Notes

Onchocerciasis in the Sudan *

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The present note is intended as a brief summary of the knowledge of onchocerciasis in the Sudan up to the present time.

Onchocerciasis in the Sudan was first reported by Bryant (1933, 1935). He was also the first to indicate that the endemic blindness that had been prevalent in the Bahr El Ghazal area was due to this disease. The disease in the Sudan had also been studied by several other workers, including McKelvie (1934, 1935), Cruickshank (1934, 1936), Kirk (1947, 1957), Woodman (1948, 1949, 1950), Bloss (1949), Lewis (1952, 1957), Satti,[a] Satti & Kirk (1957), Haseeb,[b] Morgan (1958) and Torroella Bueno.[c]

However, the first planned study of onchocerciasis in the Sudan was initiated in 1946 by Horgan (1947). The idea was to study the clinical and epidemiological aspects as well as the vector, its ecology and other relevant conditions, with a view to carrying out control of the disease combined with mass chemotherapy as well as control of, and possibly the eradication of, the vector. One of the aims of the study was to try to determine the etiology of the blindness, as there were many local factors that might be involved, especially malnutrition and fish poisons.

Distribution of the disease

The disease covers a wide area but in a patchy fashion, the heaviest incidence being in Bahr El Ghazal Province, where the disease had first been discovered and was later extensively studied. In their latest reports, Dr Torroella Bueno [c] and Dr Mohamed Sherif [d] mention a high percentage of various degrees of blindness (up to 20% among cases examined in Bahr El Ghazal Province), while very much lower figures were given in reports issued in the thirties and forties. There is evidence that the disease has increased in extent and that it has spread to new areas. The figures definitely show that there is an increase in the disease in Bahr El Ghazal—particularly in Wau where there were few infected cases in 1948-50. Hunt (quoted by Kirk, 1947) reckoned that the incidence of blindness is not more than 10% in Bahr El Ghazal. Besides Bahr El Ghazal, the disease is also found in Equatoria, Upper Nile, Blue Nile and Northern Provinces. There are no reports from the remaining four provinces of the Sudan. In Equatoria, according to Woodman (1949), the disease is known to exist in the area between and on the Bo and Sue rivers, the Mvolo region as well as sparsely in the Li Rangu area. In the course of a visit to Equatoria in 1958, one of us (M. H. S.) found cases in Meridi and a case within the precincts of Juba. This person was taken to Juba Hospital where investigation confirmed the infection and eye complications. Although there is always the possibility that such people might have become infected in the highly endemic centres, it is felt that extension is always possible if cases are imported to areas where the rivers are fast and rocky—i.e., conditions favourable for the breeding of the vector, *Simulium damnosum*. Spread of breeding Simuliidae to new areas is quite feasible during the rains, as the west of Equatoria is full of suitable streams. Over the Meridi River, a dam had been constructed to hold back water and the speed of water downstream of this reservoir had thereby been greatly accelerated; the staff working on the dam and water-pump complained bitterly of being attacked by Simuliidae. As far as we know, there were no cases of onchocerciasis in this locality, and it would be interesting to study the situation in the neighbourhood. A survey of this area might prove very fruitful. It is hoped that the team that has started work in the Sudan will have time at a later date to survey this area, as well as other suspected localities in Equatoria, and to give

* Note presented at the Second WHO Conference on Onchocerciasis, Brazzaville, June 1961.

[a] Satti, M. H. (1948) Unpublished report to the Director, Medical Services, Sudan.

[b] Haseeb, M. A. (1954) *Onchocerciasis in the Sudan* (unpublished working document WHO/Afr./Onch. Conf./10).

[c] Torroella Bueno, J. (1961) *Report on onchocerciasis control* (unpublished working document WHO/Afr./Onchocerciasis (1961)/25).

[d] Sherif, M. (1960) Unpublished report to the Director, Medical Services, Sudan.

a true picture of the status of onchocerciasis in this province. Moreover, almost nothing is known of the onchocerciasis situation on the borders of Equatoria with Ethiopia and Kenya. These frontiers are hilly and there are bound to be very many fast-flowing rivers. One of us (M. H. S.) has been informed [e] that the south-western borders of Ethiopia are infected with this disease. The area described is partly on the eastern border of Equatoria and partly east of Upper Nile Province, where it is already known that there is onchocerciasis.

In Upper Nile Province, onchocerciasis was seen in Juba Hospital from Pibor Post and Akobo (Pratt, quoted by Kirk et al., 1959). In 1949, however, Bloss described the disease as existing from Pibor Post to a point opposite the map reference Mellut. It must be pointed out that the area under consideration is either on the Ethiopian frontier or near it.

In October 1956, an area on the Yabus Bridge on the river of the same name was described by one of us and reported by Kirk et al. (1959). More cases are reported to be prevalent among the Koma south of this point, in Blue Nile Province. This area has yet to be properly surveyed for cases, eye complications, and the vector.

In Northern Province, a small focus has been described in the Abu Hamed area by Morgan (1958). No evidence of eye manifestations has been described to date. It should be recalled here that Lewis (1952) reported the prevalence of *Simulium damnosum* in this area. In Upper Nile, there are unconfirmed reports of the existence of onchocerciasis in the Kigli and Wadessa-Dago posts of Nasir District. This area is south of the Yabus Bridge area mentioned above, and is continuous southward with the Abobo-Pibor region referred to previously. It is also believed that the area between Yabus and Dago-Post is infected as well. Local missionaries report that there are many cases of blindness and epilepsy.

The vector

As far as we know, *Simulium damnosum* Theobald is the only vector of onchocerciasis in the Sudan. *Simulium naevei*, which is an important vector in other parts of Africa—namely, in Uganda and Kenya and in the Congo (Leopoldville)—has not been encountered in the Sudan (Lewis, 1959). According to Lewis, Simuliidae have been found in places where there is no onchocerciasis. This view is no longer tenable and we are now confronted with a situation where the reverse is the case—we know onchocerciasis exists but the vector has not yet been studied. This is true, for instance, of the infection on the eastern frontiers with Ethiopia. Here again, the missionaries stress the existence of Simuliidae, and the rapid flow of the water, combined with the fact that the stream-beds are very rocky, is very favourable for the breeding of these insects. A similar state of affairs exists with regard to the River Sobat, the Blue Nile upstream of Roseires, the rivers Rahad, Atbara and Dinder, as well as small rivers such as Khor Ahmer in the Fung and Setit in Kassala Province. All these rivers have their sources in Ethiopia and they run from the highlands to the plains in the Sudan with a very steep descent as they leave the Ethiopian plateau. Moreover, these areas are very hilly and therefore conditions are favourable for the breeding of Simuliidae. Further entomological work is needed here to establish the species of vector. In some of the areas near the Ethiopian frontiers, there are few or no human habitations and this may be the reason for the absence of onchocerciasis, but most of the northern areas along the rivers Dinder, Rahad, Atbara and Setit have very rich game reserves containing mammals for the flies to feed upon. Under the circumstances, one would have expected to find the vector but not the disease. This will be true for the present, but with economic development the situation will change and a different picture may then be expected. Confirmation of the possibility of importation of the disease along the rivers near the Ethiopian frontier comes from Lewis (1959), who says that the falls at Wad Arud on the River Atbara, where a few adult *Simulium damnosum* were found in November 1958, are in an uninhabited part of the Sudan.

Lewis has extensively studied the biology of the vector in the Sudan, but he feels that there are still very many unanswered problems. Qutubuddin [f] has been studying *Simulium damnosum* in the Abu Hamed area, but has found no infected flies so far. The plan for a pilot project for the eradication of the simuliidae in the Abu Hamad area on the Nile has been worked out. Besides breeding in waterfalls, rapids, cascades, rocky gorges and cataracts with well-aerated water, *S. damnosum* has also been found breeding in mud (Lewis, 1948). According to Austin (1909), the fly follows its victim for miles from the Nile and renders outdoor work impossible.

According to Lewis (1952), the range of flight of *S. damnosum* is 6-18 miles (or nearly 10-30 km) in the

[e] Y. Tsege—personal communication, 1961.

[f] Qutubuddin, M. (1960) Unpublished report to the Director, Medical Services, Sudan.

Sudan, while greater distances are recorded from other territories. Gibbins (1936) in Uganda recorded means by which the disease can be disseminated a long way away from rivers where the flies breed. Such long-distance dissemination is not in fact likely to occur, as it has been found—and corroborated by the observation of Lebied (1950)—that the thoracic stage of the microfilariae of *Onchocerca volvulus* causes impairment of flight in infected females. This, then, explains why the disease is limited to small foci. We believe that in the Southern Sudan the infection is very much localized, and that the few infected cases that we have found in villages away from breeding places are due to the people who go fishing in fly-infested rivers. We have elicited a history of fishing from most persons from such villages.

Clinical features

The principal clinical features of onchocerciasis in the Sudan are onchocercal nodules, eye manifestations sometimes progressing to blindness, various skin infections, lymphadenopathy and scrotal elephantiasis and sometimes hydrocele of the tunica vaginalis. Moreover, there are many cases combining onchocerciasis and epilepsy. The co-existence of these two has been too frequent to be dismissed as coincidental. Instances of giddiness have also been encountered; the cerebrospinal fluid of many of these persons has been examined but no microfilariae have been found. The question of epileptiform seizures and giddiness is of considerable interest and needs further study. Other patients present allergic manifestations, particularly asthma. Full descriptions of the clinical features of onchocerciasis in the Sudan have been given by various authors referred to above and there is no point in repeating them here.

Etiology of the blindness

The ocular manifestations of onchocerciasis have been described by Hissette (1932, 1938), Bryant (1933, 1935), McKelvie (1934, 1935, 1952), Ridley (1945) and many others. There is universal agreement, when speaking of the ocular manifestations, to divide the lesions into anterior and posterior manifestations, according to the part of the eye involved.

Anterior manifestations. These consist of infections of the anterior segment of the eye and present as keratitis, iritis, iridocyclitis with synechiae, nummular keratitis, and haze in the media of anterior and posterior chambers due to microfilariae and their debris. The keratitis may sometimes develop into corneal opacities with or without pannus. The opacities may be pigmented and cover the whole or part of the cornea. The synechiae may result in deformity of the pupil, which may be seclusive or occlusive, pear-shaped, circular, oval or irregular in outline. The deformity then becomes fixed and mydriatics can exert no action. These synechiae may be the cause of secondary glaucomatous changes. In some cases there is atrophy of the iris and there may be pigment deposits on its surface. There may be cataractous changes in the lens with pigment deposit on its surface. The lens was dislocated in certain cases and in one case it was completely absorbed. In some cases the lens was adherent to the posterior surface of the cornea.

There is unanimous agreement that these manifestations are due to onchocerciasis and, in fact, to the direct effect of microfilariae, as these were found on the cornea as well as in the mediae during operations on some of the persons suffering from onchocerciasis. These microfilariae were demonstrated by McKelvie during eye operations in 1948 in Bahr El Ghazal. They were also demonstrated in cases of keratitis, especially of the pigmented type.

Posterior manifestations. These mainly include the infections of the retina, optic nerve and choroid, and constitute what is known as retino-choroiditis, described in the Sudan by Bryant (1935), McKelvie (1935) and others and observed to occur with onchocerciasis. Of late, the etiology of these posterior manifestations have been the subject of great controversy. Thus, Choyce (1958) suggests three theories to explain these posterior lesions. First, that they represent a reaction to the presence of microfilariae. The difficulty with this theory is that living microfilariae do not seem to cause any reaction in the choroid, retina or optic nerve, and dead microfilariae cannot be demonstrated. Secondly, that the changes are due to a toxin liberated by microfilariae or adult worms at a distance, there being no agreement as to whether living or dead. The third theory is that these posterior segment lesions are independent of onchocerciasis, their presence being purely coincidental.

As regards the choroid retinal degenerations, Choyce (1958) says that two classical posterior segment lesions associated with onchocerciasis have been described in literature from Africa but not from that of Central and South America; one of these he called the " Ridley fundus ", a lesion looking like " a hypothetical combination of choroidal sclerosis with

retinitis pigmentosa " involving the macular area and the optic nerve which was secondarily atrophic. Nothing resembling a microfilaria, living or dead, was seen in these fundi. He then goes on to describe what he calls the " Bonjongo fundus " and argues that it is neither inflammatory nor toxic in origin. These fundi were responsible for 15% of the blindness in the Cameroons. He therefore considers that the condition was not due to onchocerciasis or to any parasitic disease or nutritional disturbance, but to an abiotrophy such as " retinitis pigmentosa ", or pigmentary degeneration of the retina as it is now known. This view has been strongly supported by Kershaw (1958, 1959) and strongly opposed by Budden (1958, 1959 and 1960). Kershaw (1959) states " as the eye-lesions are not related to the intensity of infection, they may:

" 1. not be related to infection at all, but due to a condition which is presented as an accidental association;

" 2. be related to infection in a qualitative sense;

" 3. be related to another basic condition which may be influenced by infection in a quantitative sense."

" This alternative explanation for posterior eye-lesions seen in those infected with onchocerciasis ", Kershaw maintains, is compatible with (1) and (3) and should therefore be studied by appropriate genetic and epidemiological methods.

Budden (1958) thinks that these lesions, or what Choyce (1958) calls the " Bonjongo fundus ", are due to onchocerciasis and are not genetic in any sense, although he agrees that some of them have marked similarity to familial abiotrophies in appearance.

General. In the Sudan, Bryant (1935) described the anterior and posterior manifestations in the eye and their association with onchocerciasis. McKelvie (1935) described also the changes that take place with onchocerciasis. Satti [a] in a report to the Director, Medical Services, described similar changes. He writes, " In a classical history, the blindness or defective vision starts with night-blindness which will progress to gradual dimness of vision during day, passing, sometimes, on to complete blindness. This outcome usually takes many years to develop. Some cases start with pain, lachrymacton photobia and then conjunctivitis and keratitis may develop terminating sometimes in a panophthalmitis and destruction of the eye. In certain cases, corneal scarring with or without pannus might develop ". He further states, " There are many cases with old standing nodules, while the vision is perfect and there are no abnormalities in the eye at all. If the eye is affected, the course of blindness is usually several years, if the posterior manifestations are alone in evidence; but the course may be comparatively short if both anterior and posterior infections are at work. This latter state of affairs is especially spectacular if the damage to the cornea is progressive, and is usually hastened by secondary infection. Most cases, but not every case, start with night-blindness. Night-blindness, sometimes moving shadows in front of the eyes, is therefore the danger signal of an impending late blindness, if not treated. The objective manifestations are either anterior or posterior." Previous workers in the Sudan described the posterior manifestations, among which were the full-blown pictures of what Choyce calls the " Ridley fundus " and " Bonjongo fundus ". These lesions had been particularly evident in persons who had been completely blind for a long time, and, as far as our observations go, had not been seen or described in the tribes in the Bahr El Ghazal except in association with onchocerciasis. As far as we know, there is not very much blindness among these tribes in the non-endemic areas. No family blindness of the nature described above and known as the " Sorsby fundus " or generalized choroidal sclerosis, etc., has been described in the general population, and Dr Baghir [g] informs us that in his experience of about 25 years' ophthalmic work, the occurrence of family eye disease is rare in the Sudan. In this connexion, Satti's (1959) comments in response to Kershaw (1959) at the Sixth International Congresses on Tropical Medicine and Malaria are relevant. " It is interesting to hear . . . that doubts are being cast on the etiology of the blindness and its relationship to onchocerciasis. We in the Sudan had entertained such doubts in 1948. Several factors were considered, amongst which were malnutrition and fish poisons. Fish poisons were thought to contain a rutin-like substance. All our results are so far negative. As to malnutrition, the status is the same in neighbouring localities where no blindness and no onchocerciasis are found.

" It is observed that the distribution of the blindness coincides with that of the disease, onchocerciasis, as well as with that of the vector (in the Sudan it is *Simulium damnosum*). As you have just heard from Dr Lewis, this fly is prevalent in the Abu Hamad reach of the Nile, but no onchocerciasis had

[g] I. Baghir—personal communication, 1961.

been discovered. In this connexion, it is to be pointed out that Professor Morgan last year [1957] found cases in this region. The eye manifestations in this part have yet to be sorted out, as trachoma also exists there...

" In the endemic area, the blindness decreases and so also the disease as you go away from rivers where the vector abounds."

It is important to comment that trachoma is not endemic in Bahr El Ghazal and so the eye lesions due to onchocerciasis are easier to identify.

However, as the fundal appearances of disputed etiology form a small percentage, it is considered that in the Sudan onchocerciasis is a cause of much and severe blindness and should be looked upon accordingly. From our experience we feel that the severity of infection is not necessarily linked with ocular manifestations, but the length of residence we consider important. It is also believed that we are in fact dealing with different strains of the worm and possibly different antigenic properties. Woodruff et al. (1958) noted the occurrence of ocular manifestations in Europeans with shorter residence as opposed to fewer such manifestations in the local population with longer residence; while others confirm that a long stay is an important factor in the causation of the blindness. In our opinion, these discrepancies can be explained, to a limited extent at least, by differences in strains. Individual host resistance and response, although important, do not seem to offer the full explanation.

Raper (1958) has brought up the point of the association of dwarfism, severe *Simulium* biting and onchocerciasis. This reminds us of the association of onchocerciasis, epilepsy and giddiness encountered in the endemic area in Bahr El Ghazal, where one of us (M. H. S.) performed lumbar punctures on some of the affected persons but failed to find microfilariae. No dwarfs had been seen among the tribes in the endemic areas. It is possible that microfilariae can gain access to any part of the body through the lymphatics. This takes us to what are the nutritional requirements and metabolic wastes of *Onchocerca volvulus* adult worms and their microfilariae. If we know these, we shall be able to understand many of the difficult and possibly controversial points as well. This will not be possible until some *in vitro* method of culture is devised for *O. volvulus*.

Treatment

Observations on the chemotherapy of onchocerciasis in the Sudan have been reported by Kirk (1950, 1952) and described in detail by Satti & Kirk (1957). The treatment dates from the finding of Van Hoof and his colleagues (1947) that suramin (Antrypol) has marked filaricidal activity on *Onchocerca volvulus*. Van Hoof kindly placed some of his observations and dosage at the time at our disposal and he remarked that very severe reactions, not without danger to life, might occur in the course of treatment with this drug. In fact, as a result of treatment with this drug in the doses recommended by him we had some fatalities in the Sudan. The experience in the Sudan with diethylcarbamazine (Hetrazan) is limited. In 1948, the drug was in very short supply for experimental purposes. Satti & Kirk tried some on a few patients, and although it caused the disappearance of microfilariae on about the third day of treatment it had no effect on the adult worm and in all patients microfilariae reappeared in a matter of a month.

The drawback of suramin is its severe toxicity. Satti & Kirk (1957) remarked that it " was found to be extremely toxic, sometimes even in small doses, and wide variations from case to case were observed. Some of the toxic reactions may have been due to a Herxheimer effect produced by the sudden destruction of the worms. These reactions were often so impressive and alarming that it was necessary to abandon the treatment." In the doses given by these workers, actual fatalities were reported, some of which were definitely ascribed to the direct toxicity of the drug, others being due to allergic reactions as a result of the death of the worms and their microfilariae. The doses given were very intensive, and though they were the cause of reactions, yet they were found to be extremely effective in sterilizing the infection among patients who were followed up. Those who had 4 g or more of suramin were found to be free from the infection several months later. The nodules usually swelled up, became hot and tender, and in a few that were opened up the worms were found dead and disintegrated and partially liquefied. The nodules were observed to disappear after treatment with suramin, but nothing of the sort occurred in the nodules on treatment with either diethylcarbamazine or antimony (used in the form of Pentostam).

A scheme of treatment has been suggested—and has, in fact, been tried in very few cases—involving the combined use of two drugs. In this scheme, a course of diethylcarbamazine for one week precedes treatment with suramin. This preliminary course reduces the microfilaria count, so that the severe

reactions due to the allergic effect caused by the massive death of these organisms are appreciably minimized. The reactions to which diethylcarbamazine may give rise are much reduced by the administration of antihistaminics, or, better, of cortisone (Markell & Turner, 1957). The diethylcarbamazine is given in daily doses of 3 mg/kg/body-weight in three divided doses. The suramin is given in weekly doses, starting with 0.25 g and increasing gradually to 1 g if there is no severe reaction; a course of 7 g or more is aimed at. In some cases, the diethylcarbamazine is continued, in spite of the concurrent administration of suramin, for three weeks; this is done where reactions to suramin are severe and that drug has to be given in a low dose. With these patients, as said before, a strong antihistaminic, such as promethazine or pyrilamine, is usually given.

Control

A scheme has been developed for mass treatment on the lines indicated above and for study of the eradication of *Simulium damnosum* in the many rivers in the Bahr El Ghazal area.

Mass treatment. Several schemes of treatment will be tried:

1. Denodulation alone.

2. Denodulation plus diethylcarbamazine administration.

3. Denodulation plus diethylcarbamazine administration, later to be followed by a short course of suramin for patients in whom nodules did not form or were very small and consequently missed.

4. Combined chemotherapy as described above but without denodulation.

5. Treatment with antimony compounds.

Triostam will be experimented with in our next field trials. Pentostam has already been tried, but without convincing results.

Vector control. According to Lewis (1952), the eradication of Simuliidae is not possible owing to the expense involved. It is to be pointed out that there are very many rivers in the endemic areas of the Sudan, including the Nile at the Abu Hamad reach. Of late, however, eradication has become a hopeful proposition since the work of Garnham & McMahon (1947), although the size of the rivers may be a handicap. A pilot project for vector control is due to start soon, with special reference to the Abu Hamad reach. Rivers in other endemic areas will be studied later.

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Essais de portée de deux insecticides utilisés contre les larves de *Simulium damnosum*, dans le Nord Dahomey

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Plusieurs essais d'éradication des simulies vectrices de l'onchocercose humaine ont été tentés en Afrique, notamment ceux de Garnham & McMahon,[a] au Kenya sur *Simulium neavei* Roubaud, de Wanson, Courtois & Lebied,[b] à Léopoldville, et du Service d'Hygiène mobile et de Prophylaxie d'AEF [c] (Taufflieb [d]) au Mayo Kebbi sur *S. damnosum* Theo.

En nous inspirant des techniques utilisées dans ces différents travaux, nous avons effectué dans le nord-ouest du Dahomey un essai de portée sur deux préparations insecticides: *a)* DDT 30%, densité 0,968, et DDT 30%, densité 0,995.

Ces produits ont été sélectionnés pour la lutte antilarvaire à la suite des études antérieures des hydrobiologistes du Museum national d'Histoire naturelle de Paris, et de la Section Onchocercose du Centre Muraz (Blanc & d'Aubenton, 1956; [e] Blanc, 1956; [f] d'Aubenton & Blanc, 1959; [g] Blanc, d'Aubenton, Ovazza & Valade, 1958.[h]

La rivière choisie, la Yerpao (fig. 1), présentait les avantages suivants:

1. Elle coule à proximité du centre de recherche et de la station locale de météorologie.

2. Elle est permanente et présente des gîtes à *S. damnosum* très nombreux, ce qui nous permet d'étudier la variation du nombre d'adultes piqueurs au cours de l'expérience.

3. Le relief de cette région est particulièrement mouvementé, et la Yerpao présente une variété de profils assez grande: c'est ainsi que, sur le parcours relativement court de 50 km, nous avons deux chutes, de nombreux rapides et des bassins à courant assez lent pouvant atteindre 1 à 2 km de long.

4. Dans sa partie supérieure la Yerpao est isolée des gîtes voisins par des zones montagneuses, les

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[b] Wanson, M. Courtois, L. & Lebied, B. (1949) *Ann. Soc. belge Med. trop.*, **29**, 373.

[c] L'AEF était une entité politique à la date mentionnée.

[d] Taufflieb, R. (1955) *Bull. Soc. Path. exot.*, **48**, 564.

[e] Blanc, M. & d'Aubenton, F. (1956) *Méd. trop.*, **16**, 96.

[f] Blanc, M. (1956) *Méd. trop.*, **16**, 418.

[g] d'Aubenton, F. & Blanc, M. (1959) *Méd. trop.*, **19**, 217.

[h] Blanc, M., d'Aubenton, F., Ovazza, M. & Valade, M. (1958) *Bull. Inst. franç. Afr. noire*, **20**, Série A, 634.