

# Growth in atopic eczema: a controlled study by questionnaire

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**SUMMARY** The parents of 128 children with atopic eczema and 117 healthy control children responded to a questionnaire that included measurement of their own and their children's heights at home after standardised instructions. When cases and controls were compared there was no significant difference in parental height but the children with eczema were significantly shorter than the healthy control group. This difference remained significant when children with associated asthma were excluded from the analysis.

Numerous authors have examined the growth pattern of asthmatic children but,<sup>1-7</sup> to our knowledge, only one study has addressed the question of growth in atopic eczema.<sup>8</sup> This study was not controlled and did not fully exclude the effect of concurrent asthma.

We have used a questionnaire to obtain the heights of children with eczema, matched non-eczematous controls, and the parents of these two groups. The questionnaire was also used to establish whether concurrent asthma was present in order to identify a group of non-asthmatic eczematous children for separate analysis.

## Patients and methods

The parents of all 148 children who had been seen in one consultant dermatologist's clinic at the Hospital for Sick Children, Great Ormond Street, with a diagnosis of atopic eczema between 1982 and 1985 were sent questionnaires. Each family was sent an envelope that contained two smaller sealed envelopes enclosing questionnaires for the individual child and for a control to be selected by the parents. A letter instructed the parents to give the control questionnaire to a friend with a child of approximately the same age as their own child but who had never had eczema. They were instructed to do this before opening the envelopes containing their own or the control child's questionnaire.

The questionnaire included questions about paternal employment and the presence or absence of asthma. The parents were asked to indicate the date of birth and heights of themselves and their

child. Instructions as to the measurement of height were as follows: 'It is *very important* that the heights are *very accurate*. Please take shoes and socks off. Stand straight with heels and back against a wall. Someone else should then put a large firm book on your head as flat as possible and draw a line on the wall where the book touches it. Measure the distance from the floor to the line on the wall as carefully as you can with a tape measure. You can use feet and inches or metres and centimetres. Please do not include stepmothers or stepfathers'.

Where no response was received to a questionnaire it was followed by a second letter.

Height standard deviation (SD) scores were calculated using the formula

$$\text{SD score} = \frac{x - \bar{x}}{S_x}$$

where  $x$  is the height measured,  $\bar{x}$  the mean height for the age of the individual concerned and  $S_x$  is the standard deviation for height at a given age.<sup>9 10</sup>

## Results

### RESPONSE

A total of 245 replies were received: 128 cases and 117 controls (83% of the total 296 sent). Not every reply was complete: of the replies from parents of children with eczema, 115 indicated the child's height, 125 the mother's height, and 124 the father's height; of the replies from the parents of controls 110 indicated the child's height, 110 the mother's

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Table Comparison of children with eczema and controls with respect to parental and children's SD scores for height: (a) all children, (b) children without a history of asthma, antiasthmatic medication, or oral steroids, and (c) children over 5 years without a history of asthma, antiasthmatic medication, or oral steroids

	Children with eczema		Controls		Difference by <i>t</i> test ( <i>p</i> value)
	No	Mean SD score (SE)	No	Mean SD score (SE)	
<b>(a) All children:</b>					
Mothers	125	0.2505 (0.102)	110	0.2777 (0.106)	NS
Fathers	124	0.1622 (0.094)	111	0.2025 (0.115)	NS
Children	115	-0.4505 (0.119)	110	0.0595 (0.097)	<0.0005
<b>(b) No history of asthma, antiasthmatic medication, or oral steroids:</b>					
Mothers	55	0.2429 (0.149)	108	0.2877 (0.108)	NS
Fathers	55	0.2118 (0.138)	107	0.2059 (0.116)	NS
Children	52	-0.4465 (0.177)	107	0.0518 (0.099)	<0.01
<b>(c) Aged over 5 with no history of asthma, antiasthmatic medication, or oral steroids:</b>					
Mothers	25	0.4416 (0.221)	71	0.2204 (0.132)	NS
Fathers	25	0.1408 (0.226)	70	0.1546 (0.130)	NS
Children	22	-0.7232 (0.346)	70	0.1416 (0.123)	<0.005

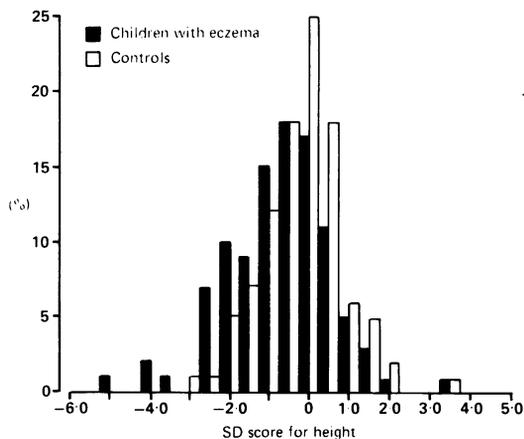


Figure Distribution of SD scores for height for children with eczema and controls.

height, and 111 the father's height. The mean age of the children with eczema where there was a response was 6.9 years (range 1.2–16.2 years) and of the controls 7.0 years (range 1.1–16.5 years), there being no significant difference between the two. Fifty percent of children with eczema and 52% of controls were boys. There was no significant difference in social class as judged by paternal employment between the two groups ( $\chi^2=1.43$ ,  $p>0.5$ ).

#### HEIGHTS

There was no significant difference between the SD score of the parents of either group. The mean SD score of children with eczema was significantly less than that of controls, which was itself close to that of the population mean (table (a) and figure). Though the difference was not significant, the parents of cases were shorter than the parents of controls (table (a)). Analysis of covariance was used to correct for this difference and the regression intercept coefficients showed a significant difference between children with eczema and controls allowing for the difference in parental height ( $p<0.0005$ , *t* test).

Fifty seven children with eczema answered negatively to questions relating to the presence of asthma and to the use of antiasthmatic medication or oral steroids at any stage in their lives. Comparison of the SD scores of these families with those of the control group who also responded negatively to these questions again produced no significant differences between parents but significant differences between the children (table (b)). When the age of this subgroup was considered, the children with eczema (mean age 5.7 years) were found to be significantly younger than controls (mean age 7.1 years) ( $p<0.05$ , *t* test). Analysis of covariance was again used confirming that the difference in SD scores between cases and controls remained significant ( $p<0.01$ ) after correction for differences in age and in parental height.

As the onset of asthma is later than that of atopic eczema and an eczematous preschool age child without respiratory symptoms might subsequently develop asthma, the same comparisons illustrated in the table (b) were made excluding children under the age of 5 years. Again a significant difference ( $p < 0.005$ ,  $t$  test) in SD scores between children with eczema and controls was found that could not be explained on the basis of an age or parental height difference (table (c)).

## Discussion

Only one study to date has assessed the growth of eczematous children.<sup>8</sup> This detailed uncontrolled cross sectional study found that 10% of 89 patients had heights below the third centile and that, of the 69 patients between 2 and 9 years of age where correction for midparental height could be performed, 22% fell below the 10th centile and only 9% had a corrected height above the 90th centile. The children also showed delayed skeletal maturity, large head circumferences, a disproportionately shorter sitting height compared with subischial leg length, and a normal skinfold thickness. Increasing disease extent, steroid potency, and asthma severity were associated with decreasing height centile but none of these three factors could be excluded.

Our own study was also cross sectional and the measurement of height was performed by parents at home in a clearly less accurate fashion. We did not assess other anthropometric data (sitting height, skinfold thickness, etc). Our numbers were greater, however, and our study included a well matched control group who followed the same instructions as to measurement as the eczematous group. Hence, although our heights are individually less reliable as absolute measurements, the significant difference found between eczematous and non-eczematous children remains valid. Furthermore, using questionnaire answers, we were able to exclude the effect of concurrent asthma, a disease that is well known to be associated with an abnormal growth pattern.<sup>1-3 6</sup> Like the study reported by Kristmundsdottir and David,<sup>8</sup> our patients came from a tertiary referral centre and were therefore likely to be more severely affected than the eczematous population as a whole.

Our findings indicate that children with eczema, who do not have asthma, are shorter than genetically expected from assessment of parental heights. While most of our eczematous group were not very short, 12 were more than 2 SDs below the mean and four were more than 3 SDs below the mean. Of these 12 very short children, six had been diagnosed as asthmatic.

The growth pattern of asthmatic children has

been shown to be that of growth delay with a delayed pubertal growth spurt and a normal adult height.<sup>3 7 11 12</sup> The cross sectional nature of our study prevents us establishing whether eczematous children follow a similar pattern. Ferguson *et al.*, comparing asthmatics with children with allergic rhinitis and finding no difference, have suggested an underlying abnormality of growth pattern related to the atopic state rather than to asthma itself,<sup>13</sup> and our findings are consistent with this hypothesis.

While growth delay may be the most likely cause of the height difference we have observed, alternative explanations for this include: (i) an effect of topical corticosteroids, (ii) nutritional factors, and (iii) an effect of sleep disturbance. It is extremely difficult to quantify lifetime topical corticosteroid use and difficult to separate its influence on growth from the influence of the eczema itself, as children with more severe eczema tend to have had greater topical corticosteroid exposure. Nutritional factors might play a part as gastrointestinal function is known to be abnormal in a proportion of eczematous children.<sup>14 15</sup> Growth failure due to transcutaneous protein loss has been reported in eczema,<sup>16</sup> and failure to thrive has been shown to result from inappropriate exclusion diets,<sup>17</sup> and eczema is frequently treated by dietary modification. The normal weight and skinfold thickness observed by Kristmundsdottir and David,<sup>8</sup> however, do not suggest a nutritional aetiology. Sleep disturbance due to pruritis is a well recognised feature of atopic eczema and might adversely affect the secretion of growth hormone and gonadotrophins which, in the normal circadian rhythm, is principally nocturnal.

Controlled longitudinal studies of non-asthmatic eczematous children are needed to establish whether the difference we have observed represent an effect of the disease or its treatment, or simply a physiological growth pattern associated with the atopic state.

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