

AFRICAN SIMULIIDAE.

Of the various species of *Simulium* recorded from Africa, *S. damnosum* would appear to be the most widely distributed.

According to Austin (1909) the species recorded from Africa are: *Simulium latipes* in Natal; *S. damnosum* from Uganda, French Guinea, French Congo, Anglo-Egyptian Sudan, Congo Free State; *S. griseicollis* in Egypt, Anglo-Egyptian Sudan; *S. nigratarsis* in Cape Colony; *S. reptans* from French Congo, *S. wellmanni* from Angola.

Mr. Bedford informs me that he has collected *S. damnosum* and *S. nigratarsis* at Onderstepoort, and in Zululand a species which may be a new one.

New species have undoubtedly been added since 1909. The simuliidae have probably not received too much attention as yet, and it is for those in a position to collect specimens to do so and have them identified.

CONTROL.

All infected animals discovered in a not badly affected area should be destroyed and the importation of infected stock should be guarded against. Cases of onchocerciasis have been reported in sheep, but would appear to be extremely rare, and the possibility of its importation into South Africa by this animal would seem very remote.

In endemic areas it may be possible to limit its devastations by an attack on the intermediate host. The use of chemicals for the destruction of the larvae of the intermediate host should be investigated.

Paper No. 12.

A BRIEF REVIEW OF THE LITERATURE DEALING WITH THE KIDNEY-WORM (*STEPHANURUS DENTATUS* DIESING, 1839) OF SWINE, TOGETHER WITH SUGGESTIONS FOR ITS CONTROL AND ULTIMATE ERADICATION.*

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It would not be out of place to allude briefly to the earlier contributions, for, from the references consulted, it is evident that there has, within recent years, been little real progress towards elucidating the life-cycle of this parasite, and, as Butler (1928) remarked:—"By losing sight of facts and principles discovered long ago, we repeat work that is entirely unnecessary."

It is lamentable that practically all the textbooks treating with Veterinary Parasitology contain rather inaccurate statements about *Stephanurus dentatus*. These inaccuracies have proved to be most virulent and resistant, for they have not only successfully crept into the more recent literature, but have continued their vicious career in many "carefully revised" editions.

The research worker who depends solely on textbooks for the latest information is often badly misled.

* It has been requested that the more recent contribution on the life-history and the control of the kidney-worm of swine should be discussed at this Conference.

DISCOVERY AND GEOGRAPHICAL DISTRIBUTION.

The kidney-worm of swine was first observed by Natterer, who in 1834 collected it from the abdominal fat of a Chinese breed of the domestic pig in Brazil. It has since been collected from pigs in South America (Brazil and the Argentine), the United States of America, the West Indies, Asia (Annam, Java, Philippine Island, and Sumatra), Australia (Queensland and New South Wales), and Africa (French Congo, Belgian Congo, Portuguese East Africa, Madagascar, etc.). Its absence from Southern Europe is remarkable.

As synonyms of *Stephanurus dentatus* Diesing, 1839, may be quoted:—

Sclerostoma dentatum (Rud.) Leidy, 1856.

Sclerostoma pingucicola Verrill, 1870.

Sclerostoma dentatus Dean, 1874.

Stephanurus natiereri Cobbold, 1879.

Strongylus (*Sclerostoma*) *pingucicola* (Verrill) Magalhaes, 1894.

Sclerostomum renium Drabble, 1922.

Stephanurus dentatus (*Sclerostomum pingucolum*) Johnstone, 1912.

Stephanuris dentatus Monnig, 1928.

White (1858) is evidently the first to record *Stephanurus dentatus* from North America, where he had recovered it from the beef lard of pigs. He observed that the parasite burrows in the fat and forms canals 3 to 4 mm. in diameter. These canals terminate in cysts filled with pus and containing a pair of worms. These cysts were located along the course of large blood vessels. White expresses the opinion that the parasites "no doubt gained their situation by boring through the circulatory system while in the embryonic state." He recovered specimens also from the liver.

Leidy (1856) records *Sclerostoma dentatum* (Rud.) from the liver of pigs, and, according to Taylor (1899), it was probably this that induced White (1858) to imply that *Stephanurus dentatus* may be identical with *Sclerostoma dentatum* (Rud.) which to-day is known as *Cesophagostomum dentatum* (the common Nodular worm of swine). Leidy's parasite was evidently the kidney-worm, for although *C. dentatum* may in one of its larval stages occur in the liver, it would appear unlikely that Leidy would have been able to identify it as *Sclerostoma dentatum* (Rud.)

Verrill (1870), Fletcher (1871), Cobbold (1871), Dean (1874), Dinwiddie (1892), and Taylor (1899) recorded it from different localities in the United States of America and contributed to our knowledge of its morphology and pathogenesis.

Fletcher observed the worms in the liver and the portal veins as well as in cysts in the lungs and in the bronchial tubes. At first he took them to be specimens of the common lung-worm of swine, but changed his view when he found them to be oviparous. He recorded that these worms are held by the American farmers to be responsible for a "paralysis of motion in the hind legs."

Cobbold (1871) reported that Fletcher had found cysts of this parasite in the pyramids of the kidney.

Dean (1874) collected specimens from the perirenal fat, the pelvis of the kidney, more especially from the depth of the walls of the first third of the ureters, and less rarely from other organs such as the spleen.

Dinwiddie confirms White's records of the worm from the liver where they were present in tracts and cavities filled with pus.

Magalhaes (1894) redescribes the species from Brazil, and concludes that the correct name is *Strongylus* (*Sclerostoma*) *pinguicola* (Verrill). Tayler (1899) gives an extensive review of the findings of the earlier workers, redescribes the species, and concludes that *Sclerostoma pinguicola* Verrill must be accepted as the correct name. Tayler notes that eggs placed in a petri dish and kept moist hatch on the fourth day, and that within a further two days the second larval stages is reached. He concludes: "From analogy, one is led to believe that no intermediate host is required, but that in all probability the embryos develop for a short time in water, casting several skins, and that they eventually gain access to the hogs either through contaminated drinking-water or food."

Cobbold (1879) reviews briefly the literature dealing with this parasite. From his lines it is interesting to note that Dr. Fletcher regarded this parasite as the cause of hog cholera, while Morris, from Sydney, in a communication to the President of the London Microscopical Society, writes:—"It is just possible that some pigs may survive the irritation such a swarm of young worms must set up, others, again, may die from peritonitis, hence the sudden deaths amongst the pigs."

Cobbold was apt to overstress the losses from Helminthiasis. His appeals for means of controlling parasites evidently met with little success, as is evident from the passage:—"The wealthy Agricultural Societies of Great Britain pay little or no regard to the subject of parasites, although thousands of valuable animals annually perish from the injurious action of entozoa."

Helleman (1911) reports on the morphology and habits of *Stephanurus dentatus* from pigs in Java and Sumatra. He also records the pathological lesions observed in infected animals. Johnstone (1912) records that *Stephanurus dentatus* (*Sclerostomum pinguiculum*) infects the ureters, kidneys, and the livers of pigs in New South Wales and Queensland.

Gedoelst (1916) mentions that Dr. Van den Branden has often observed this parasite in the livers of pigs at Leopoldville.

Boynton (1914) records the presence of *Stephanurus datatus* from the Philippine Islands, and describes the symptoms exhibited by infected animals and the localization of the parasite in the host, as follows: "The infestation is characterized by muscular pains, tenderness to pressure over the kidneys, weakness, loss of appetite, emaciation, and partial or complete paralysis of the hind quarters. The parasites may be located in the fat surrounding the kidneys, in the ureters, and encysted in the kidneys, liver, spleen, lymph glands, and muscles and connective tissues in the region of the kidneys; also, they must be free, slightly embedded, or encysted in the connective tissues of both peritoneal and thoracic cavities." He describes the macroscopic and microscopic lesions observed in the invaded organs.

The kidney-worm of pigs in New South Wales was regarded by Drabble (1922) as a species distinct from *Stephanurus dentatus*, and was accordingly named *Sclerostomum renium*. Daubney (1923) bases his redescription of the species on material from East Africa, India, and the West Indies. He alludes to incorrect statements in the

Veterinary Parasitology textbooks by Neumann, Gedoelst, Kaupp, and Underhill, but seems to accept the work of Barnard and Bauche (1914) on the life-cycle as conclusive.

Drabble (1923), having seen Daubney's communications, still maintains that the New South Wales species was distinguishable from the type species. This led Cameron and Ross (1924) to examine specimens from New South Wales. Their verdict was that Drabble's species was specifically identical with *Stephanurus dentatus*.

Dr. Ayres, of Lourenco Marques, recently informed me that he has recovered it also from bovines in Mozambique. It has also been recorded as a very rare parasite of bovines from the United States of America.

Tissie (1924) reports that it is frequent in pigs from near the lakes Aloatra and Itocy in Madagascar, and he expresses the opinion that the animals probably get infected through eating the snails inhabiting these marshy places. He further observes that pigs do not die from the infection but that the flesh is sometimes urine-tainted. On one occasion the kidneys of an infected pig weighed about ten pounds (4 kilos).

THE PROBABLE LIFE-CYCLE OF THE PARASITE.

Taylor's view has been stated. Helleman (1911) observes that the eggs pass through fine canals with the lumen of the ureters to reach the outside and to develop into rhabditiform larvae. The pig would be infected by ingesting food or water contaminated with these larvae. The larvae then penetrate the walls of the stomach or intestines and reach the circulation, to be carried to the portal vein, where they develop. Those that arrive elsewhere would die. The larvae from the portal vein finally reach the aorta where thrombi are formed. In these thrombi they develop and mout before penetrating into the adipose tissue surrounding the aorta. From here they migrate to the perirenal fat and the pelvis of the kidneys to reach the ureters. Bernard and Bauche (1914) conclude that infection of the pig is firstly via the skin and secondly via the alimentary tract, and that the specific lesions correspond to the route of infection. Perirenal and peri-urethral cysts would result from infection through the skin, whereas hypertrophic cirrhosis of the liver would follow invasion via the alimentary tract.

Their conclusions that only the larvae penetrating the skin would reach maturity while those penetrating the wall of the digestive tube reach the liver to die, cannot be accepted. These conclusions are not warranted by the experimental data furnished.

Schwartz and Price (1928), according to an abstract from a paper read at the Fourth Annual Meeting of the American Society of Parasitologists, outlined the life-history of *Stephanurus dentatus* as follows:—"Under ordinary laboratory conditions, the eggs of the so-called 'kidney-worm' of swine, *Stephanurus dentatus*, hatch in about two days, and the larvae, after undergoing two moults in the course of about three or four days, reach the infective stage. The larvae do not appear to be capable of penetrating the intact skin of swine; when placed on the scarified skin, on the nasal mucosa, injected subcutaneously or administered by mouth, infection takes place readily, the course of development from the time of the experimental administration of the larvae to the time when the worms are sexually mature in the perirenal fat and in the ureters apparently requiring about five months or longer. Apparently, irrespective of

the portal of entry into the body, the larvae reach the portal vein and the gastrohepatic artery, in which vessels they occur in thrombi. Immature worms occur in various visceral organs, notably in the liver, lungs, and pancreas, the liver being most heavily and invariably parasitized. Immature worms have also been found in thrombi in the posterior vena cava, from which their path to the lungs can be readily understood. The young worms come gradually to the surface of the liver, lying underneath the capsule. In the course of time some of them penetrate the capsule to the exterior, thus liberating themselves from the location in which many of them perish and degenerate. Migration from the liver to the kidney fat is probably an active one, the distance to be traversed being comparatively small. The means by which fistulous channels are established between the ureters and the pus pockets in which the worms lie are not yet ascertained." This mode of development seems the most likely.

From these observations on the life-cycle, it is evident that the complete development is by no means understood. It is absolutely essential that this be known before the most practical prophylactic measures can be adopted.

PREVENTION AND TREATMENT.

Taylor believes that feeding from troughs and supplying plenty of wholesome water will decrease but not exclude stephanuriasis.

Leuckart's warning to his countrymen:—"Swine should be kept in a less 'swine-like' manner," should be heeded. There is no gainsaying that most of the mortality and unproductiveness of domestic stock must be attributed to faulty sanitation, faulty feeding, and faulty husbandry.

A study of the hypothecated life-cycle and the habits of *Stephanurus dentatus* suggested that it will be with us as long as we continue to rear and keep the pig confined in filthy styes or on wet pastures. My experience is that the greater proportion of pig-owners believe that the pig will only thrive in filth, and the warnings of that eminent helminthologist, Leuckart, is even to-day not heeded in Europe. The cold climate of Northern Europe allows of sanitary methods which would spell disaster if practised in tropical or sub-tropical climates.

Since no specific for ridding pigs of this parasite is known, it is evident that the only attempts on its life should be made while it is outside its host. The attack should, therefore, be directed against the eggs passed in the urine and the larvae which may hatch from these eggs. The excreta should, therefore be treated so as to prevent the hatching of the eggs or preventing the larvae from reaching the host.

I know farms where parasitic diseases amongst the pigs are unknown. The absence of disease can be largely attributed to a few factors:—(1) The dryness of the layers of soil and manure forming the floor of the styes. (2) The absence of marshy ground or pools to which pigs will have access when allowed out for exercise. (3) The use of troughs which cannot be readily soiled with urine or faeces, and the regular cleansing of these troughs. (4) During the summer months the pigs have access to swift-running water in cement-concrete furrows. In the adjoining farms, where the pigs run in paddocks and have access to muddy pools and damp ground or where they are kept in damp styes, they suffer a lot from lung-worm, ascariasis, and oesophagostomiasis.

Although I have no personal experience with the kidney-worm of swine, I feel convinced that the control and ultimate eradication of stephanuriasis can be accomplished by the application of prophylactic measures on the following lines:—

- (1) Confine animals only in pens with cement-concrete floors which can be readily flushed or disinfected daily, or failing this, pens with a dry ground floor. In the case of the pens with concrete floors, all soiled bedding should be discarded regularly every other day or daily if possible. My experience with equines is that where the bedding is removed daily and the stalls flushed the inmates are free from worms.
- (2) Animals should only be allowed into paddocks free of marshy ground where development and hatching of the eggs and the development of the larvae into infective ones will continue. Paddocks for the use of young stock should have been left free of other pigs for at least six months or more. There are, unfortunately, no particulars as to the length of time which the infective stage larvae can exist outside the host.
- (3) Water and feed the animals from troughs which they cannot readily soil with their excreta. Piglings should be fed from troughs to which the adults will not have access.
- (4) The breeding stock should, if possible, be non-infected or only slightly. They should not be allowed to mix with the rest of the herd. Breed from strong, healthy stock.
- (5) Animals should be inspected at regular intervals, and all infected stock got rid of. All new importations should be inspected and quarantined for some time before being turned in with the main herd. Hall (1928) states that the kidney-worm is on the spread in the United States of America. This spread is attributed to the transportation of infected animals about the country.
- (6) If possible, no animal should be kept for more than a few years. Old animals would appear to be the most heavily infested, and would, therefore, be the most dangerous carriers and disseminators of this malady.
- (7) Where the animals must be allowed baths during hot weather, they should be allowed access to swift-running streams or to concrete structures from which the water can be drained regularly, every day if possible, and the bath disinfected.
- (8) Protect your stock against cold wind, wet, want, and wounds. These are the ever-faithful allies of worms.

Other measures could be appended, but they would be less practical and more costly to carry out. Those who are trying to persuade the African native to keep pigs free from worms will have to be patient and persevering.

The European stock owners are always clamouring for some drug, vaccine, or serum which, when administered, will cure their stock. Few owners are prepared to practice hygiene. Some have (fortunately) realized what good sanitation means. It is for us who are in

charge of Government experimental stock farms, etc., to practise what we preach. The public is always watching, and will adopt our methods and recommendations when these have been proved to "deliver the goods."

Gentlemen, let we African Veterinarians acquaint ourselves with local conditions and then preach what we feel can be practised.

Paper No. 13

CONTAGIOUS BOVINE PLEURO-PNEUMONIA.—CULTURE VACCINES.

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THE following is a short summary of the work carried out in the Sudan during the last three years.

INTRODUCTION.

Earlier knowledge, on which the work has been based, can be briefly set out as follows:—

1. Injections of natural lymph induce a strong immunity, but are too dangerous. By reducing the dose and introducing into selected sites mortality can be reduced, but risk has never by these means been eliminated.

2. The virus has been cultivated in various media, but most of them are rather troublesome to prepare.

3. Cultures of the virus have been used with varying success as vaccines. It has been recorded that cultures of the first and second generation are approximately as dangerous to inject as the natural lymph, but cultures of several generations are safe.

4. Regarding the best age (in generations of transplants—or length of time in artificial culture) at which to use cultures for vaccination, no very definite evidence has been recorded. From some records, however, it has been argued that to be an efficient vaccine a culture should be of such an age that it provokes a mild reaction when injected—if there is no "reaction," there is no subsequent immunity. Such a principle of vaccination entails a second vaccination of the non-reactors, which in a country such as the Sudan is in most cases impracticable, if not impossible.

Without attempting to discuss the literature in any way, it seems that evidence is either lacking or unsatisfactory on many points; those that have received attention in the Sudan are as follows:—

1. The possibility of growing the virus in very simple culture media.
2. The determination, if possible, of the exact length of time a virus must be maintained in artificial culture medium for it to become harmless when injected into cattle.
3. Assessment of the antigenic value of cultures of different ages expressed in terms of the time they have been maintained in artificial media since original isolation.