eczema appearing later to the most irritant areas. It seems reasonable to suggest that after a time, or with repeated exposure, an allergen initially producing an erythema or urticaria in the atopic patient ceases to cause urticaria but sets off itching and scratching manifesting as eczema.³ In practice, however, it is unusual to find atopic eczema starting other than per se and this perhaps offers some evidence in favour of immediate hypersensitivity to dietary allergens playing no great part in the pathogenesis of atopic eczema. Atopic eczema can, of course, worsen with an allergen that does not produce urticaria. Prevention or delayed appearance of cows' milk allergy may be helped by encouraging breast feeding in the early postnatal months, particularly in those with a strong atopic family background⁴ and in infants of low birthweight.⁵

There is obvious wisdom in withdrawing suspect, harmful dietary allergens but David¹ has illustrated the need for reintroduction to be carried out in hospital if possible. Unfortunately, prick tests and radio-allergosorbent tests cannot be relied on to obviate the occasional need for elimination diets.

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Dr David comments:

Urticaria and atopic eczema are both disorders which can occur after the ingestion of certain foods. The speed of onset of the adverse reaction is variable. Both disorders may coexist in the same patient, and the features of one may merge into those of the other. As yet, no laboratory investigation has been found to replace listening to parents. Clinicians who feel the need to equate 'allergy' with IgE mediated hypersensitivity become confused when they discover that only a few of the symptoms or signs are explicable on this basis. IgG4 antibodies to foods impress colleagues, and thereby confer a certain pseudoscientific respectability to the whole murky area of food intolerance, but their estimation is not likely to be of any practical value in the management of children with atopic eczema. The idea that allergic disease can be prevented by encouraging breast feeding is fashionable and attractive. The absence of any good supporting scientific evidence⁶ is most tiresome. It is neither possible, practical, nor rational to perform all food challenges in hospital, and to insist on this is tantamount to excluding the use of elimination diets in the treatment of atopic eczema. It would be a great shame if the small risk of severe anaphylaxis were used as yet another excuse by sceptics of food intolerance to shun elimination diets in children with severe, intractable atopic eczema.

Allergy is a minefield. Maybe the occasional explosion is not so surprising.

References

- David TJ. Anaphylactic shock during elimination diets for severe atopic eczema. *Arch Dis Child* 1984;**59**:983-6.
- Sampson HA, Jolic PL. Increased plasma histamine concentrations after food challenges in children with atopic dermatitis. *N Engl J Med* 1984;**311**:372-6.
- Sampson HA. Role of immediate food hypersensitivity in the pathogenesis of atopic dermatitis. *J Allergy Clin Immunol* 1983;**71**:473-80.
- Matthew DJ, Taylor B, Norman AP, Turner MW, Soothill JF. Prevention of eczema. *Lancet* 1977;**i**:321-4.
- Lucas A, McLaughlan P, Coombs RRA. Latent anaphylactic sensitisation of infants of low birth weight to cows' milk protein. *Br Med J* 1984;**289**:1254-6.
- Van Asperen PP, Kemp AS, Mellis CM. Relationship of diet in the development of atopy in infancy. *Clin Allergy* 1984;**8**: 525-32.

Prevalence of glucose-6-phosphate-dehydrogenase deficiency

Sir,

Glucose-6-phosphate-dehydrogenase (G6PD) deficiency is probably the most common X linked disorder in the world. It is well known to cause haemolytic crises and neonatal jaundice, and to occur more frequently in persons of Mediterranean, African, and Asian racial stock. Because we had encountered a number of patients with severe neonatal jaundice, we screened all babies (boys and girls) of African or Asian stock born at this hospital over a three month period. Consent was obtained from the mothers, 0.1 ml blood was taken by heel prick, and G6PD activity was assayed using a dye reduction method.

Altogether 234 (32%) babies were tested. Twenty two were of Asian descent and 212 were of African descent. Eleven of the 118 boys (9.3%) were G6PD deficient and four of the 116 girls (3.4%) also had the deficiency. The 15 G6PD deficient babies were followed up, and the deficiency confirmed at the age of 2 months. Five of the 15 had moderate neonatal jaundice but none required treatment and all were clinically well at 2 months of age. Of the 15 mothers of these babies, two proved to have a deficiency, but were unaware of any symptoms attributable to the condition.

A technical problem encountered was that if a baby's blood has a high reticulocyte count, the young red cell population may mask a partial (heterozygote) deficiency. Also, because the white cell count is high in neonates a deficiency may be masked; this can be resolved either by delaying the test until the white cell count is normal, or by centrifuging and discarding the buffy layer.

While we have been aware of the problem of G6PD deficiency causing neonatal jaundice we were, perhaps naively, surprised at the incidence in our health district, especially in girls. Because of the high positive yield and the implied prevention of serious complications, we now perform a G6PD screen on *any* baby, boy or girl, of African, Asian, or, Mediterranean stock who is jaundiced enough to require a serum bilirubin estimation in the newborn period.

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