beneficial effects of intravenous fat infusions in a child with cystic fibrosis. This disease is so diverse in its effects upon individuals that it is always difficult to be sure whether we are observing natural variations or results of therapy.

It was noteworthy, for instance, that the reduction in sweat sodium concentration was not maintained, despite continued infusions. Similar observations of lowered sweat electrolytes in cystic fibrosis have been attributed to treatment with anabolic steroids (Flensburg, 1970) and cloxacillin (Griffiths and Bull, 1972).

We question in particular whether the increase in essential fatty acids brought about by soya oil can account for the favourable course of the disease in this patient. The actual serum levels of linoleic and arachidonic acid are not given in the paper. Our own observations confirm that essential fatty acids levels are often below normal in cystic fibrosis, but they do not correlate with the clinical condition of the patient (J. Yassa, R. Prosser, and J. A. Dodge, unpublished). However, we did encounter frank clinical deficiency in one such infant who had been treated with an artificial diet lacking essential fatty acids, and in that child the characteristic rash, diarrhoea, and anorexia occurred only when gross deficiency had been reached. Indeed, clinical recovery occurred with treatment before any rise in serum essential fatty acids was observed. At that time the essential fatty acids accounted for only about 4% of total serum fatty acids (Dodge, Salter, and Yassa, 1975). This is in keeping with the observations of Hansen *et al.* (1963), who found that, though serum dienoic acids always accounted for more than 13% of total fatty acids in infants fed on formulae with an acceptable linoleic acid content, clinical deficiency symptoms occurred at an average blood level of only 5.6%.

It seems unlikely that minor degrees of essential fatty acid deficiency would impair prostaglandin synthesis, or give rise to the usual clinical features of cystic fibrosis. Nonetheless, the suggestion that a disorder of prostaglandin metabolism may underlie the transport abnormalities of cystic fibrosis is one which has obvious attractions, and is currently under investigation both here and elsewhere (Förström, Goldyne, and Winkelmann, 1974).

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Professor R. B. Elliott and Dr. P. G. Robinson comment as follows:

Dr. Dodge and co-workers make valid comments on the lack of correlation of essential fatty acid (EFA) deficiency with either the nature of the symptoms in cystic fibrosis or the clinical course. We agree that other treatments have altered sweat electrolytes, though in the case of Griffiths's observations, these concerned chloride, not sodium concentrations. We did not have a valid measure of EFA concentrations before starting treatment with Intralipid in the reported case. However, at the 11th month of treatment, which corresponds with the maximum rate of supplementation, the lowest sweat sodium, and the highest stool enzyme activity, the combined serum concentrations of arachidonic and linoleic acids was 37 mg/dl (our normals >29 mg/dl or approximately >12% of the total fatty acids). This contrasts with levels of 19 mg/dl EFA found in the child at 18 months of age when supplementation had been reduced. At this time the sweat sodium concentration was greater than at 11 months.

Our own more recent research (unpublished) in platelet aggregation, prostaglandin, and cyclic nucleotide production, makes us aware that simple EFA deficiency *per se* cannot be the origin of the symptoms of CF. The possibility that some other agent in the infusion mixture (which, for example, contains about 12% tocopherols) may be a contributing factor, is also under investigation. This does not, however, invalidate a possible therapeutic effect of EFA in the disease, and in particular, a partial correction of a possible disorder of prostaglandin synthesis.

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Role of house-dust mites in childhood asthma

Sir,

I should like to make two points in reference to the paper by Sarsfield and his colleagues on mite-sensitive asthma of childhood, published in the September 1974 issue.

Firstly, I agree that studies on asthmatic symptoms are notoriously difficult to control and that many factors apart from allergen exposure may influence symptoms. One very important variable that the authors have failed to control is the attention given to the child by the parents when introducing the rigorous avoidance

measures. Such attention may well make a considerable difference to the emotional component of attacks, particularly when there is an element of manipulation in the attacks.

Secondly, I would like to draw attention to Long's classic experiment (Long *et al.*, 1958) in which he exposed dust-sensitive asthmatic children while in remission in hospital to concentrated samples of house dust collected from their own houses. Their failure to provoke asthma attacks within the hospital setting surely indicates that factors additional to the allergic component are operative in activating or inhibiting bronchospasm. Purcell carried out an admirable study (Purcell *et al.*, 1969) in which he showed that when asthmatic children's parents went away and the children stayed at home, despite allergies, there were in a substantial number of cases no attacks during parental absence. Here one must similarly conclude that allergy is but one factor.

Thus, while I would agree with the author's conclusion that there is a necessity for a thorough search for offending allergens in the asthmatic child I should like to emphasize that there should also always be a full assessment of the emotional climate in the child's home.

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Dr. J. K. Sarsfield comments as follows:

On behalf of my co-authors, I should like to comment on the points made by Dr. Lask. By following our recommended avoidance measures, parental attention is directed not at the child, but at his environment, and these are preferably undertaken in his absence. We considered that any secondary psychic influences acting on the child were uncontrollable due to the nature of the measures under trial. The authors would welcome suggestions for blind placebo avoidance measures.

In the quoted 'classic experiment' of Long *et al.* (1958) over one-quarter of their asthmatic subjects had negative skin tests to house dust and an extremely crude provocation technique was employed. It is certainly possible, using recognized methods of bronchial challenge, to induce bronchospasm in hospitalized mite-sensitive subjects by inhalation of house-dust extracts (Miyamoto *et al.*, 1968). Finally, Purcell's study (Purcell *et al.*, 1969) was only able to show symptomatic improvement during parental separation in a highly selected, small subgroup of asthmatic children where emotional factors had already been predicted to play a major role.

In general paediatric practice the majority of asthmatic children will be greatly helped by appropriate therapy. Hence, the oft maligned parents can feel secure in their own homes and avoid giving excessive attention to their children.

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