Current topic

Childhood causes of adult diseases

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The search for influences in the adult environment which determine the risk of the major nonmalignant 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. 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. 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, and hypertension is a risk factor for stroke. There is increasing evidence that hypertension originates in childhood. ‘Tracking’ of blood pressure has been repeatedly observed in longitudinal studies of children and adults. 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. 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.

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. 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. 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. 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.

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. The current strategy is focused on adult lifestyles. The new one will need to address differences in the development and health of children.

References

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