Vitamin A deficiency: slow progress towards elimination

Gretchen Stevens and colleagues’ study1 in The Lancet Global Health analyses 134 reports from 83 countries of population-representative data for serum retinol concentration and shows several trends in vitamin A deficiency. First, prevalence is diminishing in a statistically significant way in east and southeast Asia and Oceania. Second, Latin America and Caribbean nations might be making progress. Third, prevalence remains unchanged in sub-Saharan Africa and south Asia. Stevens and colleagues’ most interesting conclusion is that this “evidence for both prevalence and absolute burden of vitamin A deficiency should be used to reconsider, and possibly revise, the list of priority countries for high-dose vitamin A supplementation”.

However, several caveats need to be considered, including the use of cross-sectional data, exclusion of children younger than 6 months, absence of clinical vitamin A deficiency assessments, gaps in serum retinol data for certain populations and for 55 countries, use of mortality data from randomised controlled trials of vitamin A supplementation for diarrhoea and malaria rather than population-level data, the assumption that all post-neonatal measles deaths occur in children aged 6–59 months old, and use of serum retinol as the biomarker of deficiency.

Nonetheless, we might need to focus on retinol concentration not mortality to assess and guide our efforts to eliminate vitamin A deficiency, as recommended by WHO for population-level surveys, even though this measure can be problematic in individuals because of the well known effects of acute inflammation due to infection or injury on serum retinol concentrations.2 Controlling for acute inflammation by including biomarkers for it3 might not be necessary at the population level, because adequate vitamin A status might, by itself, diminish inflammation through a reduction in the frequency or severity of infections in these populations.

Despite these limitations, the authors provide important estimates, with uncertainty distributions, for the prevalence of vitamin A deficiency, and a clear picture of trends from 1991 to 2013. The association between vitamin A deficiency and eye pathology and all-cause mortality is well known, as is the contribution of vitamin A deficiency to reduced resistance to infections, especially diarrhoea and measles, and increased mortality in children younger than 5 years.4 These benefits have driven the scale-up of vitamin A supplementation programmes as preventative public health measures around the world. However, the coverage of these programmes has not been efficient in east or southern Africa (67%) and south Asia (53%).5 Lagging coverage and continued evidence of vitamin A deficiency is the basis for the suggestion by Stevens and colleagues that future efforts refocus on these regions. This suggestion makes sense if we are confident that progress elsewhere would not be compromised as a result.

During the past two decades, mortality from diarrhoeal disease has substantially decreased and measles has been eliminated as a public health issue wherever effective immunisation programmes flourish. Continued efforts to control diarrhoeal disease and enhance measles vaccine coverage per se suggest that vitamin A supplementation programmes should now focus on the reduction of deficiency rather than diarrhoea or measles morbidity or mortality as the outcome.

What then should be done to accelerate progress towards elimination of vitamin A deficiency in children? Initiation of supplementation programmes where they do not exist and strengthening of programmes where coverage is poor is step one. However, high-dose supplementation only provides protection from hyporetinolaemia for 2–3 months in children younger than 5 years and favourably shifts the distribution of serum retinol for less than 2 months,6 indicating that biannual supplementation is not sufficient by itself to prevent vitamin A deficiency. Improvement of dietary intake of foods rich in vitamin A (eg, animal products) or beta-carotene is a more sustainable solution, but high-cost, access, and cultural dietary practices have restricted its potential to alleviate vitamin A deficiency. Other options have been tested or are under development, such as fortification of centrally processed foods (although these might not reach poor populations in rural areas); addition of vitamin A sprinkles to food in the home, day-care centres, and schools;7 promotion of beta-carotene rich foods such as sweet potatoes;8 and genetically engineered crops with high concentrations of beta-carotene such as golden rice.9 By addressing...
gaps in vitamin A sufficiency around the world, redirecting attention to areas of the world struggling to make progress while continuing to monitor other regions through systematic population-representative sampling of serum retinol, promotion of research into sustainable dietary solutions including a campaign to legitimise genetically modified crops high in vitamin A or precursors, and alignment of all of these efforts with Sustainable Development Goal 2 to “end hunger, achieve food security and improved nutrition and promote sustainable agriculture”, we can not only sustain the favourable trends described by Stevens and colleagues but also hasten progress in other parts of the world.

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We declare no competing interests.

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