the body's fitness and making it resilient to later social adversity.

What is already known on this topic

People who grow slowly in utero and during infancy remain biologically different through their lives

Such people are at increased risk of coronary heart disease

What this study adds

Among men who were thin at birth the risk of coronary heart disease is further increased if they have poor living standards in adult life

Other men tend to be resilient to the adverse effects of poor living standards

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