ABSTRACT

Background: Iron and zinc are difficult to provide in sufficient amounts in complementary foods to infants world-wide, resulting in high prevalence of both iron and zinc deficiency. These deficiency states cause anemia, delayed neurodevelopment, impaired growth, and increased susceptibility to infections such as diarrhea and respiratory infections.

Design: Two different intervention strategies; reduction of a possible inhibitor of iron and zinc absorption, i.e. phytate, or supplementation with iron and zinc, were applied to two different populations in order to improve iron and zinc nutrition:

In a high-income population (Umeå, Sweden), the amount of phytate in commonly consumed infant cereals was reduced. Healthy, term infants (n=300) were at 6 mo of age randomized to phytate-reduced infant cereals, conventional infant cereals, or infant formula and porridge.

In a low income population (Purworejo, Indonesia), daily iron and zinc supplementation was given. Healthy, term infants (n=680) were at 6 mo randomized to supplementation with iron, zinc, a combination of iron and zinc, or placebo.

Blood samples, anthropometrical measurements, and data on infant neurodevelopment and morbidity were collected. Also, in the Swedish study, detailed information on the dietary intake was recorded.

Results: In the Swedish study, the reduction of phytate had little effect on iron and zinc status, growth, development or incidence of diarrhea or respiratory infections, possibly due to the presence of high contents of ascorbic acid, which may counteract the negative effects of phytate. In the Indonesian study, significant negative interaction between iron and zinc was evident for several of the outcomes; Hb and serum ferritin improved more in the iron only group compared to placebo or the combined iron and zinc group. Further, supplementation with iron alone improved infant psychomotor development and knee-heel length, whereas supplementation with zinc alone improved weight and knee-heel length compared to placebo. Combined iron and zinc supplementation did decrease the prevalence of iron deficiency anemia and low serum zinc, but had no other positive effects. Vomiting was more common in the combined group.
Analyses of dietary intake from the Swedish study showed that dietary iron intake in the 6-11 mo period was significantly associated with Hb, but not serum ferritin at 9 and 12 mo, whereas the opposite was true in the 12-17 mo period, i.e. dietary iron intake was significantly associated with serum ferritin, but not Hb at 18 mo.

Conclusions: The phytate content of commercial infant cereals does not seem to contribute to poor iron and zinc status of Swedish infants as feared. However, the current definitions of iron and zinc deficiency in infancy may overestimate the problem, and a change in the recommended cutoffs is suggested. These studies also indicate that dietary iron is preferably channeled towards erythropoiesis during infancy, but to an increasing amount channeled towards storage in early childhood. This suggests that in evaluating dietary programs, Hb may be superior in monitoring response to dietary iron in infancy, whereas S-Ft may respond better later in childhood. However, as shown in this study, increasing Hb may not necessarily be an indicator of iron deficiency, as more dietary iron increased Hb regardless of iron status.

In the low-income setting combined supplementation with iron and zinc resulted in significant negative interaction. Thus, it is not possible to recommend routine iron-zinc supplementation at the molar concentration and mode used in this study. It is imperative that further research efforts are focused at finding cost-effective strategies to prevent iron and zinc deficiency in low-income populations.