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Correspondence

Postmeasles blindness

SIR, The title 'Postmeasles blindness' of an editorial in your journal¹ attracted my attention recently after I had read with absorbed interest the painstaking research on this subject reported by Foster and Sommer from Tanzania in the same issue.² This paper goes a long way towards resolving the problem of the nature and relative importance of the interacting factors such as vitamin A deficiency, measles, and herpes simplex infection in causing all too frequently corneal ulceration in young African children.

Imagine my astonishment when I discovered that this paper was not even mentioned in the editorial! The reason for having an editorial at all appears to have been the very slight work of Kogbe and Liotet³ on tear changes in measles. The authors of the editorial quote only three reports on measles that are by no means typical of results on the subject reported from Africa in recent years. Without quoting any supporting evidence they come to the erroneous conclusions that 'there did not appear to be any correlation between the extent and severity of measles keratitis and nutritional status' and 'suggested that genetic factors contribute to the development of postmeasles blindness.'

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**I have to reject Dr McLaren's criticism of the editorial aspect of this matter. I commissioned an editorial from Dr Monnickendam and Professor Darougar on the paper by Kogbe and Liotet. They were not shown the paper by Foster and Sommer and therefore clearly, were unable to comment on it. The decision to produce an editorial on a particular paper is something I reserve the right to choose for myself as Editor. Editorials are not chosen to reward specially meritorious papers, and I agree that the paper by Foster and Sommer is absolutely excellent. That is why I published it. But editorials are intended to bring to the readers' attention some new or unusual feature which I personally regard as novel or intriguing or otherwise worthy of special comment.

—R J H SMITH.

SIR, In response to Dr McLaren's criticism of our editorial¹ we would like to make the following points.

(1) We agree with Dr McLaren that the paper by Drs Foster and Sommer about the causes of childhood blindness in Tanzania² is very interesting. We did not refer to it in our editorial because we were not aware of it.

(2) In our editorial we attempted to discuss the factors which lead to the development of postmeasles blindness (PMB). There are only a few papers on this subject. We did not discuss measles in undernourished children, since it is well known that it is much more severe in these children than in well nourished children.

(3) Malnutrition is common in Africa, Asia, and Central and Southern America, and measles is particularly severe in malnourished children. However, PMB is found in some of these countries and not others. In a study of children with measles living in a slum area of Hyderabad, India, PMB was not seen.³ In Africa PMB is common in some countries but not others. In Tanzania² measles was not a common cause of corneal ulceration, and measles alone did not cause blindness. In Kenya⁴ 10 out of 248 children (4%) with measles developed corneal complications, seven of whom developed large corneal erosions and three of whom developed exposure ulcers. In northern Nigeria⁵ measles was the precipitating disease in 63 out of 70 children (90%) with corneal ulceration. A survey of hospital patients with corneal scarring showed that measles caused 42% of all scarring and 54% of bilateral scarring. Corneal ulceration following measles has also been reported in children of African origin living in Haiti.⁶ Why do some children develop PMB while others in the same population do not? In Kenya it was reported that 'no association between the extent of severity of keratitis and nutritional status could be found.'⁴ Nutritional status was assessed in northern Nigerian children with and without corneal ulceration following measles.⁵ There was no difference between the serum prealbumin levels in the two groups of children, while serum retinol-binding protein levels were significantly higher in the children with corneal ulcers than in those without ulcers. These results suggest that the development of PMB was not directly linked with the degree of malnutrition or vitamin A deficiency in these children.

(4) There are two possible explanations as to why there are differences in the prevalence of PMB in different countries of the Third World and why some individuals in a population with a high prevalence of PMB develop the disease while others do not. The first is that there are differences in internal or host factors—i.e., genetic factors; and the other is that there are differences in external factors. The presence of PMB in Africans and its absence in Indians

suggests that genetic differences may be important. Possible environmental differences include secondary ocular herpes simplex virus (HSV) infection, which appears to be an important factor in PMB in northern Nigeria.⁵ However, it does not appear to be important in East Africa. In Kenya HSV infection was not found,⁴ while in Tanzania² it was found that, although HSV was associated with 10 out of 48 (21%) cases of corneal ulcers following measles, it caused only one case of blindness. The use of traditional medicines is another external factor which may affect the development of PMB.² The development of dry eye appears to be important in PMB. It has been reported that the frequent application of tetracycline eye ointment prevented the development of erosions and exposure ulcers in children with measles because the greasy ointment base prevented drying of the corneal and conjunctival surfaces.⁴ Nothing is known about the factors which lead to the development of dry eye following measles, but some individuals may have a genetic predisposition and external factors may be important.

(5) We attempted to show that at present we do not have a clear understanding of the development of PMB, and to indicate that further studies are needed.

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Postmeasles blindness.

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