detection correlated significantly with the number of pyrexic episodes experienced during subsequent treatment. Such a relationship could not be detected for total immunoglobulins.

We accept that there are large variations in the distribution of immunoglobulins in children suffering from acute leukaemia, but feel our data suggests a functional importance of specific anti core glycolipid antibodies in this disease.


Relative occurrence of neuroblastoma and Wilms' tumour in ethnic subgroups in the West Midlands Health Authority Region

Str.-There have been several reports commenting on the apparent rarity or indeed absence of neuroblastoma cases in Zaire and surrounding African countries.1 The discussion has importance not only in assessing the aetiological significance of the observation for the countries concerned but also for studies of the relative rates of cancers in immigrant populations of other countries. Such studies have been revealing in adult cancers—for example, Japanese immigrants to the USA now show the spectrum of disease endemic to the American lifestyle rather than maintaining the pattern seen in their native country.2 The implication of these studies is that, in adult cancers, most of which are carcinomas, environmental as opposed to genetic factors are more important in the aetiology. Similar studies are rare in paediatric 'solid' tumours but are important because childhood tumours (being typically embryonal or tumours of the central nervous system, with carcinomas occurring as only 3% of cases) may have different causes.

We have reviewed all cases of neuroblastoma and Wilms' tumour (252 and 175 respectively) occurring in the West Midlands Health Authority Region (WMHAR) during the period 1957-86, as part of the data collection of the West Midlands Regional Children's Tumour Research Group (WMRCTRG). All cases are subjected to review of the histology material by specialist paediatric pathologists in order to confirm the diagnosis.

Included in this review is an investigation of the ethnic origin of the patients. The

| Relative occurrence of neuroblastoma and Wilms' tumour by ethnic group in the WMHAR |
|---------------------------------|----------------|
| White  | Afro-Caribbean  | Asian  | Other  | Total  |
| Neuro-  | 236  | 161  | 8  | 252  |
| blastoma  | 236  | 161  | 8  | 252  |
| Wilm's  | 10  | 5  | 175  |
| tumour  | 10  | 5  | 175  |
| *Other=Chinese/Japanese/mixed.  |
| Tests of proportions: (1) Afro-Caribbean for neuroblastoma compared with Afro-Caribbean for Wilms' tumour: p>0.01; and (2) Asian for neuroblastoma compared with Asian for Wilm's tumour: p>0.05 |

WMHAR contains a relatively large number of immigrants of Afro-Caribbean and Asian extraction, although exact childhood population figures are not available. Confirmation of ethnic group of each patient was obtained from the hospital casenotes or by communication with the general practitioner. Where these sources were unhelpful, examination of the family name of the patient denoted Asian origin and the presence of a sickle cell test provided further evidence of Afro-Caribbean origin.

The results shown in the table show that, in our 30 year series, neuroblastoma was indeed rare in Afro-Caribbeans with only two cases seen (0.8%). By comparison, neuroblastoma was proportionally five times more common in the Asian group (4.0%). Furthermore, during the same time period there were eight cases (4.6%) of Wilms' tumour in Afro-Caribbean children, proportionally slightly more than were seen in Asian children (2.9%). The results for Wilms' tumour may be seen as a control in that they confirm that cases diagnosed in black children are successfully ascertained by the Registry.

As insufficient population data are available for children by ethnic group breakdown, we are limited to expressing these results as a proportion. This difficulty in producing specific population standardised data is thus not only a problem for Third World countries but also for UK registries.

The fact that neuroblastoma is rare in our series of Afro-Caribbean children resident in the West Midlands suggests that genetic rather than environmental factors may be contributing to its aetiology. However, further, more detailed comparisons will depend upon the provision of accurate data such as that of Dr Massabi et al.,1 and on the availability of more specific population data.

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Reduced sweating in Laron's dwarfism

Str.—We have recently shown that adult patients with growth hormone deficiency have a reduced sweat secretion rate when compared with age-matched controls.1 Their sweating was correlated to the serum concentration of insulin like growth factor 1 (IGF-1).2 The question remains, however, as to whether growth hormone itself directly influences the sweat glands, or whether its action is mediated through IGF-1.

We have examined sweating in a 22 year old man with Laron's dwarfism (height: 124.2 cm, Tanner stage of puberty 3). He presented with highly raised serum growth hormone values (850 mg/l) and unmeasurable serum IGF-1. He had a sweat secretion rate of 24.9 mg/30 minutes as measured with the pilocarpine iontophoresis test.2 After treatment with subcutaneous injections of recombinant growth hormone for four days (3 IU/day, Genotropin, KabiVitrum) the measurement of serum IGF-1 and the sweat test were repeated. IGF-1 was still undetectable and the sweat secretion rate did not increase (23.6 mg/30 minutes). Both sweat secretion values are much lower than the lowest reference value for a group of 18 to 40 year old healthy men (median: 112.7, range: 59.2-259.9 mg/30 minutes, n=17) as well as for a group of healthy boys in puberty (median: 110.3, range: 49.3-202.4 mg/30 minutes, n=39).

Thus our observation is in agreement with the hypothesis that the effect of growth hormone on sweating is mediated through IGF-1.

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