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Familial risks of childhood cancer

Cancer in childhood is uncommon and leaves parents of afflicted children shocked and bewildered. Instinctively, parents seek explanations for the calamity, and their concern is often mingled with anxiety that their other children may also be at risk. Doctors will want to allay these fears, and we now have reliable information about the familial recurrence of childhood cancers that will generally allow them to do so.

Certain tumours have long been known to run in families. For example, retinoblastoma can be inherited as an autosomal dominant trait.[1] Children may inherit conditions such as neurofibromatosis or xeroderma pigmentosum, which themselves carry a high risk of malignancy.[2] Such cases, however, account for only a small fraction of childhood cancers.

Failure to find environmental causes for the bulk of cancers in children has enlivened the search for genetic factors.[3] Striking clusters of tumours in families have been reported. Li *et al*[4] recently described six families in which three or more sibs were affected. Of course, occasional clusters would be expected by chance, but these families seemed to be at special risk. Surveys of death certificates in the United States[5] have also provided evidence for familial factors.

Since 1953 in Britain most cancers occurring in children have been included in the survey started by Dr Alice Stewart. Information about most of the 20 000 patients has been culled from medical records, and the parents of 15 000 have been interviewed. Draper *et al*[6] have recently used this massive study to estimate the risk of cancer for the sib of an affected child. Two or more children were affected in just over 100 families. Excluding patients with retinoblastoma or tumours known to be associated with premalignant syndromes, they found that the risk for a sib was about 1 in 300, compared with a risk in the general population of about 1 in 600. This small increase in risk existed whether the first child had leukaemia, lymphoma, or another malignancy. The second tumour was often, but not always, of the same type as the first. The risk appeared to be much higher in a twin of the same sex as an affected child, and such twins should be examined regularly.

This small familial influence in the distribution of childhood cancers could be due either to sharing of environmental factors (before or after birth) or to shared genes, or to a combination of the two. A genetic influence in leukaemia is suggested by the predisposition in conditions such as Fanconi's anaemia, which may entail aberrations of chromosomes,[7] and by the observation that in Japan familial cases are often associated with parental consanguinity.[8] Subclinical immunodeficiency may underlie some familial clusters of lymphomas.[9] Clusters of other tumours may also sometimes be due to inherited premalignant syndromes in subclinical form: families with only minimal signs of neurofibromatosis, for example, may have a high incidence of brain tumours.[2]

Estimates of the risk in families with childhood cancer have often been based on flimsy evidence, and some estimates have been too gloomy. One textbook on genetic counselling[10] puts the risk of Wilms's tumour in sibs of an affected child at about 1 in 20, whereas Draper *et al*[6] found only one case among 2800 sibs of 1200 patients, and that was in a twin. Hence while familial risks of childhood cancer are of great interest to researchers, parents will be relieved to know that in most cases the risk is tiny.

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Pulmonary eosinophilia

Eosinophilia, an important response to immune reactions, may be initiated by "eosinophilic chemotactic factor" released from tissue mast cells.[1] On contact with this factor, the eosinophils are deactivated[2] and so are held at the site of the allergic process, where they can ingest and metabolise antigen-antibody complexes; they can also ingest the granules from mast cells and degrade the histamine contained within them.[3] Blood eosinophilia is said to be present when there are more than $0{\cdot}5 \times 10^9$ eosinophils per litre, but it is not necessarily accompanied by an increased total white cell count. Since the blood is a transient compartment for eosinophils, with considerable diurnal variation, there may be an excess of eosinophils in tissues or secretions when the blood count is normal,[4] and tissue eosinophilia cannot be excluded on the basis of an isolated blood count.

Löffler described two clinical conditions associated with blood eosinophilia: endocarditis[5] and pulmonary infiltrates.[6] Heart disease and eosinophilia were the subject of a recent review in the *British Heart Journal*.[7] Pulmonary eosinophilia, which is characterised by blood eosinophilia with pulmonary infiltrates, has been classified by Crofton and others into five kinds, of which only the first corresponds to Löffler's original