LETTERS TO THE EDITOR

Dr Filteau and Professor Tomkins comment: We regret that Dr Joel Ray interpreted our recent editorial to mean that we do not endorse vitamin A supplementation among children living in poverty. On the contrary, we are strongly supportive of improving vitamin A status of children but believe that giving capsules to infants is not the only, or necessarily the best, means of doing this. We feel that more attention should be paid to improving the vitamin A content of the diet—through fortification, nutrition education, or food processing—and to supplementing mothers which would have the added benefit of reinforcing clinical messages about the importance of breast feeding.

High dose vitamin A capsules, in association with the EPI or otherwise, continue to have a place in public health nutrition but there is some concern that there are legitimate concerns about the medicalisation of a nutritional problem and the ensuing reliance on drugs imported with foreign currency, rather than on local initiative and technology. Secondly, we maintain that issues of safety of capsules for infants have yet to be satisfactorily addressed. Although available evidence suggests that bulging fontanelle is indeed consistent with thiamine and vitamin A toxicity, some cases among young infants have been due to contaminated vitamin A oil or vitamin A capsules. Infringement of the infant's diet including breast milk with any vitamin A oil or capsules destroys the milk's vitamin A content.

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. However, the infants in this study were selected through a number of methods and were not truly representative of the population under study. The results of a randomized controlled trial on vitamin A supplementation in developing countries are dealt with in the recent meta-analysis.5 Furthermore, vitamin A is not given to infants in their first year of life. The infants may not have received adequate vitamin A before the study.


Dr Curtis and Professor Levin comment: We do not believe that it is necessary to investigate the role of superantigens in Kawasaki disease. Since our paper was submitted, conflicting data has been published concerning selective VB2 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 VB2 expression is critically dependent on the timing of the investigation with respect to the onset of disease. Our study suggests that it is impossible to detect the rise in VB2 bearing cells in patients studied early in the disease. A similar finding was observed in Choi et al's original study of staphylococcal toxic shock syndrome in which peak VB2 expansion was observed 10-14 days after onset of disease.1 Choi et al proposed that the timing of sampling explained the inability to detect VB2 expansion in three of the eight patients studied.

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Dr Filteau and Professor Tomkins comment

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