

4. Jolly SS, Prasad S, Sharma R. Endemic fluorosis in India. *J Assoc Phys India* 1970,18: 459-471.
5. Jolly SS, Singh ID, Prasad S, Sharma R, Singh BM, Mathur OC. An epidemiological study of endemic fluorosis in Punjab. *Indian J Med Res* 1969, 57: 1333-1346.
6. Krishnamachari KAVR. Further observation on the syndrome of endemic genu valgum of South India. *Indian J Med Res* 1976, 64: 284-291.
7. Ground Water Quality and Health Survey of Fluorosis in Endemic Areas of Jhabua-a Case Study. A Project Report. Department of Civil Engineering, Shri G.S. Institute of Technology and Science, Indore, 1995.
8. Krishnamachari KAVR, Krishnaswamy K. Genu valgum and osteoporosis in an area of endemic fluorosis. *Lancet* 1973, 20: 877-879.
9. Teotia SPS, Teotia M. Endemic fluorosis in India: A Challenging national health problem. *J Assoc Phys India* 1984, 32: 347-352.

## Zinc Supplementation in Severe Malnutrition

**A. Vasudevan**  
**N. Shendurnikar**  
**P.V. Kotecha**

Zinc is an essential trace element and acts as a cofactor for more than 200 enzymes in the body. In view of the widely prevalent malnutrition, it is felt that the sub-clinical zinc deficiency could be existing in developing countries, contributing to intrauterine and postnatal growth retardation<sup>(1)</sup>. The objective of the present study was to determine the effect of zinc supplementation on serum zinc levels and weight gain in children with severe malnutrition.

*From the Departments of Pediatrics and Community Medicine, Medical College, Baroda 390 001.*

*Reprint requests: Dr. N. Shendurnikar, B/142, Jagannath Puram, Near Lalbaug Crossing, Baroda 390 011.*

*Manuscript received: December 12, 1995;  
 Initial review completed: December 22, 1995;  
 Revision accepted: October 7, 1996*

## Subjects and Methods

This study was designed as a double blind placebo controlled trial. The subjects were selected from the children attending the Outpatient Division of Department of Pediatrics, Medical College, Baroda, between the ages of 8 to 24 months and suffering from protein energy malnutrition (PEM) Grades III and IV(2). Other concurrent causes of malnutrition were excluded by history, physical examination and investigations and informed consent was obtained from the parents.

A total of 72 children divided into two groups (zinc supplemented and placebo) were recruited in the study. However, only 62 cases (31 in each group) could complete the designated follow up period of 3 months. The recruited children in the two groups were matched for age (within 3 months) sex, weight for age, socio-economic status and ethnic background. The initial evaluation included weight of the child and serum zinc analysis by calorimetric method using a kit obtained from Randox Laboratories, UK(3).

Twenty apparently normal, healthy children who were not malnourished or in any way ill and who were healthy siblings

or volunteers, were also analyzed for serum zinc levels. This was done to determine the normal range of serum zinc by the calorimetric method.

Subjects in the zinc supplemented group received 6.6 mg of elemental zinc; equivalent to 20 mg of zinc sulphate once daily. Both zinc sulphate and placebo were incorporated into similar looking capsules. Nutritional counselling was given to the parents and dietary intake adjusted to 100-120 calories/kg/day by instructing the mother and the intake was assessed at the end of the study by 24 hour recall method(4). Serum zinc was again estimated at the end of three months, allowing six days after the last dose of zinc prior to analysis. Statistical methods included "t" tests (paired and Student's).

### Results

The mean serum zinc levels were  $154.4 \pm 24 \mu\text{g/dl}$  and  $98.4 \pm 26.1 \mu\text{g/dl}$  in healthy and malnourished children, respectively ( $p < 0.001$ ). Children in zinc supplemented group showed a significantly greater increase in their serum zinc levels at the end of study as compared to the controls (Table I). However, there was no difference in weight gain between the two groups. None of the children in the study showed any obvious signs of zinc deficiency. None

of the children in the zinc supplemented group developed any side effects following zinc therapy.

### Discussion

Zinc deficiency underlying PEM is being increasingly recognized and this may occur concomitantly or become manifest during the recovery phase. We observed lower plasma zinc levels in malnourished children and these may result from increased gastro-intestinal losses, high fiber and phytates in diet and increased needs for the rapid growth. Several workers have studied the effects of zinc supplementation in children with PEM(5-8) and some of them observed a beneficial effect on weight gain(5-7).

Zinc status of the children has been reported to be related to their nutritional status. A recent study from Hyderabad observed satisfactory leukocyte and plasma zinc levels in normal and undernourished children, while cases with PEM had significantly lower levels, as a part of syndrome of PEM(8). However, the need for routine zinc supplementation in the rehabilitation for these children is not clear. This aspect was recently investigated and it was observed that there were no beneficial effects of zinc supplementation on weight gains, as compared to the placebo group(8). Similar results were also observed in our study. However, lower plasma zinc values in unsupplemented (placebo group) children indicate the relative inadequacy of zinc supply through diet alone to replenish zinc stores. Dietary zinc may perhaps be adequate to bring about a biochemical response, though it is inadequate to replenish zinc stores of deficient children(8).

Children recovering from PEM have a rapid growth and increased requirement for new tissue synthesis. In this setting, the dietary zinc may limit the absolute rate of

TABLE I—Comparison of Recovery Parameters.

Parameter	Zinc supplemented (n=31)	Placebo (n=31)	p Value
Change in Zinc levels ( $\mu\text{g/dl}$ ) (before and after study)	+ 51.3	+ 16.4	<0.001
Rate of weight gain (g/kg/day)	1.4	0.98	>0.1

weight gain and composition of new tissue. It would thus be prudent to ensure an adequate zinc intake during rehabilitation from severe malnutrition to match the demands of rapid growth.

#### REFERENCES

1. Bhaskaram P. Micronutrient deficiencies in children: The problem and extent. *Indian J Pediatr* 1995, 62:145-156.
  2. Nutrition Sub-committee of Indian Academy of Pediatrics. Report of the convenor. *Indian Pediatr* 1972, 9: 360.
  3. Homsher R, Zak B. Spectrophotometric investigation of sensitive complexing agents for the determination of zinc in serum. *Clin Chem* 1985, 31:1310-1313.
  4. Kapil U, Verma D, Chaturvedi S, Nayer D, Srivastava M. Methods for assessment of dietary intake. *Indian Pediatr* 1994, 31: 477-482.
  5. Golden MNH, Golden BE. Effect of zinc supplementation on dietary intake, rate of weight gain and energy cost of tissue reposition in children recovering from severe malnutrition. *Am J Clin Nutr* 1981, 34: 900-908.
  6. Walravens PA, Hambidge M, Koepter DM. Zinc supplementation in infants with a nutritional pattern of failure to thrive: A double blind controlled study. *Pediatrics* 1989, 83: 532-538.
  7. Srivastava SP, Roy AK, Azno UK. Zinc supplementation in PEM. *Indian Pediatr* 1993,30: 779-782.
  8. Hemalatha P, Bhaskaram P, Khan MM. Role of zinc supplementation in the rehabilitation of severely malnourished children. *Eur J Clin Nutr* 1993, 47: 395-399.
-