IVACG Statement on Clustering of Xerophthalmia and Vitamin A Deficiency Within Communities and Families

Vitamin A deficiency is known to affect specific regions of the world and areas within high-risk countries. It is now recognized that vitamin A deficiency also concentrates within high-risk families and communities. Siblings of xerophthalmic children are 10 times more likely to have xerophthalmia than siblings of children who do not have xerophthalmia. Mothers of xerophthalmic children are 5 to 10 times more likely to be night blind (vitamin A deficient) than mothers of nonxerophthalmic children. In addition, neighboring children of a xerophthalmic child are twice as likely to have or develop xerophthalmia than children in neighborhoods where xerophthalmia has not been seen.

The fact that xerophthalmia "clusters" has clear relevance and application for treatment and prevention. Children presenting with xerophthalmia should be treated according to the World Health Organization's WHO/UNICEF/IVACG guidelines. Their preschool siblings should be supplemented prophylactically with vitamin A according to the WHO/UNICEF/IVACG guidelines. The family, and especially the child's mother, should be provided appropriate counseling and other assistance to improve her dietary intakes of vitamin A as well as those of her children. Communities in which xerophthalmic children present should be made aware of the problem, its consequences, and potential solutions. The local setting should be given special consideration in the context of population-based measures to prevent vitamin A deficiency.

Background
In multiple studies in Indonesia, Nepal, Malawi, Zambia, and Bangladesh, it has been demonstrated that vitamin A deficiency and xerophthalmia cluster within families, neighborhoods, and villages (1-6). A national survey of xerophthalmia in Indonesia in the 1970s demonstrated that Bitot's spots clustered by village within each of the regions surveyed (1). In a study from West Java, Indonesia, children without xerophthalmia who were from neighborhoods with xerophthalmic children had lower serum retinol levels than those from neighborhoods without xerophthalmia (2). Xerophthalmia surveys done in Sumatra, Nepal, Malawi, and Zambia showed that if one child in a household had xerophthalmia, the risk of another child in the same household having xerophthalmia was between 7.3 and 13.2 times higher than if the index child did not have xerophthalmia (5). This increased risk was not entirely explained by siblings being more likely to have the same infectious diseases that predispose children to xerophthalmia. Furthermore, mothers of xerophthalmic children in Bangladesh were 5 to 10 times more likely to have night blindness than mothers of children without xerophthalmia (6). In terms of community risk, if one child in a village had xerophthalmia, the likelihood that another child in that village had xerophthalmia was between 1.2 and 2.3 times greater than if the index child did not have xerophthalmia (5).

Although xerophthalmia is not an infectious disease, children in the same household and same community share common socioeconomic and sociocultural conditions that result in similar exposure to frequent bouts of infectious disease, food availability, and dietary habits that predispose them to xerophthalmia. The increased risk of xerophthalmia in some households and
some communities is important in improving understanding of the underlying causes of vitamin A deficiency. It also provides us with an efficient way of improving vitamin A status by targeting siblings, mothers, and neighbors of children with xerophthalmia for vitamin A prophylaxis. Health workers in areas where vitamin A deficiency is endemic should be aware that behind every child they see with xerophthalmia are siblings, mothers, and neighbors with xerophthalmia or those who are vitamin A deficient and, therefore, at high risk of developing it in the near future.

References


