

## 6 COMMON BLINDING CONDITIONS

### 6.1 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 subject will be addressed under three headings as seen in the following Table 6.1.

<p><b>A) Clinical Conditions</b></p> <ol style="list-style-type: none"><li>1. Cataract (6.2)</li><li>2. Glaucoma (6.3)</li><li>3. Diabetic retinopathy (6.4)</li><li>4. Corneal blindness (6.5)<ol style="list-style-type: none"><li>a. Trachomatous</li><li>b. Non-trachomatous<ol style="list-style-type: none"><li>i. Measles</li><li>ii. Onchocerciasis, leprosy,</li><li>iii. Traditional eye medicine (TEM) and extended wear contact lenses (EWCL)</li><li>iv. Climatic droplet keratopathy</li><li>v. Ophthalmia neonatorum</li></ol></li></ol></li><li>5. Refractive errors (6.7)</li><li>6. Recreational drugs (6.8)</li><li>7. Retinopathy of prematurity (6.9)</li><li>8. Ocular trauma (6.11)</li></ol> <p><b>B) Nutritional causes and Vitamin A Deficiency including VAD/measles/HSK synergism (6.12)</b></p> <p><b>C) Specific Important Infectious diseases</b></p> <ol style="list-style-type: none"><li>1. Trachoma (6.13)</li><li>2. Onchocerciasis (6.14)</li><li>3. Leprosy (6.15)</li><li>4. Ophthalmia neonatorum (6.16)</li><li>5. AIDS (6.17)</li></ol>
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**Table 6.1: Plan of section 2.6**

The four main causes are cataract, trachoma, onchocerciasis and xerophthalmia<sup>227-228, 286</sup> 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<sup>227-228, 286</sup>. Data on other blinding conditions, such as diabetic retinopathy in the developing countries, are scarce<sup>411</sup>.

## **6.2 Cataract**

### **6.2.1 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<sup>253, 576, 578</sup>. 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<sup>248</sup>. In the poorer countries, the prevalence is also variable<sup>154, 221, 291, 246-248</sup>. In these countries<sup>154, 246-248, 291</sup>, 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<sup>334, 578</sup>. 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<sup>576</sup>.

### **6.2.2 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. Table 6-2 lists all the possible risk factors incriminated in the aetiology of adult cataract.

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<sup>248, 291, 577</sup>. A study in Tibet<sup>266</sup> has demonstrated that cataract incidence increases with a decrease in latitude or increase in altitude due to increased solar radiation. In the surveyed areas, Zedang region, which has the highest altitude and low latitude

<b>Factors in Age Related Cataract Pathogenesis</b>		
<b>(A) Risk Factors</b>		
<b>Definite</b>	Age (all morphologies) Diabetes (posterior capsular) Gender (female) Smoking (nuclear) Steroids use (poster subcapsular) Sunlight (cortical, poster subcapsular)	
<b>Probable</b>	Dehydrational crisis/heatstroke Low social class	
<b>Possible</b>	Low height Low weight Low body-mass Limited education Rural residence Occupation	Alcohol Oestrogens Myopia Coffee Hypertension Renal failure
<b>(B) Possible protective factors</b>		
	Aspirin Antioxidant vitamins	

**Table 6-2: Factors in cataract causation**<sup>650, 665</sup>

(29 degrees 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 interven-

tions to reduce the risk of cataract seem to be to reduce ocular exposure to UV-B radiation and to stop smoking<sup>577</sup>.

### **6.2.3 Gender in Cataract Blindness**

Globally, women bear an excess of blindness compared to men accounting for 64.5% of all blind people<sup>275, 300</sup>. 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<sup>275</sup>. 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<sup>221, 573</sup>.

### **6.2.4 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<sup>200, 226</sup>. 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<sup>200</sup>.

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<sup>226</sup>.

### **6.2.5 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 prevention 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, enterovirus 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<sup>555</sup>

## **6.3 Glaucoma**

### **6.3.1 Prevalence of Glaucoma**

Glaucoma<sup>601, 636</sup> 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<sup>460, 572</sup>.

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<sup>314, 599, 600</sup>. 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<sup>285, 599, 600, 601</sup>. In these countries, fewer than 50% of those with glaucoma are aware of their disease<sup>460</sup>. Because of this, it is known as 'the little thief' in Saudi Arabia<sup>478</sup>. In the developed countries, glaucoma blindness develops at a later age leaving the patients blind for a shorter time. This pattern may be explain-

ed 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% <sup>471</sup>.

### **6.3.2 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 <sup>572, 601, 635</sup>.

### **6.3.3 Risk Factors in Glaucoma**

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 <sup>599, 601</sup>.

## **6.4 Diabetic Retinopathy**

### **6.4.1 Prevalence of Diabetic Retinopathy**

Diabetes affects nearly 3% of the population and diabetic retinopathy <sup>623, 628-630, 671-672</sup> is a leading cause of visual impairment in the working age groups in western society. 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 <sup>623, 629, 637-640</sup>. Diabetic retinopathy fulfils all the parameters for a condition that is ideal for screening <sup>615</sup>.

#### **6.4.2 Risk Factors in Diabetic Retinopathy**

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<sup>623, 628, 642</sup>. 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<sup>640</sup>. 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<sup>641, 623, 628-630, 671-672</sup>.

#### **6.4.3 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<sup>639</sup>. 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<sup>347, 623, 716, 717</sup>.

### **6.5 Corneal Blindness**

#### **6.5.1 Prevalence of Corneal Blindness in General**

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 industrialised countries <sup>126, 253</sup>.

### **6.5.2 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 <sup>253</sup>. 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) <sup>126-127, 129, 134, 253, 290, 322, 364, 579, 585, 602, 668</sup>.

## **6.6 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 <sup>227-228, 286</sup>. Data on other blinding conditions, such as diabetic retinopathy in the developing countries, are scarce <sup>411</sup>. (Refer to section 6).

## **6.7 Corneal Opacities from Non-Trachomatous Causes**

### **6.7.1 Prevalence**

Corneal blindness due to non-trachomatous causes have been reported in several parts of Africa including Nigeria <sup>300</sup>, the Niger <sup>293</sup> (7%) and Sierra Leon <sup>316</sup> (3%). These causes include measles, VAD, HSK, onchocerciasis, leprosy, TEM and EWCL.



### **6.7.2 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<sup>585</sup>. (Refer to 6.12.7 for synergism of xerophthalmia, diarrhoea and measles).

### **6.7.3 Blindness from Non-trachomatous Infections (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<sup>587</sup>, and Central African Republic<sup>581</sup>.

Leprosy can also contribute to corneal blindness by predisposing to exposure keratitis (21.3%), corneal opacities (13.5%) and chronic uveitis (10.1%)<sup>588</sup>.

### **6.7.4 Blindness from TEM and EWCL**

There is a significant association between corneal ulceration and *TEM* use and, in particular, peripheral corneal ulcerations<sup>487, 579</sup>. *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<sup>253, 487, 487, 602</sup>.

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<sup>668</sup>.

### **6.7.5 Blindness from Climatic Droplet Keratopathy**

Climatic droplet Keratopathy (CDK) is another important cause of blindness<sup>248, 285, 305, 314, 364, 602, 613, 614</sup>. 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<sup>602</sup>. 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<sup>285, 341, 602</sup>. 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<sup>602, 613, 614</sup>. Severe cases of CDK are found in the Dahlak Islands in the Red Sea where 3% of the population are blind from it<sup>602</sup>.

### **6.8 Refractive errors**

This is a major cause of recorded visual impairment in a large number of developing countries<sup>252, 271-273, 276, 295, 279-280, 287, 353, 448, 472, 451, 457, 535, 557</sup> and has recently been recognised 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<sup>287, 352</sup>. 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<sup>352</sup>.

## **6.9 Retinopathy of Prematurely**

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 <sup>79</sup>. 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 <sup>104, 121, 122</sup>. 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 <sup>97</sup>. It is also a major problem in Cuba contributing to 38.6% of childhood blindness <sup>122</sup> and has been reported in Saudi Arabia <sup>715</sup>. Its incidence in the UK is only 3% <sup>79</sup>.

## **6.10 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 <sup>79</sup>.

## **6.11 Ocular Trauma**

### **6.11.1 Incidence and Monocular Blindness**

Ocular trauma <sup>284, 621</sup> 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 <sup>621, 676</sup>.

- a) Those restricting activities for > 1 day is: 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<sup>253</sup>. The commonest cause of injury was child related from a thrown object (42%) and hazardous toys (21%)<sup>656</sup>. Risk factors in trauma are age, male gender, low socio-economic status and active life style<sup>621</sup>.

### **6.11.2 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<sup>662</sup>.

### **6.11.3 Wars and Landmine Injuries**

An important cause of bilateral blindness is landmines. These have been reported in countries such as Cambodia and Afghanistan<sup>284, 541</sup>. 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<sup>282</sup>.

## **B) Nutritional Factors and VAD**

### **6.12 Vitamin A Deficiency (VAD) (Figure 6-2)**

#### **6.12.1 Epidemiological Aspects of VAD Manifestations**

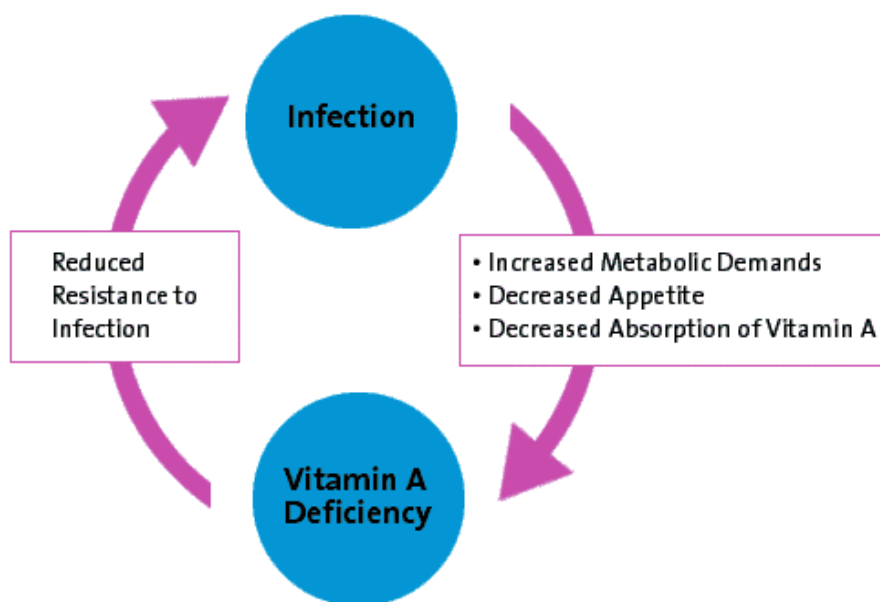
Manifestations of VAD range from night blindness (31%); conjunctival xerosis (57%); Bitot's spots (10.8%) and corneal scars (1%). Severe complications include corneal ulceration or keratomalacia.

Xerophthalmia, due to VAD, is still a major public health problem in many parts of the world, especially in Africa (Figures 6-3, 4, 5). The mother's

level of education and the likelihood of her child having xerophthalmia were significantly associated <sup>554</sup>.

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 <sup>412</sup>. The latter is considered at a serum retinol level of < 20 µg/dL (< 0.70 µmol/L), that is double that associated with clinical eye manifestations (< 10 µg/dL, < 0.35 µmol/L) according to WHO definition and cut-off value <sup>554</sup>.

For the cycle of disease and VAD refer to Figure 6-1.



**Figure 6-1: Cycle of Disease and Vitamin A Deficiency**

Adopted from WHO Website. (Accessed 25 September 2003)

Vaccines, Immunisation and Biologicals. Vitamin A <sup>531</sup>.

<http://www.who.int/vaccines-diseases/en/vitamina/science/sci01.shtml>

### 6.12.2 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) <sup>554</sup>. 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 <sup>345</sup>. 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 <sup>554</sup>.

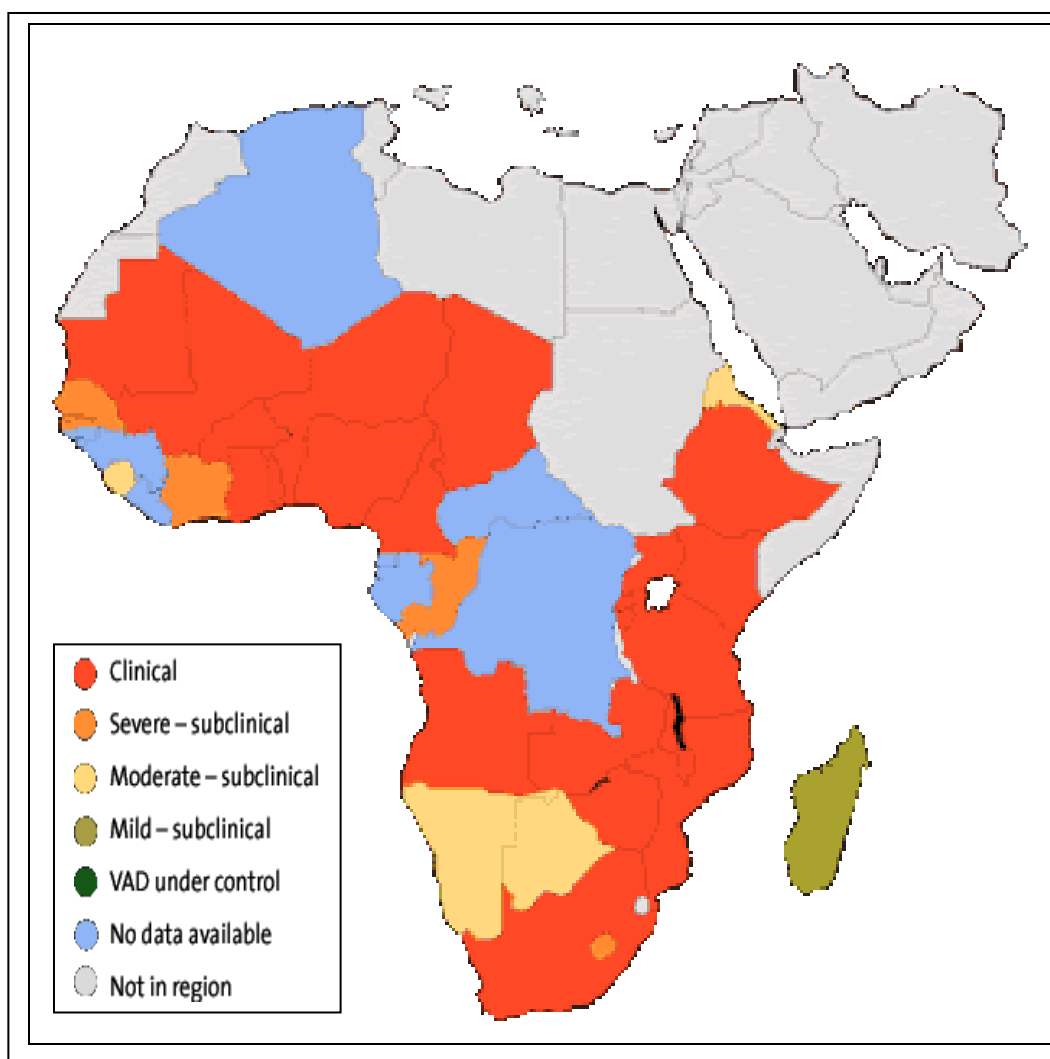
### **6.12.3 Prevalence of VAD**

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 <sup>525-530, 532-536</sup>.

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 (Figure 6-2), 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 <sup>437</sup>. 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 <sup>154</sup>.

Figure 6-3 shows the distribution of VAD in Latin America.



**Figure 6-2: VAD in Africa**

Adopted from WHO Website - Vaccines, Immunization and Biologicals Website. Vitamin A. Prevalence Maps 2/7. Africa Region <sup>525</sup>. (Accessed 25 September 2003). <http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv06.shtml>

#### 6.12.4 VAD in the MEC(Figure 6-4)

In the Eastern Mediterranean Region, VAD is present in some parts of the Arab world including Yemen, Egypt, Djibouti and Mauritania <sup>359-362</sup> and, more recently, Iraq <sup>554</sup>.

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

addition, Bitot's spots were found in 1.7%, corneal ulceration in 0.04% and corneal scars also in 0.04%.



**Figure 6-3: VAD in Latin America**

Adopted from WHO Website - Vaccines, Immunization and Biologicals Website. Vitamin A. Prevalence Maps 3/7. The Americas Region <sup>526</sup>.

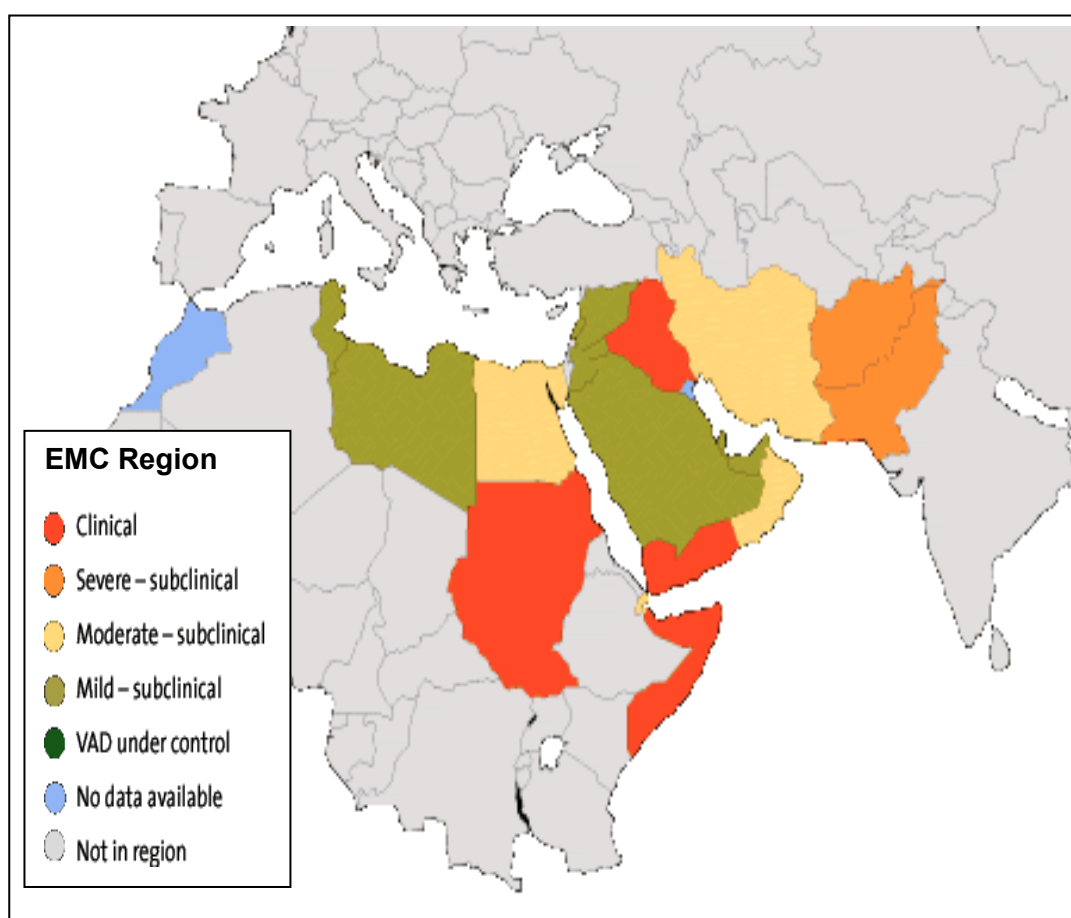
(Accessed 25 September 2003).

<http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv07.shtml>

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<sup>345</sup>. 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<sup>554</sup>.



**Figure 6-4: VAD in Eastern Mediterranean Region**

Adopted from WHO Website - Vaccines, Immunization and Biologicals Website. Vitamin A. (Accessed 25 September 2003).

Prevalence Maps 4/7. Eastern Mediterranean region<sup>526</sup>. <http://www.who.int/vaccines-diseases/en/vitamina/advocacy/adv08.shtml>

In Egypt VAD was also reported in the Beheira governorate in a study of 10,664 children<sup>359</sup>. 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 <sup>360</sup>.

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 <sup>362</sup>. Serious VAD has been periodically reported in the Republic of Djibouti <sup>361</sup> where large numbers of children, mostly in the rural area, had marginal vitamin A status and were exposed to a high level of risk <sup>361</sup>. Blindness from VAD was found in Djibouti in another study <sup>363</sup>. In the rest of the MEC, xerophthalmia has been reported in Afghani refugees in Pakistan <sup>515</sup>.

#### **6.12.5 Xerophthalmia**

Xerophthalmia, due to VAD, is still a major public health problem in many parts of the world especially in Africa (Figures 6-3, 4, 5). 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 <sup>437</sup>. 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 <sup>154</sup>.

#### **6.12.6 Xerophthalmia in the MEC**

In Egypt VAD was also reported in the Beheira governorate in a study of 10,664 children <sup>359</sup>. 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<sup>360</sup>. In addition to Yemen and Iraq, VAD existed in Mauritania<sup>362</sup> 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. In addition, serious VAD may periodically occur in the Republic of Djibouti<sup>361</sup> and blindness from VAD was reported in this country in another study<sup>363</sup>. Large numbers of children, mostly in the rural area, have a marginal vitamin A status and are exposed to a high level of risk<sup>361</sup>. In the rest of the MEC, xerophthalmia has been reported in Afghani refugees in Pakistan<sup>515</sup>.

#### **6.12.7 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<sup>241</sup>.

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<sup>586</sup>.

The synergism between xerophthalmia and diarrhoea, measles and upper respiratory infections, were statistically significant<sup>554</sup>. 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<sup>554</sup>. It was recommended that, in addition to measures such as immunisation against measles, breast-feeding, 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<sup>554</sup>.

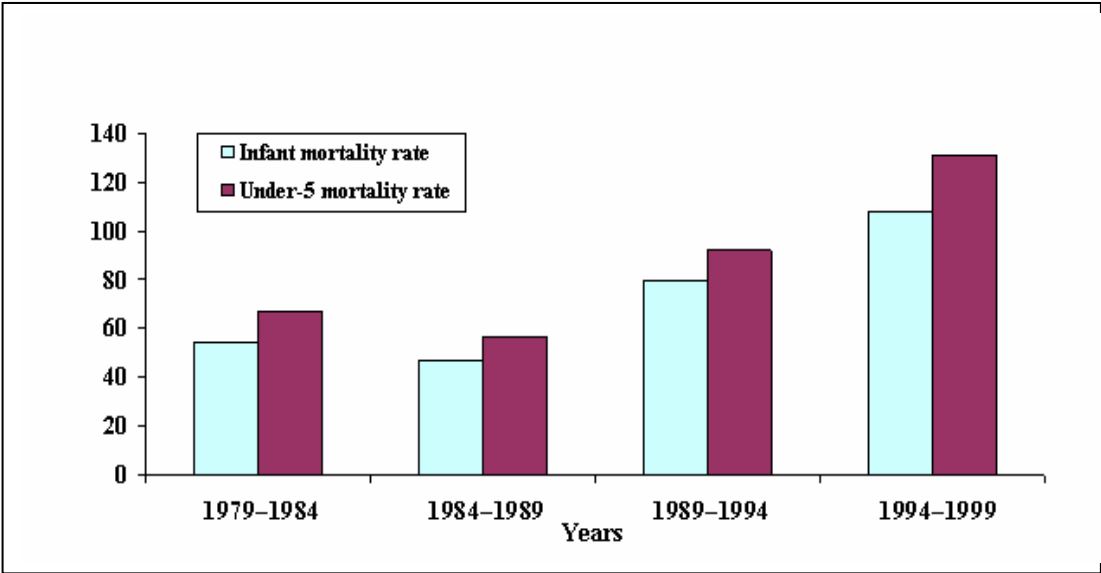
The synergism between xerophthalmia, diarrhoea and measles and upper respiratory tract infections was also demonstrated in this Iraqi study and was statistically significant. Among the 700 children in the sample population, xerophthalmia was present in 25.2% of the 481 who had diarrhoea; 60.2% of the 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<sup>554</sup>.

#### **6.12.8 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<sup>541, 544, 546, 558, 575, 711</sup>.

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 <sup>575</sup>.



**Figure 6-5: The increase of under 5 mortality in Iraq as a result of sanctions between 1991 to 2003**

Adopted from WHO Report: 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](http://www.who.int/disasters/repo/8451.doc).  
 (Updated 23 December 2003)

The effect of sanction on Iraq is well depicted by the increase of under 5 mortality rate and recent reports on the prevalence of VAD in the country <sup>558</sup>. (Figure 6-5, and Table 3-7).

## IMPORTANT BLINDING INFECTIONS

### 6.13 Trachoma

#### 6.13.1 Introduction

Trachoma<sup>84, 158, 232, 246, 251-252, 256, 289, 297, 320-321, 324, 336-338, 344, 354, 357-358, 383-384, 388, 390-392, 397-399, 401, 403-410, 416, 421-423, 426, 431-436, 489-490, 516-519, 570, 576, 589-590</sup>,

is the most common infectious cause of blindness. It has been recognised since antiquity and the first effective therapeutic modality, using copper sulphate sticks, was described by the early Egyptians thousands of years ago.

#### 6.13.2 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<sup>407</sup>. However, there is still uncertainty about how trachoma is transmitted<sup>405</sup> as *Chlamydia* is detected on only 0.5% of face flies<sup>407</sup>.

#### 6.13.3 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<sup>320</sup>. 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<sup>391</sup>. 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 <sup>409</sup>.

#### **6.13.4 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 <sup>253, 320</sup>. It is estimated there are currently about 6 million people irreversibly blind from the disease making up 15% of the world's blind <sup>574</sup>, and another 10 millions 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 <sup>570</sup>. There were 400 million cases worldwide in 1959, a very high number for the size of the world population at that time <sup>574</sup>. (Figure 6-6)

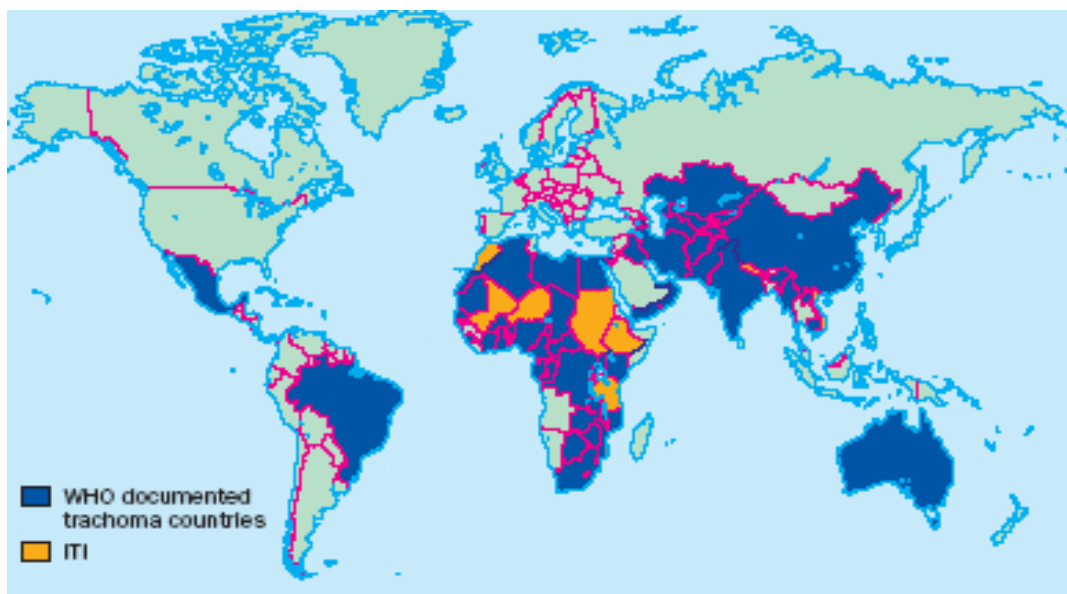
#### **6.13.5 Epidemiological Aspects and Trends of Trachoma**

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 <sup>334, 387</sup> where blinding disease is found. 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 <sup>132-133, 234, 304, 307, 408</sup>, 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. 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. Potentially blinding eyelid deformities, 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 <sup>408</sup>.

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 <sup>390</sup>.



**Figure 6-6: Global distribution of trachoma and ITI supported country programmes**

Adapted from <http://www.who.int/pbd/trachoma/img/world%20trachoma%202.jpg>  
(Accessed 25 September 2003)

- (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 <sup>402</sup>



and Gambia<sup>399</sup>. In the first two, active trachoma has diminished by over 50% and trichomatous 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<sup>399</sup>.

Improvement of economical conditions can also lead to a reduction in the prevalence of trachoma as seen in Western Nepal<sup>401, 453</sup>.

- (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<sup>408</sup>. 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<sup>401</sup>.
- (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<sup>357</sup>. It is also well established that trachoma occurs in episodes of repeated infections<sup>246</sup>.

### 6.13.6 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<sup>582, 388, 396, 723</sup> (Table 6-2).

Azithromycin, a macrolide belonging to the azide subclass<sup>393</sup>, is the antibiotic of choice in endemic areas as prolonged high levels are maintained in the conjunctival tissue following a single oral dose<sup>397</sup>. 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<sup>388, 410</sup>.

Medications used	Dosage
<b>Azithromycin</b> capsules, 250 mg or 500 mg	<u>Adults:</u> 1 g orally, in a single dose
Or <b>Doxicycline</b> capsules or tablet, 100 mg (hydrochloride)	<u>Adults:</u> 100 mg orally twice daily for 7 days
Or <b>Tetracycline</b> eye ointment, 1% hydrochloride)	<u>Adults:</u> Application twice daily for 6 weeks

**Table 6-3: WHO recommended trachoma medications<sup>582</sup>**

WHO Essential Medicines Library – EMLib. Trachoma - chlamydial infection. (Accessed 25 September 2003).

[http://mednet3.who.int/eml/disease\\_factsheet.asp?diseaseId=349](http://mednet3.who.int/eml/disease_factsheet.asp?diseaseId=349)

Considering the rate at which ocular chlamydial infection returns to a community after mass treatment, the elimination of infection in hyperendemic 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<sup>388, 396</sup> and should be restricted to children with severe or moderate intensity disease who should be monitored carefully for adverse reactions<sup>389</sup>.

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<sup>590</sup>. Frick et al found that mass treatment was as effective 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<sup>606</sup>.

On the economics of trachoma and its control, Frick et al<sup>398</sup> addressed the subject and suggested that: (1) trichiasis without visual impairment may result in an economic burden comparable to trichomatous 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.

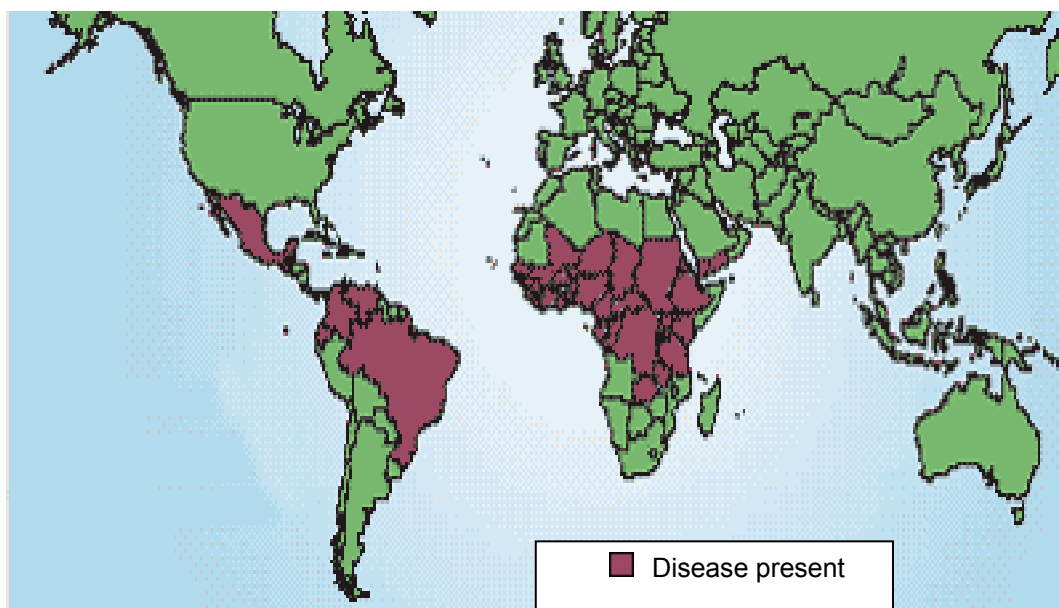
## **6.14 Onchocerciasis**

### **6.14.1 Prevalence of Onchocerciasis**

Onchocerciasis or River Blindness<sup>169, 247, 253, 277, 317, 346, 371-379, 414, 419-420, 444, 488, 491-514, 520</sup> 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, 6 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.

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 <sup>491</sup>. (Table 6-7).



**Figure 6-7: Global distribution of onchocerciasis** <sup>520</sup>

Adopted from WHO website. (Updated December 2003)

<http://www.who.int/tdr/dw/oncho2003.htm>

#### **6.14.2 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.

#### **6.14.3 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

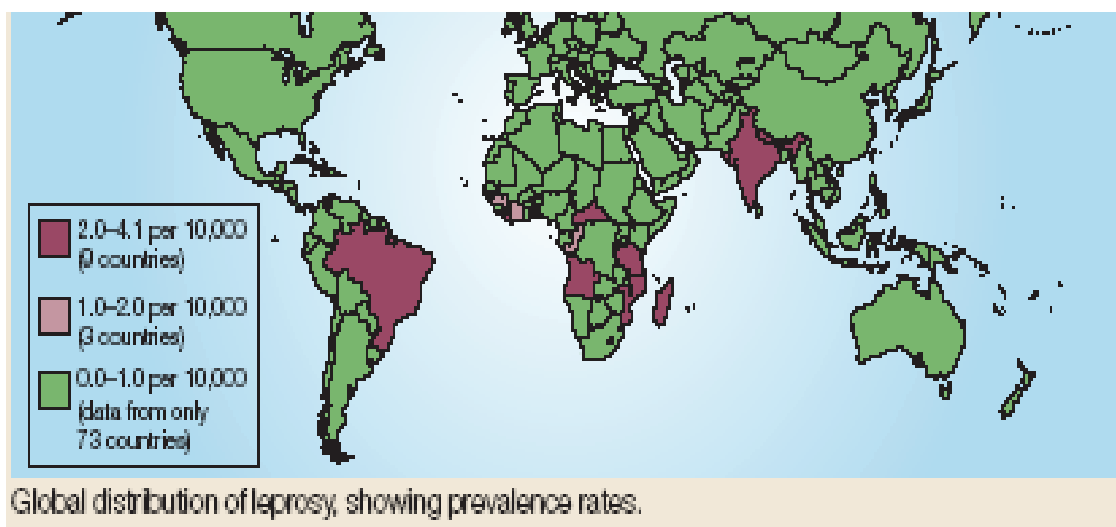
that onchocerciasis had reverted to the situation before the occurrence of ocular complications as a result of the ongoing control measures, despite the insect destruction and mass therapy not always being complete. This was thought to be sufficient to prevent blindness even if onchocerciasis is not cured<sup>443</sup>. 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<sup>522</sup>.

It is important to note the work of Murdoch et al who drew attention to the disability caused by visual fields contraction in onchocerciasis<sup>446</sup> in their large study of visual field loss in 6831 individuals aged 5 years and over in Kaduna State, northern Nigeria<sup>446</sup> which is mesoendemic for savannah onchocerciasis. 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<sup>446</sup>.

### **6.15 Leprosy**

Leprosy<sup>616, 620</sup> 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<sup>616, 620</sup>.

There are still some 10 million people with leprosy, over a half of whom live in the Indian subcontinent and Myanmar (Previously Burma) <sup>616</sup>. 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 <sup>619</sup> (Figure 6-8). 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 <sup>620</sup>. In the Arab world, reports on leprosy dates back to 1970 and 1980s <sup>617, 618</sup>, with no recent reports on the disease.



**Figure 6-8: Global distribution of Leprosy**

Adopted from Leprosy, *Nature Reviews* <sup>620</sup>

The main causes of blindness in leprosy are corneal disease, chronic uveitis and age-related cataract <sup>616</sup>.

## 6.16 Ophthalmia Neonatorum

### 6.16.1 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. gonorrhoeae*. 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. gonorrhoeae*. The predominant pathogens are bacterial and fungal infection<sup>608</sup>. In developed countries, the prevalence of gonococcal O/N ranged between 0.04/1000 live births in Belgium and the Netherlands, to 0.3/1000 in the USA<sup>602, 607</sup>.

Gonococcal O/N 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<sup>602, 607</sup>.

### **6.16.2 Causative Factors**

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

### **6.16.3 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 O/N<sup>602, 607</sup>. The second approach, of treating infection in pregnant women, can only be carried out in places where medical care is well organised 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<sup>607</sup>.

## 6.17 AIDS

HIV/AIDS<sup>622</sup> affects every country in the world and in many infection rates are increasing rapidly. 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 globalisation, 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<sup>603-604</sup>. 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<sup>598</sup>.