disease may have greatly increased energy requirements because of their abnormal neuromuscular state. The findings presented here show that energy expenditure can vary at different stages of the day. Nevertheless, it seems that increased energy requirements may be an important contributor to the clinical syndrome of thinness in Parkinson's disease and we suggest that this should be considered in the nutritional management of such patients.

We are grateful for a research fellowship from the Parkinson's Disease Society.

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(Accepted 15 August 1990)

Retinoblastoma in grandchildren of workers at Sellafield nuclear plant

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Br Med J 1990;**301**:1257

Evidence indicates that exposure of workers to radiation at Sellafield nuclear reprocessing plant increases the risk of acute lymphoblastic leukaemia in the next generation.[1] This could be due to a germinal mutation occurring in the workers or to an increased rate of somatic mutation occurring in their children during intrauterine or early extrauterine life.[2] One way to investigate this is to study the incidence of retinoblastoma in the offspring of those who work in the nuclear industry. This is because more is known about the genetic basis of retinoblastoma than of leukaemia[3] and it is often possible to work out the stage at which mutation has occurred. For instance, if there is an increased rate of germinal mutation this would lead to an increased frequency of bilateral retinoblastoma, and an increased rate of somatic mutation would lead to an increased incidence of unilateral retinoblastoma.

We report three cases of retinoblastoma that are relevant to this debate. These cases were recognised not as a result of a systematic study but because the mother of the index child knew of the two other cases and brought them to our attention.

Case reports

The maternal grandfathers of all three children worked at Sellafield, and the mothers had lived in Seascale, a small village less than 3 km from Sellafield, during part of their childhood: one mother had been born there and the two others had moved there at the age of 6 months and 11 years of age. None of the children, however, had been born in Seascale. One child (case 1) developed a unilateral retinoblastoma at 6 months of age, which was treated by enucleation of the eye. At the age of 5 years she was well with no evidence of tumour in the remaining eye. The constitution of her chromosomes was 46,XX. The second child (case 2) developed a unilateral retinoblastoma at 12 months of age, which was treated by enucleation. The other eye was still free of tumour when she was seen at the age of 3. The constitution of her chromosomes was 46,XX. The third child (case 3) developed retinoblastoma in one eye at 3 months of age, which was treated by enucleation, and a tumour in the other eye at 8 months, which was treated with a cobalt plaque. At the age of 12 she is well. The constitution of her chromosomes was 46,XX with a partial deletion of one chromosome 13 (del 13 [q12–q21]); this defect was not present in the cells of either parent.

The diagnosis of retinoblastoma was confirmed by histological examination of the enucleated eyes in all three cases. There was no history of retinoblastoma in any other member of these families. In addition, the fundi of the parents of the three children showed no evidence of a healed retinoblastoma. The fathers of the children had not had any contact with the nuclear industry and had never worked in Cumbria.

Comment

There was no evidence of a germinal mutation derived from the maternal grandfather in any of the three cases. In case 3 there was evidence of a germinal mutation derived from one of the parents. In the two other cases the evidence pointed to somatic mutation, although the possibility of germinal mutation derived from the parents cannot be completely excluded.

It is, of course, possible that this cluster of cases is a chance occurrence unrelated to exposure to radiation. Retinoblastoma, however, is extremely rare, occurring with a frequency of 1 in 20 000 live births and being 10 times less common than acute lymphoblastic leukaemia. To calculate the probability of a cluster of cases reliably it is necessary to define in advance the total population from which the cases have arisen. This is not possible if the cluster is observed in retrospect. In 1987, however, Gardner *et al* calculated that roughly 2600 children had been resident in Seascale at some time between 1950 and 1983.[4,5] This cohort is unlikely to have produced more than 5000 children. The expected number of cases of retinoblastoma in 5000 children is 0·25 and the chance of three occurring is 0·002.

If these cases are related to exposure to radiation then one possible explanation is that the mothers, who had spent part of their childhood in Seascale, had accumulated radionuclides and had an increased rate of cell mutation. This could then lead to an increased risk of a germinal mutation being passed on to the next generation and to an increased rate of somatic mutation affecting their children during intrauterine life. The mothers could have been exposed to radiation in the general environment or in contaminated homes. If this is the correct explanation it has important implications for the pathogenesis of other childhood tumours in this locality, and possible ways in which nuclear power workers contaminate their homes need urgent investigation. A large scale study of the incidence of retinoblastoma in the offspring of those who work in the nuclear industry is also required. This study will need to include not only the workers' children but also their children's children.

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(Accepted 10 July 1990)