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Biting Ceratopogonids as Vectors of Human and Animal Diseases.

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Four families of lower diptera have been incriminated as vectors of human and animal diseases. They are the Culicidae, Psychodidae, Simulidae and Ceratopogonidae. The last family has received relatively little attention because it does not transmit a fatal disease to man although it does to his animals. This review will draw attention to the economic importance of the Ceratopogonidae and may, it is hoped, encourage work on their biology and bionomics.

Most Ceratopogonids feed on other invertebrates but three genera have evolved the habit of feeding on warm blooded birds and mammals. They are *Culicoides, Leptoconops* and *Lasiohelea.* As with other nematocera the blood-sucking habit is confined to the female, the male feeding on nectar. They are tiny flies, whose wing lengths rarely exceed 2 mm, which hold their short probosces vertically and fold their wings scissor-like over the abdomen at rest.

The wings of most *Culicoides* have a pattern of light and dark marks, those of *Leptoconops* are milky white contrasting sharply with the black body while those of *Lasiohelea* are densely hairy and without pattern. Several hundred species of *Culicoides* have been described and they are to be found in all parts of the world. *Leptoconops* has fewer species (about 30) and is not so widely distributed but *Lasiohelea,* with about the same number of species as *Leptoconops,* is restricted to the tropics and subtropics.

Published work on *Lasiohelea* is almost entirely taxonomic, while there is some information available on the biology and control of *Leptoconops.* Only for *Culicoides* is there a modest amount of information on their bionomics but even this is scanty compared with other genera of comparable importance. This aspect has already been reviewed (KETTLE, 1962) and the present paper will concentrate on the role of Ceratopogonids as vectors of human and animal diseases. So far only *Culicoides* have been incriminated and they have been shown to be concerned in the spread of three groups of pathogens: Nematoda (Filarioidea), Protozoa (Haemosporidiidea) and Viruses.

Nematoda transmitted by Culicoides.

Culicoides transmit five species of filarial worms to man and his animals. In 1927 Dyce SHARP reported that *C. grahami* could transmit *Acanthocheilonema perstans,* a non-pathogenic parasite of man in East and West Africa. The following year he showed in detail that *C. austeni* could also act as a vector (SHARP, 1928). Twenty years later these findings were challenged by HENRARD & PEEL (1949) and CHARDOME & PEEL (1949), who drew attention to the fact that two parasites were involved, *A. perstans* and *Dipetalonema streptocerca.* They claimed that SHARP had followed the development of *D. streptocerca* in *C. grahami* and hinted that the same was true for *C. austeni.* The Belgian workers obtained a 23% experimental infection rate with *D. streptocerca* in *C. grahami* and provided evidence to suggest that *C. grahami* did not ingest microfilariae of *A. perstans.* The microfilariae of *A. perstans* occur in the circulating blood while those of *D. streptocerca* are in the skin. Unfortunately SHARP did not check the identity of the ingested microfilariae.

Transmission experiments at that time laboured under the handicap of having to use wild-caught flies, which may already be infected. It is essential to use bred, uninfected flies and this became possible when HOPKINS (1952) located the breeding site of both *C. austeni* and *C. grahami* in the decomposing cut stumps of banana plants. Using clean, bred flies and hosts only infected with *A. perstans,* HOPKINS & NICHOLAS (1952) obtained 41% infection (29/70) of *C. austeni.* In a parallel experiment with wild-caught *C. grahami* the flies took up fewer *A. perstans* microfilariae than was to be expected from their density in the circulating blood, whereas *C. austeni* took up more than expected (*C. austeni* 40 cf. 15; *C. grahami* 2 cf. 26). DUKE (1954 and 1956) confirmed this result for *C. grahami* and also showed that this species readily acquired infection with *D. streptocerca.*

In the Caribbean man is host to another non-pathogenic filaria, *Mansonella ozzardi.* BUCKLEY (1934), working on St. Vincent where about one third of the people were infected, investigated a number of possible vectors but only in *C. furens* did the microfilariae develop. Using wild-caught flies the infection rate was increased from 5 to 27½% after being fed on an infected host.

GIBSON & ASCOLI (1952) investigated the potentiality of *Culicoides* as vectors of *Onchocerca volvulus* in Guatemala and found that although *C. paraensis* and *C. stigmalis* ingest the microfilariae, none complete their development and the longevity of the host fly is reduced.

Onchocerca reticulata (= cervicalis), which is associated with a condition known as fistulous withers in horses, was shown by STEWARD (1933) to be transmitted by *Culicoides.* He had no success with *Simulium* or *Haematopota* but obtained infections in *C. obsoletus* and *C. nubeculosus.* STEWARD followed the entire development of the worm in *C. nubeculosus,* which was surprisingly long, taking 25 days from ingestion of microfilariae to the attainment of head infectivity. This factor must limit transmission in nature.

In Australia and Malaya heavy infection rates of *O. gibsoni* have been found in cattle and these may lead to carcases being condemned. In his investigations to find the vector BUCKLEY (1938) collected 20 species of *Culicoides* and *Lasiohelea* off cattle. Of these *C. pungens* and *C. oxystoma* were the most abundant but the natural infection rate was very low (0.3%). Even after feeding on infected cattle the rate did not rise above 1%. However, as *Culicoides* can be collected off cattle at 500 per hour a very low infection rate in the vector would be adequate to maintain a high parasite rate in the host.

Protozoa transmitted by Culicoides.

The incrimination of *Culicoides* as vectors of protozoa is a fairly recent discovery, which was made by FALLIS & WOOD (1957) when they observed the development of *Haemoproteus nettionis* in an unidentified species of *Culicoides.* This was followed by FALLIS & BENNETT (1960) observing the sporogony of *H. canachites* in *C. sphagnumensis.* These findings were supplemented by epidemiological studies in which it was found that the peak abundance of *Culicoides* preceded heavy parasitaemia of *Haemoproteus* spp. in immature birds (BENNETT & FALLIS, 1960; BENNETT, 1960). Unpublished work by BENNETT indicates that avian trypanosomes will develop in, and can be transmitted by *Culicoides.*

The involvement of *Culicoides* in the transmission of blood-dwelling protozoa was rapidly followed up by workers in Japan and Kenya. In Japan, AKIBA and his co-workers showed that *Leucocytozoon caulleryi* could be transmitted by *Culicoides arakawae* (AKIBA et al., 1959; AKIBA, 1960). Leucocytozoonosis is economically a very important disease of poultry in S.E. Asia

and Japan. *C. arakawae* was considered, on very little evidence, to breed in chicken manure, which would have given a very close vector/host relationship. This is not so. *C. arakawae* breeds in paddy fields (TOKUNAGA et al., 1961; KITAOKA & MORII, 1963).

In Kenya, GARNHAM finally solved the long standing mystery of the transmission of *Hepatocystis (Plasmodium) kochi,* a malaria like parasite of *Cercopithecus* monkeys. GARNHAM and his co-workers followed the development of the parasite in *Culicoides adersi* (GARNHAM et al., 1961). The unusual feature of this cycle is that the sporocyst lies free in the haemocoele and is not attached to the midgut wall of the vector.

Viruses transmitted by Culicoides.

It is as vectors of viruses that *Culicoides* are becoming more and more important economically. DU TOIT (1944) recovered the viruses of Bluetongue and Horse Sickness from wild-caught *Culicoides* at Onderstepoort in South Africa. Specimens collected in a modified New Jersey light trap were ground up and the resulting suspension injected into clean sheep and horses. Three sheep developed typical symptoms of Bluetongue and one died from the disease. The two sheep which recovered were challenged with Bluetongue virus and resisted infection. Inoculation with wild-caught *Culicoides* had produced immunity to Bluetongue virus. The virus could have been present in undigested blood in the gut of the fly, and therefore it is necessary to show that the virus can be transmitted by biting.

DU TOIT allowed other wild-caught *Culicoides* to feed on a sheep suffering from Bluetongue. They were kept for ten days and then fed on a healthy sheep. Two female *C. pallidipennis* survived to feed and seven days later the sheep developed a febrile reaction which lasted five days. This suggests that Bluetongue can be transmitted by the bite of *Culicoides.*

Bluetongue is endemic to East and Southern Africa. It was recognised in the U.S.A. in 1948 when an epizootic of Bluetongue occurred in Texas (PRICE & HARDY, 1954). In 1956 it appeared in Spain and Portugal and a similar disease has been recorded recently in cattle in Japan. Cattle are susceptible to Bluetongue but the disease runs a milder course.

Laboratory experimental work on transmission awaited the development of a technique to rear large numbers of clean *Culicoides.*

MEGAHED (1956) managed to keep a self-maintaining colony but it was JONES (1957, 1960) who finally bred *C. variipennis* in large enough numbers (1,000 per day) for experiment. This in turn led to the transmission of Bluetongue by the bite of *C. variipennis* in the laboratory, using clean bred flies and the most stringent precautions against accidental infection (FOSTER, JONES & MCCRORY, 1963). A minimum incubation period of 10 days is required at 21-24°C. No transmission is obtained 8 days after feeding. In view of all this experimental work it is of interest to learn that it was not until 1961 (JONES, 1961) that field observations confirmed that *C. variipennis* did, in fact, feed on sheep under natural conditions.

DU TOIT (1944) recovered the virus of Horse Sickness by intravenous injection of an emulsion of wild-caught *Culicoides* into a horse. Subinoculation into another horse gave rise to a fever which terminated fatally ten days later. Horse sickness is endemic in Eastern and Southern Africa and the Sudan but rare in West Africa. Periodically in the past (1928, 1943) there have been epizootics in Egypt and Israel but in the last five years the disease has erupted dramatically into new areas. In 1959 it spread to Iran, Afghanistan and Western Pakistan. The following year it continued its eastward expansion

TABLE 1.

A summary of the parasitic organisms spread by Culicoides.

Organism	Vector	Author
Nematoda (Filarioidea).		
Acanthocheilonema perstans	*C. grahami*	SHARP (1927)
Acanthocheilonema perstans	*C. austeni*	SHARP (1928)
Acanthocheilonema perstans	*C. austeni*	HOPKINS & NICHOLAS (1952)
Dipetalonema streptocerca	*C. grahami*	CHARDOME & PEEL (1949)
Dipetalonema streptocerca	*C. grahami*	HENRARD & PEEL (1949)
Dipetalonema streptocerca	*C. grahami*	DUKE (1954)
Mansonella ozzardi	*C. furens*	BUCKLEY (1934)
Onchocerca reticulata	*C. nubeculosus*	STEWARD (1933)
Onchocerca reticulata	*C. obsoletus*	STEWARD (1933)
Onchocerca gibsoni	*C. pungens*	BUCKLEY (1938)
Protozoa (Haemosporidiidea).		
Haemoproteus nettionis	*C. sp.?*	FALLIS & WOOD (1957)
Haemoproteus canachites	*C. sphagnumensis*	FALLIS & BENNETT (1960)
Leucocytozoon caulleryi	*C. arakawae*	AKIBA, KITAOKA & YAJIMA (1959)
Leucocytozoon caulleryi	*C. arakawae*	AKIBA (1960)
Hepatocystis kochi	*C. adersi*	GARNHAM, HEISCH & MINTER (1961)
Viruses.		
Bluetongue	*C. pallidipennis*	DU TOIT (1944)
Bluetongue	*C. variipennis*	PRICE & HARDY (1954)
Bluetongue	*C. variipennis*	FOSTER, JONES & MCCRORY (1963)
Horse Sickness	*C. sp.?*	DU TOIT (1944)
Three Day Fever	*C. sp.?*	LEE, REYE & DYCE (1962)
Eastern Equine Encephalomyelitis	*C. sp.?*	KARSTAD, FLETCHER, SPALATIN, ROBERTS & HANSON (1957)
Venezuelan Equine Encephalomyelitis	*C. sp.?*	LEVI-CASTILLO reported in KARSTAD et al. (1957)
Japanese B encephalitis	*Lasiohelea taiwana*	WU & WU (1957)
Organism?		
Queensland Itch	*C. brevitarsis*	RIEK (1954)

into India and westwards to Turkey and Cyprus, putting at risk about thirteen million horses and mules and killing two to three hundred thousand animals (HUQ, 1961). Control measures have been restricted to immunisation of threatened animals but studies are wanted on the vector before it will be possible to understand the epidemiology of the disease and the reason for this particular outburst.

On circumstantial evidence, *Culicoides* have been accused of transmitting three day fever (LEE et al., 1962), a disease of cattle which is endemic in Kenya where in recent weeks there has been an outbreak in the Nairobi area. RIEK (1954) has attributed Queensland Itch to the bites of *Culicoides brevitarsis (= robertsi).* It is an allergic condition of horses. KARSTAD et al. (1957) have recorded the recovery of the virus of Eastern equine encephalomyelitis from *Culicoides* in South Georgia but experimental work in Maryland using *C. obsoletus* failed to transmit this virus (SCANLON, 1960). Levi-Castillo is also reported as having recovered Venezuelan equine encephalomyelitis virus from *Culicoides* (KARSTAD et al., 1957).

The first incrimination of the genus *Lasiohelea* in the transmission of a human disease is that of WU & WU (1957), who recovered the virus of Japanese B encephalitis from *L. taiwana.*

Conclusions.

The substance of the material presented in the preceding pages is summarised in Table 1. It will be clear from the evidence presented here that the bloodsucking Ceratopogonids, and especially *Culicoides,* are worthy subjects for research. This comment is particularly relevant to East Africa where so many of the conditions listed in Table 1 are endemic. For a variety of reasons virtually no epidemiological work has yet been carried out. It is intended to make a start on this subject in the near future at the University College Nairobi.

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