

Blinding Conditions: A review of the Literature

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Introduction

In this section, the common and important preventable and treatable blinding conditions are reviewed with emphasis on those that form major public health problems in developing countries.

The four main causes are cataract, trachoma, onchocerciasis and xerophthalmia in most developing countries where it occurs at ten times the rate of that seen in the developed countries; in over 80% of cases is either preventable or curable. ^{(1), (2), (3)} Data on other blinding conditions, such as diabetic retinopathy in the developing countries, are scarce. ⁽⁴⁾

1. Cataract

Prevalence of Cataract

Cataract is the leading cause of blindness worldwide in both developed and developing

countries as a result of the increasing ageing population. It is responsible for half the blindness in the world, with an estimated 20 million affected. ^{(5), (6), (7)} Figures range from as low as 0.05% in North America, Europe, the former USSR, and Oceania, to 0.15% in Central and South America and reaching as high as 0.3% in Asia, and 0.5% in Africa. ⁽⁸⁾ In the poorer countries, the prevalence is also variable. ^{(9), (10), (11), (12), (13), (14)} In these countries, ^{(8), (9), (11), (12), (13)} financial and cultural barriers often exist to accessing surgical services even when available. This is combined with low surgical throughput and limited resources, together with a shortage of ophthalmic specialists. ^{(15), (16)} An additional problem in these countries is the very low use of intraocular lens implants (IOLs); in India for example, IOLs are used in only 11% of all cataract surgery. Rapid progress, however, is taking place by producing cheap locally manufactured IOLs combined with the necessary surgical training. ⁽⁶⁾

Aetiology and Risk Factors

Cataract has a complex multifactorial aetiology. A number of risk factors have been suggested for its formation, with age at the top of the list, followed by exposure to ultraviolet light.

Additional definite risk factors are prolonged exposure to ultraviolet-B radiation (UV-B, sunlight), diabetes, smoking, steroids and being female. Other possible factors are: alcohol, oestrogen, low education, low body mass and weight, low social class, rural residence, severe diarrhoea or dehydration, renal failure and one ocular condition which is myopia. On the other hand, the use of dietary antioxidant vitamins such as vitamin E and aspirin are thought to have a protective effect.^{(8), (11), (17)} A study in Tibet has demonstrated that cataract incidence increases with a decrease in latitude or increase in altitude due to increased solar radiation.⁽¹⁸⁾ In the surveyed areas, Zedang region, which has the highest altitude and low latitude (29° north), harbours the highest incidence of cataract at 1.32%, followed by Aleitai at 0.25% and Zhongshan at 0.23%. On the other hand, the incidence in lowland areas are between 0.12 and 0.14%, and in the lowest surveyed, Zhongmou, the incidence is as low as 0.066%. Until the results of current studies into the effectiveness of antioxidant vitamin supplements become available, the only effective protective interventions to reduce the risk of cataract seem to be to reduce ocular exposure to UV-B radiation and to stop smoking.⁽¹⁷⁾

Gender in Cataract Blindness

Globally, women bear an excess of blindness compared to men accounting for 64.5% of all blind people.^{(19), (20)} There is a good homogeneity of findings from Africa, Asia, and the industrialised countries, being marked among the elderly. The overall ratio of blind women to men is 1.43:1, ranging from 1.39:1 to 2:1 in Africa, 1.41:1 in Asia, and 1.63:1 in industrialised countries. The difference is not only due to longer life expectancy, but is likely to be due to a number of factors which are different in industrialised countries compared to developing countries.⁽¹⁹⁾ In the

latter, the prohibitive cost of surgery (cost, transportation, loss of income, and living expenses for the guardian, etc.), the lower disposable income or control of finances available to women, and the perceived “value” of cataract surgery, especially as community-based education about cataract has not been undertaken in most areas, all play a part.^{(10), (21)}

Cataract Blindness in Children

In children, cataract is also the most important cause of treatable childhood blindness with an estimated 200,000 children blind from cataract worldwide, representing 14% of the total number of childhood blindness. Each year some 20,000 to 40,000 children are born with congenital bilateral cataract.^{(22), (23)} The incidence varies from 1 to 3/10,000 live births in low-income countries. It has been shown that when babies are screened in maternity wards, much earlier referrals for surgery are achieved in comparison to well-baby clinics or no formal screening. The need for standardised protocols for screening babies for ocular anomalies, in particular cataract, before discharge from maternity units has been advocated.⁽²²⁾

Rubella remains an important cause of congenital cataract, deafness and congenital anomalies in many developing countries where there is a need to improve early case detection and referral services to specialised centres geared to provide expert management of this condition.⁽²³⁾

Congenital Rubella Syndrome

The WHO has provided recommendations for prevention of CRS, and, encouragingly, the number of countries introducing rubella vaccination programmes has risen. Maternal rubella is now rare in many developed countries that have rubella vaccination programmes. However, declining uptake rates due to concerns about the measles-mumps-rubella vaccine in the UK, and increasing numbers of cases in some European countries, coupled with poor uptake rates, might jeopardise this progress. Surveillance of postnatally and congenitally acquired infection is an essential component of CRS prev-

ention since rubella is difficult to diagnose on clinical grounds alone. Laboratory differentiation of rubella from other rash-causing infections, such as measles, parvovirus B19, human herpes virus 6, enter in developed countries, and various endemic arboviruses is essential. Reverse transcriptase PCR and sequencing for diagnosis, molecular epidemiological investigation, and detection of rubella-specific IgG and IgM salivary antibody responses in oral fluid are now available.⁽²⁴⁾

2. Glaucoma

Prevalence of Glaucoma

Glaucoma^{(25), (26)} is the second leading cause of blindness in developed countries and a major cause of blindness worldwide; one person in 10 will eventually develop glaucoma. The estimated number of people affected by the condition is about 66.8 million, with 6.7 million suffering from bilateral blindness.^{(27), (28)}

Glaucoma is also a major cause of blindness in developing countries with a much higher proportion of the narrow angle type than in the Western hemisphere, especially in East Asia.^{(29), (30), (31)} Foster et al found that 9.7% of patients examined in China had either manifest, latent, or suspected glaucoma and believed that the same proportion may exist in neighbouring populations such as Mongolia because of the shared genetic heritage.^{(32), (30), (31), (25)} In these countries, fewer than 50% of those with glaucoma are aware of their disease.⁽²⁷⁾ Because of this, it is known as 'the little thief' in Saudi Arabia.⁽³³⁾ In the developed countries, glaucoma blindness develops at a later age leaving the patients blind for a shorter time. This pattern may be explained by improved health services and advances in screening and management of the condition, although the possibility of a change in the course of the disease has also been suggested. The percentage of glaucoma blindness in the population at risk was estimated to be 4-5%.

Glaucoma Screening

New advances in psychophysical testing, such as frequency doubling perimetry, and developments in optic nerve imaging, such as the SLO offer the potential to develop new screening modalities that are both more sensitive and more specific for use in community based screening programmes for glaucoma. They, together with the targeting of those with a positive family history of glaucoma, are likely to significantly decrease the proportion of people with glaucoma who are undiagnosed and not on treatment. In industrialised countries, screening for glaucoma is well established.^{(25), (28), (34)}

Risk Factors

Risk factors incriminated in the aetiology of POAG are: age, family history, being African in origin, height of IOP, diabetes, hypertension, evidence of vascular spasm such as migraine and one ocular factor which is myopia. People with a family history of glaucoma have an approximately four times increased risk of developing glaucoma. The identification of the first genes associated with glaucoma was a major breakthrough. Although these genes only account for a small percentage of cases, they clearly indicate that a family history is important.^{(25), (30)}

3. Diabetic Retinopathy

Prevalence

Diabetes affects nearly 3% of the population and diabetic retinopathy is a leading cause of visual impairment in the working age groups in western society.^{(35), (36), (37), (38)} The need for earlier diagnosis and the success of the early management of diabetic retinopathy is now well recognised; blindness can be reduced by 50% by Argon laser treatment. This concept was boosted by the St Vincent Declaration in 1989.^{(38), (37), (39), (40), (41), (42)} Diabetic retinopathy fulfils all the parameters for a condition that is ideal for screening.⁽⁴³⁾

Risk

The possible risk factors for diabetic retinopathy

includes: duration of diabetes, glycaemic control (blood glucose and glycosylated haemoglobin), type of diabetes (age at onset), diabetic treatment (insulin, oral hypoglycaemic, dietary), systemic hypertension, renal function/nephropathy, body mass, sex, HLA status, cigarette smoking and elevated blood lipids. Risk factors in diabetic maculopathy have received less attention in the literature. These include: duration of diabetes, glycaemic control, age, sex, age of diagnosis, insulin use, higher glycosylated haemoglobin, use of diuretic, systemic hypertension, plasma lipids level and proteinuria have been associated with diabetic macular oedema.^{(35), (36), (44)} Some of the risk factors such as renal disease and age (< 50 years) were identified as risk factors for non-regression after pan retinal photocoagulation (PRP) making these patients in need of a higher dose of treatment than that suggested by earlier studies.⁽⁴²⁾ New associations between serum lipoproteins and severity of retinopathy in type-1 diabetes, has been found which also points to the role of dyslipoproteinemia involving lipoprotein subclasses in the pathogenesis of diabetic retinopathy.^{(35), (36), (37), (38), (45), (46), (47)}

Blindness from Diabetes

The incidence rate of blindness in the diabetic population has come down by only 17% in Germany in 1977 indicating the need for more vigorous attention to preventive measures for microvascular complications especially in the younger age-group.⁽⁴¹⁾ There are no estimates of the prevalence of diabetic blindness in the developing countries; however, there is evidence that this, together with other diabetic complications, is a major public health problem in these countries as a result of lack of screening programmes, health education and patients' awareness together to poor compliance with therapy and diet.^{(35), (48), (49), (50)}

4. Corneal Blindness

Prevalence of Corneal Blindness

Blindness resulting in corneal scarring is a major cause of bilateral and monocular blindness in

both adults and children in developing countries, second only to cataract in overall importance. In high-risk areas in some parts of Africa and Asia, the incidence of childhood cornea-related visual loss is 20-times higher than in industrialized countries.^{(5), (51)}

Causes of Corneal Blindness

The epidemiology of corneal blindness is complicated and encompasses a wide variety of infectious and inflammatory eye diseases that cause corneal scarring which ultimately leads to blindness. In addition, the prevalence of corneal disease varies from country to country and even from one population to another being largely dependent on the ocular conditions in that particular area.⁽⁵⁾ Diseases that predispose to corneal scarring are trachoma, onchocerciasis, leprosy, ophthalmia neonatorum, and xerophthalmia. These conditions remain important causes of blindness. However, the recent progress in combating onchocerciasis and leprosy, as well as the gradual worldwide decline in the number of cases of trachoma, has shifted attention to other causes of corneal blindness including ocular trauma, corneal ulceration, and complications from the use of traditional eye medicines (TEM).^{(51), (52), (53), (54), (5), (55), (56), (57), (58), (59), (60), (61)}

Corneal Blindness from trachoma

Corneal blindness from trachoma is the second major cause in the adult population after cataract in most developing countries where blindness occurs at ten times the rate of that seen in the developed countries; in over 80% of cases is either preventable or curable. The four main causes are cataract, trachoma, onchocerciasis and xerophthalmia.^{(1), (2), (3)} Data on other blinding conditions, such as diabetic retinopathy in the developing countries, are scarce.⁽⁴⁾

Corneal Opacities from Non-Trachomatous Causes

Prevalence

Corneal blindness due to non-trachomatous causes have been reported in several parts of Africa including Nigeria, ⁽²⁰⁾ the Niger (7%) ⁽⁶²⁾ and Sierra Leon (3%). ⁽⁶³⁾ These causes include measles, VAD, HSK, onchocerciasis, leprosy, TEM and EWCL.

Measles Blindness

Measles blindness is the single leading cause of blindness among children in low-income countries, especially Africa, where it affects an estimated 30 million children and causes up to one million deaths a year. Every year some 15,000 to 60,000 children go blind from the condition. Stanford-Smith and Whittle demonstrated acute corneal ulceration in malnourished children which is rarely produced by other severe diseases. They demonstrated that 69% of the children were blind from corneal disease, and a survey of children with corneal scars showed that at least 42% were caused by ulceration after measles. The clinical appearance of the active ulcers was very varied. ⁽⁵⁹⁾ (Refer to 6.12.7 for synergism of xerophthalmia, diarrhoea and measles).

Corneal Blindness from Onchocerciasis and Leprosy

Another cause of corneal scarring is *onchocerciasis* in the endemic areas as has been reported in the savannah region of southern Nigeria, and Central African Republic. ⁽⁶⁴⁾

Leprosy can also contribute to corneal blindness by predisposing to exposure keratitis (21.3%), corneal opacities (13.5%) and chronic uveitis (10.1%). ⁽⁶⁵⁾

Corneal Blindness from TEM and EWCL

There is a significant association between corneal ulceration and TEM use and, in particular, peripheral corneal ulcerations. ^{(7), (66)} TEM is an important cause of blindness and has been implicated as the most important cause of corneal blindness in the current epidemic of corneal ulceration in developing countries. ^{(5), (60), (66)}

Corneal infection secondary to EWCL is becoming an increasing hazard in some regions such as in the Arab countries. There is considerable ignorance of the dangers of wearing these lenses for long period, a problem that is not helped by the failure of local opticians to provide the necessary advice to their patients. ⁽⁶¹⁾

Corneal Blindness from Climatic Droplet Keratopathy

Climatic droplet Keratopathy (CDK) is another important cause of blindness, ^{(14), (32), (29), (57), (60), (67), (68), (69)} The disease can progress rapidly resulting in spontaneous sterile ulceration and secondary microbial keratitis. CDK is common in many parts of the world where there is high exposure to UV light and is significantly more predominant in males than females. In the northern hemisphere, it is found in areas where there is a high reflection of UV light, where snow persists for long periods of the year, and in the southern hemisphere in deserts and areas of white sand, particularly if mixed with crystalline sea salt. ⁽⁶⁰⁾ The commonest prevalence are in northern Cameroon, where 60% of the population from both genders are affected; Australian Aborigines, where it affects 41% of male but only 8% of females and, in Somalia and Mongolia, CDK is the third commonest cause of blindness. In Mongolia, it was responsible for 7.2% of blindness and 19.3% of visual impairment in semi-nomadic cattle breeder's communities. ^{(32), (70), (60)} It has been reported in Labrador in people above the age of 40 years where it was found in 61% of males and 13.5% of females, among the Inuit population of Greenland (12%), in Somalia (12%), in Saudi Arabia, Iran, Pakistan, and India. ⁽⁶⁰⁾ Severe cases of CDK are found in the Dahlak Islands in the Red Sea where 3% of the population are blind from it. ⁽⁶⁰⁾

5. Refractive errors

This is a major cause of recorded visual impairment in a large number of developing countries, ^{(12), (71), (72), (73), (74), (7), (75), (76), (77), (78), (79), (80), (81), (82),}

^{(83), (84)} and has recently been recognized as an important problem due to the lack of refractive correction and inadequate refractive correction of aphakia after cataract surgery. This reflects the inadequacy of eye care services in general, although cultural barriers may be a factor in some countries. ^{(77), (85)} Addressing this simple problem requires several measures such as large scale screening programmes, sufficient numbers of well trained personnel to perform reasonable quality refraction and the development of adequate infrastructures to facilitate the logistics of providing affordable, reasonable quality spectacles. ⁽⁸⁵⁾

6. Retinopathy of Prematurity

ROP had been the major cause of childhood blindness during the 1940s and 1950s in the industrialised countries, accounting for 50% of childhood blindness during this period. ⁽⁸⁶⁾ It now accounts for only 6-18% of blind registrations in many developed countries as a consequence of active screening programmes and early treatment. It is, however, an emerging problem in middle-income countries. ^{(87), (88), (89)} In the PSE, ROP is a major cause of preventable childhood blindness where it accounts for e.g. for 42% of childhood blindness in the Czech Republic. ⁽⁹⁰⁾ It is also a major problem in Cuba contributing to 38.6% of childhood blindness ⁽⁸⁹⁾ and has been reported in Saudi Arabia. ⁽⁹¹⁾ Its incidence in the UK is only 3%. ⁽⁸⁶⁾

7. Recreational Drugs

The intake of drugs, alcohol and cocaine is a contributory factor to childhood blindness and is becoming an increasingly important cause of blindness from optic atrophy and optic disc anomalies in the USA. Ocular involvement is usually part of other systemic manifestation of the conditions resulting from drug intake such as fetal alcohol syndrome and cocaine embryopathy. ⁽⁸⁶⁾

8. Ocular Trauma

Incidence and Monocular Blindness

Ocular trauma ^{(92), (93)} is a major cause of monocular blindness in both the developed and developing world, but is not seen as a significant cause of bilateral blindness. The size of the problem is difficult to assess in view of the lack of informative surveillance systems and population based studies on the subject. Negrel, based on a review of literature on the subject, estimated that the incidence of eye injuries are as follows. ^{(93), (94)}

- a) Those restricting activities for > 1 day are: 900-1000 /100,000.
- b) Those requiring medical attention is: 400-1000 / 100,000.
- c) Those requiring hospitalisation is: 13/100,000.

Figures on ocular trauma from the developing countries are lacking. However, in view of the difficulties of surgical treatment of ocular trauma in many developing countries, long-term management is usually aimed at prevention, e.g. by improving safety standards in the workplace. It may be responsible for 1.5-2.0 million new cases of monocular blindness every year. ⁽⁵⁾ The commonest cause of injury was child related from a thrown object (42%) and hazardous toys (21%). ⁽⁹⁵⁾ Risk factors in trauma are age, male gender, low socio-economic status and active life style. ⁽⁹³⁾

Eye Injuries in Children

The frequency of the various types of eye injuries in children were reported by LaRoche as; contusions (51%); penetrating lacerations (28%); foreign bodies and burns (5%); and non-penetrating lacerations (16%). Male predominance was significant in all age groups, with an average M/F ratio of 3.5:1. ⁽⁹⁶⁾

Wars and Landmine Injuries

An important cause of bilateral blindness is landmines. These have been reported in countries such as Cambodia and Afghanistan^{284 (92), 541 (97)}. The former has high landmine densities and penetrating ocular trauma is a significant cause of bilateral blindness in this country, predominantly affecting young men. Cambodia has an estimated 4-10 million landmines that cause significant morbidity and mortality and it is estimated that it would take 250 years to clear the landmines at the current activity levels. One report found 14 cases of bilateral blindness as a result of landmines out of 17 cases of blindness from ocular trauma in Cambodia.⁽⁹⁸⁾

9. Vitamin A Deficiency (VAD)

Epidemiological Aspects of VAD Manifestations

Manifestations of VAD range from night blindness (31%); conjunctival xerosis (57%); Bitot's spots (10.8%) and corneal scars (1%). Severe complications include corneal ulceration or keratomalacia.

Xerophthalmia, due to VAD, is still a major public health problem in many parts of the world, especially in Africa.

The mother's level of education and the likelihood of her child having xerophthalmia were significantly associated.⁽⁹⁹⁾

Subclinical forms of VAD are also well recognised as having a negative effect on metabolic functions, with a great impact on childhood morbidity and mortality^{412 (100)}. The latter is considered at a serum retinol level of $< 20 \mu\text{g/dL}$ ($< 0.70 \mu\text{mol/L}$), that is double that associated with clinical eye manifestations ($< 10 \mu\text{g/dL}$, $< 0.35 \mu\text{mol/L}$) according to WHO definition.⁽⁹⁹⁾

Age and Gender in VAD

The age of developing VAD manifestations varied within the study. Age and xerophthalmia prevalence were highest among children 1-2 years of age (82 children, 40.4% of this age group) and the lowest among those aged 4-5

years (8 children, 16.7% of this age group).⁽⁹⁹⁾ However, in another study in Yemen children aged 4-5 years were more likely to develop xerophthalmia than those under the age of 4 with no child aged 12-23 months having the condition.^{345 (101)} Most xerophthalmia cases (77.8%) had Bitot's spots. Those exhibiting Bitot's spots tended to have the condition in both eyes (71%) with 66.6% of them in the 4-5 years age range. Boys were more likely to have xerophthalmia than girls.⁽⁹⁹⁾

Prevalence of VAD

Vitamin A deficiency remains prevalent in many countries in the world with an estimated 100 to 140 million children being vitamin A deficient and with some 250,000 to 500,000 of these children becoming blind every year; half of them dying within 12 months of losing their sight. The condition is the leading cause of preventable blindness in children and, untreated, raises the risk of disease and death from severe infections. It is also worth noting that nearly 600,000 women die from childbirth-related causes each year, the vast majority of them from complications, which could be reduced through better nutrition, including provision of vitamin A. In pregnant women VAD causes night blindness and may increase the risk of maternal mortality.^{(102), (103), (104), (105), (106), (107), (108), (109), (110), (83), (111)}

Vitamin A deficiency is a public health problem in 118 countries, especially in Africa and South-East Asia, once again hitting hardest young children and pregnant women in low-income countries. In Africa, hunger and malnutrition have been on the increase since the 1960s. During the 1970s, it is estimated that 30 million people were directly affected by famine and malnutrition. In the 1983-1984 famine, about 5 million children died in 1984 alone and in Mozambique about 100,000 people perished. In Ethiopia, Sudan, Somalia, Liberia, and Angola, armed conflicts compounded the problem; Ethiopia alone had 9 million famine victims in 1983. In the early 1990s, VAD affected some 10 million Africans.⁽¹¹²⁾ The visual loss, low resistance to disease and increased mortality caused

by the disease can be simply prevented by vitamin A distribution programmes /education.⁽⁹⁾

VAD in MENA

In the Eastern Mediterranean Region, VAD is present in some parts of the Arab world including Yemen, Egypt, Djibouti and Mauritania,^{(113), (114), (115), (116)} and more recently, Iraq following the sanctions and wars.⁽⁹⁹⁾

In Yemeni children, xerophthalmia and VAD is a public health problem. In a study of 2438 children aged 1-5 years in the 18 districts of western Yemen, night blindness was found in 0.5% of the children.

In Iraq, a study of the prevalence of xerophthalmia after the recent international sanctions (1991-2003) has shown how health conditions can regress when economic conditions change and also demonstrates the wider effects of VAD on health. The study was conducted in Diyala Province, north east of Baghdad, where 700 randomly selected preschool children (M/F ratio = 1:3.1; age range 0–6 years) who had been admitted to Saddam Paediatric Hospital in Diyala for different illnesses during May to August 1995, were examined. Xerophthalmia is a common problem among sick Iraqi children with active xerophthalmia found in 2.21% of the children.⁽¹⁰¹⁾ Its prevalence of xerophthalmia after the sanctions was 29%, mostly among children aged 1–3 years. However, more had keratomalacia or corneal ulceration. The study also highlighted the inverse relationship between breastfeeding and the association of VAD with common childhood infections such as measles, diarrhoea and respiratory tract infections.⁽⁹⁹⁾

In Egypt VAD was also reported in the Beheira governorate in a study of 10,664 children.⁽¹¹³⁾ This, however, does not appear to be a public health problem. Ocular signs of VAD were more prevalent among older children, suggesting an improvement in socioeconomic conditions and health care over the past few years. It was also noted that children from lower socio-economic class had significantly lower mean vitamin A intake compared with the respective mean intake obtained in children from higher socioeconomic background.⁽¹¹⁴⁾

Vitamin A deficiency also existed in Mauritania as a public health problem as reported in the 1973 and 1983 droughts and a programme was set up by the Mauritanian Health Ministry in 1989 to tackle this problem.^{362 (116)} Serious VAD has been periodically reported in the Republic of Djibouti where large numbers of children, mostly in the rural area, had marginal vitamin A status and were exposed to a high level of risk.⁽¹¹⁵⁾ Blindness from VAD was found in Djibouti in another study.⁽¹¹⁷⁾ In the rest of the MEC, xerophthalmia has been reported in Afghani refugees in Pakistan.⁽¹¹⁸⁾

Xerophthalmia

Xerophthalmia, due to VAD, is still a major public health problem in many parts of the world especially in Africa. Hunger and malnutrition in Africa have been on the increase since the 1960s. During the 1970s, it is estimated that 30 million people were directly affected by famine and malnutrition. About 5 million children died in 1984 alone. In Mozambique during the 1983-84 famine, about 100,000 people perished in the famine. In Ethiopia, Sudan, Somalia, Liberia, and Angola armed conflicts compounded the problem. Ethiopia alone had 9 million famine victims in 1983. It is, therefore, not surprising that VAD affected some 10 million Africans in the 1990s.⁽¹¹²⁾ The visual loss, low resistance to disease and increased mortality caused by the disease can be simply prevented by vitamin A distribution programmes or education.⁽⁹⁾

Xerophthalmia in the MEC

In Egypt VAD was also reported in the Beheira governorate in a study of 10,664 children.⁽¹¹³⁾ This, however, does not appear to be a public health problem. Ocular signs of VAD were more prevalent among older children, suggesting an improvement in socioeconomic conditions and health care over the past few years. It was also noted that children from lower socio-economic class had significantly lower mean vitamin A intake compared with the respective mean intake obtained with children from higher socioeconomic background.⁽¹¹⁴⁾ In addition to Yemen and

Iraq, VAD existed in Mauritania where it was found to be a public health problem in studies conducted after the 1973 and 1983 droughts, and a programme was set up by the Mauritanian Health Ministry in 1989 to tackle this problem.⁽¹¹⁶⁾ In addition, serious VAD may periodically occur in the Republic of Djibouti and blindness from VAD was reported in this country in another study.^{(117), (119)} Large numbers of children, mostly in the rural area, have a marginal vitamin A status and are exposed to a high level of risk.⁽¹¹⁵⁾ In the rest of the MEC, xerophthalmia has been reported in Afghani refugees in Pakistan.⁽¹¹⁸⁾

VAD/Measles//HSK Synergism

There is a close synergism between measles and VAD that can result in xerophthalmia, with corneal ulceration, keratomalacia, and subsequent corneal scarring or phthisis bulbi. High-dose oral vitamin A supplements are recommended for all children with measles in developing countries. Higher measles immunisation coverage to interrupt measles transmission, and interventions aimed at improving vitamin A intake of children are the main strategies to prevent measles blindness.⁽¹²⁰⁾

In addition, malnourished children who had had a severe attack of measles are prone to deep ulcers of the mouth and eyes. Herpes simplex virus was isolated from 17 of 25 of the mouth ulcers which were erosive, slow to heal and caused much suffering and loss of weight. Herpetic corneal ulcers in these patients heal slowly in two to six weeks leaving damaging scars which impaired vision and cause blindness in some cases. It is suggested that measles leads to profound depression of cell-mediated immunity in malnourished children with the consequence that secondary herpes simplex infections become abnormally severe and erosive.⁽¹²¹⁾

The synergism between xerophthalmia and diarrhoea, measles and upper respiratory infections, were statistically significant.⁽⁹⁹⁾ Among the 700 children in the sample population, xerophthalmia was present in 25.2% of the 481 who had diarrhoea; 60.2% of the 103 who had

measles, and 17.2% of the 116 who had upper respiratory tract infections.⁽⁹⁹⁾ It was recommended that, in addition to measures such as immunisation against measles, breastfeeding, environmental sanitation, food safety and personal hygiene were also necessary to reduce the incidence of diarrhoea. The supplementation of vitamin A upon diagnosis of measles in areas where xerophthalmia is prevalent was also recommended.⁽⁹⁹⁾

The synergism between xerophthalmia, diarrhoea and measles and upper respiratory tract infections was also demonstrated in this Iraqi study and was statistically significant. Among the 700 children in the sample population, xerophthalmia was present in 25.2% of the 481 who had diarrhoea; 60.2% of the 103 who had measles, and 17.2% of the 116 who had upper respiratory tract infections. It was recommended that, in addition to measures such as immunisation against measles, breastfeeding, environmental sanitation, food safety and personal hygiene were also necessary to reduce the incidence of diarrhoea. The supplementation of vitamin A upon diagnosis of measles in areas where xerophthalmia is prevalent was also recommended.⁽⁹⁹⁾

10. Wars and Sanctions

The WHO highlighted the catastrophic impact of disasters and sanctions on the health and well-being of nations and that these events have caused more mortality and disability than any major disease. War can destroy communities and families and disrupt the development of the social and economic fabric of a nation with long-term physical and psychological harm to children and adults. All of the countries of the EMC have been exposed over the past century to war, disasters or international sanctions. At present, Afghanistan, Palestine, Somalia and Sudan are experiencing long-term protracted social conflict. Palestine is being subjected to brutal and unprecedented aggression. During the 1990s, sanctions were imposed on Afghanistan, Iraq, Libya and Somalia. The growing body of information about the adverse effects of sanctions on the health and livelihoods of the

people in these countries has prompted international debate and review of the effectiveness and appropriateness of international sanctions. (97), (122), (123), (124), (125), (126)

Natural disasters, refugee crises, drain of health personnel, economic collapse and ongoing violence are all determinants of ill health. Health indicators in certain countries of the EMC reflect the problems inherent in trying to improve the overall health status and delivering health care under difficult circumstances. Despite a lack of in-depth research and data analysis, it is clear that countries with ongoing difficult circumstances, such as Afghanistan, Palestine, Somalia, Iraq and Sudan, face complex challenges. WHO must continue to invest in and advocate for health under difficult circumstances. Moreover, ensuring immediate equitable access to basic quality health care will lay a foundation for future investments in development.

The effect of sanction on Iraq is well depicted by the increase of <5 mortality rate and recent reports on the prevalence of VAD in the country. (124)

COMMON BLINDING INFECTIONS

11. Trachoma

Introduction

Trachoma is the most common infectious cause of blindness. It has been recognized since antiquity and the first effective therapeutic modality, using copper sulphate sticks, was described by the early Egyptians thousands of years ago. (127), (128), (129), (130), (131), (132), (133), (134), (135), (136), (137), (138), (139), (140), (141), (142), (143), (144), (145), (146), (147), (148), (149), (150), (151), (152), (153), (154), (155), (156), (157), (158), (159), (160), (161), (162), (163), (164), (165), (166), (167), (168), (169), (170), (171), (172), (173), (174), (175), (176), (177), (178), (179), (180)

Causative Factors in Trachoma

The disease is caused by ocular serovars of *Chlamydia trachomatis*, an intracellular epithelial

gram-negative bacterium. Transmission is favoured in poor communities, where crowding is common and access to water and sanitation is inadequate. *Musca sorbens* are vectors blamed for the transmission of the organism along with fingers and fomites. Eyes of young children are considered to be the main reservoir of *Chlamydia trachomatis*, collections of eye-seeking flies from children showed frequent fly-eye contacts averaging 3 (1.5-7) every 15 minutes. Children with potentially infective ocular or nasal discharge had twice as many fly-eye contacts than children with no discharge. (407) (158) However, there is still uncertainty about how trachoma is transmitted as *Chlamydia* is detected on only 0.5% of face flies. (156), (158)

Factors in Trachoma Blindness

Trachomatous blindness follows frequent episodes of reinfection, which can be prevented by simple hygienic measures. The host immune response is probably, at least, partly the cause of this process. (135) However, the continual inflammation in trachoma may not be due to repeated exposure to chlamydial surface antigen(s) but rather to a labile product released by the living organisms. (391) (148) The resultant inversion of the lashes abrades the eyeball, and the abrasion leads to corneal opacification and visual impairment. It is worth noting that chlamydial infection at the time of surgery and at follow-up is a significant risk factor for postoperative failure and recurrence of trichiasis. (160)

Prevalence of Trachoma

In 1950 trachoma was a major cause of blindness in all developing countries of the world and the second major cause of blindness. (5), (135) It is estimated there are currently about 6 million people irreversibly blind from the disease making up 15% of the world's blind, (181) and another 10 million at high risk, mainly as a result of corneal scarring and vascularisation. This is the tip of an iceberg of 146 million active cases that need treatment and 500 million afflicted by the disease, making it the most common of all human chronic infections, and the most common

cause of preventable blindness today. ⁽¹⁷⁸⁾ There were 400 million cases worldwide in 1959, a very high number for the size of the world population at that time. ⁽¹⁸¹⁾

Epidemiology and Trends

Trachoma occurs worldwide, most often in poor rural communities in developing countries. It is endemic in 49 countries, mostly in Africa (both Sub-Saharan and North Africa), but also in the Eastern Mediterranean, Southeast Asia and the Western Pacific where blinding disease is found. ^{(15), (182)} It also exists in parts of the Indian subcontinent, Southern Asia and China. Pockets of blinding trachoma occur in Latin America, Australia (among native Australians) and the Pacific Islands. In these communities, women and children bear the brunt of the burden.

In Kenya the prevalence showed significant regional variations which were found not only in seasonal variation (see below) but also in age-specific prevalence (28% in <3 years, 11% in >60 years), and severity of the disease within the high-risk regions. ^{(183), (184), (185), (186), (187), (188)}

Active trachoma was present in 19% of 13,805 cases from 8 regions and 50% of all those with trachoma were found to have moderate to severe inflammation. A potentially blinding eyelid deformity, secondary to chronic trachoma occurred in 5% of the rural population and was more prevalent in females of all ages than in males. The prevalence rate of visual impairment from the condition (< 6/18) was 7.2/1000. ⁽¹⁵⁹⁾

Several observations have been made on the trends of trachoma and the variation in its prevalence as follows:-

(a) Trachoma is not disappearing in many of these areas because in many of the hyperendemic regions, neither the standard of living nor hygiene conditions are improving. Some are of the opinion that blindness is likely to increase as more people are being exposed to trachoma at childhood and more will survive to old age when trachomatous blindness develops. ⁽¹⁸⁹⁾

(b) In other parts of the developing world, secular changes in the form of improvements in

sanitation, water supply, education and access to health care in villages after the initiation of health, water and hygiene programmes have resulted in a decrease in the prevalence of the disease in these areas. This has occurred without any trachoma-specific intervention, suggesting that sustained reductions in active trachoma can be achieved without community-based antibiotic distribution and little change in socioeconomic status as demonstrated in Malawi ^{402 (149)} and Gambia. ⁽¹⁵²⁾ In the first two, active trachoma has diminished by over 50% and trachomatous trichiasis by over 80% compared to 1983. In the latter, it has diminished significantly as noted in a study encompassing three age cohorts: 0-9 years old children, 10-19 years old teenagers and 20+ adults. The figures came down between 1959 and 1996 from 65.7%, 52.5% and 52.5% respectively in 1959, to 2.4%, 1.4% and 0% in 1996 respectively. ⁽¹⁵²⁾

Improvement of economical conditions can also lead to a reduction in the prevalence of trachoma as seen in Western Nepal. ^{(153), (190)}

(c) Climatic factors and seasonal variation: - Prevalence of trachoma while a wet climate with greater rain fall, and sustainable agriculture is associated with lower prevalence of the disease. In Kenya, for example, higher prevalence (57-63%) was found in areas with high climatic aridity with a lower prevalence in regions of greater rainfall, sustainable agriculture, and a higher general standard of living. ⁽¹⁵⁹⁾ In addition, the seasonality of trachoma has been thought to be important in many geographic areas, including Morocco, Tunisia, Egypt, and Nepal. A 20% seasonal fluctuation in prevalence between the spring and the autumn, with the peak prevalence in the former was also found. It has been suggested that it is preferable to administer antibiotics in the peak trachoma season (spring, before the monsoon rains), when there is the most infection, or in the trough season (Autumn), when programmes might have the best chance to locally eradicate infection from households or even small communities. ⁽¹⁵³⁾

(d) It is important to remember when conducting epidemiological studies on trachoma that

clinically active trachoma is not always a reliable marker of infection, particularly in teenagers and after treatment where it leads to a clinical picture in transition. Of children with clinically active trachoma aged 1-10 years, 31% did not have infection and conversely, 31% of infected children were not clinically active; 78% of clinically active children aged 1-5 years were infected, versus 17% of those aged 11-15 years.⁽¹⁴²⁾ It is also well established that trachoma occurs in episodes of repeated infections.⁽¹²⁾

Preventive Measures and Strategies in Trachoma

Considerable efforts have been made to tackle this blinding condition in recent years. Prevention and intervention programmes in endemic areas have been introduced and are based on mass therapy in the form of topical application of antibiotics with or without oral antibiotics administered as a supplement to topical therapy.⁽¹⁹¹⁾

Azithromycin, a macrolide belonging to the azide subclass,⁽¹⁹²⁾ is the antibiotic of choice in endemic areas as prolonged high levels are maintained in the conjunctival tissue following a single oral dose.⁽¹⁵⁰⁾ This has been demonstrated in conjunctival biopsy specimens obtained from adult patients for up to 2 weeks after a single oral dose of the drug. No difference in the efficacy of 1-6 doses of Azithromycin and 30 days of topical oxytetracycline / polymyxin ointment therapy have been found.^{(146), (161)}

Considering the rate at which ocular chlamydial infection returns to a community after mass treatment, the elimination of infection in hyper-endemic areas is believed to be feasible with biannual mass antibiotic administrations. The use of oral antibiotics in selected cases is found to be a cost-effective strategy, particularly in communities where less than 20% of children have active trachoma and should be restricted to children with severe or moderate intensity disease who should be monitored carefully for adverse reactions.^{(146), (193), (194)}

Two strategies have been described in treating trachoma in children. One is to treat children

who have clinically active trachoma and their households, and the other to treat all children, regardless of clinical activity. Both strategies reduced the prevalence of active trachoma at 6 months, and there was no significant difference between them.⁽¹⁸⁰⁾ Frick et al found that mass treatment was as effective as and no more expensive than targeted household treatment. Although it was felt that less expensive targeting methods are required to improve the cost-effectiveness of targeted household treatment.⁽¹⁹⁵⁾

On the economics of trachoma and its control, Frick et al addressed the subject and suggested that: (1) trichiasis without visual impairment may result in an economic burden comparable to trachomatous low vision and blindness so that; (2) the monetary burden of trachoma may be 50% higher than conservative, published figures; (3) within some regions more productive economies are associated with less national blindness from trachoma and; (4) the ability to achieve a positive net benefit of trachoma control depends importantly on the cost per dose of antibiotic.⁽¹⁹⁶⁾

12. Onchocerciasis

Prevalence of Onchocerciasis

Onchocerciasis or River Blindness is still an endemic disease in 35 countries despite health success stories. It is endemic in African countries - in the savannah as well as in the forest zone - from Senegal to Malawi, six countries in Latin America and in the Yemen. At present, the WHO estimates that there are more than 17.7 million people infected with the disease with nearly 500,000 with visual impairments, 270,000 of whom are blind, 99% of the cases being found in Africa.^{(197), (13), (5), (198), (199), (200), (201), (202), (203), (204), (205), (206), (207), (208), (209), (210), (211), (212), (213), (214), (215), (216), (217), (218), (219), (220), (221), (222), (223), (224), (225), (226), (227), (228), (229), (230), (231), (232), (233), (234), (235), (236), (237), (238)}

The prevalence of blindness in hyperendemic areas is 20% among those with skin loads of 100 microfilariae snip where the lifetime risk of becoming blind is more than twice as high in areas

of hyperendemicity of onchocerciasis than in areas of mesoendemicity of onchocerciasis.⁽²¹⁵⁾

Transmission of Onchocerciasis

As it is transmitted by black flies that breed in rapidly flowing rivers and streams, hence eradication programmes have focused on the control of onchocerciasis transmission. There is a direct relationship between microfilarial load and the incidence of blindness was significantly and positively associated with increasing microfilarial burden.

Eradication of Onchocerciasis

It has been predicted that only a few of the present-day patients with river blindness will still be left by 2030. This was based on the conclusion that onchocerciasis had reverted to the situation before the occurrence of ocular complications as a result of the ongoing control measures, despite the insect destruction and mass therapy not always being complete. This was thought to be sufficient to prevent blindness even if onchocerciasis is not cured.⁽²³⁹⁾ On the other hand, Winnen et al argued that, although elimination of onchocerciasis from most endemic foci in Africa by mass treatment was possible, requirements dictated by duration, coverage, and frequency of treatment might be prohibitive in highly endemic areas. They pointed out that as the duration of treatment required depended on the endemicity and treatment programme, annual mass treatments with 65% coverage for at least 25 years were necessary in areas with medium to high levels of infection. This time could, however, be halved if treatment intervals were reduced from 12 to 6 months. The authors doubted the feasibility of such long-term high coverage levels needed to achieve worldwide eradication.⁽²⁴⁰⁾

It is important to note the work of Murdoch et al who drew attention to the disability caused by visual fields contraction in onchocerciasis in their large study of visual field loss in 6831 individuals aged 5 years and over in Kaduna State, northern Nigeria which is mesoendemic for savannah onchocerciasis.⁽²⁴¹⁾ A total of 185

(2.7%) were bilaterally blind by acuity and an additional 28 (0.4%) were blind by visual field constriction. Also 118 (1.7%) individuals were visually impaired by acuity criteria. The authors highlight that the current WHO definition of blindness includes visual field damage criteria for blindness but not for visual impairment at a time when visual field loss is a major disability. The need for the development of satisfactory definitions for visual impairment by visual field constriction was put forward by the authors.⁽²⁴¹⁾

13. Leprosy

Leprosy is a chronic disease caused by the noncultivable, slow-growing, acid-fast bacterium *Mycobacterium leprae*. It is thought to be transmitted from human to human by nasal droplets, as distinct from the transmission of *Mycobacterium tuberculosis*. Leprosy lesions have been reported to develop at the site of skin abrasions.^{(242), (243)}

There are still some 10 million people with leprosy, over a half of whom live in the Indian subcontinent and Myanmar (Previously Burma).⁽²⁴²⁾ The condition remains prevalent in 15 countries and territories in Africa, Asia and Latin America, which is a great reduction in comparison to 1985 when it was prevalent in 122 countries.⁽²⁴⁴⁾ In the early 1980s, multi-drug therapy (MDT) was introduced to treat leprosy and since then, over 12 million leprosy patients have been cured by MDT treatment with no drug resistance or significant relapses.⁽²⁴³⁾ In the Arab world, reports on leprosy date back to 1970 and, but no recent reports on the disease in the 1980s.^{(245), (246)}

14. Ophthalmia Neonatorum

Prevalence

Ophthalmia neonatorum (O/N) remains a source of childhood blindness in developing countries with some 1000–4000 newborn babies becoming blind every year from the condition. The condition prevails in various proportions and is caused by different pathogens worldwide. In

Kenya for e.g., 28.5% of mothers were found to be infected with *Chlamydia trachomatis* and 9.5% with *N. gonorrhoea*. Neonates with clinical signs of conjunctivitis had *Chlamydia* in 28.7% of cases and gonorrhoea in 20.2%; however the latter is the predominant cause of conjunctivitis in STD clinics. In Al Ain, UAE, 81.5% of children with O/N showed bacterial or fungal infections but only 5% of all cases were caused by *C. trachomatis* or *N. gonorrhoea*. The predominant pathogens are bacterial and fungal infection. In developed countries, the prevalence of gonococcal ophthalmia neonatorum ranged between 0.04/1000 live births in Belgium and the Netherlands, to 0.3/1000 in the USA.^{(60), (247)}

Gonococcal ophthalmia neonatorum has reappeared and been reported in Denmark, Florida and Sweden. This took place only a few years after discontinuing the general use of Crede's prophylaxis in 1985.^{(60), (247)}

Causative Factors

Neisseria gonorrhoea as a cause for O/N has now been replaced by a range of agents which include *C. trachomatis*, *Staphylococcus* spp., *Streptococcus* sp., *Haemophilus influenza* and *Enterobacteriaceae*, together with chemical agents. The former is the most frequent sexually transmitted disease (STD) in industrialized countries, with prevalence rates ranging from 4/1000, 5-60/1000, and 40/1000 live births in the UK, USA, and Belgium respectively.

Prophylaxis

Prophylaxis could be either by treating the neonates at birth, or treating the pregnant mothers. The former has been discontinued in some countries such as the UK, Denmark and Sweden. In others, the use of silver nitrate 1% has been abandoned because of its ineffectiveness against the *C. trachomatis* and the risk of chemical conjunctivitis. Instead 0.5% erythromycin or 1% tetracycline ointment are used. More recently, povidone-iodine, a much cheaper preparation, has been introduced and shown to be effective in preventing ophthalmia neonatorum.^{(60), (247)} The second approach, of treating

infection in pregnant women, can only be carried out in places where medical care is well organized so that pregnant women at risk can easily be screened for STDs and treated accordingly. However, screening pregnant women for chlamydial infection is not easily implemented, and reinfection is common. In addition, general screening would be too costly.⁽²⁴⁷⁾

15. AIDS

HIV/AIDS affects every country in the world and in many infection rates are increasing rapidly.⁽²⁴⁸⁾ In 2004, an estimated 34-46 million people were afflicted with AIDS. Sub-Saharan Africa is the region of the world worst hit with nearly three-quarters of all those affected globally. It is argued that the rapid spread of HIV / AIDS is linked with globalization, which makes it easier for people to travel and may promote some risk behaviors. The disease has reversed the improvement in life expectancy witnessed between 1960 and 1990, and has hindered economic growth in some sub-Saharan African countries due to the continuing loss of skilled and unskilled workers in the prime of life. In South Africa HIV / AIDS may depress GDP by as much as 17% over the next decade.^{(249), (250)} Guex-Crosier and Telenti speculated that, based on experience in higher income countries, epidemics of blindness might hit hard in regions where HIV care and life expectancy progressively improves. This condition will be a new challenge for Vision 2020.⁽²⁵¹⁾

References

1. Johnson GJ, Minassian DC. Prevalence of blindness and eye disease: discussion paper. *J Roy Soc Med* 1989; 82: 351-4.
2. Weale R. Introduction. in: Johnson GJ, Minassian DC, Weale R (eds.) *the Epidemiology of Eye Disease*. Chapman & Hall 1998, pp 1-30.
3. Narita AS, Taylor HR. Blindness in the Tropics. *Med J Aust* 1993; 159: 416-20.
4. Thylefors B, Negrel AD, Pararajasegaram R,

- Dadzie KY. Global data on blindness. *Bull World Health Organ* 1995; 73: 115-21.
5. Whitcher JP, Srinivasan M, Upadhyay MP. Corneal blindness: a global perspective. *Bull World Health Organ* 2001; 79: 214-21.
 6. Thylefors B. Avoidable blindness (Editorial). *Bull World Health Organ* 1999; 77: 453.
 7. Lewallen S, Courtright P. Peripheral corneal ulcers associated with use of African traditional eye medicines. *Br J Ophthalmol* 1995; 79: 343-6.
 8. Dolin P. The epidemiology of cataract. In: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*. Chapman & Hall 1998, pp 103-18.
 9. McLaren DS. The Epidemiology of Vitamin A Deficiency Disorders. In: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*. Chapman & Hall 1998: 209-25.
 10. Rabi MM. Cataract blindness and barriers to uptake of cataract surgery in a rural community of northern Nigeria. *Br J Ophthalmol* 2001; 85: 776-80.
 11. Hyman L. Epidemiology of eye disease in the elderly. *Eye* 1987; 1: 330-41.
 12. West S, Munoz B. Epidemiology of Trachoma. In: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*. Chapman & Hall 1998, pp 119-35.
 13. Duke BOL. Onchocerciasis. In: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*. Chapman & Hall 1998, pp 227-47.
 14. Dolin P. the epidemiology of cataract. in: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*. Chapman & Hall 1998, pp 103-18.
 15. Tabbara KF, Ross-Degnan D. Blindness in Saudi Arabia. *JAMA* 1986; 255: 3378-84.
 16. Brian G, Taylor H. Cataract blindness – challenges for the 21st century. *Bull World Health Organ* 2001; 79: 249–56.
 17. Taylor HR. Epidemiology of age-related cataract. *Eye* 1999; 13: 445-8.
 18. Hu TS, Lao YX. An epidemiologic survey of senile cataract in China. *Dev Ophthalmol* 1987; 15: 42-51.
 19. Abou-Gareeb I, Lewallen S, Bassett K, Courtright P. Gender and blindness: a meta-analysis of population-based prevalence surveys. *Ophthalmic Epidemiol* 2001; 8: 39-56.
 20. Fafowora OF. Prevalence of blindness in a rural ophthalmically underserved Nigerian community. *West Afr J Med* 1996; 15: 228-31.
 21. Lewallen S, Courtright P. Gender and use of cataract surgical services in developing countries. *Bull World Health Organ* 2002; 80: 300-3.
 22. Foster A, Gilbert C. Cataract in children. *Acta Paediatr* 2003; 92: 1376-8.
 23. Foster A, Gilbert C, Rahi J. Epidemiology of cataract in childhood: a global perspective. *J cataract Refract Surg* 1997; 23: S601-4.
 24. Banatvala JE, Brown DW. Rubella. *Lancet* 2004; 363: 1127-37.
 25. Johnson GJ. The Glaucomas. in: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*, Chapman & Hall 1998, pp 159-80.
 26. Grierson I. What is open angle glaucoma? *Eye* 1987; 1: 15-28.
 27. Quigley HA. Number of people with glaucoma worldwide. *Br J Ophthalmol* 1996; 80: 389-93.
 28. Taylor HR, Keeffe JE. World blindness: a 2st century perspective. *Br J Ophthalmol* 2001; 85: 261-6.
 29. Kayembe L. Common causes of blindness in Zaire. *Br J Ophthalmol* 1985; 69: 389-91.
 30. Foster PJ, Johnson GJ. Glaucoma in China: how big is the problem? *Br J Ophthalmol* 2001; 85: 1277–82.
 31. Foster PJ, Baasanhu J, Alsbirk PH, Munkhbayar D, Uranchimeg D, Johnson GJ. Glaucoma in Mongolia. A population-based survey in Hovsgol province, northern Mongolia. *Arch Ophthalmol* 1996; 114: 1235-41.
 32. Baasanhu J, Johnson GJ, Burendei G, Minassian DC. Prevalence and causes of blindness and visual impairment in Mongolia: a survey of populations aged 40 years and older. *Bull World Health Organ* 1994; 72: 771-6.
 33. Sommer A, Tielsch JM, Katz J, Quigley HA, Gottsch JD, Javitt JC, Martone JF, Royall RM, Witt KA, Ezrine S. Racial differences in the cause-specific prevalence of blindness in East Baltimore. *N Engl J Med* 1991; 325: 1412-7.
 34. Johnson CA, Samuels SJ. Screening for Glaucomatous visual field loss with frequency-

- doubling perimetry. *Invest Ophthalmol Vis Sci* 1997; 38: 413–25.
35. Klein BEK, Klein R. Diabetic Retinopathy. In: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*. Chapman & Hall 1998, pp 311-24.
36. Sparrow JM, McLeod BK, Smith TDW, Birch M, Rosenthal R. The prevalence of Diabetic Retinopathy and Maculopathy and their risk factors in the non-insulin treated diabetic patients of an English town. *Eye* 1993; 7: 158-63.
37. Saint Vincent Declaration. Diabetes Mellitus in Europe: a problem at all ages in all countries: a model for prevention and self care. Saint Vincent (Italy), 1989. <http://www.show.scot.nhs.uk/crag/topics/diabetes/vinc ent.htm>. (Accessed 15 September 2004).
38. Klein R. Diabetic retinopathy: an end of the century perspective. *Eye* 1999; 13: 133-35.
39. No Author. Proliferative diabetic retinopathy: treatment with xenon-arc photocoagulation. Interim report of multicentre randomised controlled trial. *Br Med J* 1977; 1: 739-41.
40. Harding S, Greenwood R, Aldington S, Gibson J, Owens D, Taylor R, Kohner E, Scanlon P, Leese G. Diabetic Retinopathy Grading and Disease Management Working Party. Grading and disease management in national screening for diabetic retinopathy in England and Wales. *Diabet Med* 2003; 20: 965-71.
41. Trautner C, Icks A, Haastert B, Plum F, Berger M. Incidence of blindness in relation to diabetes. A population-based study. *Diabetes Care* 1997; 20, 1147-53.
42. Cordeiro MF, Stanford MR, Phillips PM, Shilling JS. Relationship of diabetic microvascular complications to outcome in panretinal photocoagulation treatment of proliferative diabetic retinopathy. *Eye* 1997; 11: 531-6.
43. Wormald R. Screening in Ophthalmology. In: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of eye disease*, Chapman & Hall 1998, pp 83-100.
44. Davis MD, Fisher MR, Gangnon RE, Barton F, Aiello LM, Chew EY, Ferris FL 3rd, Knatterud GL. Risk factors for high-risk proliferative diabetic retinopathy and severe visual loss: Early Treatment Diabetic Retinopathy Study Report #18. *Invest Ophthalmol Vis Sci* 1998; 39: 233-52.
45. Lyons TJ, Jenkins AJ, Zheng D, Lackland DT, McGee D, Garvey WT, Klein RL. Diabetic retinopathy and serum lipoprotein subclasses in the DCCT/EDIC cohort. *Invest Ophthalmol Vis Sci* 2004; 45: 910-8.
46. Cormack TGM, Grant B, Macdonald MJ, Steel J, Campbell IW. Incidence of blindness due to diabetic eye disease in Fife 1990-9. *Br J Ophthalmol* 2001; 85: 354-56.
47. Taylor RH, Jones HS, Dodson PM, Hamilton AP, Kritzinger EE. Diabetic eye disease: a natural history study. *Eye* 1997; 11: 547-53.
48. Kassir MS. [Ophthalmic pathology at a Lebanese clinic: the example of Sidon]. *Sante* 2000; 10: 237-42.
49. Resnikoff S, Pascolini D, Etya'ale D, Kocur I, Parajasegaram R, Pokharel GP, Marioti SP. Global data on visual impairment in the year 2002. *Bull World Health Organ* 1994; 82: 844-51.
50. Herman WH, Aubert RE, Engelgau MM, Thompson TJ, Ali MA, Sous ES, Hegazy M, Badran A, Kenny SJ, unter EW, Malarcher AM, Brechner RJ, Wetterman SF, DeStefano F, Smith PJ, Haib M, abd el Shakour S, Ibrahim AS, el Behairy M. Diabetes mellitus in Egypt, glycaemic control and microvascular and neuropathic complications. *Diab Med* 2004; 15: 1045-51.
51. Foster A, Sommer A. Childhood blindness from corneal ulceration in Africa: causes, prevention and treatment. *Bull World Health Organ* 1986; 64: 619-23.
52. Foster A, Johnson GJ. Measles, corneal ulcerations and childhood blindness: prevention and treatment. *Trop Doct* 1988; 18: 74-8.
53. Foster A, Yosten D. Corneal ulceration in Tanzanian children: relationship between measles and vitamin A deficiency. *Trans Roy Soc Trop Med Hyg* 1992; 86: 454-5.
54. Foster A, Sommer A. Corneal ulceration, measles, and childhood blindness in Tanzania. *Br J Ophthalmol* 1987; 71: 331-43.
55. Khoo CY. Corneal blindness in Singapore and its prevention. *Ann Acad Med Singapore*

- 1989; 18: 123-30.
56. Whitcher JP, Srinivasan M, Upadhyay MP. Prevention of corneal ulceration in the developing world. *Int Ophthalmol Clin* 2002; 42: 71-7.
57. Resnikoff S, Filliard G, Dell'Aquila B. [Corneal complications of climatic keratopathy]. *Rev Int Trach Pathol Ocul Trop Subtrop Sante Publique* 1990; 67: 207-12.
58. Kini MM, Leibowitz HM, Colton T, Nickerson RJ, Ganley J, Dawber TR. Prevalence of senile cataract, diabetic retinopathy, senile macular degeneration, and open-angle glaucoma in the Framingham Eye study. *Am J Ophthalmol* 1978; 85: 28-34.
59. Sandford-Smith JH, Whittle HC. Corneal ulceration following measles in Nigerian children. *Br J Ophthalmol* 1979; 63: 720-4.
60. Klauss V, Schwartz EC. Other conditions of the outer eye. In: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*, Chapman & Hall 1998; 137-58.
61. Tabbara KF. Eye disease induced by traditional eye practices. *Int Ophthalmol Clin* 1990; 30: 23-27.
62. Abdu L. Prevalence and causes of blindness and low vision in Dambatta local government area, Kano State, Nigeria. *Niger J Med* 2002; 11: 108-12.
63. Stilma JS, Bridger S. Causes and prevalence of blindness in the Northern Province of Sierra Leone. *Doc Ophthalmol* 1983; 56: 115-22.
64. Schwartz EC, Huss R, Hopkins A, Dadjim B, Madjitouloum P, Hénault C, Klauss V. Blindness and visual impairment in a region endemic for onchocerciasis in the Central African Republic. *Br J Ophthalmol* 1997; 81: 443-7. .
65. Malu KN, Malu AO. Blindness in Leprosy patients of Kaduna State, Northern Nigeria. *Trop Doct* 1995; 25: 181-3.
66. Tabbara KF. Blindness in the Eastern Mediterranean Countries. *Br J Ophthalmol* 2001; 85: 771-5.
67. Tabe Tambi F. Causes of blindness in the western province of Cameroon. *Rev Int Trach Pathol Ocul Trop Subtrop Sante Publique* 1993; 70: 185-97.
68. Tabbara KF. Climatic droplet keratopathy. *Int Ophthalmol Clin* 1986; 26: 63-8.
69. Matta CS, Tabbara KF, Cameron JA, Hidayat AA, al-Rajhi AA. Climatic droplet keratopathy with corneal amyloidosis. *Ophthalmology* 1991; 98: 192-5.
70. Ormerod LD, Dahan E, Hagele JE, Guzek JP. Serious occurrences in the natural history of advanced climatic keratopathy. *Ophthalmology* 1994; 101: 448-53.
71. Murthy GV, Gupta S, Ellwein LB, Munoz SR, Bachani D, Dada VK. A population-based eye survey of older adults in a rural district of Rajasthan: I. Central vision impairment, blindness, and cataract surgery. *Ophthalmology* 2001; 108: 679-85.
72. Saw SM, Husain R, Gazzard GM, Koh D, Widjaja D, Tan DT. Causes of low vision and blindness in rural Indonesia. *Br J Ophthalmol* 2003; 87: 1075-8.
73. Dineen BP, Bourne RR, Ali SM, Huq DM, Johnson GJ. Prevalence and causes of blindness and visual impairment in Bangladeshi adults: results of the National Blindness and Low Vision Survey of Bangladesh. *Br J Ophthalmol* 2003; 87: 820-8.
74. Zainal M, Masran L, Ropilah AR. Blindness and visual impairment amongst rural Malays in Kuala Selangor, Selangor. *Med J Malaysia* 1998; 53: 46-50.
75. See JL, Wong TY, Yeo KT. Trends in the pattern of blindness and major ocular diseases in Singapore and Asia. *Ann Acad Med Singapore* 1998; 27: 540-46.
76. Lim KH. Singapore National Eye Centre, Singapore. Registration of new blindness in Singapore for 1985-1995. *Singapore Med J* 1998; 39: 104-6.
77. Rekhi GS, Kulshreshtha OP. Common causes of blindness: a pilot survey in Jaipur, Rajasthan. *Indian J Ophthalmol* 1991; 39: 108-11.
78. World Health Organization. WHO Regional Office for the East Mediterranean. Vision 2020 Regional Planning Workshop and Launching of Vision 2020 in Egypt, Cairo, 14-17 December 2003. Press Release No. 23 10 December 2003. <http://www.emro.who.int/pressreleases/2003/no23.htm>. (Accessed January 2005)
79. Thulasiraj RD, Nirmalan PK, Ramakrishnan R, Krishnadas R, Manimekalai TK, Baburajan NP, Katz J, Tielsch JM, Robin AL. Blindness and vision impairment in a rural south Indian

- population: the Aravind Comprehensive Eye Survey. *Ophthalmology* 2003; 110: 1491-8.
80. Cheraskin E. Macular degeneration: how big is the problem? *J Natl Med Assoc* 1992; 84: 873-6.
81. Ho T, Law NM, Goh LG, Yoong T. Eye diseases in the elderly in Singapore. *Singapore Med J* 1997; 38: 149-55.
82. Reference Channel. Allrefer.com Website. Blue Mountains, Australia, Australian And New Zealand Physical Geography. <http://reference.allrefer.com/encyclopedia/B/BlueMtnsAus.html> (Accessed 12 September 2004).
83. Humphrey JH, West KP Jr, Sommer A. Vitamin A deficiency and attributable mortality among under-5-year-olds. *Bull World Health Organ* 1992; 70: 225-32.
84. Zainal M, Ismail SM, Ropilah AR, Elias H, Arumugam G, Alias D, Fathilah J, Lim TO, Ding LM, Goh PP. Prevalence of blindness and low vision in Malaysian population: results from the National Eye Survey 1996. *Br J Ophthalmol* 2002; 86: 951-6.
85. Dandona R, Dandona L. Refractive error blindness. *Bull World Health Organ* 2001; 79: 237-43. .
86. Gilbert C. Childhood blindness. In: Johnson GJ, Minassian DC, Weale R (EDS) *The Epidemiology of eye disease*, Chapman & Hall 1998; 181-207.
87. Steinkuller PG, Du L, Gilbert C, Foster A, Collins ML, Coats DK. Childhood blindness. *J AAPOS* 1999; 3: 26-32.
88. Gilbert CE, Canovas R, Kocksch de Canovas R, Foster A. Causes of blindness and severe visual impairment in children in Chile. *Dev Med Child Neurol* 1994; 36: 326-33.
89. Gilbert C, Rahi J, Eckstein M, O'Sullivan J, Foster A. Retinopathy of prematurity in middle-income countries. *Lancet* 1997; 350: 12-4.
90. Kocur I, Kuchymka P, Rodney S, Barakova D, Schwartz EC. Causes of severe visual impairment and blindness in children attending school for the handicapped in the Czech Republic. *Br J Ophthalmol* 2001; 85: 1149-52.
91. Tabbara FK, El-Shaikh MF, Shawaf SS. Pattern of childhood blindness at a referral centre in Saudi Arabia. *Ann Saudi Med* 2005; 25: 18-21.
92. Jackson H. Bilateral blindness due to trauma in Cambodia. *Eye* 1996; 10: 517-20.
93. Negrel AD. An approach to the Epidemiology of eye injuries. In: Johnson GJ, Minassian DC, Weale R (EDS) *The Epidemiology of eye disease*. Chapman & Hall 1998: 265-87.
94. Negrel AD, Thylefors B. The global impact of eye injuries. *Ophthalmic Epidemiol* 1998; 5: 143-69.
95. Chan T, O'Keefe M, Bowell R, Lanigan B. Childhood penetrating eye injuries. *Ir Med J* 1995; 88: 168-70.
96. LaRoche GR, McIntyre L, Schertzer RM. Epidemiology of severe eye injuries in childhood. *Ophthalmology* 1988; 95: 1603-7. .
97. Muzaffar W, Khan MD, Akbar MK, Malik AM, Durrani OM. Mine blast injuries: ocular and social aspects. *Br J Ophthalmol* 2000; 84: 626-30.
98. Jackson H, Foster A. Causes of blindness in northwest Cambodia. *Ophthalmic Epidemiol* 1997; 4: 27-32.
99. Al-Kubaisy W, Al-Rubaiy MG, Nassief HA. Xerophthalmia among hospitalized Iraqi children. *East Mediterr Health J* 2002; 8: 496. www.emro.who.int/publications/emhj/0804_5/Xerophthalmia.htm.
100. Ramalho RA, Flores H, Saunders C. [Hypovitaminosis A in Brazil: a public health problem]. *Rev Panam Salud Publica* 2002; 12: 117-22.
101. Rosen DS, al Sharif Z, Bashir M, al Shabooti A, Pizzarello LD. Vitamin A deficiency and xerophthalmia in western Yemen. *Eur J Clin Nutr* 1996; 50: 54-7.
102. WHO. Vaccines, Immunization and Biologicals Website. vitamin a. prevalence maps 2/7. africa region. <http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv06.shtml> (accessed 12 september 2004).
103. WHO. Vaccines, immunization and biologicals website. vitamin a. prevalence maps 3/7. the americas region. <http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv07.shtml> (accessed 25 august 2004).
104. WHO. Vaccines, Immunization and Biologicals Website. vitamin a. prevalence maps

- 4/7. eastern mediterranean region.
<http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv08.shtml>
 (Accessed 25 August 2004).
105. WHO. Vaccines, Immunization and Biologicals Website. Vitamin A. Prevalence. Prevalence Maps 5/7. European Region.
<http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv09.shtml>
 (Accessed 25 August 2004).
106. WHO. Vaccines, Immunization and Biologicals Website. Vitamin A. Prevalence Maps 6/7. Southeast Asian Region.
<http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv10.shtml>
 (Accessed 25 August 2004).
107. WHO. Vaccines, Immunization and Biologicals Website. Vitamin A. Prevalence Maps 7/7. Southeast Asian Region.
<http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv11.shtml>
 (Accessed 25 August 2004).
108. World Health Organization Website. Nutrition. micronutrient deficiencies. combating vitamin a deficiency. the challenge.
www.who.int/nut/vad.htm (updated 13/09/2004).
109. WHO. Children and Vitamin A.
<http://www.who.int/vaccines-diseases/en/vitamina/science/sci02.shtml>
 (Accessed 15 September 2004).
110. Fawzi WW, Chalmers TC, Herrera MG, Mosteller F. Vitamin A supplementation and child mortality: A meta-analysis. *JAMA* 1993; 269: 898-903.
111. Katz J, Khattry SK, West KP, Humphrey JH, Leclercq SC, Kimbrough E, Pohkrel PR, Sommer A. Night blindness is prevalent during pregnancy and lactation in rural Nepal. *J Nutr* 1995; 125: 2122-7.
112. Maletnlema TN. Hunger and malnutrition: the determinant of development: the case for Africa and its food and nutrition workers. *East Afr Med J* 1992; 69: 424-7.
113. Curtale F, Tammam H, Hammoud ES, Aloï A. Prevalence of xerophthalmia among children in Beheira governorate, Egypt. *East Mediterr Health J* 1999; 5: 984-91.
114. el-Arab AE, Khalil F, Hussein L. Vitamin A deficiency among preschool children in a rural area of Egypt: the results of dietary assessment and biochemical assay. *Int J Food Sci Nutr* 2002; 53: 465-74.
115. Resnikoff S, Filliard G, Carlier C, Luzeau R, Amedee-Manesme O. Assessment of vitamin A deficiency in the Republic of Djibouti. *Eur J Clin Nutr* 1992; 46: 25-30.
116. Chassot P, Barry AK. [Campaign against hypovitaminosis in Mauritania]. *Rev Int Trach Pathol Ocul Trop Subtrop Sante Publique* 1989; 66: 73-84.
117. De Groulard M, Le Bras J. [Nutritional disorders and primary health care. Analysis and strategic approach in the Mauritanian Adrar region]. *Med Trop (Mars)* 1991; 51: 71-5.
118. Awan HR, Ihsan T. Prevalence of visual impairment and eye diseases in Afghan refugees in Pakistan. *East Mediterr Health J* 1998; 4: 560-6.
119. Resnikoff S, Filliard G, Carlier C, Luzeau R, Amedee-Manesme O. Assessment of vitamin A deficiency in the Republic of Djibouti. *Eur J Clin Nutr* 1992; 46: 25-30.
120. Semba RD, Bloem MW. Measles blindness. *Surv Ophthalmol* 2004; 49: 243-55.
121. Whittle HC, Smith JS, Kogbe OI, Dossetor J, Duggan M. Severe ulcerative herpes of mouth and eye following measles. *Trans R Soc Trop Med Hyg* 1979; 73: 66-9.
122. Pillgram-Larsen J, Mellesmo S, Peck R. [Injuries from mines]. *Tidsskr Nor Laegeforen* 1992; 112: 2183-7.
123. Kugoeva EE, Aslanova AF, Kulieva ZT. [Characteristics of the clinical picture of combined injuries of the organ of vision and eye appendages under conditions of peace-time and war injuries]. *Vestn Oftalmol* 2002; 118: 11-3.
124. WHO. Regional Office for the Eastern Mediterranean. The WHO collaborative programme. Noncommunicable Diseases including blindness and deafness. Situation analysis. <http://www.emro.who.int/jordan/CollaborativeProg-NCD.HTM> (Accessed 29 August 2004).
125. WHO. Health Under Difficult Circumstances: The Impact of War, Disasters, and Sanctions on the Health of Population. Executive summary. Document Reference: RC49/Tech.Disc.1.

www.who.int/disasters/repo/8451.doc (Accessed 29 August 2004).

126. Abdu Z, Hashim K, Muhi MA, Gilbert C. Prevalence and causes of childhood blindness in camps for persons displaced in Khartoum, Sudan: results of a household survey. Personal communication.
127. Chumbley LC, Thomson IM. Epidemiology of trachoma in the West Bank and Gaza Strip. *Eye* 1988; 2: 463-70.
128. Katz J, West KP Jr, Khattry SK, LeClerq SC, Padhan EK, Thapa MD, Ram Shrestha S, Taylor HR. Prevalence and risk factors for trachoma in Sarlahi district, Nepal. *Br J Ophthalmol* 1996; 80: 0137-41.
129. Whitfield R Jr. Dealing with Cataract Blindness. Part III: Paramedical Cataract surgery in Africa. *Ophthalmic Surg* 1987; 18: 765-7.
130. Kumaresan JA, Mecaskey JW. The global elimination of blinding Trachoma: progress and promise. *Am J Trop Med Hyg* 2003; 69: s24-28.
131. West SK. Blinding trachoma: prevention with the safe strategy. *Am J Trop Med Hyg* 2003; 69: S18-23.
132. Rostkowski L, Szmyt J. [New method of campaigning against trachoma in Poland; shock action]. *Rev Int Trach* 1950; 27: 161-5.
133. Verin P, Hoang TL, Do TN, Polak C. [Epidemiology of trachoma and vision disorders in central Viet Nam]. *Rev Int Trach Pathol Ocul Trop Subtrop Sante Publique* 1991; 68: 107-13.
134. Dolin PJ, Faal H, Johnson GJ, Ajewole J, Mohamed AA, Lee PS. Trachoma in The Gambia. *Br J Ophthalmol* 1998; 82: 930-3.
135. Mabey DC, Solomon AW, Foster A. Trachoma. *Lancet* 2003; 362: 223-9.
136. Diamond J. Demography of the Arab World. Lecture. ST203, 21.10.02.
137. Tabbara KF, al-Omar OM. Trachoma in Saudi Arabia. *Ophthalmic Epidemiol* 1997; 4: 127-40.
138. al Faran MF. Low prevalence of trachoma in the south western part of Saudi Arabia, results of a population based study. *Int Ophthalmol* 1994-95; 18: 379-82.
139. Chandra G. Trachoma in eastern province of Saudi Arabia. *Rev Int Trach Pathol Ocul Trop Subtrop Sante Publique* 1992; 69: 118-32.
140. Graz B. Trachoma: possibilities of prevention. A study in the Sultanate of Oman. *Eur J Ophthalmol* 1993; 3: 127-31.
141. Ezz al Arab G, Tawfik N, El Gendy R, Anwar W, Courtright P. The burden of Trachoma in the Rural Nile Delta of Egypt: a survey of Menofiya Governorate. *Br J Ophthalmol* 2001; 85: 1406-10.
142. Bird M, Dawson CR, Schachter JS, Miao Y, Shama A, Osman A, Bassem A, Lietman TM. Does the diagnosis of trachoma adequately identify ocular chlamydial infection in trachoma-endemic areas? *J Infect Dis* 2003; 187: 1669-73.
143. Barsoum IS, Mostafa MS, Shihab AA, el Alamy M, Habib MA, Colley DG. Prevalence of trachoma in school children and ophthalmological outpatients in rural Egypt. *Am J Trop Med Hyg* 1987; 36: 97-101.
144. Salim AR, Sheikh HA. Trachoma in the Sudan. an Epidemiological study. *Br J Ophthalmol* 1975; 59: 600-4.
145. Mahmoud EA, Sheikh AH, Domeika MA, Mardh PA. Prevalence of trachoma among displaced persons in the Sudan: a clinical and sero-epidemiological study. *Eye* 1994; 8: 130-3.
146. Dawson CR, Schachter J, Sallam S, Sheta A, Rubinstein RA, Washton H. A comparison of Oral Azithromycin with Topical Oxytetracycline/Polymyxin for the treatment of trachoma in children. *Clin Infect Dis* 1997; 24: 363-8.
147. Schachter J, Dawson CR. The Epidemiology of Trachoma predicts more blindness in the future. *Scand J Infect Dis* 1990; 69: s55-62.
148. Taylor HR, Johnson SL, Schachter J, Caldwell HD, Prendergast RA. Pathogenesis of Trachoma: the stimulus for inflammation. *J Immunol* 1987; 138: 3023-7.
149. Courtright P, Sheppard J, Lane S, Sadek A, Schachter J, Dawson CR. Latrine ownership as a protective factor in inflammatory Trachoma in Egypt. *Br J Ophthalmol* 1991; 75: 322-5.
150. Tabbara KF. Trachoma: a review. *J Chemother* 2001; 13: S18-S22.
151. Frick KD, Hanson CL, Jacobson GA. Global burden of trachoma and economics of the disease. *Am J Trop Med Hyg* 2003; 69: 1-10.
152. Hoehsmann A, Metcalfe N, Kanjaloti S, Godia H, Mtambo O, Chipeta T, Barrows J,

- Witte C, Courtright P. Reduction of trachoma in the absence of antibiotic treatment: evidence from a population-based survey in Malawi. *Ophthalmic Epidemiol* 2001; 8: 145-53.
153. Jha H, Chaudary JS, Bhatta R, Miao Y, Osaki-Holm S, Gaynor B, Zegans M, Bird M, Yi E, Holbrook K, Whitcher JP, Lietman T. Disappearance of trachoma from Western Nepal. *Clin Infect Dis* 2002; 35: 765-68.
154. Dolin PJ, Faal H, Johnson GJ, Minassian D, Sowa S, Day S, Ajewole J, Mohamed AA, Foster A. Reduction of trachoma in a sub-Saharan village in absence of a disease control programme. *Lancet* 1997; 349: 1511-2.
155. Ballard RC, Fehler HG, Fotheringham P, Sutter EE, Treharne JD. Trachoma in South Africa. *Soc Sci Med* 1983; 17: 1755-65.
156. Melese M, Alemayehu W, Gaynor B, Yi E, Whitcher JP, Lietman TM. What more is there to learn about trachoma? *Br J Ophthalmol* 2003; 87: 521-2.
157. Lietman T, Fry A. Can we eliminate trachoma? *Br J Ophthalmol* 2001; 85: 385-7.
158. Emerson PM, Bailey RL, Mahdi OS, Walraven GE, Lindsay SW. Transmission ecology of the fly *Musca sorbens*, a putative vector of trachoma. *Trans R Soc Trop Med Hyg* 2000; 94: 28-32.
159. Schwab L, Whitfield R Jr, Ross-Degnan D, Steinkuller P, Swartwood J. The epidemiology of trachoma in rural Kenya. variation in prevalence with lifestyle and environment. study survey group. *Ophthalmology* 1995; 102: 475-82.
160. Zhang H, Kandel RP, Sharma B, Dean D. Risk factors for recurrence of postoperative trichiasis: implications for trachoma blindness prevention. *Arch Ophthalmol* 2004; 122: 511-6.
161. Mabey D, Fraser-Hurt N. Antibiotics for trachoma. *Cochrane Database Syst Rev* 2002; 1: CD001860.
162. Alene GD, Abebe S. Prevalence of risk factors for Trachoma in a rural locality of North-Western Ethiopia. *East Afr Med J* 2000; 77: 308-12.
163. Paula JS, Medina NH, Cruz AA. Trachoma among the Yanomami Indians. *Braz J Med Biol Res* 2002; 35: 1153-7.
164. Vastine DW, Dawson CR, Daghfous T, Messadi M, Hoshiwara I, Yoneda C, Nataf R. Severe Endemic Trachoma in Tunisia. I. effect of topical Chemotherapy on Conjunctivitis and Ocular Bacteria. *Br J Ophthalmol* 1974; 58: 833-42.
165. Dawson CR, Daghfous T, Messadi M, Hoshiwara I, Schachter J. Severe Endemic Trachoma in Tunisia. *Br J Ophthalmol* 1976; 60: 245-52.
166. Ayed S, Jeddi A, Daghfous F, Ben Osman N, Darghouth K. [Senile Cataract and Trachoma in Tunisia]. *Rev Int Trach Pathol Ocul Trop Subtrop Sante Publique* 1992; 69: 139-46.
167. Emerson PM, Cairncross S, Bailey RL, Mabey DC. Review of the evidence base for the 'F' and 'E' components of the SAFE strategy for trachoma control. *Trop Med Int Health* 2000; 5: 515-27.
168. Negrel AD, Khazraji YC, Akalay O. [Trachoma in the Province of Ouarzazate, Morocco]. *Bull World Health Organ* 1992; 70: 451-6.
169. Ferriman A. Blinding Trachoma almost eliminated from Morocco. *Br Med J* 2001; 323: 1387.
170. Takourt B, Milad A, Radouani F, Boura H, Guinet R, Benslimane A. [Isolation of *Chlamydia trachomatis* in trachomatous Moroccan patients]. *J Fr Ophtalmol* 1996; 19: 527-32.
171. Negrel AD, Chami-Khazraji Y, Arrache ML, Ottmani S, Mahjour J. [The quality of trichiasis surgery in the kingdom of Morocco]. *Sante* 2000; 10: 81-92.
172. Kmietowicz Z. Who steps up efforts against Trachoma. *Br Med J* 1996; 313: 1428.
173. Thylefors B, Negrel AD. Developments for a global approach to Trachoma control. *Rev Int Trach Pathol Ocul Trop Subtrop Sante Publique* 1994; 71: 63-7, 69-77.
174. Mecaskey JW, Ngirwamungu E, Kilima PM. Integration of Trachoma control into primary health care: the Tanzanian experience. *Am J Trop Med Hyg* 2003; 69: 29-32.
175. Schemann JF, Sacko D, Banou A, Bamani S, Bore B, Coulibaly S, el Mouchtahide MA. [Cartography of Trachoma in Mali: results of a national survey]. *Bull World Health Organ* 1988; 76: 599-606.
176. Schemann JF, Sacko D. [Strategies to control Trachoma]. *Sante* 1998; 8: 150-6.

177. Huguet P. [Trachoma in Niger (results of a sample survey in the department of Tahoua)]. *Rev Int Trach Pathol Ocul Trop Subtrop Sante Publique* 1989; 66: 31-41.
178. Tabbara KF. Blinding trachoma: the forgotten problem (editorial). *Br J Ophthalmol* 2001; 85: 1397-1401.
179. Cunningham Jr ET, Lietman TM, Whitcher JP. Blindness: a global priority for the twenty-first century (editorial). *Bull World Health Organ* 2001, 79: 180.
180. Holm SO, Jha HC, Bhatta RC, Chaudhary JSB, Thapa BB, Davis D, Pokhrel RP, Yinghui M, Zegans M. Comparison of two Azithromycin distribution strategies for controlling Trachoma in Nepal. *Bull World Health Organ* 2001; 78: 194-200.
181. Report of the Regional Director to the Regional Committee for the Western Pacific. Fifty Years of the World Health Organization in the Western Pacific Region 1998. Chapter 28. Trachoma and other causes of blindness. http://www.wpro.who.int/public/policy/50TH/Ch_28.html.
182. World Health Organization. Water-related diseases. Trachoma: the disease and how it affects people. http://www.who.int/water_sanitation_health/diseases/trachoma/en/print.html (Accessed 24 August 2004).
183. Loewenthal R, Pe'er J. A prevalence survey of ophthalmic diseases among the Turkana tribe in northwest Kenya. *Br J Ophthalmol* 1990; 75: 84-8.
184. Jaques J, Sauter M. Xerophthalmia and measles in Kenya. *Doc Ophthalmol* 1976; 42: 1-235.
185. Whitfield R Jr, Schwab L, Bakker NJ, Bisley GG, Ross-Degnan D. Cataract and corneal opacity are the main causes of blindness in the Samburu tribe of Kenya. *Ophthalmic Surg* 1983; 14: 139-44.
186. Cook CD, Knight SE, Crofton-Briggs I. Prevalence and causes of low vision and blindness in northern KwaZulu. *S Afr Med J* 1993; 83: 590-3.
187. Whitfield R, Schwab L, Ross-Degnan D, Steinkuller P, Swartwood J. Blindness and eye disease in Kenya: ocular status survey results from the Kenya Rural Blindness Prevention Project. *Br J Ophthalmol* 1990; 74: 333-40.
188. Schwab L, Whitfield R Jr, Ross-Degnan D, Steinkuller P, Swartwood J. The epidemiology of Trachoma in Rural Kenya. variation in prevalence with lifestyle and environment. study survey group. *Ophthalmology* 1995; 102: 475-82.
189. Schachter J, Dawson CR. The epidemiology of trachoma predicts more blindness in the future. *Scand J Infect Dis* 1990; 69: s55-62.
190. Brilliant LB, Pokhrel RP, Grasset NC, Lepkowski JM, Kolstad A, Hawks W, Pararajasegaram R, Brilliant GE, Gilbert S, Shrestha SR, et al. Epidemiology of blindness in Nepal. *Bull World Health Organ* 1985; 63: 375-86.
191. WHO. Essential Medicines Library – EMLib. Trachoma - chlamydial infection. http://mednet3.who.int/eml/disease_factsheet.asp?diseaseId=349 (Accessed 29 August 2004).
192. Asaka T, Manaka A, Sugiyama H. Recent developments in Macrolide Antimicrobial research. *Curr Top Med Chem* 2003; 3: 961-89.
193. Melese M, Chidambaram JD, Alemayehu W, Lee DC, Yi EH, Cevallos V, Zhou Z, Donnellan C, Saidel M, Whitcher JP, Gaynor BD, Lietman TM. Feasibility of eliminating ocular Chlamydia trachomatis with repeat mass antibiotic treatments. *JAMA* 2004; 292: 721.
194. Dawson CR, Schachter J. Strategies for treatment and control of Blinding Trachoma: cost-effectiveness of topical or systemic antibiotics. *Rev Infect Dis* 1985; 7: 768-73.
195. Frick KD, Lietman TM, Holm SO, Jha HC, Chaudhary JDP, Bhatta RC. Cost-effectiveness of trachoma control measures: comparing targeted household treatment and mass treatment of children. *Bull World Health Organ* 2001, 79: 201-7.
196. Frick KD, Hanson CL, Jacobson GA. Global burden of trachoma and economics of the disease. *Am J Trop Med Hyg* 2003; 69: 1-10.
197. Richards F, Klein RE, Gonzales-Peralta C, Flores RZ, Ramirez JC. Knowledge, attitudes and perceptions (KAP) of onchocerciasis: a survey among residents in an endemic area in Guatemala targeted for mass chemotherapy with

- ivermectin. *Soc Sci Med* 1991; 32:
198. Richards F, Hopkins D, Cupp E. Programmatic goals and approaches to Onchocerciasis. *Lancet* 2000; 355: 1663-64.
199. Frentzel-Beyme RR. Visual impairment and incidence of blindness in Liberia and their relation to onchocerciasis. *Tropenmed Parasitol* 1975; 26: 469-88.
200. Richard-Lenoble D, al Qubati Y, Toe L, Pisella PJ, Gaxotte P, al Kohlani A. [Human onchocerciasis and "sowda" in the Republic of Yemen]. *Bull Acad Natl Med* 2001; 185: 1447-59.
201. McMahon JE, Sowa SI, Maude GH, Hudson CM, Kirkwood BR. Epidemiological studies of onchocerciasis in savanna villages of Sierra Leone. *Trop Med Parasitol* 1988; 39: 260-8.
202. Ibrahim AA. [Onchocerciasis in Sudan: epidemiological situation in the south-west]. *Med Trop (Mars)* 1987; 47: 333-7.
203. Mackenzie CD, Williams JF, O'Day J, Ghalal I, Flockhart HA, Sisley BM. Onchocerciasis in southwestern Sudan: parasitological and clinical characteristics. *Am J Trop Med Hyg* 1987; 36: 371-82.
204. Baker RH, Abdelnur OM. Onchocerciasis in Sudan: the distribution of the disease and its vectors. *Trop Med Parasitol* 1986; 37: 341-55.
205. Bird AC, Anderson J, Fuglsang H. Morphology of posterior segment lesions of the eye in patients with onchocerciasis. *Br J Ophthalmol* 1976; 60: 2-20.
206. el Sheikh H, Ghalib H, Hussein SM, Barbiero V, Mustafa MB, Williams JF. Onchocerciasis in Sudan: the Southern Darfur focus. *Trans R Soc Trop Med Hyg* 1986; 80: 902-5.
207. Mustafa KY, Turunen U, Gumaa KA. Serum vitamin A levels of patients with onchocerciasis from two areas of the Sudan. *J Trop Med Hyg* 1979; 82: 122-7.
208. Renz A, Wenk P, Anderson J, Fuglsang H. Studies on the dynamics of transmission of onchocerciasis in a Sudan-savanna area of North Cameroon V. What is a tolerable level of Annual Transmission Potential? *Ann Trop Med Parasitol* 1987; 81: 263-74.
209. Picq JJ, Albert JP. [Sudan-savanna and rain-forest onchocerciasis in West Africa: an epidemiological problem]. *Rev Epidemiol Sante Publique* 1979; 27: 483-98.
210. Ndyomugenyi R. Onchocerciasis control in Uganda. *World Health Forum* 1998; 19: 192-5.
211. Sa MR, Maia-Herzog M. [Overseas disease: comparative studies of onchocerciasis in Latin America and Africa]. *Hist Cienc Saude Manguinhos* 2003; 10: 251-58.
212. Guderian RH, Shelley AJ. Onchocerciasis in Ecuador: the situation in 1989. *Mem Inst Oswaldo Cruz* 1992; 87: 405-15.
213. Whitworth JA, Gilbert CE, Mabey DM, Morgan D, Foster A. Visual loss in an onchocerciasis endemic community in Sierra Leone. *Br J Ophthalmol* 1993; 77: 30-2. .
214. Homeida M, Braide E, Elhassan E, Amazigo UV, Liese B, Benton B, Noma M, Etya'ale D, Dadzie KY, Kale OO, Seketeli A. Apoc's strategy of community-directed treatment with ivermectin (CDTI) and its potential for providing additional health services to the poorest populations. *African programme for Onchocerciasis control. Ann Trop Med Parasitol* 2002; 96: s93-104.
215. Little MP, Basanez MG, Breitling LP, Boatman BA, Alley ES. Incidence of blindness during the Onchocerciasis control programme in Western Africa, 1971-2002. *J Infect Dis* 2004; 189: 1932-41.
216. Burnham G. Onchocerciasis. *Lancet* 1998; 351: 1341-6.
217. Vivas-Martinez S, Basanez MG, Botto C, Villegas L, Garcia M, Curtis CF. Parasitological indicators of Onchocerciasis relevant to ivermectin control programmes in the Amazonian focus of Aouthern Venezuela. *Parasitology* 2000; 121: 527-34.
218. Okhuysen PC. Onchocerciasis in an expatriate living in Cameroon. *J Travel Med* 1997; 4: 11-13.
219. Ochoa JO, Castro JC, Barrios VM, Juarez EL, Tada I. Successful control of Onchocerciasis vectors in San Vicente Pacaya, Guatemala, 1984-1989. *Ann Trop Med Parasitol* 1997; 91: 471-9.
220. Vieta F. River Blindness. Protection for 54 cents a year. *Un Chron* 1998; 1: 12-3.
221. Hougard JM, Yameogo L, Seketeli A,

- Boatin B, Dadzie KY. Twenty-two years of blackfly control in the Onchocerciasis control programme in West Africa. *Parasitol Today* 1997; 13: 425-31.
222. Molyneux DH, Davies JB. Onchocerciasis control: moving towards the millennium. *Parasitol Today* 1997; 13: 418-25.
223. Tielsch JM, Beeche A. Impact of ivermectin on illness and disability associated with Onchocerciasis. *Trop Med Int Health* 2004; 9: a45-56.
224. Calamari D, Crosa G. Long-term ecological assessment of West African rivers treated with insecticides: methodological considerations on quantitative analyses. *Toxicol Lett* 2003; 140-141: 379-89.
225. Pitroipa X, Sankara D, Konan L, Sylla M, Doannio JM, Traore S. [Evaluation of cocoa oil for individual protection against simulum damnosum s.i.]. *Med Trop (mars)* 2002; 62: 511-6.
226. Dadzie Y, Neira M, Hopkins D. Final report of the conference on the eradicability of Onchocerciasis. *Filaria J* 2003; 2: 2.
227. Editoria. River blindness: NGOS agree Africa strategy in Geneva. *Essent Drugs Monit* 1993; 16: 4.
228. Hougard JM, Yameogo L, Philippon B. Onchocerciasis in West Africa after 2002: a challenge to take up. *Parasite* 2002; 9: 105-11.
229. Benton B, Bump J, Seketeli A, Liese B. Partnership and promise: evolution of the african river-blindness campaigns. *Ann Trop Med Parasitol* 2002; 96: s5-14.
230. Chovet M, Carlier C, Queguiner P, Mariko S. [Mass treatment of Onchocerciasis in 1996]. *Med Trop (mars)* 1995; 55: 425-8.
231. Seketeli A, Guillet P, Coloussa B, Philippon B, Quillevere D, Samba EM. [National entomological teams of the Western extension zone of the Onchocerciasis control program (OCP) in West Africa from 1986 to 1990]. *Bull World Health Organ* 1993; 71: 737.
232. De Sole G, Remme J. Onchocerciasis infection in children born during 14 years of simulum control in West Africa. *Trans R Soc Trop Med Hyg* 1991; 85: 385-90.
233. Agoua H, Quillevere D, Back C, Poudiougou P, Guillet P, Zerbo DG, Henderickx JE, Seketeli A, Sowah S. [Evaluation of control measures against simuliidae in the framework of the OCP (Onchocerciasis Control Program)]. *Ann Soc Belg Med Trop* 1991; 71: s49-63.
234. Le Berre R, Walsh JF, Philippon B, Poudiougou P, Henderickx JE, Guillet P, Seketeli A, Quillevere D, Grunewald J, Cheke RA. The who Onchocerciasis control programme: retrospect and prospects. *Philos Trans R Soc Lond B Biol Sci* 1990; 328: 721-7.
235. Quarcoopome CO. Onchocerciasis: a major social problem in West Africa. *Soc Sci Med* 1983; 17: 1703-7.
236. Evans TG, Murray CJ. A critical re-examination of the economics of blindness prevention under the Onchocerciasis control programme. *Soc Sci Med* 1987; 25: 241-9.
237. Dadzie KY, Remme J, Rolland A, Thylefors B. The effect of 7-8 years of vector control on the evolution of ocular Onchocerciasis in West African Savanna. *Trop Med Parasitol* 1986; 37: 263-70.
238. Lazdins-Helds JK, Remme JHF, Boakye N. WHO Website. research results: Onchocerciasis. <http://www.who.int/tdr/dw/oncho2003.htm>. (Updated December 2003).
239. Kluxen G. [Vision 2020: 100 years of river blindness research]. *Klin Monatsbl Augenheilkd* 2002; 219: 149-55.
240. Winnen M, Plaisier AP, Alley ES, Nagelkerke NJD, Van Oortmarsen G, Boatin BA, Habbema JDF. Can Ivermectin mass treatments eliminate onchocerciasis in Africa? *Bull World Health Organ* 2002; 80: 384-390.
241. Murdoch IE, Jones BR, Cousens S, Liman I, Babalola OE, Dauda J, Abiose A. Visual field constriction as a cause of blindness or visual impairment. *Bull World Health Organ* 1997; 75: 141-46.
242. Courtright P, Lewallen S. Ocular manifestations of leprosy. In: Johnson GJ, Minassian DC, Weale R (eds.) *The Epidemiology of Eye Disease*. Chapman & Hall 1998: 249-64.
243. No Author. Leprosy. *Nature Reviews* 2003; 1: 4-5.
244. World Health Organization. *The World Health Report 2002: Reducing Risks, Promoting Healthy Life*. <http://www.who.int/whr/2002/en/>. (Accessed 15 September 2004).

245. Ibrahim MA, Kordy MN, Aiderous AH, Bahnassy A. Leprosy in Saudi Arabia, 1986-89. *Lepr Rev* 1990; 61: 379-85.
246. Lavrik AU, Obadi Akhmad Said, Bochanov EA. [Initial measures in studying the distribution of leprosy in Yemen]. *Vestn Dermatol Venerol* 1970; 44: 56-8.
247. Schaller UC, Klauss V. Is Crede' s prophylaxis for ophthalmia neonatorum still valid? *Bull World Health Organ* 2001; 79: 236.
248. Kestelyn O. HIV/Aids and the eye. In: Johnson GJ, Minassian DC, Weale R (EDS) *The Epidemiology of Eye Disease*. Chapman & Hall 1998: 289-309.
249. World Health Organization. *The World Health Report 2004. Changing history*. www.who.int/whr/en/ (Accessed 15 September 2004).
250. Kestelyn PG, Cunningham Jr ET. HIV/AIDS and blindness. *Fjj*.
251. Guex-Crosier Y, Telenti A. An epidemic of blindness: a consequence of improved HIV care? *Bull World Health Organ* 2001; 79: 181