LETTERS TO THE EDITOR

Vitamin A supplementation in developing countries

EDITOR—I was frustrated by the recent annotation on vitamin A supplementation in developing countries.1 My main complaint is the inability of Dr Filteau and Professor Tomkins to endorse vitamin A supplementation among children living in poverty.

The authors are concerned that 15 mg (50 000 IU) of supplemental retinol given at about 1-5, 2-5, and 3-5 months has an associated 11% increase incidence of bulging fontanelle among young infants.2 However, Dr infants in this study received more frequent doses of vitamin A than is likely necessary for clinical benefit 3; there was a higher chance of having unnecessary side effects among them. In a similar trial, using a single dose of 50 000 IU retinol among neonates, failed to demonstrate any significant adverse effects.3 Furthermore, Filteau and Tomkins incorrectly state that the bulging fontanelle represents vitamin A ‘toxicity’ when, in fact, this phenomenon is transient, and has no adverse effect on a baby (or parents for that matter). Do fever and injection site erythema represent toxicity from the diphtheria, pertussis and tetanus vaccine, or are they simply acceptable side effects of a beneficial treatment?

Most importantly, Filteau and Tomkins failed to cite two recent rigorous cumulative meta-analyses that demonstrated clear cut benefits of vitamin A supplementation.3,4 Both publications showed reduced childhood morbidity and mortality related to respiratory and diarrhoeal diseases among children in ‘developing’ countries. These data prove, through trial consensus, that supplemental vitamin A is safe, efficacious, and cost effective. Retinol supplements should be a part of the Expanded Programme on Immunisation (EPI). The longer we sit on the fence of inconclusiveness, the more children will suffer and die from preventable illness.

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5 West KP, Khayat SK, LeClerq SC, et al. T-cell receptor variable beta (V beta) chain repertoire in patients with Kawasaki disease. We are concerned that readers may reach the premature conclusion that the involvement of superantigens in the aetiopathogenesis of Kawasaki disease has been proved fact. The authors ignored a substantial body of evidence that does not support this view. The results of Abe et al implicating a superantigen in Kawasaki disease could not be confirmed by several laboratories.6–8 In our recent unpublished study we found no increase in the percentage of V beta2 cells in patients with Kawasaki disease; in addition, our analysis of T-cell activation markers in Kawasaki disease and superantigen-stimulated purified samples collected at different intervals showed no changes in the expression of HLA-DR or interleukin-2 receptor. Thus, we could demonstrate no evidence that our patients had been exposed to a superantigen.

Subsequently, a multicentre study confirmed our observations.4 Another recent study also found no evidence of exposure to superantigens in patients with Kawasaki disease,5 and several groups reported their inability to reproduce the results of Leung et al regarding the isolation of a new, toxic shock syndrome toxin-secreting Staphylococcus aureus in patients with Kawasaki disease.6,7 Even in the report by Curtis et al the majority of patients during the acute phase had a percentage of V beta2 cells in the normal range.

The possible involvement of superantigen in the aetiopathogenesis of Kawasaki disease is far from resolved, and a more balanced discussion of the evidence for and against it would have been helpful.

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Dr Curtis and Professor Levin comment: We share the concern that our study may be interpreted to investigate the role of superantigens in Kawasaki disease. Since our paper was submitted, conflicting data has been published concerning selective Vbeta usage in the disease. We believe this conflict is due to methodological differences, in particular the different time at which samples were taken in other studies. We observed that the detection of increased Vbeta expression is critically dependent on the timing of the investigation with respect to the onset of disease. Our study suggests that it is not possible to detect the rise in Vbeta2 bearing cells in patients studied early in the disease course. A similar finding was observed in Choi et al original study of staphylococcal toxic shock syndrome in which peak Vbeta2 expansion was observed 10–14 days after onset of disease. Choi et al proposed that the timing of sampling explained the inability to detect Vbeta2 expansion in three of the eight patients studied.

None of the studies cited by De Inocencio and Hirsh were the results from the acute patients analysed with respect to time after disease onset. In Sakaguchi et al study, patients in the ‘acute’ group (defined as under nine days) may have been studied too early to detect a change in Vbeta repertoire.