

survey, our casual observation was that response rates to both the eye and serosurveys benefited from the eye surveys.

Finally, we have some reservations about applying the rates observed in this study to the whole of Uganda since residents in the study area did not suffer from eye diseases that are common in other parts of Uganda, for example, trachoma (in northern Uganda) and vitamin A deficiency (in eastern Uganda). Therefore, it is likely that the observed rates underestimate the true incidence of visual loss (and the projected number of incident cases) in Uganda overall. Excluding people with visual impairment in R1 from the denominator for calculation of incidence rates (see Methods) may also make our reported incidence rates appear low. For example, four people who progressed from bilateral visual impairment to bilateral blindness (from vision category 4 to 5 in table 2) did not contribute to the estimate of the incidence of bilateral blindness.

In summary, these results demonstrate that the incidence of visual loss is high in southwest Uganda and that the causes of the majority of cases are treatable. Planners need to take into account such data when planning for sustainable eye services.

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ECHO

High and low chromosome instability separates retinoblastomas



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Evidence from a cytogenetic study supports the idea that retinoblastoma may exist in two distinct forms which differ clinically.

Eleven of 13 children in the study had chromosome abnormalities in their tumour—an array of gains/losses in various chromosomes—including those other than in chromosome 13q where the retinoblastoma gene maps. Frequency of the abnormalities was bimodal, with tumours showing low level chromosomal instability and 0–3 abnormal events (seven children) or high level instability and eight plus events (six). Children with low level chromosomal instability showed some similar traits. Their mean age was half that of the other children; there were fewer males (male to female ratio 1.3:1 v 5:1) and undifferentiated tumours (57% v 83%); and hereditary retinoblastoma was commoner (57% v 33%), as was disease in both eyes (57% v 17%). The control showed no chromosome abnormalities.

The study was performed on 13 retinoblastomas from 13 consecutive children aged 0–45 months. Chromosome instability was found by comparative genomic hybridisation of DNA extracted from frozen sections of tumour biopsy specimens and control lymphocyte DNA from a healthy donor with metaphase spreads in cultured donor lymphocytes.

Retinoblastoma is the commonest malignancy in the eye in children. About 40% of cases are hereditary, involving chromosome 13. Changes in other chromosomes—extra copies, deletions and gains, ring chromosomes—have been noted. Comparative gene hybridisation is useful way of testing for chromosomal instability but has not been used much in retinoblastoma.

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