common in patients with coeliac disease.¹⁴ Even without reliable figures on the incidence of thyroid disorders, these also appear to be more common in patients with coeliac disease. Similarly, the number of patients with ulcerative colitis was greater than expected. The other individual associations may well have been fortuitous. No patients were seen with vitiligo,⁸ Addison's disease,^{15 16} Sjögren's syndrome,¹⁷⁻¹⁹ or amyloid²⁰ or idiopathic pulmonary haemosiderosis,²¹ apart from one patient with Addison's disease who died before the introduction of jejunal biopsy and was therefore excluded from the study. Also among the excluded patients were six with chronic rheumatic heart disease.²² The association between coeliac disease and scleroderma has not been reported, that with Crohn's disease has been reported twice,^{23 24} and that with diabetes and thyrotoxicosis only once.²

These associations may possibly be explained by a genetic link through HLA genes, since coeliac disease is associated with HLA-B8^{25 26} as also are diabetes mellitus,^{27 28} thyroid disease,²⁹ and allergic alveolitis,³⁰ although other disorders such as inflammatory bowel disease are not.³¹ Various autoantibodies are common in the serum of patients with coeliac disease^{7 8 32 33} and may be related to the jejunal mucosal damage. Scott and Losowsky,¹ using as an analogy the lesions of dermatitis herpetiformis, suggested that circulating immune complexes originating in the small intestine might cause other disorders by deposition in the appropriate organ, the antigen being either a product of gluten or derived from the abnormal jejunal mucosa. In the present series the time spent on a normal diet was much greater than that spent on a gluten-free diet; thus not surprisingly most of the autoimmune disorders developed while the patients were on a normal diet and when the small bowel was damaged. Some did develop, however, when the jejunal mucosa was macroscopically normal. There was no evidence that the autoimmune disorders other than insulin-dependent diabetes¹⁴ improved with a gluten-free diet, as has been suggested.¹

Hodgson *et al*⁷ suggested that atopy in coeliac disease is due to either the passage of antigens across the damaged mucosa or predisposition as a result of abnormal mucosal IgA responses, for it has been claimed that selective IgA deficiency predisposes to atopy.^{34 35} Although in our series atopy was not common, the dramatic and persistent relief of eczema with a gluten-free diet³⁶ seen in two patients suggests that jejunal mucosal abnormalities do predispose to atopy in some cases.

Our experience suggests that jejunal biopsy should not be performed routinely in cases of autoimmune disorders, as has been recommended,¹ but should be performed in any patient who also has folate-deficiency refractory anaemia or diarrhoea.

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(Accepted 13 December 1977)

Diet and retarded growth

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British Medical Journal, 1978, **1**, 539-542

Summary and conclusions

The diets of 36 children below the third centile for height but with no organic disease were compared with the diets of a control group. In most cases retarded growth was associated with a long-continued deficiency in calorie intake. When the diets were reassessed about a year later the shortfall in calorie intake was significantly reduced.

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This improvement, which tended to be followed by an increase in the rate of growth in height, might have been due to alteration in the child's circumstances or improvement in the family attitudes and feeding habits or both. Advice given at the clinic is thought to have played a part in bringing these changes about.

Introduction

We previously discussed our hypothesis¹ that short stature in otherwise healthy children is associated with a long-continued deficiency in calorie intake. We now report on a new series of children under the third centile for height and compare their diets with those of a control sample representative of Bristol children. Most of the children were followed up over several years.

There are two reasons why short children might eat too little.

Either not enough food is available, because of poverty or mismanagement; or the child does not eat enough of what is available, because of poor appetite or other disorder in eating habits. In our series the second reason predominated.

Several findings support our hypothesis. Few of the many tests for metabolic or endocrinological disorder that we have applied have indicated disease,¹ and most of the mothers have told us of the small, selective, and capricious appetites of their children. The mothers recorded for us systematically, at the time of feeds or immediately after, everything their children ate during one week; assessment of the calorie content of what was eaten each day gave low totals in most cases. Catching up in height, increase in the calorie content of the diet, and reported improvement in appetite tended to be associated.

Since tallness and shortness tend to run in families, height has been widely assumed to be determined genetically. The higher coefficient for intra-pair correlation for height in monozygotic than dizygotic twins and the similarity of the coefficients in separated and non-separated monozygotic twins have been claimed as support for this assumption.² On the other hand, the increase in mean height in successive generations of children and adults in rich countries, the sometimes dramatic increase in mean height in the second generation of immigrants into rich countries, and the greater mean height of adults and children in the more affluent social classes³ all argue in favour of the view that improvement in general health and perhaps especially nutrition brings with it an increase in height. Infancy is probably one of the critical periods when environmental programming of human growth takes place.⁴

Organic disease—Illnesses and disorders of many kinds may retard growth.⁵ They include diseases affecting absorption from the gut, renal infection, heart lesions, fibrocystic disease, and chromosomal or genetic disorders—for example, Turner's syndrome. Growth-hormone deficiency or other endocrine dysfunctions have been incriminated in a few cases. Anyone studying short children must look for an organic disorder, especially in children at or below the first centile for height. Nevertheless, demonstrable organic causes are rare. In the large Newcastle study⁶ organic disease was found in only a minority of cases, and of the 23 such children detected only three had asymptomatic disease. One had a chromosomal abnormality, one chronic pyelonephritis, and one probable growth-hormone deficiency. True growth-hormone deficiency occurs once in 30 000 births and is therefore extremely rare, even when both idiopathic deficiency and that due to intracranial disease are included.⁷ We have excluded from our series any child whose short stature might have been caused by organic disease.

Emotional deprivation—Emotional deprivation has been accepted as a cause of retarded growth. Various mechanisms have been suggested, including dietary deficiency. The children in our series came from families of many kinds; we refer below to some of their common characteristics. None of the children were as deprived as those described in papers on severe emotional deprivation as a cause of stunting.⁸ The poor social conditions suggested as playing an important part in some cases of short stature⁹ were not systematically investigated in our series, but only a few of the children were judged to have been so affected.

Subjects and methods

Clinical series—All the children attending our growth clinic between June 1972 and March 1977 were included in the study if at the time of the first dietary assessment they were aged over 2 and under 15 years and were below the third centile for height according to the standards of Tanner and Whitehouse.¹⁰ Most of the children had already been investigated and those with obvious organic disease excluded. We further excluded one with Turner's syndrome, one with fibrocystic disease, one with a subthyroid disorder, one with microcephaly, one with hypochromic anaemia, and one who had been taking prednisolone for asthma for several years. There was no case of coeliac disease, although this had been expected and looked for. Thirty-six children satisfied our criteria, of whom 25 were boys and

11 girls. The preponderance of boys probably resulted from the complex processes concerned in referral to a growth clinic. Shortness in boys is likely to cause more worry for child and parent than that in girls.

Control sample—A representative sample of 75 children aged over 2 and under 15 was assembled from a register kept by the Bristol health department. A letter and forms for recording diet were sent to the parents. Forty-five forms were returned but five were incomplete and were therefore excluded.

Investigations—The children in the clinical series were examined with special regard to signs of puberty. In 35 cases bone age was estimated from radiographs of the wrist according to the standards of Tanner and Whitehouse.¹⁰ Other tests carried out as routine included urinary investigations and measurement of serum thyroxine, serum and haemoglobin folate, blood urea, and blood electrolyte concentrations. Buccal smears were examined for sex determination (with chromatin counts when necessary). Growth hormone, measured systematically in our first series, was measured only occasionally, in atypical cases especially. Any hint of organic disease was investigated further—for example, by means of jejunal biopsy for suspected coeliac disease. Nothing of interest emerged except in the cases listed above.

Dietary assessment—Each mother in the clinical series was interviewed by the dietitian to get a description of the child's appetite and eating habits. She was then told how to record immediately everything the child ate, whether at mealtimes or not, during seven consecutive days. A considered estimate of quantity was requested, such as number of tablespoons or slices, but not weighing of food. From the record the dietitian estimated the mean daily calorie content of the diet and intake of protein in grams. The diets of the children in the control sample were assessed in the same way from the records returned. In the clinical series any dietary shortfall was moderate; the obvious clinical effects described in severe malnutrition in developing countries were not relevant, though such malnutrition may have lasting inhibitory effects on body size and slow the rate of skeletal maturation.¹¹ The children in our series, although small for age, presented as healthy and lively; many were good-looking. Their deficit was in height for age, not weight for height,¹² although their position on the percentile scale for weight was in general similar to that for height.

Results

Comparison of clinical series with control sample—Table I gives the mean daily calorie intakes in the two groups of children. The method of dietary assessment was calibrated by calculating from the findings in the control sample a regression equation for age and calories—namely, expected calories (1325 kcal) + 2.8 × age in months (1000 kcal is 4.18 MJ.) This equation was applied to each child in the clinical series to give the calories expected for a child of his age. Student's *t* test was then used to see whether the values for observed minus expected calories differed significantly from zero. The overall result for the clinical series was as follows: observed minus expected calories daily, mean −246 ± SD 445 kcal; *t* = 3.64; *P* < 0.01. The null hypothesis was thus disproved—that is, observed calories fell significantly short of expected calories. The mean difference was about 15%.

First and second dietary assessments—The diet was reassessed from time to time, a second assessment being made in 23 cases. The mean interval between the first and second assessments was 18 months. The mean shortfall in calories at the first assessment was 295 kcal, and at the second assessment 45.5 kcal. This reduction (249.5 ± SD 275.2 kcal) was significant (*t* = 4.19; *P* < 0.01), and the difference between the clinical series and the control sample was no longer significant. Statistical tests did not support the hypothesis that this improvement in diet was attributable to the increase in age. The increase in height alone accounted for only a small part of the increase in calories.

Rate of growth—The mean increase in height in the 12 months

TABLE 1—Mean daily calorie intakes in control and clinical groups

	No in group	Mean age (years) ± SD	Mean daily calorie intake (kcal) ± SD
Controls	40	7.6 ± 3.2	1579 ± 333
Clinical series	36	8.4 ± 3.3	1358 ± 350

Conversion: traditional to SI units—1000 kcal ≈ 4.18 MJ.

TABLE II—Details of children showing pronounced growth spurt and mothers' comments on appetite

Case No	Age at spurt	Growth in two-year period (cm)	Growth in previous two years (cm)	Dietary calorie assessments (kcal)			Mother's comments on child's appetite	
				1st	2nd	3rd	Appetite at time of spurt	Factor accounting for change
1	4y 1m	15.8		1245	1445		"Lazy"	"Tongue-tie operation"
2	9y 0m	17.0	12.6	<1000	1900		"Whole platefuls"	
3	11y 9m	18.8	9.1	1750	1950		"Good now," "enormous"	"Since seeing the dietitian" "Since his father's death" "Since moving away from her father"
4	12y 3m	17.9	10.0	1550	1950		"Better"	
5	13y 1m	18.3	7.2	1400	2460	2300	"Eats more"	
6	13y 8m	14.8	8.3	850	1350	1550	"Like a horse"	
7	13y 10m	15.3	7.8	1150	1335	1920		

Conversion: traditional to SI units—1000 kcal ≈ 4.18 MJ.

after the first dietary assessment was 5.8 cm (SD 1.7; n=23). A child on the third centile would be expected to gain 4 cm a year before the growth spurt of puberty. We think that the greater the calorie content of the diet the more rapid would be the rate of growth in children previously showing retarded growth. Coefficients of correlation for increase in height over various periods and dietary calories (observed minus expected) fell short of significance, however, although the trends were in the expected direction. One reason for the low value of the coefficients lies in the low reliability of the increase in height in successive six-month periods as shown by correlation coefficients.

Spurts in growth—Table II gives the details of seven children who showed a pronounced spurt in growth, which was arbitrarily defined as a gain in height in any two-year period of more than 11.6 cm—that is, twice the mean increase in the year after the first assessment. In each case the second dietary assessment (and in some cases a third) was made during the period of the spurt. In every case the diet had improved. We had thought it likely that a spurt in height would be preceded by some catching up in weight, but catching up in height and weight appeared to have been contemporaneous in these children. The figure shows the growth chart in case 3. This boy's growth spurted when he was 11, soon after he had started at a comprehensive school, which he said he liked, especially the dinners. The first signs of puberty, a few pubic hairs, were noted at 12 years 3 months. Until his start at the new school his appetite had been poor. Feeding difficulties had begun at 18 months of age with his refusal to accept solids; his growth had slowed at that time.

Puberty—In case 3 the child's eating habits probably changed before the onset of puberty. In cases 5 and 7, on the other hand, the growth spurt probably reflected the onset of puberty, since other signs of puberty appeared soon afterwards—for example, growth of pubic hair and, in case 7, enlargement of breasts and the menarche. In case 6 other signs of puberty did not appear for another two years. There was a general tendency in our series for puberty to be delayed, which allowed growth to go on longer. The growth of a girl in an earlier series, one of monozygotic twins, whom we continued to follow up, spurted when she was 14 years 9 months. She gained 17 cm in the next two years, which took her from below the third centile to above the 25th centile and within 2 cm of her co-twin, whose growth above the 10th centile had spurted at 13 years 6 months. The smaller twin had consistently eaten less than her co-twin.

Growth chart in case 3 (see table II).

Bone age—Of the 35 children in the clinical series whose bone age was known from radiographs of the wrist, 23 were below the 10th centile, three were below the 25th centile, four were below the 50th centile, and five were above the 50th centile. We found no other difference between the children, either retarded or not retarded in bone age. There was slender evidence to suggest that the growth of those not retarded in bone age slowed up relatively late. Retarded bone age is favourable, since it indicates that growth will continue longer.

Other factors—Three of the 36 children had been fostered, and in three other cases there was no father figure in the household. The birth weight of three children had been less than 2500 g. Six children had IQs below 85 (62, 65, 70, 70, 77, and 83), and five others had shown some delay in developing speech. Bedwetting had been a problem in three cases, and in one of them soiling as well. Poor sleep was reported in six cases. Eczema, asthma, or hay fever had been a problem in five cases. None of these factors was present in nine children.

Discussion

Feeding difficulties—Most of the mothers volunteered or readily agreed that there had been persistent feeding difficulties, displayed in a poor appetite, slowness in eating, or refusal to eat at mealtimes or refusal of all but a narrow range of foods. Most mothers dated fairly precisely the onset of the feeding difficulties and the slowing of growth, mentioning such exigencies as establishing bottle-feeding, weaning on to solid food, or getting the child to take food for himself; or illness (especially stomach or bowel upsets), removal to a new home, or other change in the home environment. About a third of the mothers reported that feeding difficulties had begun soon after birth. As a rule the dating of the slowing of growth was confirmed as reasonably accurate whenever information from hospital, clinic, or school records was available. Weights were much easier to get than heights. The significance of the events mentioned by the mothers as related to the onset of the slowing of growth is uncertain. In some cases the event mentioned was one of several occurring at about the same time. The mother who attributed a pronounced change in appetite to a tongue-tie operation (case 2; table II), for instance, had brought a new stepfather into the home a month earlier.

Diet—The quality of the diet varied. The protein content was poor in some cases but relatively good in others. There was no clinical or radiological evidence of any vitamin deficiency. There was a tendency to eat variable amounts between meals, crisps, chips, sweets, and chocolate often being mentioned. A few children told of a revulsion from food, like that in cases of anorexia nervosa. A few mothers had strange ideas about the nutritional needs of children. The food provided by most mothers was appropriate but the children did not eat it. The mothers had mostly tried hard in sensible ways to improve the child's diet.

Mother-child relationship—In most cases there had been other difficulties in bringing up the child, as noted above. The relationship of mother and child was disturbed to some degree in most cases, but the degree of disturbance was not noticeably different from that common among attenders at hospital clinics. As a group the mothers were highly anxious and insecure. Many of the children impressed us as unduly anxious and active, often

to the point of restlessness, or were flitting in play. Exchanges between mother and child tended to be reduced and to lack warmth. The mothers tended to express dissatisfaction with the child, although there was also some concern over the difficulties in feeding and his shortness. This was often attributed to inheritance, a short parent or grandparent being mentioned. The decision to seek advice had usually been made when comparison with other children had shown up the degree to which growth had lagged.

Advice given—At the first attendance we sought information about appetite, feeding habits, and diet. Once the dietary assessment had been made we discussed these matters at each visit in as relaxed a way as we could. We encouraged the mothers to give the child as wide a choice of food as possible at each meal in cafeteria or smörgåsbord style. Occasionally we gave advice about supplementing the diet, recommending special high-energy foods when appropriate. At one stage we embarked on a placebo-controlled trial of cyproheptadine 4 mg twice daily for six months as an appetite stimulant,¹³ but the results were poor and the trial was abandoned. We think that the increase in calorie content of the diets recorded at the second assessment was due, in part at least, to a change in the mothers' and children's attitudes as a result of the discussions at the clinic.

Conclusions

The hypothesis that retarded growth in otherwise healthy children is due to a long-continued deficiency in calorie intake gains some support from our findings within the limits set by the scope and methods of the study. The following recommendations are based on our experience over several years.

(1) The monitoring of development during childhood should include regular measurement of height and its recording on a chart.

(2) If growth lags, inquiries should be made into the child's feeding habits. Details of the diet should be obtained and

recorded, even when an accurate assessment of the calorie content cannot be made.

(3) Interest in these matters should not divert attention from the indications and investigation of organic disease, and the physician should be alert for other causes. An endless repetition of investigations for a physical cause of stunted growth, however, is likely to increase anxiety and be self-defeating.

(4) Relaxed, repeated discussion of any feeding problems or deficiency in the diet may be expected to bring about an improvement in many cases, though it may be many months before this shows. A sustained increase in the calorie content of the diet should result in more rapid growth.

(5) Some catching up often takes place. The pubertal growth spurt tends to occur, although it may be delayed. A spurt of this kind may be associated with a change in feeding habits and improvement of the diet. Among the many facets of general management this encouraging expectation should be discussed with the parents and child.

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(Accepted 13 December 1977)

Human leucocyte antigens and mixed lymphocyte reaction in severe pre-eclampsia

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British Medical Journal, 1978, **1**, 542-544

Summary and conclusions

Human leucocyte antigens (HLA) and mixed lymphocyte reactions (MLR) were studied in 38 women with severe pre-eclampsia and their husbands. Thirty-nine women with normal pregnancies and their husbands served as

controls. Thirty-three of the control women were matched for age and parity with members of the study group. Infants were studied when possible. HLA compatibility was increased in the pre-eclamptic group compared with matched controls and with theoretical estimates for possible matings.

The one-way MLR at delivery showed diminished response of maternal to paternal and cord cells in pre-eclamptic women. This reduced maternal reactivity in women with pre-eclampsia may have a role in the illness, and paternal/maternal histocompatibility may be a feature of the severe form.

Introduction

The fetus, which inherits half of its histocompatibility antigens from the father, is an allograft on the maternal host. Its survival remains an immunological paradox, and the cause of the major disease in pregnancy, pre-eclampsia, is still unknown. As immunogenetic factors may be relevant¹ we undertook a con-

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