fact that reaginic antibodies (IgE) are particularly produced in helminthic infections and become fixed on mast cells in the capillaries of the skin and mucosa. There they react with reagin, and histamine is liberated, immediately producing a wheal.

Considerable trouble has been taken to standardize the skin test. We find it best to use the antigen in high dilution, in which state our evidence indicates that cross-reactions with other helminthic infections are rare but that sensitivity is retained (Woodruff & Thacker 1964, Woodruff 1970).

In the fluorescent antibody test which we have developed we use second stage larvæ of *T. canis* and the results correlate very well with those of the skin test. Thus, in the 40 patients with toxocariasis to which reference has just been made, the skin test was positive in 38 and negative in 2, in whom, however, the fluorescent antibody test was positive. Among these 40 the fluorescent antibody test was carried out in 29 and was positive in 21.

In those with clinical manifestations suggestive of ophthalmic toxocariasis a positive skin test and/or fluorescent antibody test, along with eosinophilia, make a diagnosis of a toxocaral etiology highly probable.

Management: There is evidence from both laboratory animals and man (Wiseman *et al.* 1971) that treatment with diethylcarbamazine cuts short the infection. Indeed, of 19 patients with ophthalmic toxocariasis who have been treated with diethylcarbamazine in our unit and followed up for periods of up to ten years, none has developed further lesions elsewhere, nor has the original lesion progressed once the initial period following treatment has been passed.

Epidemiology and pathogenesis of toxocariasis: Some of the most fascinating questions concerning toxocariasis relate to its prevalence and pathogenicity. When we first became interested in the subject we had to go back in the literature for forty years before we could find a reference to a prevalence study of the infection carried out in British dogs. We have now examined well over 1000 dogs and cats in and around London and the overall prevalence rate among them is around 15%. It is suggested that from this large reservoir infection is likely to be contracted by man with greater frequency than indicated by the relatively small number of cases of toxocariasis in which an eye is involved. It was largely to explore this prevalence and to study the pathogenesis of the infection that we worked on the skin and fluorescent antibody tests, and we have found that toxocariasis has implications which spread considerably beyond the field of ophthalmology. The larvæ, migrating as they do from the lumen of the bowel into many tissues, can cause a wide variety of clinical conditions and are almost certainly also capable of transmitting or predisposing to heavy infection with organisms present in the bowel (Woodruff 1970), including, from time to time, poliomyelitis. The granulomata which the larvæ produce in the brain may also account for some cases of epilepsy (Woodruff *et al.* 1966).

Among the important pieces of information which have recently been uncovered with the assistance of Dr O Borg, who is working in the Department of Clinical Tropical Medicine, is the fact that the infection may be transmitted without direct contact with infected dogs or cats. Samples of soil recovered from public parks in Britain have been examined and a considerable number of these contain *T. canis* ova. Thus a history of close contact with a dog is not essential in the diagnosis of toxocariasis. The public health implications of an infection which can be so widespread are considerable. I have recently heard from New York that, in the light of work which has been done in our Unit, it was decided to set up an organization which has taken the colourful name 'Children Before Dogs'. One of the recent successes of this organization has been to win a high court action in New Jersey, where the court has ruled that owners of dogs must now 'pick up after their animals'. I believe that in the interests of ophthalmic health similar legislation is now overdue in this country.

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Kerato-uveal Changes in Leprosy and Onchocerciasis: A Question of Immunity

Most of my work over the past twenty years at the Hospital for Tropical Diseases, London, has been connected with the ocular complications of leprosy and filarial disease, especially onchocerciasis, and I wish to develop, even if I cannot absolutely prove, the concept that these ocular changes are conditioned by the immunological state of the patient or population at risk. Table 1 suggests that leprosy and onchocerciasis are

Table 1
World blindness, 1971(possible total = 20 million)

	Condition		
	Trachoma	*Leprosy*	*Onchocerciasis*
Total affected	100–400 million	16 million	30–50 million
Total blind	3% or 3–12 million	5% or 800 000	1% or 300 000–500 000

important causative factors in world blindness. So far as the United Kingdom is concerned, since leprosy has an incubation period of from two to seven years, it would seem that we are assured of a steady supply of new cases in years to come so long as the present level of immigration from endemic areas continues. Other major causes of blindness involving millions of disabled persons include cataract and malnutrition.

LEPROSY

The key to the understanding of clinical leprosy, including the ocular complications, is to realize that its manifestations depend on the immunological resistance demonstrated by the individual patient, as shown by Fig 1 (*after* Jopling 1971). At one end of the spectrum of resistance is lepromatous leprosy, which includes most of the serious eye complications. Here the infected person has a low resistance and enormous numbers of leprosy bacilli develop, the nerves, skin, eyes, reticuloendothelial system (this includes lymph glands and specialized cells of liver, spleen and bone marrow), mucosa of mouth, nose, pharynx, larynx and trachea and the testes bearing the brunt of this infection. At the other end of the spectrum is tuberculoid leprosy in persons with a good resistance. Here the Schwann cells which engulf intraneural bacilli become fixed epithelioid cells and, in due course, groups of epithelioid cells become giant cells. Bacilli are destroyed in the process. Here we have cell-mediated immunity at work. In such a patient no skin lesions are present and we are dealing with pure neural tuberculoid leprosy. The affected nerve or nerves become thickened and, as this takes place where the temperature is coolest, nerves nearest the surface of the body bear the brunt and can easily be palpated. Various intermediate forms are present, borderline lepromatous, middle borderline and borderline tuberculoid, and the classification of any given variety can change from time to time according to the degree of resistance or immunity within this middle zone, but it is very rare for the two polar types of lepromatous and tuberculoid leprosy to change places.

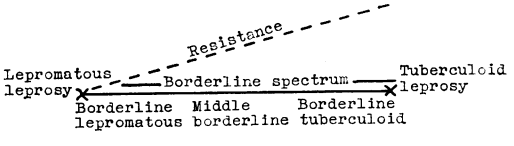

Fig 1 *Spectrum of immunological resistance in leprosy*

Cause of Blindness

The eyes may become involved in leprosy in three ways: as a complication of involvement of the facial and occasionally the trigeminal nerve(s) in both lepromatous and tuberculoid leprosy; by invasion of the eyeball by large numbers of acid-fast bacilli in lepromatous leprosy; and by participation in the generalized allergic reaction, known as the reactive phase. It is curious that the eyeball is rarely, if ever, involved by direct spread from neighbouring lepromatous lesions. Bilateral symmetry of the eye lesions is common.

Assuming that leprosy is acting on its own it may cause blindness in the following manner:

(1) Chronic insidious iridocyclitis, due to destruction of the ciliary body by leprosy bacilli, which leads to gradual failure of ocular physiology, resulting in complicated cataract and phthisis bulbi.
(2) Neglected cases of lagophthalmos, due to involvement of the VII nerve, which may or may not be associated with corneal anæsthesia due to the involvement of the ophthalmic division of the V nerve. This combination will cause exposure and/or neuroparalytic keratitis, with the attendant danger of perforation of the eye, which will be brought forward by secondary bacterial or viral infection.
(3) Leprous keratitis may cause some interference with vision, but this is not usually serious unless the corneal deposits are very substantial, as occurs with the development of sclerosing keratitis late in the disease.
(4) A smaller group due to acute plastic iridocyclitis with or without a secondary rise in intraocular pressure (secondary glaucoma) as part of the lepra reaction. If untreated, severe damage to the sight can be caused in 24–72 hours.
(5) A still smaller group in which blindness is due, not directly to leprosy, but to the presence of intercurrent eye disease, such as senile cataract and chronic open-angle glaucoma.

Incidence of Blindness Due to Leprosy

The most serious ocular complications are due to lepromatous or borderline lepromatous cases of leprosy and it is generally accepted that the proportion of these two groups to tuberculoid cases, in which the incidence of blindness is less, varies from country to country and continent to continent. Generally speaking, the further east one goes the higher the incidence of lepromatous cases.

A survey in October 1969 at the Utale Leprosarium, Malawi, Central Africa (Choyce 1970)

Table 2

Incidence of leprous keratitis and leprous iridocyclitis at the Utale Leprosarium, Malawi, in 1969

Type of leprosy	*Percentage with leprous keratitis*	*Percentage with leprous iridocyclitis*
Lepromatous	6	20
Borderline	14	28
Tuberculoid	–	–

Table 3

Incidence of blindness at the Utale Leprosarium, Malawi, 1969

Type of leprosy	*Percentage blind in one eye*	*Percentage blind in both eyes*
Lepromatous	6	7.5
Borderline lepromatous	–	–
Tuberculoid	3	3
Other causes	1	1.5
Total	3	3

gives the typical findings for Africa. The 344 patients surveyed comprised 112 with lepromatous leprosy, 7 borderline lepromatous, 104 tuberculoid aged over 10 years, and 121 tuberculoid aged under 10. Twenty-one per cent had leprous ocular disease, or 32% if the 121 children with unaffected eyes are excluded from the total. Leprous keratitis and leprous iridocyclitis were seen only in lepromatous and borderline lepromatous cases (Table 2). Lagophthalmos and exposure keratitis were seen in both lepromatous (6%) and tuberculoid (30%) cases. The actual incidence of blindness in one and both eyes is given in Table 3, which shows the total to be 3% in both cases.

In the Far East, Hobbs (Hobbs & Choyce 1971) has recently undertaken a similar survey in the Sungei Buloh Leprosarium in Malaysia. Hobbs examined 507 patients, 297 male and 210 female. The incidence of ocular lesions of all types (pterygium, senile cataract, primary glaucoma and leprotic lesions) was 32.5%, of which 50% were leprotic eye lesions (lagophthalmos, exposure and intrinsic keratitis, corneal leproma, iridocyclitis and its complications). Fifty per cent of the leprotic eye lesions were due to iritis (56% in males, 44% in females). Thirty-six patients were blind (7.1% of the sample); of these, 18 (50%) were blind from leprotic lesions, and 11 of these (61%) from leprotic iritis.

The important statistic here is that 7.1% of the sample were blind. The well-known association of lepromatous leprosy with iritis was confirmed – 35 out of a total of 38 cases of iritis (90%) arose in lepromatous patients. No doubt the higher incidence of blindness in Hobbs's series (7.1%) was due to the higher incidence of lepromatous and borderline lepromatous cases in Malaysia.

We are now in a position to estimate the actual amount of leprosy blindness in the world today. Assuming 16 million leprosy sufferers and a median figure of 5% for the incidence of blindness, we arrive at a grand total of 800 000, which makes it a major cause of blindness in the world today alongside such well-known scourges as malnutrition, trachoma, onchocerciasis, &c. All the published surveys confirm that the incidence of blindness tends to increase with age, e.g. from 4.9% in the 30–39 age group to 11.7% in the 70+ age group in Hobbs's series.

Prevention of Blindness Due to Leprosy

The basic factors in the prevention of leprosy have been well summarized by Jopling (1971) as follows: (1) case-finding and prompt treatment of all cases found; (2) keeping the families of patients under surveillance and giving prophylactic dapsone where it is considered advisable and can be properly supervised; (3) giving BCG vaccination to all infants newly born into leprous families; (4) improvement in living conditions, especially housing, in the endemic regions so that members of families do not have to live in close contact; and (5) education and propaganda about leprosy.

On the basis that tuberculosis provides cross-immunity to leprosy, BCG vaccination has been advocated as a public health measure against leprosy, and many trials involving children have been carried out. The majority have given encouraging results, such as the large scale ones in East Africa, but a recent trial in Burma has proved disappointing.

Lagophthalmos leading to exposure keratitis: This can readily be brought under control by timely tarsorrhaphy involving the outer third or half of the lid margins. Paramedical personnel can and should be trained in the indications for and the technique of this essential measure.

Recognition and treatment of the chronic insidious iridocyclitis: A great advantage is the availability of lightweight corneal microscopes which can be run off the batteries of a land-rover and therefore taken into the bush if necessary, thus enabling the bound-down pupil of chronic iridocyclitis to be identified before the onset of blinding sequelæ. For those pupils which will not dilate with mydriatic drops, sector iridectomy has been shown to be highly beneficial. Unfortunately there is a lack of competent eye surgeons to carry out this important procedure and the same lack hinders the proper management of the less common cases of acute iridocyclitis associated with a secondary rise of intraocular pressure complicating the lepra reaction.

Two observations may be made: (1) Prevention should be aimed at reducing the incidence of leprosy as a whole and at endeavouring to alter the immunological state of susceptible persons so that if leprosy develops it takes the tuberculoid

form, which is associated with a lower incidence of blinding lesions, rather than the lepromatous form. (2) Regarding the management of lagophthalmos and chronic iritis, in the absence of trained ophthalmologists much good preventive and therapeutic work can and should be done by leprologists without formal ophthalmological training and by paramedical workers.

ONCHOCERCIASIS

In Africa filaria *Loa loa* and filaria *Onchocerca volvulus* often coexist, but loiasis is confined to Africa because the intermediate host (chrysops) is not found outside that continent, and microfilaria *Loa loa* is blood-borne and as such has been found to cause neurological and choroidoretinal vascular disorders, including meningoencephalitis (which may be fatal) and occlusions of the retinal arteries and veins. Microfilaria *O. volvulus*, however, is never found in the blood. This coexistence can lead to confusion in the interpretation of choroidoretinal and optic nerve lesions found in individual patients.

History

In 1875 the Royal Naval surgeon O'Neil identified microfilaria *O. volvulus* as causing a chronic irritative skin eruption in West Africa. The connexion between the nodes or onchocercomata and the chronic blinding kerato-iridocyclitis was established in Guatemala in 1915 by the work of Pacheco-Luna and Robles (Pacheco-Luna 1918). They also saw the connexion between the nodules and erysipelas of the coast (mal morado) and rightly attributed this to a state of anaphylactic shock induced by the sudden death of millions of microfilariæ – an aspect of the disease almost totally absent from the African variety. However, it was not until the slit-lamp became available that Torroella (1931) saw the microfilariæ in the aqueous humour. In 1932 this physical sign was confirmed by Hissette in the Belgian Congo.

It may well be asked what connexion there is between the African and Central American versions of the disease. If it had arisen spontaneously in Central America one would have expected to find references to it in the history and folklore of preconquest Central America, but this is not the case. Shortly after the capitulation of 1524 slave labour was introduced in Central America, much of it from Africa, but it now seems probable that the disease described by Pacheco-Luna in 1918 had in fact been incubating for no more than fifty to sixty years. Torroella (the son of the original observer of microfilariæ in the aqueous) & Carmen Portillo (1971) have shown that in early January 1862 the 2nd Battalion of the Sudanese Rifles was transported from French Sudan to Nantes (France–Loire) and from there to Mexico, where it arrived on August 23 of the same year, at a time when British, French and Spanish contingents were being sent to Mexico to safeguard the lives and property of their nationals during international unrest. In 1863 the French installed Maximilian of Austria as Emperor of Mexico and in 1967 he was forced to abdicate and was shot, which was the end of the European role in Mexican affairs. During those five years some of the Sudanese soldiers (who came from the Bahr-el-Ghazal area of the Sudan) were in contact, for instance in Chiapas, with various species of simulium, some of which, notably *S. ochraceum*, could act as onchocercal vectors. This provides a most interesting example of the influence of politics on the spread of disease.

Clinical Ocular Findings in Onchocerciasis

So far as is known, adult *O. volvulus* is harmless, alive or dead. Living microfilariæ, however, which have a sucker at one extremity, are apt to become adherent to iris, ciliary processes or Descemet's membrane, thus causing localized but cumulative

Table 4

Comparison of onchocerciasis in Central America, Africa and the British Isles

	Central America	*Africa*	*British Isles*
Erysipela de la costa	++	?Nil	Nil
Intensity of infection:			
General	++	+++	+
Periocular	+++	++	+
Location of nodules	Two-thirds above waist	Two-thirds below waist	Two-thirds below waist
Total number infected	200 000	30–50 million	2000
Total number blind	2000 (1%)	300 000–500 000 (1%)	None
Age of blind patients	Usually over 50	Children and adults	–
Main cause of onchocercal blindness	Anterior segment	Anterior and posterior segment, the ratio varying from place to place	–
Treatment	Nodulectomy	Control of simulium	Nodulectomy, filaricidal drugs
Other filarial diseases	Nil	Common	Common
Nutrition	Generally good. Some lack of vitamin A and animal protein	Variable, may be deficient in vitamin A, vitamin B complex and animal protein, especially in savannah regions	Excellent

Table 5

Ocular onchocerciasis in Central America, Africa and the British Isles: anterior and posterior segment lesions

	Central America	*Africa*		*British Isles*
		Savannah	*Forest*	
Anterior segment lesions				
Trachoma	Rare	+ +	Rare	Rare
Conjunctival lesions	+	+ +	+	Slight injection
Keratitis	+ +	+ + +	+	+
Iridocyclitis	+ + +	+ +	+	+
Posterior segment lesions				
Primary optic atrophy	Not seen	+ +	+	Not seen
Secondary optic atrophy	Possibly +	+ +	+	Not seen
Postinflammatory choroidoiretnitis:				
Anterior	Probably +	+ +	+	Not seen
Posterior	+	+ +	+	Not seen
Posterior degenerative lesions	Few and ill-defined	+ +	+	Not seen

mechanical damage; when dead they excite an exudative and cellular reaction of varying intensity, increasing the trauma to the ocular tissues. Choyce (1964) summarized the ocular findings in 800 onchocercal cases at the Hospital for Tropical Diseases, London, 1952–63. Of these 800 cases 48% had onchokeratitis, 8% had microfilariæ in the anterior chamber, and 3% had mild anterior uveitis. There were no cases of posterior uveitis, choroidoretinal degenerations or loss of vision. In the period 1964–71, cases at the Hospital for Tropical Diseases have been less numerous, but a higher proportion have been those exposed to the disease since birth who have since come to the United Kingdom for educational, business or other purposes. This may explain why the incidence of onchokeratitis has declined while the incidence of pretreatment microfilariæ in the anterior chamber has increased (48% to 35% and 8% to 10% respectively).

I have paid a number of visits to the Central American and African areas where onchocerciasis is endemic and am therefore in a position to compare the findings, which I have attempted to do in Table 4. Table 5 compares the anterior and posterior segment lesions in the three localities.

Clinical impressions may count for little, but some of mine are:

(1) The remarkable manner in which numerous microfilariæ can circulate in the aqueous of African eyes which are normal in every other respect. In Central American and European eyes the microfilariæ, when seen, are always accompanied by a greater or lesser degree of onchokeratitis and iridocyclitis. We are still not absolutely certain how the microfilariæ get into the anterior chamber. Almost certainly they do so by their own motility at the limbus, but this process has not yet been actually observed. Tagging the living microfilariæ to render them visible seems to be the answer and I am assured by M E Langham (1971, personal communication) that he is well on the way to solving this problem. Painstaking observation has established that once inside the anterior chamber the microfilariæ can live for eight to nine months. The administration of 50 mg diethylcarbamazine by mouth clears the anterior chamber of them within twenty-four hours, producing a severe iritic reaction if they were very numerous. Is some naturally-occurring immune reaction the cause of both mal morado and the intracameral death of microfilariæ in Central American patients, these reactions not being present in affected Africans?

(2) The severity of the Central American keratoiridocyclitis with its typical down-drawn pinpoint pupil.

(3) The comparative immunity of rain-forest onchocercal Africans and expatriate Europeans to fundus lesions, compared with the presence of some in the Central American patients and more in the African savannah patients.

DISCUSSION

How can one bring together in one workable hypothesis the considerable variations in behaviour listed above? I believe it to be a matter of immunity, both individual and racial. Fig 2 attempts to illustrate that onchocerciasis can be interpreted on an immunological basis, as shown in the first section of this paper concerning leprosy. At one pole is the apparent immunity noted in rain-forest Africans to onchocerciasis and at the other the greatly heightened reactivity observed in Central Americans. Torroella's work quoted above, pinpointing the introduction of the disease not much more than 100 years ago, would indeed fit in with the likely effect of a fresh disease on a susceptible

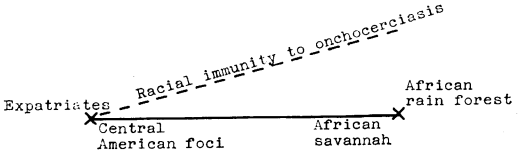

Fig 2 *Spectrum of immunity to onchocerciasis*

population, in contrast to millions of Africans who have built up a racial immunity to the same disease over thousands of years. One suspects that the true explanation of the apparently different behaviour in the African savannah is related to other factors, especially nutrition. It is not easy accurately to fit the expatriate European cases into this scheme because their infections are so minuscule compared with the African and Central American groups. But again, I have the impression that, if anything, they are the most susceptible of all, and at the Hospital for Tropical Diseases I have seen numerous patients with very well marked onchokeratitis who had only been exposed to infected simulia on one occasion several years ago. Some authorities consider that the intensity of the Central American disease is slowly lessening as racial immunity builds up, but it could be, as suggested by Torroella & Carmen Portillo (1971), that its novelty is just wearing off.

Acknowledgments: I am indebted to Mr H E Hobbs, for permission to quote his findings at Sungei Buloh, and to Professor A W Woodruff for the constant stimulation which he has provided, both in London and when we have been together in Africa and Central America.

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Trachoma [*Summary*]

The organism responsible for trachoma is an atypical virus of the genus *Chlamydia* (*C. trachomatis*) or TRIC agent (trachoma inclusion conjunctivitis agent). The organism is of the PLT group (psittacosis, lymphogranuloma venereum, trachoma), midway between true viruses and bacteria. They are filtrable yet divide by binary fission, have both DNA and RNA, a cell wall with muramic acid as do bacteria, and a complexity of structure approaching that of bacteria.

Man, and perhaps his very near relatives the apes, may be the only natural hosts of lymphogranuloma venereum and TRIC agent. TRIC agent, however, affects very much more than just the conjunctival mucous membrane for it is found in the nose, throat and rectum, and is probably a major cause of vaginitis, cervicitis, salpingitis and undiagnosed pelvic disease in the female (Jones 1964, Dunlop *et al.* 1965). TRIC agent can be found in 44% of patients with nonspecific urethritis (Dunlop *et al.* 1971), a condition rapidly increasing in frequency and importance.

In the eye *C. trachomatis* gives rise to four clinical entities: trachoma, inclusion conjunctivitis, inclusion blennorrhœa of the newborn and TRIC agent keratoconjunctivitis.

Trachoma

In trachoma-endemic countries the trachoma usually starts in childhood, indeed often at birth, but can occur at any age. The onset may be insidious but there is usually an initial period of about five to six days during which time the conjunctivitis is indistinguishable from a simple bacterial conjunctivitis. In hot dry climates the onset is frequently accompanied by a bacterial conjunctivitis often caused by *Hæmophilus aegyptius* (Koch-Weeks bacillus), which increases the discharge from the eye.

In 1908 MacCallan produced the classical description of the disease. He recognized four clinically distinguishable stages of the disease. Stage 4 lasts for a lifetime but the time in the other stages varies very considerably from as little as a few days to several months or even years.

Stage 1: A lymphoid hyperplasia of all the conjunctiva manifests itself as a diffuse hyperæmia, papillary hypertrophy and follicle formation. Vessels start to grow into the cornea usually from above (pannus), preceded or accompanied by a diffuse punctate keratitis.

Stage 2: The follicles which are particularly noticeable on the upper tarsal conjunctiva age and become necrotic in the centre. Follicles also occur in both fornices, on the plica and on the lower tarsus. Indeed, the first signs of the disease are often to be found in the upper fornix.

Stage 3: The earliest manifestation is the appearance of star-shaped scars among the fleshy follicles.

Stage 4: The conjunctiva is healed and no inflammatory signs are found but conjunctival scarring turns the lashes inwards. The lashes then rub on the cornea giving rise to so much of the trouble seen in the cornea in trachoma.

Corneal Changes

The changes in the cornea are as characteristic as those in the conjunctiva. The first change in the