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## Efficancy multi-micronutrient supplementation on somatic growth and episodes and duration of morbidity among infants aged 6 - 12 months: a randomized placebo controlled trial

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## **Abstrak**

Nowadays, many children in developing countries are suffering from micro nutrient deficiencies (Lartey et at., 2000b; Zhao et al., 1998; van Stuijvenberg et al, 1999; Sempertegui et at, 1995). And the most prevalent ones are VAD (Vitamin A Deficiency), IDA (Iron Deficiency Anemia), IDD (Iodine Deficiency Disorder), and lately is zinc deficiency (Sandstead, 1991).

The causes of micro nutrient deficiencies are varied and include inadequate dietary intakes (Robert et al, 2000), repeated infections (Khanum et al, 1998) and poor bioavailability from foods due to the presence of inhibitors or inadequate intake of dietary enhancers (Berdanier, 1998; Lunnerdal, 2000; de Pee et al, 1998; Donnen et al, 1996; Lartey et al, 2000a).

Due to the roles of micronutrients in metabolic process, immune competence and taste acuity (Golden, 1995), previous findings showed that micronutrient deficiencies might impaired growth (Allen, 1994b; Rosado, 1999; Krieger et al, 1986; Simondon et al, 1996; Hambidge, 2000; Golden & Golden, 1981) and immune system (Black, 1998; Khanum et al, 1998; Semba et al, 1993). The reversibility of impairments caused by micronutrient deficiencies depends on the severity, duration, and stage of development. In some cases, micronutrient supplementation can correct the impairment right after a certain period of supplementation.

Ninh et al (1996) stated that zinc deficiency among nutritionally deprived children may limit growth because the growth stimulating effects of zinc might be mediated through changes in circulating insulin-like growth factor (IGF). And after 5 months supplementation with daily-10 mg zinc, weight and height of growth-retarded children in supplemented group significantly increased compared to those of placebo group. In Uganda, zinc supplementation had a short-term effect (within 3 months) on weight gain and MUAC increment only among children from the school with the highest socioeconomic status (Kikafunda et al, 1998). Clinical vitamin A deficiency has been associated with poor child growth (Tarwotjo et al, 1992). Study in Zaire among moderately malnourished preschoolers found that high dose vitamin A supplementation (60 mg of oily solution of retinal palmitate, 30 mg if aged <12 months) increased MUAC and weight significantly compared to control group although without deforming at baseline (Donnen et al, 1998). In Indonesia, the intervention using vitamin A-monosodium glutamate did not merely result on increment of serum vitamin A level, but it also increased the linear growth of supplemented children compared to children in control group at every age (Muhilal et al, 1988). Similar with zinc and vitamin A, iron deficiency may also lead to slowing of growth in regarding to the increment of iron demands during periods of rapid growth and the adverse effects of morbidity.