LETTERS TO THE EDITOR

Prevalence of asthma and wheeze in the Highlands of Scotland

EDITOR,—In their study on asthma and wheeze in the Highlands of Scotland Austin et al.1 performed prevalence tests on children in six geographical regions between April and June 1992.1 The prevalence of exercise induced bronchospasm was particularly high on the Isle of Skye which was one of the most rural of the areas studied. However, data from the weekly returns service of the Royal College of General Practitioners showed that new episodes of asthma in 5–14 year olds were almost three times greater in June compared with April in 1992. Both asthma and hay fever show annual and seasonal variation2 and are partly affected by the severity of the prevailing pollen season.

Clear-cut pollen the exercise testing was carried out simultaneously in each region, comparisons are likely to be unreliable.

A M ROSS
D M FLEMING
Royal College of General Practitioners
Birmingham Research Unit,
Lordswood House, 54 Lordswood Road,
Horbours, Birmingham B17 9DB

Dr Austin and Russell comment:
Dr Ross and Fleming raise an interesting point in relation to the exercise testing which we used to support our questionnaire survey carried out in March and April 1992. We know of no epidemiological study which has correlated the prevalence of bronchial hyperactivity and wheeze in the Highlands of Scotland. Arch Dis Child 1994; 71: 211-6.

Drs Ross and Fleming note that exercise-induced bronchoconstriction might therefore be influenced by the data of study.

We have therefore reanalysed our data to look for these effects. If exercise testing performed in June are omitted, the overall prevalence of exercise induced bronchial hyperactivity rises to 9-6% compared with 8.9% when data for June tests are included.

Although weekly general practitioner returns for new asthma episodes in school aged children show a rise in late June/July that is small compared with the peak rise in September.1 In Highland Region, figures from one general practice suggest that there is little difference for out of hours consultations for childhood asthma between April and June and although the numbers are low (four calls in April, one in May, and two in June) (figures

courtesy of Ardlanich Medical Practice, Inverness).

We believe it is unlikely that pollen levels had any major influence on our results. In the north of Scotland the pollen count is because of bad climate (pollen tends not to hang in the air of the prevailing winds, and pollen production is lower and occurs later than elsewhere in UK) and the relatively sparse vegetation, especially in Skye. In any case, the tests in Skye were performed on 8th May 1992, too soon to be influenced by the seasonal effects proposed by Ross and Fleming.

1 Asthma Information Agency. Seasonal variations in asthma. Factsheet 93/4. Available from Department of Environmental Sciences, St George's Hospital Medical School, London. (Data 1987/92 quoted from Royal College of General Practitioners weekly returns service.)

Cardiovascular malformations in Turner's syndrome

EDITOR,—We were interested to read the findings of Gotzsche et al who reported the prevalence of cardiovascular malformations and karyotype abnormality in Turner’s syndrome.1 We have recently reviewed the karyotype and phenotypes of 63 Turner’s syndrome patients who have been referred to our department. Cardiovascular malformations have only been recorded in seven of them (2/14 45 X, 2/4 45 X/47 XXX, 2/2 45 X/46 XY, and 1/3 45 X 46 X(X) patients). Like Gotzsche et al we did not see cardiovascular malformations in any patient without a 45 X cell line and the chromosomal abnormalities accounted for most of the cases. If, as Gotzsche suggests, around 26% of Turner patients have cardiovascular abnormalities then it is likely that some of our older patients who have not undergone routine echocardiography have as yet unrecognised cardiac problems. This is an important point, as minor aortic abnormalities may be associated with dissections of the aorta in Turner’s syndrome.2 Adults3 and this complication could be screened for in susceptible individuals.

Gotzsche et al concluded that ‘no patient with structural abnormalities of the X chromosome had cardiovascular malformations’. This is true for their 13 non-mosaic patients but in fact 4/46 of their patients with cardiovascular problems did have a cell line containing a ring or t(X). It is likely therefore that these four individuals began life with a non-mosaic karyotype and that a 45 X cell line arose later as the abnormal X chromosomes were lost during cell division. It seems, therefore, that it is the presence of a 45 X cell line at an early stage of development that might influence whether a cardiovascular abnormality is likely to be present or not.

Why should this be so? It has been suggested that a critical determinant of both the X and the Y chromosome and which escapes X inactivation may be responsible for the somatic features of Turner’s syndrome.2 An individual must possess two functioning copies of this gene in order not to manifest Turner’s syndrome characteristics. Structural abnormal X chromosomes may still express this gene which is positioned to lie on the long arm of the X at Xq13, close to the centromere and the X inactivation centre. Only a single copy of this gene can be expressed in cells which contain a 45 X cell line. This may play a role in the causation of cardiovascular malformations or a secondary role because its absence gives rise to lymphoedema.4 In our study lymphoedema was documented in 79% patients with only a 45 X cell line and in 33% patients with structural X chromosome abnormalities.

We are aware of a report by Gotzsche and other authors who have suggested an increased prevalence of cardiovascular malformations in Turner’s syndrome patients with a 45 X karyotype. However, although the incidence of cardiovascular abnormalities is low in patients with non-mosaic structural abnormalities, patients who are mosaic for ring (X) or iso (X) with a 45 X cell line or who have X chromosomes or markers lacking the Xq13 region may still be at an important risk of heart problems and should be screened for these.

CATHarine PARCHMENT
University of Manchester Medical School, Oxford Road, Manchester M13 9PT

Growth hormone and hypophosphataemic rickets

EDITOR,—There has been some interest in the use of growth hormone in children with hypophosphataemic rickets who have short stature as a consequence of their metabolic bone disease. In addition to its growth promoting role there is the theoretical advantage that growth hormone by increasing the tubular reabsorption of phosphate may lead to a reduction in phosphate supplement requirements which have been linked to the development of nephrocalcinosis.

We would like to report our observations on the relationship of renal phosphate handling and markers of bone turnover in a 7 year old girl with hypophosphataemic rickets treated with growth hormone over an initial seven month period. She was originally referred for an endocrine assessment because of her short stature. Although she was exceedingly short for her age (height SD score −3.9), there was no evidence of deformity of her legs and no rickets. An invasive bone test with adequate hypoglycaemia demonstrated a peak growth hormone response of 7.5 μg/l (15 mU/l). In view of this and her short stature it was decided to give her a therapeutic trial of growth hormone in a dose of 0.6 IU/kg/day which increased to 0.75 IU/kg/week after three months given as six daily injections/week.

Over a seven month period she gained 4.4 cm in height, her height SD score improved from −3.9 to −3.7 and height velocity SD score from −1.8 to −0.1. On the same doses of phosphate and vitamin D her plasma phosphate rose from 0.99 mmol/l to 1.37 mmol/l in three months and then dropped to 1.02 mmol/l at seven months. The level of TmPO4/glomerular filtration rate increased

Drs Austin and Russell comment

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