

## Supplementation with Beef or Milk Markedly Improves Vitamin B<sub>12</sub> Status of Kenya Schoolers

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*Based on food intake data collected on rural Kenyan children in Embu, Kenya in 1984-1987, a high prevalence of vitamin B<sub>12</sub> deficiency was found. Similarly, in 1998 baseline values for an intervention study showed a high prevalence of vitamin B<sub>12</sub> deficiency based on food intake and biochemical measures. In 1998, a two year controlled intervention with animal source foods was initiated in this same population to improve the micronutrient status of 6-9 year old rural Kenyan school children and test if animal source foods improve growth and cognitive function as well. Twelve schools were randomly assigned to three different but equicaloric food supplements. A local maize and bean stew (githeri) was used as the basic dish to which was added oil, beef, and milk respectively for each of the groups. Feedings were provided as a morning school snack 5 days/week. A control group received no school feeding. At baseline, 38% of children had severe and 30% of children had moderate vitamin B<sub>12</sub> deficiency. In years 1 and 2 respectively, plasma B<sub>12</sub> (ng/L) increased by 55 and 201 ng/L ( $p < 0.0013 - 0.0001$ ) in the meat group; 84 and 126 ng/L ( $p < 0.0013 - 0.0001$ ) in the milk group; and did not change appreciably in the added oil group or control groups. The increase of vitamin B<sub>12</sub> concentration in the meat group was significantly greater than in the milk group at the end of year 2 ( $p = 0.0017$ ). After 2 years of school feeding, severe deficiency in the meat group was nearly eliminated and greatly reduced in the milk group. Because vitamin B<sub>12</sub> was alleviated by supplementation with animal products, the cause of B<sub>12</sub> deficiency appears to be due mainly to low intake of animal source foods rather than to malabsorption due to infection.*

### Background

Vitamin B<sub>12</sub> deficiency is being found increasingly in a number of low income countries (LICs) (Allen et al, 1995). Vitamin B<sub>12</sub> deficiency in LICs may be caused by:

1. Low intake of animal source foods – Vitamin B<sub>12</sub> concentration is high in meat and milk and virtually non-existent in plant-based foods, which are the predominant staples in LICs (Institute of Medicine, 1998).
2. Malabsorption – Bacterial overgrowth, particularly with *Helicobacter pylori* infection, which are more common in LICs and may impair B<sub>12</sub> absorption from food (Allen et al, 1995).

Vitamin B<sub>12</sub> plays a key role in nervous system development and brain function (Graham 1992), in red blood cell formation and other functions, and is almost exclusively furnished by animal source foods (Institute of Medicine, 1998). Food consumption data collected quantitatively in Embu, Kenya from 1984-1987 revealed that vitamin B<sub>12</sub> and other micronutrients were deficient in the diet of school children (Murphy et al, 1995).

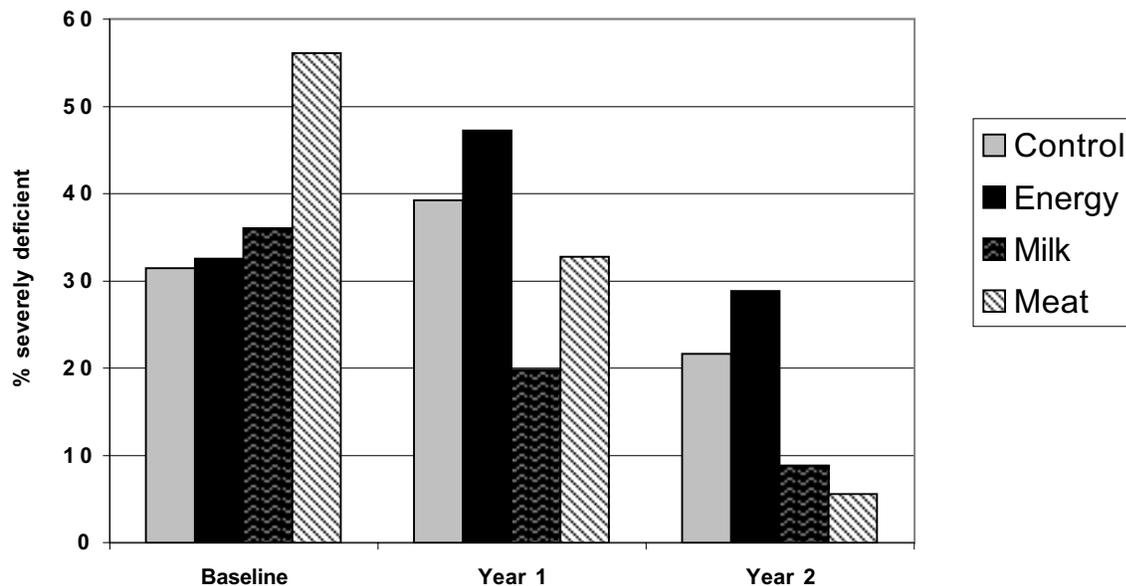
### Intervention Study: Design and Method

In 1998 a controlled school feeding intervention study was initiated to determine whether animal products, fed daily in school, could reduce micronutrient malnutrition in these rural Kenyan children. The main objectives of the study are to assess whether there exists a casual link between meat and with milk and micronutrient status, cognitive development, and behavior, physical growth and health of schoolers. Reported here is the effect of the dietary interventions on vitamin B<sub>12</sub> status. The hypothesis tested here is whether or not vitamin B<sub>12</sub> deficiency can be improved by supplementation with animal source foods, both meat and milk.

Twelve Grade I classes in 12 different schools (555 children) were randomized at the school level to one of four groups:

1. Non-intervention Control
2. Githeri (a maize and bean stew) with added energy (oil)
3. Githeri with milk (200 mL of whole cow's milk for the first year, then 250 mL/d in the second year)
4. Githeri with beef (60 g for the first year, then 85 g/d for the second year)

Figure 1. Severe Vitamin B<sub>12</sub> Deficiency at Baseline and 1 and 2 years after the intervention.



The milk provided 1/3 RDA for vitamin B<sub>12</sub> and the beef doubled the RDA for 6-10 year old children. Meals were delivered to the school in the individual containers, 5 days per week for the three 3-month school terms per year. Leftovers were weighed and recorded. Over 99% of supplemental food was consumed when children were present. Mean school attendance was 84.5% and did not differ significantly between supplemented groups. Ages ranged from 5-14 years (mean  $\pm$  SD, 7.4  $\pm$  1.2y).

Blood samples were collected prior to the intervention (baseline) and at the end of one and two years. Plasma vitamin B<sub>12</sub> was assessed in duplicate by radioimmunoassay. Severe B<sub>12</sub> deficiency was defined as plasma vitamin B<sub>12</sub> < 200 ng/L and marginal deficiency as 200-300 ng/L (Institute of Medicine 1998).

Changes in vitamin B<sub>12</sub> concentrations were compared among the group using analysis of covariance, controlling for baseline values. Differences in the prevalence of vitamin B<sub>12</sub> deficiency between groups were also determined. Some attrition was seen at follow-up blood drawings with children at the end of Year One and ~300 children at the end of Year Two. Results from children with evidence of infection as indicated by C-reactive protein and malaria were excluded, as infection lowers vitamin B<sub>12</sub> concentration (Neumann et al, 2001).

Compared to the control group in years one and two respectively, plasma B<sub>12</sub> increased by :

- 84 ng/L (p=0.0013) and 126 ng/L (p=0.0001) respectively in the milk group
- 55 ng/L (p=0.0145) and 201 ng/L (p=0.0001) respectively in the meat group (Figure 1)

These differences were highly significant compared to the Energy group and Control groups for both the Meat (p=0.0014, p< 0.0001) and Milk groups (p=0.0002, p< 0.0001) at the end of years one and two respectively. After Year Two of feeding, the increase in plasma vitamin B<sub>12</sub> was significantly greater in the Meat group than in the Milk group (0.0017). Thus, compared to the Control and Energy groups, the prevalence of B<sub>12</sub> deficiency decreased significantly in the Meat (p< 0.0001, p< 0.0001) and Milk (p= 0.0006, p<0.0001) groups after two years of intervention (Table 1) with a decrease of severe vitamin B<sub>12</sub> deficiency to 8.8% in the milk group and 5.5% in the meat group (see table and figure).

### Practical Implications

School feeding is sporadic, or non-existent in many rural areas of Africa and other poor countries (UNU-WFP, 2001). Where present, the feedings are usually devoid of or extremely low in animal source foods, which are a compact source of energy as well as multiple readily absorbed micronutrients. Moreover, meat and milk from a variety of

species are the near exclusive sources of vitamin B<sub>12</sub>. This nutrient plays a key role in nervous system development, brain function, and red blood cell formation. Several studies have implicated vitamin B<sub>12</sub> deficiency in decreased cognitive function in affluent countries among children raised on strict vegetarian diets (Dagnelie et al, 1989) and more recently in poor countries (Allen et al, 1999). Modest amounts of milk and meat can greatly improve vitamin B<sub>12</sub> status and eliminate severe vitamin B<sub>12</sub> deficiency overall as diet greatly improves (Neumann, 2002).

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Table 1. Percent of Children with Mild and Severe Vitamin B<sub>12</sub> Deficiency at Baseline and after One and Two Years of Intervention.

<b>Timepoint</b>	<b>Group</b>	<b>Mild deficiency</b>	<b>Severe deficiency</b>
Baseline	Control	28.4	31.4
	Energy	30.9	32.5
	Milk	33.3	36.0
	Meat	25.5	56.1
Year 1	Control	27.5	39.5
	Energy	27.6	47.2
	Milk	27.0	19.8
	Meat	23.6	32.7
Year 2	Control	35.1	21.6
	Energy	40.0	28.8
	Milk	26.3	8.8
	Meat	29.6	5.5

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The GL-CRSP Child Nutrition Project (CNP) was established in 1997 and is built on a decade of research conducted by the Nutrition CRSP (USAID) in the 1980s. The Child Nutrition Project research addresses food-based approaches to micronutrient deficiencies, particularly of children with respect to both the quantity and quality of food intake. The study is centered on a controlled intervention feeding trial of school children in Embu, Kenya. The project is directed by Dr. Charlotte Neumann and Professor Nimrod Bwibo as Principal Investigators and Suzanne Murphy, Marion Sigman, Shannon Whaley, and Lindsay Allen as Co-Investigators. Email contact for Dr. C. Neumann is: [cneumann@ucla.edu](mailto:cneumann@ucla.edu).



The Global Livestock CRSP is comprised of multidisciplinary, collaborative projects focused on human nutrition, economic growth, environment and policy related to animal agriculture and linked by a global theme of risk in a changing environment. The program is active in East Africa, Central Asia and Latin America.

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