Current topic

Childhood causes of adult diseases

D J P BARKER

Medical Research Council Environmental Epidemiology Unit

The search for influences in the adult environment which determine the risk of the major non-malignant diseases, cardiovascular disease, and chronic bronchitis has met with limited success. Cigarette smoking has been implicated; evidence on dietary fat has accumulated to the point where a public health policy of reduced intake is considered prudent, even if the case is not proven. Much, however, remains unexplained.

A puzzling aspect of the epidemiology of ischaemic heart disease, stroke, and chronic bronchitis in Britain is that they are more common in poorer areas and in lower income groups.¹ Differences are large, greater than twofold. For ischaemic heart disease they are also paradoxical in that its steep rise in Britain and elsewhere has been associated with rising prosperity. Why should its rates be lowest in the most prosperous places, such as London and the home counties? Variations in cigarette smoking and adult diet do not explain these differences.

There is increasing evidence that they result from geographical and social class differences in child development and health 60 and more years ago. Past differences in child health were reflected in the wide range of infant mortality. For example in 1921-5 infant mortality ranged from 44 per 1000 births in rural West Sussex to 114 in Burnley. The highest rates were generally in northern counties where large manufacturing towns had grown up around the coal seams. Rates were also high in poor rural areas such as north Wales. They were lowest in counties in the south and east, which have the best agricultural land and are historically the wealthiest. A series of government inquiries on child and maternal mortality from 1910 onwards, prompted by revelations of the poor physique of military recruits, showed how these differences in infant mortality were related to differences in maternal physique and health, infant feeding, housing, and overcrowding.²⁻⁴

Mortality statistics for England and Wales can be used to compare the present distribution of adult death rates from specific causes with the past

geographical distribution of causes of infant mortality. These comparisons can be made with the country divided into large towns and groupings of small towns and rural areas within counties, totalling 212 areas—a division of the country used in routine statistics since the turn of the century.

Mortality from chronic bronchitis and emphysema is concentrated in the large towns. Its geographical distribution correlates remarkably closely with infant mortality from bronchitis and pneumonia in the early years of the century.⁵ The coefficient of correlation is 0.85. A similarly high coefficient is seen for each sex separately and if comparisons are made between large towns only, or small towns or rural areas.

Suspicion that respiratory infection in early childhood might be a major cause of chronic bronchitis in adult life has been reinforced by follow up studies which show that after bronchiolitis, bronchitis, or pneumonia abnormalities of pulmonary function may persist through childhood.⁶⁻⁹ In the national sample of children born in Britain during 1946 there was a strong association between the occurrence of one or more lower respiratory infections before 2 years of age and the prevalence of cough during the day, or at night in the winter, at the age of 25 years.¹⁰

The close geographical relation between infant mortality from respiratory infection and adult mortality from bronchitis may be interpreted as strong evidence for a direct causal link between respiratory infection in early childhood and chronic bronchitis. Regression analysis of bronchitis and lung cancer mortality suggests that childhood infection is more important than cigarette smoking in determining the prevalence of bronchitis.⁵ The high rates of chronic bronchitis in Britain, and its distribution within the country, are therefore a legacy of poor social conditions in the past. Particular adverse influences which are implicated include inadequate housing, large family size, overcrowding in the home, and the abandonment of breast feeding. This interpretation is consistent with the progressive

decline in mortality from chronic bronchitis over the past 50 years.

Another close geographical relation in England and Wales is that between current mortality from stroke and maternal mortality from causes other than puerperal fever in the early years of the century.¹¹ Toxaemia was the commonest cause of these maternal deaths. Hypertension may be the mechanism linking maternal health and risk of stroke in the offspring. Maternal hypertension increases the risk of hypertension in the children,¹² and hypertension is a risk factor for stroke. There is increasing evidence that hypertension originates in childhood.¹³ 'Tracking' of blood pressure has been repeatedly observed in longitudinal studies of children and adults.¹⁴⁻¹⁷ A link between the intrauterine environment and hypertension is suggested by the negative relation between birth weight and blood pressure at age 36 in the 1946 cohort.¹⁸ The distribution of stroke in Britain may therefore reflect the past distribution of poor living standards which accompanied industrialisation or economic depression in certain areas. These adversely affected the development of girls and increased the risk of stroke in their offspring, possibly through mechanisms associated with blood pressure and rates of growth in early life.¹¹

The geographical distribution of ischaemic heart disease in Britain is similar to that of stroke. A study in 24 British towns has shown that the mean blood pressure of adult men in a town correlates with the prevalence of ischaemic heart disease.¹⁹ Childhood influences associated with blood pressure could determine the distribution of both diseases. Their time-trends, however, are opposite. Mortality from stroke has fallen in Britain over the past 40 years, which is consistent with past improvements in maternal health and physique. Ischaemic heart disease mortality, however, has risen steeply. This is attributed to increasing affluence, in particular diet, during adult life. Ischaemic heart disease may therefore have two groups of causes. One acting through the mother or in infancy, which is associated with poor living standards.²⁰ The other acting in adult life, which is associated with affluence. Further evidence for the existence of the former comes from the inverse relation between ischaemic heart disease and height, which is largely determined by growth in childhood.²¹ Longterm 'programming' of lipid metabolism during infancy, as a result of infant feeding, is a mechanism for which there is increasing evidence in experimental animals, and which may be a link between childhood and the risk of ischaemic heart disease.²²

A prediction from this is that the improvements in maternal and child health in generations born before

the second world war will now be reflected in a fall in ischaemic heart disease. Rates are beginning to fall in many parts of Britain and there have already been substantial falls in the United States, Canada, Australia, and New Zealand.

Epidemiological investigation of the effect of childhood influences on the risk of cardiovascular disease and chronic bronchitis is being pursued in a variety of ways – through prospective and retrospective studies of individuals, detailed analyses of time-trends and studies of migrants. If they show major effects we will need a new national strategy for reducing inequalities of health in Britain.¹ The current strategy is focused on adult lifestyles. The new one will need to address differences in the development and health of children.

References

- 1 Barker DJP, Osmond C. Inequalities in health in Britain: specific explanations in three Lancashire towns. *Br Med J* 1987;**294**:749–52.
- 2 Local Government Board. *Forty second annual report, 1912–13. Supplement in continuation of the report of the medical officer of the board for 1912–13*. London: HMSO, 1913.
- 3 Local Government Board. *Forty-fourth annual report 1914–15. Supplement: maternal mortality in connection with childbearing*. London: HMSO, 1916.
- 4 Campbell JM, Cameron D, Jones DM. *High maternal mortality in certain areas*. London: HMSO, 1932. (Ministry of Health Reports on Public Health and Medical Subjects, No 68.)
- 5 Barker DJP, Osmond C. Childhood respiratory infection and adult chronic bronchitis in England and Wales. *Br Med J* 1986;**293**:1271–5.
- 6 Kattan M, Keens TG, Lapierre JG, Levison H, Bryan AC, Reilly BJ. Pulmonary function abnormalities in symptom-free children after bronchiolitis. *Pediatrics* 1977;**59**:683–8.
- 7 Gurwitz D, Mindorff C, Levison H. Increased incidence of bronchial reactivity in children with a history of bronchiolitis. *Pediatrics* 1981;**98**:551–5.
- 8 Pullan CR, Hey EN. Wheezing, asthma, and pulmonary dysfunction 10 years after infection with respiratory syncytial virus in infancy. *Br Med J* 1982;**284**:1665–9.
- 9 Mok JYQ, Simpson H. Outcome for acute bronchitis, bronchiolitis, and pneumonia in infancy. *Arch Dis Child* 1984;**59**:306–9.
- 10 Kiernan KE, Colley JRT, Douglas JWB, Reid DD. Chronic cough in young adults in relation to smoking habits, childhood environment and chest illness. *Respiration* 1976;**33**:236–44.
- 11 Barker DJP, Osmond C. Death rates from strokes in England and Wales predicted from past maternal mortality. *Br Med J* 1987;**295**:83–6.
- 12 Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Family (parental) history and prevalence of hypertension. Results of a nationwide screening programme. *JAMA* 1979;**241**:43–6.
- 13 Hofman A. Blood pressure in childhood: an epidemiological approach to the aetiology of hypertension. *J Hypertens* 1984;**2**:323–8.
- 14 de Swiet M, Fayers P, Shinebourne EA. Blood pressure survey in a population of newborn infants. *Br Med J* 1976;**iii**:9–11.
- 15 Beaglehole R, Salmond CE, Eyles EF. A longitudinal study of blood pressure in Polynesian children. *Am J Epidemiol* 1977;**105**:87–9.
- 16 Clarke WR, Schrott H, Leaverton PE, Connor WE, Laver RM.

- Tracking of blood lipids and blood pressure in school age children: the Muscatine study. *Circulation* 1978;**58**:626–34.
- ¹⁷ Voors W, Webber LS, Berenson GS. Time course studies of blood pressure in children: the Bogalusa heart study. *Am J Epidemiol* 1979;**109**:320–34.
- ¹⁸ Wadsworth MEJ, Cripps HA, Midwinter RE, Colley JRT. Blood pressure in a national birth cohort at the age of 36 related to social and familial factors, smoking, and body mass. *Br Med J* 1985;**291**:1534–8.
- ¹⁹ Shaper AG, Pocock SJ, Walker M, Cohen NM, Wale CJ, Thomson AG. British Regional Heart Study: cardiovascular risk factors in middle-aged men in 24 towns. *Br Med J* 1981;**283**: 179–86.
- ²⁰ Barker DJP, Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet* 1986;**i**:1077–81.
- ²¹ Marmot MG, Shipley MJ, Rose G. Inequalities in death-specific explanations of a general pattern? *Lancet* 1984;**i**:1003–6.
- ²² Mott GE. Deferred effects of breastfeeding versus formula feeding on serum lipoprotein concentrations and cholesterol metabolism in baboons. In: Filer LJ Jr, Fomon SJ, eds. *The breastfed infant: a model for performance. Report of the ninety-first Ross conference on paediatric research*. Columbus, Ohio: Ross Laboratories, 1986: 144–9.

Correspondence to Professor DJP Barker, MRC Environmental Epidemiology Unit, Southampton General Hospital, Southampton SO9 4XY.