Causes of visual loss and their risk factors: an incidence summary from the Barbados Eye Studies

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ABSTRACT

Objectives. *To summarize incidence and risk factors for each main cause of visual loss in an African-Caribbean population and discuss the implications of these data from a public health perspective.*

Methods. *A nationally representative cohort (*n = *4 709; ages 40–84 years at baseline) had ophthalmic and other examinations over 9 years. Incidence rates were estimated by the product-limit approach. Risk factors were evaluated from Cox regression models.*

Results. *Average incidence was ~ 0.1% per year for blindness (< 6/120) and 0.7% per year for low vision (< 6/18 to 6/120), increasing steeply with age (*P < *0.05) and affecting related quality of life (*P < *0.05). Age-related cataract and open-angle glaucoma (OAG) accounted for 73.2% of blindness and diabetic retinopathy (DR) for 8.9%; cataract caused two-thirds of low vision. Average incidence was 5.1% per year for all lens changes (gradable/ungradable opacities or aphakia) and 0.4% per year for cataract surgery. Incidence of definite OAG was 0.5% per year (0.9% for suspect or probable); 53% of the affected were unaware. Persons with diabetes mellitus (DM) had a DR incidence of 4.4% per year. Age-related macular degeneration was rare (0.08% per year). Main cataract risk factors were age and DM. OAG incidence increased with age, intraocular pressure, family history, low ocular perfusion pressures, and thinner corneas. DR risk increased with early DM onset, DM duration, oral/insulin treatment, increased systolic and diastolic blood pressures, and hyperglycemia. Antihypertensive treatment halved DR risk.*

Conclusions. *Incidence of visual impairment was high and significantly affected quality of life. Age-related cataract and OAG caused ~ 75% of blindness, indicating the need for public health action to increase appropriate cataract surgery and early OAG detection and treatment. Controlling DM and hypertension would help prevent DR-related complications and could lower cataract risk, further decreasing visual loss.*

Key words Vision disorders; vision, low; incidence; risk factors; Barbados.

Visual loss is a major public health problem. This condition has severe social and economic impacts, not only on individuals and their families but also on society at large (1–4). The extent of this effect was documented in recent evaluations, which found that visual disorders are associated with significant financial burden, a related loss in quality-adjusted

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life years, and disparities in access to medical care (1–4). Given its serious repercussions, loss of sight is a universally held concern, as confirmed by a recent multicountry study in which twice as many people reported fearing blindness more than early death (5). The magnitude of visual loss is very high worldwide and will increase as populations age. According to World Health Organization (WHO) estimates, more than 161 million persons were visually impaired in 2004 and more than 37 million were blind (6). Most (82%) were > 50 years of age and 90% lived in developing areas of the world. Globally, one person is estimated to go blind every 5 seconds, further underscoring the problem (7).

While adults of African descent are particularly at risk of blindness, limited data on eye diseases have been available for this group. The Barbados Eye Studies were designed to address this gap and to collect key information based on an African-Caribbean population (8). The main goals were to determine: (1) the frequency of visual impairment, thus obtaining data needed for public health planning; (2) its causes and risk factors, thus expanding knowledge on etiology; and (3) ways to reduce risk of visual loss, thus opening avenues to possible prevention and control. To date, the Barbados studies remain the main source of data on all major eye diseases in populations of African ancestry.

While the information provided by the Barbados studies has been presented in a number of publications, which address specific topics, no previous document has provided a summary overview of the latest incidence and risk factor data from the studies. The goals of this paper are the following:

- Provide a summary overview of the incidence and risk factors for each of the main causes of visual loss in the Barbados Eye Studies, based on long-term follow-up of the cohort.
- Discuss the implications of these data from a public health perspective.

MATERIALS AND METHODS

The Barbados Eye Studies were funded by the National Eye Institute, U.S. National Institutes of Health, with the overall goal of determining prevalence, incidence, and risk factors for all main eye diseases (age-related cataract, open-angle glaucoma (OAG), diabetic retinopathy (DR), and age-related macular degeneration (AMD)) in a population of African origin. After a pilot study in 1986, the Barbados Eye Studies began with a prevalence phase, which was based on a random sample of citizens of Barbados, West Indies, ages 40–84 years (n = 4 709; ~ 95% African descent by self-report; 84% participation) (8, 9). From 1992 to 2003, the cohort was followed to collect 4- and 9-year incidence data, also with high participation (81% to 85%). An additional group of probands with OAG and their families (n = 1 286) were examined as part of the Barbados Family Study of Open-Angle Glaucoma, which was designed to evaluate genetic factors for that condition.

Protocol

All study examinations followed the same standardized protocol, reported in detail in the references that follow. It included best corrected visual acuity (VA) (Ferris-Bailey chart, following a modified Early Treatment of Diabetic Retinopathy Study protocol), Goldmann tonometry, Humphrey perimetry, slit lamp lens gradings with the Lens Opacities Classification System II (LOCS II), color stereo fundus photography, automated refraction, a comprehensive interview, blood pressure (random zero sphygmomanometer), body circumferences and other measurements, and an ophthalmologic examination with dilatation and glycosylated hemoglobin testing. Fundus photographs were evaluated by masked graders at a reading center to determine possible glaucomatous optic neuropathy, diabetic changes, or macular changes. Vision-targeted quality of life was assessed by the National Eye Institute's 25-item visual functioning questionnaire (NEI-VFQ-25) at the 9-year examination (10–13). The NEI-VFQ-25 is a well-validated instrument designed to measure vision-targeted health-related quality of life (10). The questionnaire contains one general health rating question and vision-related subscales for overall vision, difficulties with near and distance vision activities, limitations in social functioning due to vision, role limitations due to vision, dependency on others due to vision, mental health symptoms due to vision, driving difficulties, limitations with peripheral and color vision, and ocular pain. Scores for all subscales and a composite score were calculated based on a 0 to 100 scale, with higher scores representing better vision-targeted quality of life (10–13).

Definitions

The VA component of the WHO criteria was used to define blindness (VA < 6/120) and low vision (VA from 6/120 to < 6/18), both in the better eye. Vision loss was defined as a decrease of ≥ 15 letters read correctly (better eye) between baseline and follow-up examinations (e.g., corresponding to a change in VA from 6/15 to 6/30) (14). Lens opacity types (cortical, nuclear, posterior subcapsular (PSC)) were defined by a LOCS II score of ≥ 2 in either eye (15). In addition, a broad definition of "any lens changes" included gradable or ungradable opacities or aphakia. OAG was defined by specific criteria that required the presence of glaucomatous visual field defects in either eye (by Humphrey C64 suprathreshold (three-zone strategy): C24-2 and C30-2 full-threshold tests), plus optic disc damage (horizontal or vertical cup-to-disc ratio ≥ 0.7, narrowest remaining retinal rim ≤ 0.1 disc diameter, notching, cup-to-disc ratio asymmetry > 0.2, and disc hemorrhages), in the absence of other possible causes, as determined by ophthalmologic evaluation (8, 16). Participants who partially met these strict criteria were classified as having probable or suspect OAG. DR was defined by specific fundus changes in the worse eye (≥ 3 microaneurysms, hard and soft exudates, intraretinal microvascular abnormalities (IRMA), new vessels within 1 disc diameter of the disc (NVD), new vessels originating elsewhere (NVE), and other abnormalities) in persons with diabetes mellitus (DM: physician diagnosis and/or glycosylated hemoglobin > 10%). Other features graded included clinically significant macular edema (CSME), defined by specific signs of retinal thickening or hard exudates near the center of the macula. Sight-threatening DR (STDR) was defined as severe/proliferative retinopathy (NVE/NVD) or CSME (17). AMD was assessed by the presence of atrophic/nonexudative (macular drusen and disturbance of the pigment epithelium) or atrophic/exudative (hemorrhage, fluid, lipid, or disciform scar) features in fundus photographs (18). Refractive errors were represented by the spherical equivalent (< –0.5 diopter (D) for myopia; > +0.5 D for hyperopia) (19).

Incidence and risk factor evaluation

The 9-year incidence was estimated by the product-limit approach (20). Incidence was defined by the presence of a given condition in either eye during the 9-year follow-up, based on persons without the condition at baseline. Risk factors evaluated varied as appropriate for each eye condition but generally were categorized into: (1) demographic: for example, age, sex, education, occupation; (2) medical: for example, hypertension (blood pressure > 140/90 mmHg or antihypertensive medication), ocular perfusion pressure (blood pressure minus intraocular pressure (IOP)), DM, body size (e.g., obesity = body mass index ≥ 30 kilograms per meter squared); (3) environmental: for example, smoking, alcohol use, sunlight exposure; (4) familial: for example, family history; and (5) ocular: for example, iris color, IOP, central corneal thickness, refractive error, and coexisting eye conditions. Relative risk (RR) or hazard ratio (HR) estimates were based on Cox proportional hazards regression models with discrete-time data (21). Odds ratio (OR) estimates were based on logistic regression models for cross-sectional data. The Statistical Analysis System (SAS; Institute Inc., Cary, NC) was used for the analyses. Results presented are limited to African-Caribbean participants, given the small number of persons in other groups.

RESULTS

Baseline data on the population-based random sample indicated nationwide representation, as compared to census information. The median age of participants was 58 years, 57% were female, and median educational duration was 10 years. Hypertension was present in 55% of participants, DM in 18%, and obesity in 33% of women and 11% of men.

TABLE 1. Nine-year incidence of blindness and low vision[a] by age and sex and estimated average annual incidence in individuals of African descent, Barbados, 1992–2003

	Blindness			Low vision		
	No.[b]	%	95% CI[c]	No.[b]	%	95% CI[c]
Age (years)						
40–49	1 065	0.0	NA[d]	1 062	0.9	0.4, 1.7
50–59	894	0.1	0.0, 1.0	878	2.8	1.8, 4.2
60–69	772	1.2	0.6, 2.5	747	8.1	6.2, 10.6
≥70	563	4.3	2.7, 6.9	486	23.0	18.8, 28.0
Sex						
Male	1 369	1.1	0.6, 1.9	1 311	7.1	5.8, 8.8
Female	1 925	0.9	0.5, 1.4	1 862	5.2	4.2, 6.3
Overall 9-year incidence	3 294	1.0	0.7, 1.4	3 173	6.0	5.1, 6.9
Average annual incidence		~ 0.1			~ 0.7	

[a] Blindness: visual acuity < 6/120 in the better eye; low vision: 6/120 ≤ visual acuity < 6/18) in the better eye.
[b] No. = number of persons at risk.
[c] CI = confidence interval.
[d] NA = not applicable.

Table 1 provides blindness and low-vision incidence by age and sex. The 9-year incidence of bilateral blindness was 1.0%, or an average estimate of ~ 0.1% per year. Incidence increased steeply with age ($P < 0.05$) and tended to be higher in men than in women. While no new cases occurred in persons aged 40–49 years, comparisons with persons aged 50–59 years indicated a 38-fold increase in blindness (HR = 38.4 (95% confidence interval (CI): 5.1, 288.0)) for persons ≥ 70 years and a 10-fold increase in the 60- to 69-year age group (HR = 9.9 (95% CI: 1.2, 79.5)).

As also indicated in Table 1, the overall 9-year incidence of low vision in the better eye was 6.0% or 0.7% per year. With the frequently used cutoff of VA ≤ 6/12 in the better eye, the 9-year incidence was 10.1% (95% CI: 9.0, 11.3), or 1.1% per year. Rates increased with age ($P < 0.05$) and were significantly higher in men ($P < 0.05$, adjusting for age). Additional details on unilateral and bilateral incidence of VA loss are provided in a previous report (14).

Table 2 presents the 9-year incidence of vision loss (doubling of the visual angle), which was 5.5% or 0.6% per year. Rates increased with age ($P < 0.05$) and, compared with persons aged 40–49 years, those ≥ 70 years had a 16-fold increase (HR =16.0 (95% CI: 8.8, 29.3)) and those aged 60–69 years had a 6-fold increase (HR = 5.8 (95% CI: 3.1, 10.8)). Men tended to have higher rates than women, with statistically significant differences among participants aged 60–69 years ($P < 0.05$).

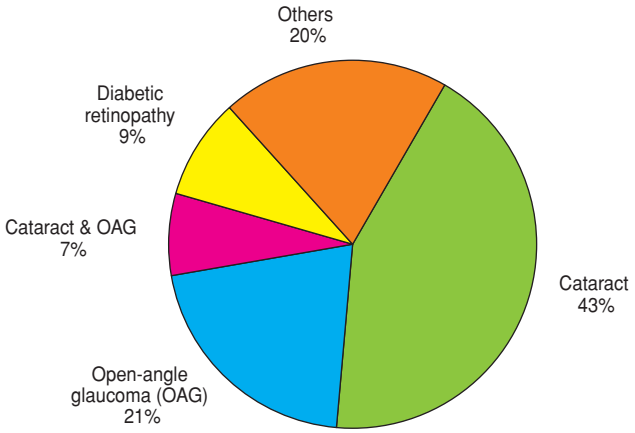
Impaired vision had a major adverse impact on self-perceived quality of life, as determined by the NEI-VFQ-25 (12). Persons with decreased vision had significantly ($P < 0.01$) lower scores in multidimensional aspects of vision-related

TABLE 2. Nine-year incidence of vision loss (doubling of the visual angle)[a] by age and sex and estimated average annual incidence in individuals of African descent, Barbados, 1992–2003

	Male			Female			Total		
	No.[b]	%	95% CI[c]	No.[b]	%	95% CI[c]	No.[b]	%	95% CI[c]
Age (years)									
40–49	471	1.3	0.5, 3.0	594	1.5	0.8, 0.3	1 065	1.4	0.8, 2.4
50–59	345	3.0	1.6, 5.7	548	2.8	1.6, 4.8	893	2.9	1.9, 4.4
60–69	299	10.7	7.3, 15.7	470	5.9	3.9, 8.8	769	7.7	0.6, 10.2
≥70	256	19.4	13.7, 27.1	316	15.5	11.3, 21.1	572	17.1	13.5, 21.5
Overall 9-year incidence	1 371	6.3	5.0, 7.9	1 928	4.9	4.0, 6.1	3 299	5.5	4.7, 6.4
Average annual incidence		0.7			0.5			0.6	

[a] Decrease of 15 letters or more read correctly in the better eye.
[b] No. = number of persons at risk.
[c] CI = confidence interval.

FIGURE 1. Primary causes of incident bilateral blindness (visual acuity < 6/120) in the Barbados Eye Studies; 9-year follow-up (*n* = 56 eyes), 1992–2003



quality of life, particularly when the impairment was noncorrectable with refraction. Thus, the presence of OAG, PSC lens opacities, or aphakic cataract surgery had a marked impact on visual functioning and related quality of life (11).

The primary causes of incident blindness are shown in Figure 1, based on 56 bilaterally affected eyes. Cataract and glaucoma were responsible for nearly three-fourths of all blindness. The most frequent causes were cataract alone (42.9%) and OAG alone (21.4%), with some participants being blind from cataract and OAG combined (7.1%). Overall, frequencies were 71.4% for cataract and OAG, or 73.2% when also including secondary glaucoma. Optic atrophy (12.5%) and DR (8.9%) were additional causes; other conditions were infrequent. As to low vision, cataract accounted for 65% or almost two-thirds of cases, with retinal conditions and OAG being less frequent causes. Most (81%) visual impairment was not correctable with refraction.

From Table 3, the overall incidence of any type of lens change (gradable or ungradable opacities or aphakia) was 46.0%, or 5.1% per year. Rates increased markedly with age, from 22.3% at ages 40–49 years to 83.6% at ages ≥ 70 years and tended to be higher in women than in men (P = 0.07 after age adjustment; data not shown). When considering specific types of opacities, cortical-only types were the most frequently developing (23.2% or 2.6% per year), followed by nuclear-only types (17.1% or 1.9% per year) and mixed opacities (15.3% or 1.7% per year); PSC-only opacities were infrequent (0.6% or 0.07% per year). Cortical-only opacities affected mainly younger participants, while nuclear-only and mixed opacities were most frequent in older age groups; the latter were also more frequent in women than in men (P < 0.01 after age adjustment), without significant gender differences for other types (15).

Over the 9-year follow-up period, the incidence of cataract surgery was 4.2% (95% CI: 3.5%, 5.0%), or 0.4% per year. The 9-year incidence increased with age, ranging from 0.4% at ages 40–49 years to 14.4% at 70 years or older; rates were similar for men (4.4% (95% CI: 3.2%, 5.6%)) and women (4.1% (95% CI: 3.2%, 5.1%)). A slight increase in surgery was noted over the 9 years of follow-up, primarily among persons 80 years or older, where rates increased from 12.9% at baseline to 15.2% at 4 years and 21.8% at 9 years (15).

In African-Caribbean participants, the age- and sex-adjusted frequency of any lens changes was almost double that of white participants (RR = 1.8 (95% CI: 1.2%, 2.8%); data not shown), a difference that was mainly due to a 3-fold increased risk of cortical opacities (RR = 3.2 (95% CI: 1.7%, 6.2%)). In addition to age, a major risk factor for all types of lens opacities was DM, which doubled the risk for cortical opacities (RR = 2.4 (95% CI: 1.8, 3.2)), tripled the risk of PSC opacities (RR = 2.9 (95% CI: 1.9, 4.5)), and also influenced the risk of nuclear opacities (RR = 1.6 (95% CI: 1.1, 2.4)). The incidence of cortical opacities was increased in women and in those with low socioeconomic status, while aspirin decreased risk; for nuclear opacities, related factors were dark iris color, myopia, leaner body mass, and treatment to lower IOP (22, 23).

The 9-year incidence of definite OAG was 4.4%, or 0.5% per year, and, as shown in Table 4, it increased considerably with age (P < 0.0001). When jointly considering definite plus suspect or probable disease, the overall OAG incidence was 8.4%, or 0.9% per year (16).

TABLE 3. Nine-year incidence of single and mixed types of lens opacities, and all lens changes, and estimated average annual incidence in individuals of African descent, Barbados, 1992–2003

	Cortical only		Nuclear only	PSC[b] only	Mixed	All lens changes	
	No.[a]	%	(%)	(%)	(%)	No.[a]	%
Age (years)							
40–49	1 000	15.1	4.5	0.7	4.6	1 022	22.3
50–59	651	29.9	24.1	0.5	17.6	685	59.5
60–69	305	36.3	42.3	0.7	41.6	336	86.1
≥70	52	19.2	47.3	0.0	60.9	71	83.6
Overall 9-year	2 008	23.2	17.1	0.6	15.3	2 114	46.0
incidence (95% CI)[c]		(21.3, 25.2)	(15.3, 18.9)	(0.2, 0.9)	(13.6, 17.1)		(43.8, 48.3)
Average annual incidence		2.6	1.9	0.07	1.7		5.1

[a] No. = number of persons at risk.
[b] PSC = posterior subcapsular.
[c] CI = confidence interval.

TABLE 4. Nine-year incidence of definite, suspect or probable, and all open-angle glaucoma, by age, and estimated average annual incidence in individuals of African descent, Barbados, 1992–2003

	Definite OAG[a]			Suspect OAG[a]			Definite and suspect OAG[a]		
	No.[b]	Incidence (%)	95% CI[c]	No.[b]	Incidence (%)	95% CI[c]	No.[b]	Incidence (%)	95% CI[c]
Age (years)									
40–49	1 060	2.2	1.5, 3.4	1 036	3.6	2.6, 5.0	1 036	5.3	4.0, 6.9
50–59	878	3.6	2.5, 5.1	850	5.1	3.8, 6.9	850	7.0	5.4, 9.1
60–69	742	6.6	4.9, 8.9	705	7.7	5.8, 10.2	705	12.1	9.7, 15.1
≥70	542	7.9	5.6, 11.1	510	8.4	5.9, 11.8	510	13.4	10.2, 17.5
Overall 9-year incidence	3 222	4.4	3.7, 5.2	3 101	5.6	4.8, 6.5	3 101	8.4	7.4, 9.5
Average annual incidence		0.5			0.6			0.9	

[a] OAG = open-angle glaucoma.
[b] No. = number of persons at risk.
[c] CI = confidence interval.

The frequency of undiagnosed disease was high, as more than half of participants with incident OAG were unaware of their diagnosis. Lack of OAG awareness was related to lower IOP at baseline (OR = 0.86 (95% CI: 0.8, 0.9)), hyperopia (OR 2.7 (95% CI: 1.1, 6.7)), and patterns of eye care utilization. The unaware group had less frequent visits for eye care than those who were aware of their OAG diagnosis (33.4% and 64.4%) and mainly for eyeglasses (71.4% and 12.5%). They were four times more likely to consult optometrists or opticians than private ophthalmologists (OR = 4.2 (95% CI: 1.0, 7.7)) and much less likely to consult a public ophthalmologic clinic (OR = 0.2 (95% CI: 0.04, 0.9)) (24).

Risk factors for definite OAG, in addition to age, were IOP (RR = 1.1 (95% CI: 1.1, 1.2) per mmHg); glaucoma family history (RR = 2.4 (95% CI: 1.3, 4.6)); lower systolic, diastolic, and mean perfusion pressures (RR = 0.9, 0.8, and 0.7, respectively, all $P < 0.05$); and thinner corneas (OR = 1.4 (95% CI: 1.0, 2.0)). Lower systolic blood pressure was marginally significant ($P = 0.05$) (25).

Almost 20% of the population met the criteria for DM at baseline, mainly having type 2 diabetes, as type 1 is infrequent in this population (26). Table 5 presents the incidence of DR among persons with DM, based on masked photogradings in the worse eye. Overall 9-year incidence was 39.6%, or about 4.4% per year. Most persons were classified as having minimum DR (≥ 3 microaneurysms, soft or hard exudates, retinal hemorrhages); moderate (IRMA or venous beading) and severe/proliferative DR (NVE or NVD) were less frequent. The estimated average incidence of STDR was 0.9% and was very similar to that of CSME (1.1%), which was its major component. No specific patterns in incidence were observed by age or gender.

Of the participants with minimum or moderate retinopathy at baseline, 8.2%, or 0.9% per year, progressed to proliferative DR. After including individuals who had pan-retinal photocoagulation during the 9-year period, the rate of progression would increase to 12.9% (95% CI: 6.1%, 19.6%).

The incidence of DR, CSME, and STDR tended to increase with duration of DM and varied with DM treatment type. Compared with persons with none or diet-only treatment, users of oral or insulin therapy were 2.4 times (95% CI: 1.4, 3.9) more likely to develop any DR and 3.6 times (95% CI: 1.1, 12.2) more likely to develop CSME. The 9-year risk of DR was also related to age at DM onset, with a 30% decrease in risk for each 10 years of age at DM diagnosis (RR = 0.7 (95% CI: 0.6, 0.96)). In contrast, the risk of DR increased by 30% for each 10 mmHg higher systolic blood pressure at baseline (RR = 1.3 (95% CI: 1.1, 1.4)). High diastolic blood pressure was also related to increased DR risk ($P < 0.05$), while antihypertensive medication halved the risk of DR compared with no treatment (RR = 0.5; 95% CI: 0.3, 0.9) (27).

DISCUSSION

The Barbados Eye Studies provide extensive data on the prevalence, incidence, progression, and risk factors for all major causes of blindness in adults of African descent (≥ 40 years). The wealth of information provided by these 9-year studies has been reported in more than 140 published papers and abstracts and has assisted in developing population estimates for visual impairment and its causes (e.g., among African-Americans) (28–32). This communication summarizes these data and their implications, thus providing a public health perspective on the findings.

Table 6 summarizes average annual incidence rates. Overall, the results show a high frequency of conditions causing visual impairment. In addition to its known deleterious effects, such impair-

TABLE 5. Nine-year incidence and progression of diabetic retinopathy in participants with diabetes mellitus and estimated average annual incidence in individuals of African descent, Barbados, 1992–2003

Incidence	No.[a]	Nine-year incidence or progression (%)	95% CI[b]	Average annual incidence or progression (%)
Any DR[c]	324	39.6	33.6, 45.5	4.4
Minimum DR	324	38.0	32.1, 43.9	4.2
Moderate DR	400	9.0	5.5, 12.4	1.0
Proliferative DR	433	2.6	1.0, 4.3	0.3
Sight-threatening DR	387	8.3	5.1, 11.5	0.9
CSME[d]	377	8.7	5.4, 12.0	1.1
Progression to proliferative DR (NVD/NVE)[e]	109	8.2	2.5, 13.9	0.9

[a] No. = number of persons at risk.
[b] CI = confidence interval
[c] DR = diabetic retinopathy.
[d] CSME = clinically significant macular edema.
[e] NVD = new vessels of the disc, NVE = new vessels originating elsewhere.

TABLE 6. Summary of average annual incidence for visual impairment and major causes in individuals of African descent, Barbados, 1992–2003

Condition	Incidence, % per year
Blindness (< 6/120)	0.1
Low vision (< 6/18 to 6/120)	0.7
Vision loss (doubling of visual angle)	0.6
All lens changes (gradable/nongradable opacities and aphakia)	5.1
Definite open-angle glaucoma	0.5
All open-angle glaucoma (including suspect/probable)	0.9
Diabetic retinopathy (among persons with diabetes mellitus)	4.4
Late age-related macular degeneration	< 0.1

ment had an important negative impact on overall visual functioning and related quality of life (11, 12). These findings highlight the urgent need for appropriate public health policies and actions to decrease visual loss.

Blindness and low vision

The prevalence of blindness at baseline, 1.7%, was substantially higher than rates in other populations, which are usually well below 1% (33). A similar pattern was observed at follow-up, with higher incidence and progression of vision loss than found elsewhere. Long-term studies in other populations (which defined VA impairment as ≤ 6/60) have reported annual rates between 0.04% and 0.1% (34–37), while the comparable estimated yearly rate (0.2%) in the Barbados studies was at least double that rate (14). Reasons for these large discrepancies are likely multifactorial, including access to care, cultural factors, and economic factors.

To decrease the burden of visual loss, it is important to address its underlying causes. Almost three-fourths of all blindness was due to cataract and OAG (Figure 1), which clearly identifies these two conditions as the main targets for intervention in this and similar populations. Approximately 2 of every 5 cases of incident blindness were due to age-related cataract alone, 1 in 5 was attributable to OAG, and 1 in 15 was due to both conditions. Furthermore, cataract was responsible for about two-thirds of incident low vision (14).

Cataract and OAG

It is unclear why opacities are twice as frequent in participants of African origin versus those of European descent, but this disparity could be influenced by the 3-fold higher frequency of cortical opacities (15). Gene–environment interactions are likely responsible for these findings. The disparity in cortical opacities could be related to the strong association of these opacities with DM (22), which was frequent in the study population, but DM was also related to PSC (22) and nuclear opacities (23). Control of DM may thus decrease the risk of all opacity types and perhaps lead to a reduction in cataract-induced vision loss.

Since cataract is successfully treated with appropriate surgery, most blindness from this cause could be prevented. Despite the impact of lens opacities on visual status, the frequency of cataract surgery over the study period was only 0.4% per year, which seems insufficient to address the high volume of incident cases. While the trend toward a modest increase in rates over time was encouraging, a substantial increase in cataract surgery would be needed to eliminate the backlog of existing cases, as well as to keep pace with new cases.

The importance of cataract as a cause of impairment is not surprising. Worldwide, cataract predominates as the prime cause of blindness, followed by glaucoma (6). However, OAG is a key reason for visual impairment in populations of African descent, as documented by our incidence and prevalence data. At baseline, OAG had a high prevalence of 7% in the study population of African ancestry, while prevalence was 0.8% in people of European ancestry and 3.3% among people with mixed (black and white) ancestry (8). The follow-up data also confirmed the high OAG risk, estimated at 0.5% per year for definite cases and almost 1% per year when suspect or probable cases were included. In contrast, the few studies of OAG incidence report annual rates of 0.10% to 0.24% in Australian and European populations (38–40). Furthermore, OAG was also a major cause of irreversible visual loss at baseline, as 28% of blindness was caused by OAG, 28% by cataract, and 4% by both conditions (33). The high OAG rates are a major concern, as the disease cannot be prevented, at present.

In addition to age, the high OAG risk in our study population was strongly related to family history, with ancestral genetic factors probably underlying this risk. Findings from the Barbados Family Study of Open-Angle Glaucoma, based on probands with OAG and their family members, suggest that the disease likely follows a Mendelian codominant inheritance pattern. These and other results from the Family Study have been published (41–44) and analyses are ongoing to discover causative genes for OAG. Recently, these genetic analyses have identified a locus with a major impact on glaucoma susceptibility (45).

IOP was a major factor influencing the risk of OAG (25). In this regard, the IOP in study participants (mean = 18.0 (standard deviation 4.1) at baseline) was higher than that reported in other populations (46). While IOP was strongly related to OAG incidence, most incident cases arose in persons without elevated IOP at baseline, thus highlighting the limitations of IOP as a predictor of OAG risk (47). As found in our study, persons of African descent have thicker corneas than Europeans, a trait that may cause underestimation of IOP (48). An association between central corneal thickness and OAG has been reported and the presence of thin corneas was also related to OAG in our study participants (25).

The etiology of OAG is multifactorial and likely influenced by gene–environment interactions. Our analyses also suggested a role for vascular factors, with low ocular perfusion pressures showing a strong association with OAG risk (25). The relationships with low perfusion pressure have been consistent in other large epidemiologic studies, further confirming a vascular involvement (49). The role of blood pressure alone is controversial, however, as evidence in this regard has been inconsistent (50–56). Therefore, the high frequency of hypertension in this population of African descent is not the explanation for the high OAG risk.

Given the importance of OAG in the study population, a major concern is lack of awareness of the disease, as more than half of those affected did not know their diagnosis. This finding has been reported in other studies, as OAG has no symptoms until an advanced stage (49). Our results strongly suggest the need for programs to increase awareness of OAG. The major strategy to reduce visual loss from glaucoma is to enhance early detection and treatment. To maximize effectiveness, these efforts should target the

high-risk groups identified from risk factor analyses, in particular the relatives of OAG patients. After diagnosis, appropriate clinical management is essential for early and sustained treatment.

Diabetic retinopathy and macular degeneration

Another important finding was the high prevalence of DM in the study population, which affected 17.5% or almost one-fifth of adults (57), leading to DR complications and an increase in cataract risk. Approximately two-fifths of participants with DM and free of DR at baseline developed DR during the 9-year follow-up, based on a standardized photograph grading system. The DR-related rates represent the first such documentation in an African-descent population and limited data are available for comparison. The overall DR incidence and that of proliferative DR (2.6%) appear somewhat lower than rates in the Wisconsin Epidemiologic Study of DR (58). The discrepancies could be due to study design, photographic protocol, and population differences; for example, the Wisconsin study was based on persons with a DM diagnosis and not on an unselected population. In contrast, our 9-year incidence of CSME (8.7%) seemed higher and suggested an elevated CSME risk (17).

The evaluation of risk factors indicated a role for age and duration of DM onset as well as for hyperglycemia (27). These results are consistent with previous research (59–63) and suggest that control of DM would result in decreased development of DR. The association of DR with high blood pressure, as found in our study, has been inconsistent (63). However, our finding that antihypertensive treatment reduced DR by half was particularly encouraging, as it appears to open a path to preventing DR and its complications.

Of interest, although visual loss from AMD is the main cause of blindness in European-descent populations (6, 28, 30), it was extremely infrequent in our study population (18). Early macular changes (e.g., drusen) were as frequent as in other populations, but exudative changes were rare and few persons lost vision from that cause. The low frequency of age-related maculopathy in populations of African origin has been confirmed in other studies as well as in our previous reports (64–67).

Strengths and weaknesses

The strengths of the Barbados studies include the population-based, national representation as well as the extended length of follow-up. Also, a good participation was achieved at follow-up, with examinations completed on 85% and 81% of those remaining eligible at 4 years and 9 years, respectively. The study design included many features to ensure accurate results. Standardized protocols were used throughout, allowing comparability during the study period and including ongoing quality control assessments. Disease definitions were based on rigorous, preset criteria, using specific and objective measures (e.g., automated perimetry, masked photogradings). Given the strict definitions and standardization, misclassification was likely to be minimal. All personnel were certified in study procedures, and reproducibility evaluations were performed during all phases of the study period. As an additional quality control measure, a comprehensive ophthalmologic examination was completed on every 10th participant, regardless of screening results, with no false negatives detected.

As expected in all cohort studies of an older population, the main study limitation concerned losses to follow-up, which were inevitable, with death being a main reason for nonparticipation in later examinations. Persons lost to follow-up were older and more likely to have been hypertensive at baseline but had a similar ancestry distribution. Biases due to selective mortality or the competing risk of death could have led to possible underestimates—for example, of the magnitude of the incidence rates. We attempted to adjust for such risk in our glaucoma estimates, however, with negligible effect on the rates (16). Risk factor evaluations could have been affected by unadjusted confounding, which cannot be discounted in epidemiologic analyses. Finally, interpretation of our results must consider the specific characteristics of our study population, which limit extrapolation to other populations.

Conclusions

This article has summarized incidence estimates and risk factors for the main causes of visual impairment in a population of African origin and discussed the public health implications of these data. The information is based on a long-term cohort study of a representative national sample. Such information is of substantial public health value, as the burden of blindness and visual impairment, which is already considerable, will increase further as the population ages.

The Barbados Eye Studies data, summarized here, will allow health planners to estimate the magnitude of visual impairment in similar populations; it will also allow the identification of high-risk groups for targeting interventions. Few populations have such rich data at their disposal. These data indicate the need for a much broader dissemination of prevention and control strategies, especially aimed to reduce visual loss from cataract, OAG, and DR. In particular, study findings suggest the following recommendations:

- Increase resources for the provision of cataract surgery.
- Enhance early detection and treatment of OAG—for example, by targeting high-risk groups such as relatives of OAG patients and providing appropriate clinical management after diagnosis.
- Decrease the development of DR by controlling DM and blood pressure.

To realize the full potential of these research findings, the results now need to be applied and public health strategies implemented on the basis of the information gained.

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RESUMEN

Causas de la pérdida visual y sus factores de riesgo: resumen de la incidencia a partir de los Estudios de Oftalmología de Barbados

Objetivos. Presentar un resumen de la incidencia y los factores de riesgo de cada causa principal de pérdida de la visión en una población afrocaribeña y examinar las implicaciones de estos datos desde una perspectiva de salud pública.

Métodos. En una cohorte representativa al nivel nacional (n = 4 709; edades de 40 a 84 años al inicio) se hicieron exploraciones oftálmicas y de otros tipos durante nueve años. Se calcularon las tasas de incidencia mediante el método del producto-límite. Los factores de riesgo se evaluaron mediante modelos de regresión de Cox.

Resultados. La incidencia promedio fue ~ 0,1% al año para la ceguera (< 6/120) y de 0,7% al año para la visión deficiente (< 6/18 a 6/120), que aumentó de manera pronunciada con la edad (P < 0,05) y afectó a la calidad de vida relacionada (P < 0,05). Las cataratas y el glaucoma de ángulo abierto relacionados con la edad representaron 73,2% de los casos de ceguera, y 8,9% de los casos de retinopatía diabética; las cataratas causaron dos tercios de los casos de visión deficiente. La incidencia media fue de 5,1% al año en todos los cambios del cristalino (opacidades graduables o no graduables o afaquia), y de 0,4% al año en la cirugía de cataratas. La incidencia del glaucoma de ángulo abierto definitivo fue de 0,5% al año (0,9% en el caso de la sospecha o la probabilidad); 53% de los pacientes afectados no era conciente. Las personas que padecían diabetes tenían una incidencia de retinopatía diabética de 4,4% al año. La degeneración macular relacionada con la edad fue muy infrecuente (0,08% al año). Los principales factores de riesgo de las cataratas fueron la edad y la diabetes. La incidencia de glaucoma de ángulo abierto aumentó con la edad, la presión intraocular, los antecedentes familiares, las presiones bajas de perfusión ocular y el grosor más fino de la córnea. El riesgo de retinopatía diabética aumentó con la diabetes de inicio temprano, la duración de la diabetes, el tratamiento con antidiabéticos orales o insulina, el aumento de la presión sistólica o diastólica, y la hiperglucemia. El tratamiento hipotensor redujo el riesgo de retinopatía diabética a la mitad.

Conclusiones. La incidencia de trastornos visuales fue alta y afectó de manera significativa a la calidad de vida. Las cataratas relacionadas con la edad y el glaucoma de ángulo abierto causaron aproximadamente 75% de los casos de ceguera, lo que indica la necesidad de adoptar medidas de salud pública destinadas a aumentar la cirugía de cataratas adecuada y la detección y el tratamiento tempranos del glaucoma de ángulo abierto. El control de la diabetes y la hipertensión ayudaría a prevenir complicaciones relacionadas con la retinopatía diabética y podría reducir el riesgo de cataratas, lo que disminuirá aún más la pérdida de la visión.

Palabras clave Trastornos de la visión; baja visión; incidencia; factores de riesgo; Barbados.