Night blindness—the inability to see after dusk or at night—is the most common ocular manifestation of moderate to severe vitamin A deficiency. Poor dark adaptation leading to night blindness occurs when there is decreased production of a vitamin A–dependent photosensitive pigment, rhodopsin, in the retinal receptors responsible for seeing under low levels of illumination (rods). Normally, when these photoreceptor cells are stimulated by light, rhodopsin is transformed, initiating neural signals that are transmitted to the brain, resulting in vision in dim illumination. In vitamin A deficiency, less rhodopsin is transformed and the level of light needed for vision rises. This results in delayed dark adaptation or, when sufficiently severe, in night blindness. Where prevalent, night blindness may be known by local terms that refer to evening or twilight blindness or, in some cultures, to “chicken eyes” or “chicken blindness” (chickens lack rod cells).

Night blindness is frequently reported in young children in developing countries, but only recently has it been recognized as a public health problem in women of reproductive age. Two small studies in India in the 1960s suggested that night blindness was common in poor pregnant women, often occurring in the third trimester, and that it responded to treatment with high-potency vitamin A. In some cultures, night blindness is thought to be a normal consequence of pregnancy, given its common occurrence and tendency to disappear without treatment shortly after childbirth. Recent reports from poor populations in different regions of the world suggest that ~10% of women experience night blindness during pregnancy. Extrapolations suggest that 6 million women become night-blind during pregnancy each year.
Risk Factors

A population-based case-control study in Nepal found that maternal night blindness was associated with low to deficient vitamin A status. Serum retinol of night-blind women was 0.3 µmol/L lower than non-night-blind controls matched for gestational age of pregnancy (0.72 versus 1.03 µmol/L). Night-blind women were four times more likely to have low serum retinol concentrations (< 0.7 µmol/L) than controls, and were only half as likely to consume dietary sources of vitamin A. Their mean hemoglobin (Hb) was 7 g/L lower, and they were three times more likely to be severely anemic (Hb < 70 g/L). They had a lower mean weight (–2.6 kg), arm circumference (–9 mm), and height (~1.3 cm) than their peers, reflecting a higher level of malnutrition. Pregnant night-blind women were two to three times more likely than controls to report symptoms of urinary or reproductive tract infections (i.e., lower abdominal pain, painful urination, or vaginal discharge), preeclampsia/eclampsia (i.e., convulsions or swelling of hands or face), diarrhea or dysentery, and symptoms of nausea, poor appetite, and vomiting.

Mortality Consequences of Maternal Night Blindness

The mortality rates of Nepali women followed prospectively for up to 2 years postpartum as part of a randomized trial were found to be different among those who did and did not experience night blindness during pregnancy. Mortality of night-blind women in the placebo group was nearly four times that of non-night-blind women (3,601 versus 950, respectively, per 100,000 pregnancies). In night-blind and non-night-blind women who received weekly vitamin A or β-carotene, the mortality rate was only 1,163 and 631, respectively, per 100,000 pregnancies. This indicates that vitamin A or β-carotene supplementation lowered the excess risk of mortality by 68% in the night-blind women and by 34% in the non-night-blind women. Night-blind women were five times more likely to die from infections than were non-night-blind women. Similarly, 6-month mortality in infants of women who had night blindness during pregnancy was higher than that of non-night-blind women in the placebo group. Maternal receipt of vitamin A reduced this risk to some extent.

Prevention and Treatment of Maternal Night Blindness

A large, community-based randomized trial in Nepal showed that regular weekly doses of vitamin A (25,000 IU—a weekly “Recommended Dietary Allowance”) could reduce the incidence of maternal night blindness by 67%, although β-carotene was not efficacious in this regard. However, low compliance (<40% of intended doses taken) provided no protection against night blindness, suggesting that a minimum prophylactic dose of vitamin A was required to prevent the condition. At present, the World Health Organization and IVACG recommend treating maternal night blindness with daily oral doses of 10,000 IU or weekly doses of 25,000 IU vitamin A for 4–8 weeks. Based on the Nepal trial, weekly supplementation prevented only 67% of maternal night blindness at this dose.
Maternal Night Blindness as an Indicator of Vitamin A Deficiency

The common occurrence of maternal night blindness and its extensive associated health risks, ease of assessment, and clinical responsiveness to vitamin A make a history of the condition during pregnancy a useful indicator of vitamin A deficiency in women of reproductive age. In women, night blindness is strongly associated with other biochemical and functional indicators of vitamin A deficiency such as serum retinol concentration, conjunctival impression cytology, dark adaptation, and breast milk vitamin A. It was recently recommended that maternal night blindness be adopted as an indicator of vitamin A deficiency in the community as a whole, at least for populations in which intervention programs directed toward children have not been initiated. Where such programs have been initiated, the vitamin A status (and prevalence of night blindness) in women may say little about status among children, nor would it be useful for purposes of monitoring or evaluating the impact of such programs.

To determine night blindness prevalence, a history is elicited from women for a previous pregnancy that ended in a live birth in the past 3 years. It is preferable to use a local term for night blindness whenever possible. Effort should be made to exclude from the estimate of prevalence (i.e., both from the numerator and denominator) women whose night blindness during pregnancy was probably due to visual impairment from other causes.

A maternal night blindness prevalence of $\geq 5\%$ is recommended as a cut-off at which vitamin A deficiency may be considered a problem of public health significance within a community. This cut-off was chosen because existing data suggest that misclassification of self-reported night blindness may account for a prevalence of $\leq 3\%$. The higher cut-off of $5\%$ includes this potential false-positive prevalence and thereby improves specificity.*

In populations where vitamin A deficiency is endemic and vitamin A programs are not in place, rates of maternal night blindness correlate highly with rates of childhood xerophthalmia. In India, for example, a maternal historical night blindness rate during pregnancy correctly identified 14 out of 16 states as having a vitamin A deficiency problem based on xerophthalmia rates in children. Rates of maternal night blindness should be routinely investigated in nutrition and health surveys to further define its extent and to develop effective strategies for its treatment and prevention.

Summary

Maternal night blindness occurs commonly during pregnancy in vitamin A–deficient regions of the world. Night blindness during pregnancy, caused by vitamin A deficiency, is also a marker of increased reproductive morbidity, protein-energy malnutrition, anemia, and elevated mortality in women and their infants. Vitamin A supplementation in the recommended daily amounts reduces the risk of night blindness to a large extent. Because a history of night blindness is easy and simple to obtain, it is recommended for use as an indicator of vitamin A deficiency in communities where interventions are not in place. A prevalence of maternal night blindness of $5\%$ or greater is suggested for considering vitamin A deficiency to be a public health problem.

*Recently, Demographic Health Survey data from six countries in Africa found that $4–17\%$ of women who gave birth in the previous 5 years reported having night blindness during their last pregnancy. However, after exclusion of women who also reported daytime vision problems (as currently recommended), the prevalence rate of maternal night blindness dropped to $1–4.8\%$, all below the recommended cutoff of $5\%$. Further work is being undertaken to refine methods of collecting information on maternal night blindness in Africa and on the adjustment factor for daytime vision problems. Until then, maternal night blindness rates in these countries should be reported unadjusted as well as adjusted for “daytime blindness.”

A maternal night blindness prevalence of $\geq 5\%$ is recommended as the cut-off at which vitamin A deficiency may be considered a problem of public health significance for the community.

Rates of maternal night blindness should be routinely investigated in nutrition and health surveys.
References


About IVACG

Established in 1975, the International Vitamin A Consultative Group guides international activities for reducing vitamin A deficiency in the world. IVACG concentrates its efforts on stimulating and disseminating new knowledge, translating that new knowledge to enable its practical application, and providing authoritative policy statements and recommendations that others can use to develop appropriate prevention and control programs.

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<table>
<thead>
<tr>
<th>IVACG Secretariat</th>
<th>Tel: 202-659-9024</th>
</tr>
</thead>
<tbody>
<tr>
<td>ILSI Research Foundation</td>
<td>Fax: 202-659-3617</td>
</tr>
<tr>
<td>One Thomas Circle, NW, 9th floor</td>
<td>E-mail: <a href="mailto:hni@ilsi.org">hni@ilsi.org</a></td>
</tr>
<tr>
<td>Washington, DC 20005-5802, USA</td>
<td>Internet: <a href="http://ivacg.ilsi.org">http://ivacg.ilsi.org</a></td>
</tr>
</tbody>
</table>

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