Role of diet in treating atopic eczema: dietary manipulation has no value

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The eczema that so often occurs as part of the atopic state usually has its onset between the ages of 4 and 24 months. Thus the affected infant is born healthy and becomes ill so distressed mothers naturally ask what they have done to harm their child. Environmental factors probably do play a part in the expression of eczema as there is, for example, a low incidence in poor tropical countries¹ and the highest incidence in Britain is in socioeconomic group I.² Understandably therefore dietary factors come under scrutiny, although it is clear that they are not the fundamental cause of the problem. Even the most enthusiastic advocates of dietary manipulation claim that it is a suitable approach in only about 10% of children with eczema.³

Adverse reactions to food

If diet were significant there are two mechanisms by which it could exert an influence: it might be in some way deficient or the eczema could constitute an adverse reaction to something eaten. For both mechanisms it is easy to argue logically that breast feeding should be beneficial, but, although this argument was first put forward over 50 years ago,⁴ there is still no persuasive evidence that it is correct, with many trials producing contrary evidence.^{5,6} During the same decade Hansen claimed that infantile eczema was associated with a deficiency of the newly discovered essential fatty acids.⁷ This hypothesis has not, however, stood the test of time, although it has had something of a resurrection with the suggestion that patients with atopic eczema may be deficient in Δ -6 desaturase.⁸ This enzyme converts linolenic acid into γ -linolenic acid, allowing its further metabolism and incorporation into the phospholipid of the cell wall. γ -Linolenic acid is not plentiful in a normal diet but it is found in breast milk⁹ and oil from the seeds of the evening primrose (*Oenothera biennis*).¹⁰ Trials of the oil as a dietary supplement in atopic eczema have yielded conflicting results^{11,12} so that currently there is no conclusive evidence of its benefit.

Many people, both lay and medical, who advocate dietary manipulation assume that eczema is being caused by a food "allergy." Unfortunately the subject of food associated disease is bedevilled by unsubstantiated claims and often a complete misuse of the word "allergy," which should be restricted to describing only those reactions in which an immune response can be shown. Otherwise the term "adverse reaction" or "intolerance" should be used.¹³ That breast feeding

does not prevent the development of true food allergy has been clearly shown in a study following for seven years 1753 children fed initially on breast, soy, or cows' milk: the occurrence of allergy was similar in each group.¹⁴ The only immunological reaction that has been unequivocally shown to occur from food is a type I reagenic response mediated by IgE. Its characteristics are swelling like angio-oedema of immediate onset around the lips, burning of the mouth and throat, widespread urticaria, and an immediate recurrence on challenge with the offending substance. Undoubtedly children with atopic eczema do show a high incidence of true food allergy of this type and many have eczema¹⁵; the two conditions are, however, separate and there is neither a diagnostic nor therapeutic problem as the offending food is easily identified and excluded from the diet: indeed even infants will spit it out because of the intense burning sensation it often causes in the mouth.

Can IgE mediated reactions result in eczema?

No evidence exists that a response mediated by IgE gives rise to an eczematous type of eruption.¹³ Thus the hypothesis in infants who subsequently develop eczema that the gut wall provides a defective barrier against the absorption of macromolecules that give rise to IgE antibodies which in turn cause eczema seems to be flawed because eczema has never been shown to occur. In an investigation of 541 patients with atopic dermatitis 84 gave histories of cutaneous symptoms related to food, but whereas in the group with urticaria a good correlation was found among the history, the results of radioallergosorbent tests, and the results of a challenge, no such correlation was found in those who believed that their eczema was made worse.⁵ Unfortunately most studies of diet in eczema rely heavily on the apparent improvement after excluding an item, whereas the true test of an adverse reaction is the development of an exacerbation on double blind challenge.¹³ Theoretically an adverse reaction to food may be caused by a non-immunological mechanism. The commonest of these is psychogenic,¹³ which in the case of children with atopic eczema afflicts their parents. Local irritation of the lips can certainly be caused by some foods, such as tomatoes, citrus fruit, and cheese, but as with the reactions mediated by IgE, identification and exclusion does not present difficulties, although misinterpretation of the reaction

continued on page 1460

EDITORIAL COMMENT

The diagnosis of type I contact urticaria mediated by IgE is straightforward. Identifying a clear relation between one particular type of food and atopic eczema is much more difficult. There is very limited evidence in the form of double blind rechallenge that certain foodstuffs can affect atopic eczema. Such a relation might, however, be the exception rather than the rule. In most cases in which diet has been implicated in causing atopic eczema rechallenge has not taken place, although the double blind crossover

study described by Dr Atherton certainly gives suggestive evidence that in some patients diet may be implicated. Both authors emphasise that dietary manipulation may result in inadequate nutrition and therefore does not constitute a form of management that is without risk. Although the case remains unproved, when conventional management has not succeeded a place for dietary manipulation seems reasonable, but under careful supervision. As with all other forms of treatment if there is no clear improvement then the diet should revert to normal.

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Controversies in Therapeutics

David J Atherton
continued from page 1458

vocative role of a wider variety of foods. Hathaway and Warner reported their experience in a group of 40 eczematous children who were responsive to diet.⁷ Those not helped by simple empirical diets had been given increasingly restrictive diets until improvement occurred. Seven of the 40 children responded to excluding egg and milk alone, 30 responded to more restricted diets, and three improved only when given an elemental feed. An attempt was then made to identify the provocative foods, checked by double blind challenges. In addition to milk and eggs, commonly implicated foods included citrus fruits, colourings and preservatives, nuts, fish, wheat, tomatoes, lamb, chicken and soya, showing that unresponsiveness to a simple diet does not necessarily exclude a provocative role for foods in individual patients.

Conclusions

The evidence from published studies and our own experience, suggests that a proportion of children with atopic eczema will benefit from dietary elimination of selected foods. Several foods may need to be excluded, and currently available tests will not reliably identify them. The proportion of children likely to benefit is probably not less than a quarter and not greater than a

half. Whether this rate of response could be improved by developing more accurate methods for identifying provocative foods is an important but unanswered question. How long the benefit of dietary modification may be maintained is also unclear. Our practice is to consider a trial of a simple, empirical exclusion diet in any children below the age of 8 years in whom adequate topical treatment has been insufficiently beneficial.⁸ Children with atopic eczema should not be treated with diets as a matter of routine. Elimination diets should not be continued long term without good evidence of benefit, and their nutritional sufficiency should be carefully checked.

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Roger Allen
continued from page 1459

may persuade the parents to exclude unnecessarily from the diet any prepared foods containing such ingredients.

Food additives have also been blamed for exacerbating eczema, but as Lessof *et al* pointed out there is no logic in considering additives as a unified group, such is the wide range of chemicals included under this heading.¹⁵ The dye tartrazine has been incriminated most frequently in view of its effect in urticaria, but, although it releases histamine, a survey of the prevalence of reactions to food additives in 18 582 people failed to identify one in whom the challenge produced eczema.¹⁶

Conclusion

Dietary factors are not the cause of atopic eczema, and I think that any substantial evidence that they cause it to worsen is lacking. There is a risk that exclusion diets inexpertly used in children will in providing inadequate nutrition do harm,¹⁷ and I therefore see no grounds on which to recommend manipulating the diet as a form of treatment and strongly discourage my patients from doing so.

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ANY QUESTIONS

Sodium is said to increase the urinary excretion of calcium, and osteoporosis has been attributed partly to the salt content of the Western diet. If so can sodium bicarbonate be justified as an ingredient of a calcium supplement?

There is good evidence both in man and in rats that increased sodium chloride intake increases urinary calcium excretion and is associated with raised serum concentrations of parathyroid hormone. The initial effect is mediated at a renal level, where tubular reabsorption of calcium is closely linked to that of sodium. Susceptibility to osteoporosis is determined by age, sex, race, and a host of local bone and systemic factors, which include urinary calcium excretion and calcium intake.¹ While salt loading leads to osteopenia in animals,² there is as yet no direct evidence in man linking sodium intake to accelerated bone loss. It is illogical, however, to prescribe

calcium in a preparation that is likely to affect adversely calcium balance. Furthermore, sodium intake in Western communities is excessive. It probably contributes to an increased blood pressure with age³ and is likely in salt sensitive individuals to raise blood pressure in the short term. It seems unwise, therefore, to prescribe sodium bicarbonate as an ingredient of a calcium supplement when alternative preparations are available. — J PERRY, research registrar, D G BEEVERS, consultant physician, Birmingham

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