Incidence of visual loss in rural southwest Uganda

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Background: Surveys have been conducted to measure prevalence of eye disease in Africa, but not of incidence, which is needed to forecast trends. The incidence of visual loss is reported in southwest Uganda.

Methods: A rural population residing in 15 neighbouring villages was followed between 1994-5 (R1) and 1997-8 (R2). Survey staff screened adult residents (13 years or older) for visual acuity using laminated Snellen’s E optotype cards at each survey. Those who failed (VA >6/18) were evaluated by an ophthalmic clinical officer and an ophthalmologist. Incidence of visual loss (per 1000 person years (PY)) was calculated among those who had normal vision at R1.

Results: 2124 people were studied at both survey rounds (60.9% of those screened at R1); 48% were male. Participants in R1 were older (34.7 versus 31.5 years at R2, p<0.001). Visual loss in R2 occurred in 56 (2.8%) of 1997, yielding a crude incidence rate of 9.9, and an age standardised incidence rate of 13.2, per 1000 PY. Incidence of visual loss increased with age from 1.21 per 1000 PY among people aged 13–34 to 64.2 per 1000 PY in those aged 65 years or older (p for trend >0.001). The six commonest causes of visual loss were: cataract, refractive error, macular degeneration, choroid retinitis, glaucoma, and corneal opacity. If similar rates are assumed for the whole of Uganda, it is estimated that 30 348 people would develop bilateral blindness or bilateral visual impairment, per year.

Conclusions: Cataract and refractive error were the major causes of incident visual loss in south west Uganda. These data are valuable for forecasting and planning eye services.
conditions (NVIC) were all referred to an ophthalmic clinical officer (OCO; a medical assistant trained in community eye care). During the R1 survey, the OCO worked from a fixed clinic held regularly on one day of the week at the survey headquarters, but many of those referred did not attend the clinic.

On the basis of this experience from the R1 survey, additional resources were made available for the R2 survey so that the OCO regularly worked alongside survey teams to improve coverage and to reduce the period between screening by field staff and the more detailed testing of visual acuity by the OCO. The OCO measured visual acuity using a full Snellen illiterate E chart (lines ranging from 6/5 to 6/60) and performed a general eye examination to determine the main cause of visual loss, the cause most amenable to prevention was assigned.

A general eye examination was then performed to determine visual acuity. During the R1 survey, the OCO worked from a fixed clinic held regularly on one day of the week at the survey headquarters, but many of those referred did not attend the clinic. On the basis of this experience from the R1 survey, additional resources were made available for the R2 survey so that the OCO regularly worked alongside survey teams to improve coverage and to reduce the period between screening by field staff and the more detailed testing of visual acuity by the OCO. The OCO measured visual acuity using a full Snellen illiterate E chart (lines ranging from 6/5 to 6/60) and performed a general eye examination. He treated people who had simple non-vision impairing (NVIC) conditions and referred complex cases to the ophthalmologist for specialist evaluation. All patients thought to have low vision on screening were referred to the ophthalmologist regardless of the findings by the OCO.

**Diagnosis**

The ophthalmologist confirmed the visual acuity at fortnightly clinics; if there was discordance in the visual acuity measurements, those made by the ophthalmologist were used. A general eye examination was then performed to determine the causes of low vision for the eye and for the person. In determining the main cause of visual loss, the cause most amenable to prevention was assigned. Cataracts were diagnosed as any lens opacity visible to the ophthalmologist by direct ophthalmoscopy against the red reflex. When refractive error was suspected to be the cause of loss of vision, visual acuity was measured with a pinhole and any significant refractive error was confirmed using lens power readings from the ophthalmoscope. Central visual loss was attributed to glaucoma on observation of the following, when no other cause could be discerned: (a) a pathological optic disc (marked pallor of the nerve head or vertical cupping > 0.5 mm) in the presence of intraocular pressure >21 mm Hg (using a Shiotz tonometer); (b) markedly raised intraocular pressure (>26 mm Hg) even without pathological disc; (c) a history of glaucoma surgery or treatment when no other cause could be discerned.

Age related disorders such as macular degeneration and retinal abnormalities were diagnosed on clinical grounds. All patients received treatment at the clinic except when specialised treatment was needed, in which case they were referred to the regional eye centre about 35 km away in the district capital. Travel expenses were paid by the study. Spectacles were provided at a subsidised cost (around $5.00) to those who needed them.

**Statistical methods**

Data were recorded using a modified WHO/PBL eye examination form. Records were checked for completeness and accuracy before being double entered into a computer. Analyzes were performed using Stata 6.0 (Stata Corporation, College Park, TX, USA) after carrying out consistency checks. The incidence analyses reported here are based on people who were present at both rounds.

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**Table 1** Comparison of people screened in round 1, round 2, and both rounds 1 and 2

<table>
<thead>
<tr>
<th>Screening results</th>
<th>All people screened in round 1 (n=3489)</th>
<th>All people screened in round 2 (n=4075)</th>
<th>People screened in rounds 1 and 2 (n=2124)</th>
<th>People screened only in round 1 (n=1365)</th>
<th>People screened only in round 2 (n=1951)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Passed both eyes</td>
<td>3308 (94.8%)</td>
<td>2007 (94.5%)†</td>
<td>1301 (95.3%)§</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Failed one eye</td>
<td>91 (2.6%)</td>
<td>61 (2.9%)</td>
<td>30 (2.2%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Failed both eyes</td>
<td>90 (2.6%)</td>
<td>56 (2.6%)</td>
<td>34 (2.5%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 2** Visual acuity results, in rounds 1 and 2, as assessed by an ophthalmic clinical officer who validated the visual acuity results of those who failed screening

<table>
<thead>
<tr>
<th>Round 2</th>
<th>Round 1</th>
<th>Passed screen, both eyes assumed to be better than 6/18</th>
<th>0</th>
<th>1*</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>9</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1906</td>
<td>27 12 21 1 8</td>
<td>1997</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>1962</td>
<td>24 1 0 0 0 0</td>
<td>9 9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>90</td>
<td>9 2 4 1 0 1 16</td>
<td>6 6</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>31.5 (17.6)†</td>
<td>36.0 (18.0)‡</td>
<td>32.6 (18.2)‡</td>
<td>26.6 (18.8)§</td>
<td>960 (49.2%)‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>1671 (47.9)†</td>
<td>1988 (48.8%)‡</td>
<td>1028 (48.4%)‡</td>
<td>643 (47.1%)‡</td>
<td>960 (49.2%)‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*The proportion of people passing in round 1 who were also screened in round 2 was not significantly lower than the proportion passing in round 1 who were not screened in round 2.†The proportion of people passing in round 2 who were also screened in round 1 was significantly lower than the proportion passing in round 2 who were not screened in round 1 (χ² = 39.7, df = 2, p<0.0001).‡Age was missing for 1 patient.§Mean age of patients screened in both rounds was significantly higher than patients screened in round 1 only (difference = −9.40 years, 95% CI = −10.4 to −8.4, p<0.0001).
In order to estimate incidence of visual loss in Uganda, we used person years of observation to calculate incidence rates.

Person years of observation were estimated from the time of first screening to the time of second screening for people who did not develop visual loss. For those who developed visual loss, it was assumed that the times of onset of visual loss were distributed evenly throughout the study period; therefore only half of person years of observation between the first and second survey dates were included. People who failed screening or who were identified as having visual loss at R1 were not included in the person years of observation to calculate incidence rates.

Three categories were used to classify the vision in each eye: vision equal to or better than 6/18 was categorised as normal vision; vision worse than 6/18 but better than or equal to 3/60 was categorised as “visual impairment”; vision worse than 3/60 was categorised as blindness. Classification in either of the two latter categories constituted visual loss. Using these three categories, five levels of visual loss in individual participants were derived, combining information for both eyes: (1) normal vision equal to or better than 6/18 in both eyes; (2) unilateral visual impairment in one eye and normal vision in the fellow eye; (3) unilateral blindness in one eye and normal vision in the fellow eye; (4) bilateral visual impairment, including participants who had one blind eye; and (5) bilateral blindness.

In order to estimate incidence of visual loss in Uganda, we used person years of observation to calculate incidence rates. Person years of observation were estimated from the time of first screening to the time of second screening for people who did not develop visual loss. For those who developed visual loss, it was assumed that the times of onset of visual loss were distributed evenly throughout the study period; therefore only half of person years of observation between the first and second survey dates were included. People who failed screening or who were identified as having visual loss at R1 were not considered to be “at risk” of developing visual loss during the study period. However, since some causes of visual loss are treatable, and the provision of treatment when appropriate was an aim of the first survey, the results of visual assessments in these individuals are also described. Age standardised projected numbers of people with incident visual impairment in Uganda per year were calculated by combining observed rates of visual impairment with age stratified population estimates.16 17

RESULTS

Table 1 shows the screening results, and the age and sex of the population for both surveys. A total of 3489 adults were screened during the R1 eye survey; 48% were males and 181 (5.2%) people failed visual screening in one or both eyes. There were 4075 people in the R2 eye survey; 49% were male and 191 (4.7%) people failed visual screening in one or both eyes. A total of 2124 people were screened at both survey rounds (60.9% of those screened at R1); 48% were male. Of the 2124, 127 (6.0%) and 175 (8.2%) failed visual acuity screening at R1 and R2 eye surveys, respectively. The mean age of participants in the R1 survey was higher than in the R2 survey (34.7 years versus 31.5 years). The mean age of individuals screened in both surveys was higher than those present only in the R1 survey (difference −3.33 years, 95% CI −4.6 to −2.1) or only in the R2 survey (difference −9.4 years, 95% CI −10.4 to −8.4; all p<0.0001). The percentage of participants who were male was similar (48%) among those who were screened in both surveys and among those screened in only one survey (47% and 49% for R1 and R2 respectively). The proportion of screen failures was higher among individuals present at both rounds than those present only at R2 (6.7% versus 2.5%; p<0.0001) but not higher than those present only at R1 (4.7%).

Table 2 shows the R1 and R2 screening results for people who were present at both survey rounds. Of the 1997 (94.0%) who were presumed to have normal vision at R1, 56 (2.8%) developed visual impairment—that is, failed screening, and were validated as having visual loss by the OCO. An additional 27 (1.4%) failed screening at R2 but were found to have normal vision by the OCO, and eight (0.4%) who failed R2 screening did not attend for evaluation by the OCO. The distribution of incident visual loss was 22 (1.1%) unilateral vision impairment; 12 (0.6%) blind in one eye; 21 (1.1%) vision impairment in two eyes; one (0.05%) was blind in both eyes.

Table 3  Distribution of causes of visual loss by visual loss category at round 2 among people who passed screening at round 1: estimated incidence rates stratified by age and by visual loss category and cause of visual loss

<table>
<thead>
<tr>
<th>Causes</th>
<th>Bilateral blindness</th>
<th>Bilateral visual impairment</th>
<th>Unilateral blindness</th>
<th>Unilateral visual impairment</th>
<th>Total (PY)</th>
<th>Rate/1000 PY*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age stratified incidence:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13–34†</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>4 (3301)</td>
<td>1.21 (0.33 to 3.10)</td>
</tr>
<tr>
<td>35–54 years</td>
<td>0</td>
<td>3</td>
<td>2</td>
<td>6</td>
<td>11 (1482)</td>
<td>7.42 (3.71 to 13.3)</td>
</tr>
<tr>
<td>55–64 years</td>
<td>0</td>
<td>7</td>
<td>1</td>
<td>5</td>
<td>13 (462)</td>
<td>28.1 (14.5 to 48.1)</td>
</tr>
<tr>
<td>65 years and older</td>
<td>1</td>
<td>11</td>
<td>7</td>
<td>9</td>
<td>28 (436)</td>
<td>64.2 (42.7 to 92.7)</td>
</tr>
<tr>
<td>Specific causes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cataract</td>
<td>0</td>
<td>11</td>
<td>6</td>
<td>6</td>
<td>23 (5682)</td>
<td>4.05 (2.57 to 6.07)</td>
</tr>
<tr>
<td>Refractive error</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>5</td>
<td>7 (5682)</td>
<td>1.23 (0.50 to 2.54)</td>
</tr>
<tr>
<td>Macular degeneration</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>2</td>
<td>3 (5682)</td>
<td>0.53 (0.11 to 1.54)</td>
</tr>
<tr>
<td>Choriororetinitis</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>3 (5682)</td>
<td>0.53 (0.11 to 1.54)</td>
</tr>
<tr>
<td>Glaucoma</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2 (5682)</td>
<td>0.35 (0.04 to 1.27)</td>
</tr>
<tr>
<td>Corneal opacity</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2 (5682)</td>
<td>0.35 (0.04 to 1.27)</td>
</tr>
<tr>
<td>Other cause†</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>7 (5682)</td>
<td>1.23 (0.50 to 2.54)</td>
</tr>
<tr>
<td>Cause not determined</td>
<td>0</td>
<td>4</td>
<td>1</td>
<td>4</td>
<td>9 (5682)</td>
<td>1.58 (0.72 to 3.01)</td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
<td>21</td>
<td>12</td>
<td>22</td>
<td>56 (5682)</td>
<td>9.86 (7.44 to 12.8)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Expected cases/year†</th>
<th>Bilateral blindness and bilateral visual impairment</th>
<th>Unilateral blindness</th>
<th>Unilateral visual impairment</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>15–34†</td>
<td>0</td>
<td>4940</td>
<td>4940</td>
<td>9880</td>
</tr>
<tr>
<td>35–54</td>
<td>5476</td>
<td>3651</td>
<td>10952</td>
<td>20078</td>
</tr>
<tr>
<td>55–64 years</td>
<td>10654</td>
<td>1522</td>
<td>7610</td>
<td>19786</td>
</tr>
<tr>
<td>65 years and older</td>
<td>14218</td>
<td>8294</td>
<td>10664</td>
<td>33179</td>
</tr>
<tr>
<td>Total</td>
<td>30348</td>
<td>18407</td>
<td>34166</td>
<td>82920</td>
</tr>
</tbody>
</table>

* Rates based on 1997 people who passed screening test at round 1 and 56 cases identified at round 2.
† Expected number of cases based on (a) an estimated total population for Uganda of 24.6 million (CIA-World Fact Book, updated Jan 2002) and (b) the proportion of the population in each age stratum reported in The Uganda Health and Demographic Survey of 1991. The single incident case of bilateral blindness is included with incident cases of bilateral visual impairment because of the imprecision of any estimate based on a single case.
" Up to date population estimates were not available for exactly the same strata as those used for the study; the tabulated estimates are based on incidence rates for the age stratum 13–34 years observed in the study and a denominator derived from the sources described above for the age stratum 15–34.
Visual assessments were concordant in the two surveys for 1941/2124 (91.4%), of whom 32 had the same level of visual loss in both surveys (an additional three people failed screening for both surveys but were not tested further). Visual loss of five people worsened between the two survey rounds; four with one eye blind and the other impaired at R1 became bilaterally blind at R2; another person with unilateral visual impairment became blind in one eye and visually impaired in the other. The vision of 26 people (1.2%) appeared to have improved between the R1 and R2 surveys. Eleven people who had vision impairment in one eye at R1 passed screening (nine people) or were found to have normal vision by the OCO (two people) in R2. Seven of 12 people who were blind in one eye (with or without visual impairment in the fellow eye) in R1 passed screening (four people) or were found to have normal vision by the OCO (three people) in R2; in the remaining five people, the worst affected eye had apparently improved from being blind to having visual impairment. One eye in each of three people found to be blind in both eyes in R1 was found to have improved to visual impairment in R2. In addition, 45/59 people who failed screening and who were not tested any further in R1 passed screening (30 people) or were found to have normal vision by the OCO (15 people) on R2; the other 14 people were found to have varying degrees of visual loss.

Table 3 also shows the distribution of incident cases by age group, causes of visual loss, and category of visual loss and estimated incidence rates. The crude incidence rate for visual loss was 9.86 (95% CI 7.44 to 12.8) per 1000 person years. The incidence rate increased dramatically across age groups; compared to the youngest age group (13–34 years), rate ratios were 6.13 (95% CI 1.81 to 26.4), 23.2 (95% CI 7.17 to 97.8), and 53.0 (95% CI 18.5 to 208) for the age groups 35–54 years, 55–64 years, and ≥65 years, respectively (p for trend <0.001).

Unilateral and bilateral visual impairment were the most frequent categories of visual loss, with unilateral blindness slightly less frequent and bilateral blindness much less frequent. Cataract was the commonest cause of visual loss for all categories except bilateral blindness and accounted for 41% of incident cases. Refractive error was responsible for 13% of incident cases of visual loss. Other causes of incident visual loss were macular degeneration (3.4% of cases), chorioretinitis (5.4%), glaucoma (3.6%), and corneal opacity (3.6%). Overall, the six commonest causes of visual loss were cataract, refractive error, macular degeneration, chorioretinitis, glaucoma, and corneal opacity.

Table 3 also shows the expected total number of cases of visual loss by age group, and separately for each category of visual loss based on census population estimates for Uganda and assuming that the rates of visual loss observed in this study were applicable to the whole of Uganda. About 83 000 new cases of visual loss per year would be expected, including 30 348 with bilateral visual impairment.

**DISCUSSION**

Our survey indicates that over 50% of incident visual loss are the result of easily treatable causes (cataract and refractive error). If we assume that the same incidence rate for Uganda, we estimate that close to 30 000 people per year in Uganda develop disabling (bilateral) visual loss. If so, this number represents a significant and unsustainable addition to already stretched eye care services. Thus, while prevalence studies are useful for planning eye services, our study highlights the value of incidence data in forecasting future needs. Unfortunately, logistical constraints and the need for highly trained staff have precluded surveys of the incidence of visual loss and its causes from being carried out in sub-Saharan Africa. We demonstrate that eye surveys can be conducted cheaply using E-optotypes operated by non-medically trained staff backed by a small staff of ophthalmic clinical officers and ophthalmologists.

The six commonest causes of incident visual loss were cataract, refractive error, macular degeneration, chorioretinitis, glaucoma and corneal opacity. Visual loss due to corneal opacity was observed in the unilateral visual impairment or blindness category, consistent with traumatic aetiology, a common cause of monocular visual loss. As previously reported from this population, corneal opacity due to trachoma was rare. Trachoma is more common in settings where water is scarce and hand washing is rarely practised. Glaucoma, a difficult disease to recognise, was the fifth commonest cause of visual loss, although we acknowledge that the definition used for this study was weak. Data from several studies in Africa indicate that glaucoma may be as common in Africa as in African-Americans.

We estimated that glaucoma had an incidence rate of 0.4 per 1000 person years, accounting for 3.6% of incident cases. However, our estimate of glaucoma is probably conservative since we included only people with established visual loss, a relatively late sequel of glaucoma. Furthermore, we did not examine for characteristic visual field defects to confirm the presence of glaucoma. Our results highlight the need for further studies to clarify the epidemiology of glaucoma in sub-Saharan Africa.

There are no comparative data on incidence of visual loss from Africa. However, a recent review of studies conducted in Africa broadly summarised the causes of visual loss into three groups:

1. Those which occur universally and have successful treatments, such as refractive error as found in this study
2. Those which occur in specific populations, and which can be prevented by cheap medicines or interventions such as vitamin A deficiency, trachoma, or river blindness. These conditions were absent in our population
3. Major blinding diseases that are less well defined, and which do not have cheap cost-effective means of screening that are applicable in Africa currently, such as glaucoma, identified as one of the top six causes of visual loss in our population.

This study strengthens the rationale of developing sight saving plans based on these simple categories, and also provides a means to evaluate the effectiveness of such interventions.

We note several limitations of our study. The study population is homogeneous and lives in a small geographical area. However, it is typical of rural Uganda, particularly in the central, south, and west. Although the population size is small size, this allowed us to achieve a high follow up of the population (61%) over 3 year period, but meant that, with median duration of follow up of less than 3 years, our estimates of incidence had poor precision. Necessarily, the incidence estimates are based on people who were surveyed on both occasions, who tended to be older than those who were surveyed on only one occasion. Loss to follow up is a concern in cohort studies. Because visual loss tends to incapacitate people, and these people are in turn more likely to be found at home, prevalence studies are likely to be biased by overestimating visual loss. Similarly, the sample from which incidence was estimated may have been selectively less mobile or less healthy, causing us slightly to overestimate the incidence of visual loss in this population. Finally, accurately identifying people who develop visual loss depends on the sensitivity of the E-optotype screening procedure. However, E-optotypes were associated with a high sensitivity and specificity, estimated to be 93% and 99%, respectively.

The eye surveys were done as part of a longitudinal study into the population dynamics of HIV infection in rural southwest Uganda, and were viewed as useful by the population because they provided an immediate additional health benefit to the population. Specifically, eye evaluation that could be done only in the district capital about 35 km away was now possible within their village. Thus, while it is possible that the serosurveys would have affected the response to the eye...
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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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References


References


High and low chromosome instability separates retinoblastomas

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.