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Sir,

I would like to take issue with Dr Taitz's recommendations with regard to the use of soy based infant milk formulae.[1] I do not think Dr Taitz can have any idea how common gastrointestinal symptoms caused by milk intolerance are. This is because he demands 'partial or subtotal villous atrophy' and failure to thrive. I have performed many biopsies on children with this condition and I find that it is more often a cause of chronic gastrointestinal symptoms in children who are thriving and have normal villous morphology. I realise there is the problem of patchy morphological change, but in practice the villi are essentially normal in the thriving milk intolerant child. Sometimes colic is the predominant symptom, but this is not 'evening colic'—these infants get colic after every feed. Usually the children grow out of their symptoms, but there is much misery on the way and it is not adequate to say 'the diagnosis of this condition should not be made without careful evaluation by an expert in the field'. Many tests can be helpful in the diagnosis of milk intolerance, ie radioallergosorbent test, milk antibodies, jejunal biopsy etc, but I think the final arbiter is still the milk challenge, preferably more than one if the mother will permit this.

Soy protein is probably just as allergenic as cows' milk protein, but since it is introduced later, ie as treatment, it is not common in practice to get a second intolerance to the soy protein, though this has to be watched for. Its palatability is superior to that of the hydrolysates which is an important consideration especially with the older infants. Now that cheap soy based formulae are available and are known to be nutritionally satisfactory the management of these infants is much easier.

If an unweaned infant has symptoms due possibly to milk intolerance and is failing to thrive he should be referred to a consultant paediatrician for further evaluation. If the infant has such symptoms but is thriving, then the feed should be changed for 2 weeks to a soy based formula under a doctor's supervision (general practitioner or consultant). If there is no improvement the child should be put back on the original formula. If there is improvement or the situation is doubtful, then milk intolerance is possible and the infant should be referred to a consultant paediatrician at that point if the GP has begun the trial of the soy based formula. After one month a cows' milk challenge should be given preceded by a lactose tolerance test. Some cases are very clear and one challenge is enough to make the diagnosis; others are more difficult and two challenges or other tests may be necessary.

We are still waiting for a good test for milk intolerance comparable with the jejunal biopsy in coeliac disease or the sweat test in cystic fibrosis, but I fear we may wait a long time. Considering the variation in symptoms and the differing response times after milk challenge, there may be several immunological mechanisms at work.

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Dr Taitz comments:

The three letters written in response to my annotation present powerful arguments against my conclusions. On reflection, however, I do not believe that they fundamentally weaken the case against widespread use of soy feeds, or rather and more importantly, the over diagnosis of cows' milk intolerance.

Babies who fail to thrive warrant jejunal biopsy before beginning treatment. Proved cows' milk intolerance with failure to thrive and jejunal mucosal atrophy warrants optimal treatment. This condition is rare and the use of hydrolysed formula seems justified by the well documented reports cited in my review of soy intolerance including anaphylaxis which may occur particularly in these infants. Jejunal changes in the absence of failure to thrive but with gastrointestinal symptoms are in the same category.

Infants who have symptoms which may be due to milk intolerance but who are thriving and show no evidence of intolerance such as positive RAST or raised IgE values continue to pose problems. Most children who cry, are restless, have wind, colic and possetting but are thriving, do not have cows' milk intolerance and become normal in time. My annotation is intended as a corrective to a growing use of soy feeds as reflex action.

In an atmosphere of growing anxiety—some might say hysteria—about food allergies, we have some responsibilities not to add unnecessarily to the trend. Hence my plea for careful evaluation of all babies before diagnosing milk intolerance. The mere improvement of a symptom on changing feeds in itself proves nothing as there is such a high probability of spontaneous improvement or a placebo effect. Symptoms which are highly subjective in both nature and severity, cannot be confirmed by investigation, are self limiting, and may be due to multiple causes, are easily misdiagnosed with delay in identification of the basic problem. I have had to deal recently with the potentially serious consequences of maternal depression treated by changing the baby's feed from breast to cows' milk formula to soy. Even allowing Dr Miles's generous calculation of incidence, a general practitioner is likely to see one case of cows' milk intolerance in three years. Even paediatricians are not likely to encounter more than five cases a year. These considerations argue for circumspection in ascribing symptoms to milk intolerance with consequent over investigation, medical

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intervention and the labelling of a large number of normal infants with a condition they do not have.

Before embarking on such a course the baby should at the very least be observed, handled, and fed by experienced staff in a calm atmosphere, perhaps in a day care unit. This often resolves the problem and relieves the parents of much unnecessary anxiety. If this fails to resolve symptoms I concede that a trial with soy might be worthwhile in those few babies whose symptoms remain, but there is no convincing objective evidence that this is beneficial. Perhaps someone should try to find out.

Screening for cystic fibrosis

Sir,

In his commentary on our recent review paper[1] in which we advocate a programme of neonatal screening for cystic fibrosis (CF), Professor Peter Phelan rightly points out that data in the published reports are inconclusive. This is partly because of the difficulty in obtaining data which compare like populations. Professor Phelan himself compares the survival figures for 30 years ago (fewer than 10% alive at 5 years) when definitive diagnosis was often made at necropsy only, with major contemporary US and Australian statistics showing 80% survival to late adolescence. The Gibson-Cooke standardised sweat test was not available before 1959, and its widespread application has resulted in identification of milder cases and better understanding of the considerable variations in the natural history of CF which are independent of particular treatment regimens.

Our support for a neonatal screening programme is based as much on the value of a pre-symptomatic diagnosis to the individual parents and child as on the scanty useful incidence and mortality data in the reports.[2] The fact that the majority of newly diagnosed children with CF are born into families without a previously affected child is of no help to those parents whose eldest CF child is diagnosed only after the birth of the second. We are less sanguine about the long term outlook for CF patients than Professor Phelan and conscious of the physical and emotional cost at which the improving prognosis is achieved. New approaches to treatment are needed, perhaps with particular attention to early nutritional support. A recent paper from another Australian centre indicates how, even in patients with advanced CF, substantial benefit can be achieved by an aggressive nutritional policy.[3] We have not, contrary to the fear expressed by Professor Phelan, encountered parental rejection on the grounds of the diagnosis of CF. Our experience is that parents told (we hope sensitively, optimistically, but realistically) that their new born infant has CF nearly always form a very close bond with their baby: nor is rejection an observed phenomenon in the 15% or so who present with meconium ileus and are effectively self selected for neonatal screening.

We agree that measures of the progress of lung disease in early life are insensitive, and feel that Professor Phelan can not really be sure that his 39 patients with a family history, who were therefore detected early, were ascertained 'before the development of lung disease'. We also agree that the benefits of early diagnosis in a long term prospective study of survival may be small, but this is not a valid argument against such a study since only an unselected series can unequivocally show differences between centres and treatment regimens—the methods of selection for treatment at a particular centre being otherwise questionable.

The existence of a screening programme implies the existence of a regional centre for diagnosis with which a treatment centre is likely to be associated. Although Professor Phelan claims that 95% of all patients in Victoria are treated at his own (justly renowned) centre, simple population statistics suggest that this may not be so. In our own centre, which treats all patients from South Glamorgan (population approximately 400 000) and an equal number from other health authorities, about 55 are resident in the area. This gives a prevalence of 1:7500. The population of Victoria is about 4 000 000 and the clinic in Melbourne has 336 patients, ie a prevalence of 1:12 000 in a comparable ethnic group. Either the Cardiff survival figures are much better than those of Victoria (which we doubt) or about 30% of the Australian patients are being cared for, or dying undiagnosed, without reference to the regional centre. Perhaps there is therefore a particularly strong case for neonatal screening in Victoria, the results of which might well surprise us all.

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Professor Phelan comments:

In their letter Dodge and Ryley raise a number of important issues that warrant further comment. I am pleased that they agree that great difficulties are likely to be experienced in showing an improved outlook as a result of newborn screening for cystic fibrosis and they are almost certainly correct in predicting that demonstrable long term benefits of early diagnosis will, at the best, be small.

They suggest that parents appreciate early pre-symptomatic diagnosis. However, this remains to be proved by a properly conducted study. It is not reasonable to compare ill babies with meconium ileus with those diagnosed pre-symptomatically. It seems crucial to show, before newborn screening is widely introduced, that pre-symptomatic diagnosis is beneficial to parents and child and that it does not disturb developing family

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