If we hope to gain any knowledge about who may be safely given a normal diet, it is vital that we have at least a 'comparative' diagnosis based upon a standardized study.

We thank Dr. Hudson and Miss Clothier for their comments and also for the opportunity to reply to them.

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Urinary tract infection in children of preschool age

Sir,

The high prevalence of urinary tract infection reported in preschool children in Glasgow (Boothman, Laidlaw, and Richards, 1974) is interesting and also surprising. It contrasts with the lower prevalence found in a slightly larger Leeds survey published in the *Lancet* last year (Davies *et al.*, 1974) and also a London survey (Saxena, Collis, and Laurance, 1975).

The reason for the apparently high prevalence in Glasgow rests mainly on the finding of 6 boys with urinary tract infection out of 81 boys tested in the age range 3-23 months (prevalence 7.4%). We found only one case of definite infection confirmed by suprapubic aspiration, in 334 Leeds boys of similar age (prevalence 0.3%). Was the urinary tract infection of the 6 Glasgow boys confirmed by suprapubic aspiration and culture? If not we suggest the results may be misleading. Have the authors any other explanation for this high prevalence in Glasgow boys? A prevalence of over 7% in boys of 3-24 months would be most surprising. It does not fit in with the known prevalence of urinary tract infection in boys of other ages, ranging from 2.5% in neonates (Littlewood, Kite, and Kite, 1969) to less than 0.05% after the age of 4 years (Kohler, Fritz, and Scherstén, 1972; Kunin, Zacha, and Paquin, 1962).

We agree with Boothman *et al.* that voided urine specimens from children under the age of 1 year contain a large proportion of false significant or doubtful colony counts. In screening over 500 children aged 1-12 months we found that 6.5% had 3 or more consecutive significant or doubtful colony counts. However, this apparently high prevalence of bacteriuria was shown to be false in that suprapubic aspiration showed only one of those 32 children with apparent persistent bacteriuria to have true bladder bacteriuria. It is significant that we found more contamination in boy

infants than girls. Without suprapubic aspiration, urine bacteriology reports in infants are misleading.

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The authors reply as follows:

The high incidence of significant bacteriuria in small boys in our study is indeed surprising but there is little to suggest the possibility of false positive cultures except, perhaps, in Case 6 (Table V) (Boothman, Laidlaw, and Richards, 1974). When investigated at the renal clinic, 4 of the 6 boys had positive cultures and these were confirmed in 3 cases by suprapubic aspiration. Reviewing the 2 cases where positive cultures were not obtained at the clinic, it is interesting that one child had serious renal impairment and has since had recurrent infections. This case, together with Case 8 who was admitted with acute symptomatic bacterial pyelonephritis within a fortnight of having one positive and one negative culture, led us to wonder whether some cases of urinary tract infection with intermittent bacteriuria were being missed.

These doubts regarding the possible occurrence of false negative or false positive cultures only serve to highlight the problems which surround the definition, diagnosis, and prognosis of urinary tract infection.

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Essential fatty acids in cystic fibrosis

Sir,

We have read with interest the report by Elliott and Robinson (*Archives*, 1975) concerning the apparent

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