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HEARTWATER IN HOSTS OTHER THAN DOMESTIC RUMINANTS

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ABSTRACT

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The importance of further research on the susceptibility of wild hosts to *Cowdria ruminantium* infection is discussed. The literature is surveyed and an attempt is made to divide the various species described into susceptible and refractory hosts. The reasons for the numerous apparently conflicting reports are considered and it is suggested that those making further inquiries in this field of work take these factors into account.

INTRODUCTION

Heartwater is of considerable importance to the livestock industry and it is to be expected that the greater part of the research effort centres on the occurrence of the disease in domestic animals. Most reports on heartwater in wild animals have been incidental and of a cursory nature. There are a number of reasons why more work should be done on the susceptibility of wild hosts to heartwater.

Introduction of game

The most obvious of these is the tremendous increase in game farming activities. Farmers wishing to re-introduce ruminant game species into heartwater endemic areas need to know whether the animals involved are susceptible and whether they require vaccination or not.

The role of game in the maintenance of heartwater

Less obvious, but perhaps far more important, is a knowledge of the role of wild animals in maintaining the disease in certain areas. This will assist in an understanding of the epizootiology of the disease. Although Neitz (1967) stated that heartwater does not require a wildlife host to maintain itself, there is little doubt that these animals can play a part in the maintenance of the vector, especially where tick control on domestic stock is exercised. Bezuidenhout (1985) placed the situation in perspective when he studied the biology and life cycle of the vector, and asked how, with such little overlapping between larval and adult peaks, the ticks could become infected on domestic stock. While many authors (Brassey-Edwards, 1929; Norval & Lawrence, 1979; MacKenzie & Norval, 1980; Uilenberg, Barré, Camus, Burridge & Garris, 1984) have considered wild animals as possible reservoirs of the causative organism of heartwater, Uilenberg (1983) gave thought to the "real problem" posed by wild ungulates where control of the disease through vector eradication is attempted. Still others (Ilemobade, 1976; Ilemobade & Leeflang, 1977; MacKenzie & Van Rooyen, 1981) have speculated on the role of rodents in maintaining heartwater in certain areas. While Uilenberg (1983) thought this unlikely, the possibility should be further investigated in the light of the fact that transovarial transmission of *C. ruminantium* does occur (Bezuidenhout & Jacobsz, 1986), that nymphal and adult *Amblyomma* ticks do occasionally feed on rodents (Theiler, 1962) and that the organism has been shown to circulate in the blood of these animals for up to 14 days (Daubney, 1941; Hudson & Henderson, 1941). These facts indicate that rodents could, in certain circumstances, play a role in maintaining the disease. Other wild animals, apart from wild ruminants, which want attention are lagomorphs, such as the scrub hare *Lepus saxatilis*, which is a very good host for both larval and nymphal stages of *Amblyomma* ticks (I. G. Horak, personal communication, 1986). Many ground birds are also good hosts of *Amblyomma* larvae and nymphae (Theiler, 1962; Horak & Williams, 1986).

The role played by wild hosts in the maintenance of the disease in an area has 3 clear features: First, the immature *Amblyomma* ticks are generally found on small mammals and ground birds which are therefore in a position to spread the ticks to new areas or re-introduce the ticks into areas from which they had been previously eradicated. Secondly, wild hosts help maintain the vector especially where the domestic stock is dipped regularly while the vector itself may act as a reservoir of the infection said to survive in a single tick generation for over three years (Neitz, 1968). Thirdly, wild hosts may circulate the organism for considerable lengths of time, e.g. 30 days in the black wildebeest (Neitz, 1944), so acting as possible sources of infection for *Amblyomma* larvae or nymphs which may happen to parasitize them.

Suitable laboratory hosts

Until the discovery of the mouse infective strains (Du Plessis & Kümm, 1971; MacKenzie & Van Rooyen, 1981; Du Plessis, 1985a) one of the major obstacles to progress in heartwater research was the lack of suitable laboratory hosts of *Cowdria*. It was in the search for such hosts that Swiss mice, rats, ferrets and a number of wild rodents were examined for susceptibility to heartwater.

Better vaccine strains

The possibility of finding a strain of *Cowdria* which though still immunogenic is less virulent to domestic stock than the available strains cannot be discounted.

PRESENT STATE OF OUR KNOWLEDGE

Table 1 is a summary of the literature available including some unpublished data. The findings of various authors have been used to list the animals as being clinical, subclinical or refractory hosts of *Cowdria ruminantium*.

Not included in the table are early reports on the susceptibility of horses (Edington, 1904; Coutts, 1905; Van Saceghem, 1918) and dogs (D. T. Mitchell, 1919, cited in Alexander, 1931) which were later shown to be incorrect by Theiler, 1905 and Alexander, 1931.

Refractory hosts are those exposed to the disease, usually by intravenous injection, which show no clinical signs of infection nor give any indication of the circulation of *C. ruminantium* in the blood of the animal.

It must be emphasized that, in most cases, the hosts were not examined for the presence of circulating organisms in the blood, and the temperature was monitored in only a few cases. Clearly some of the animals listed as refractory could, in fact, be subclinical hosts.

Hosts which show only mild pyrexia or no signs at all but in which *C. ruminantium* are found to circulate in the blood are classified as subclinical hosts. This is usually confirmed either by subinoculation of blood into susceptible hosts or by xenodiagnosis.

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TABLE 1 The susceptibility of various non-domesticated hosts to heartwater*: Summary of the literature

Host		Susceptibility†		
Common name	Scientific name	Clinical	Subclinical	Refractory
Laboratory hosts:				
Mice		10[a], 25[a]	15	34
Hamsters				44
Albino rats			7,17	23,34,35
Guinea pigs				2,11,34,44
Rabbits		18[b]		2,11,23,34,44
Domestic ferrets	*Mustela furo*	1	23,40	
Wild ruminants:				
Giraffe	*Giraffa camelopardalis*		9	13
Black wildebeest	*Connochaetes gnu*	9,27	8,28,30	9
Blue wildebeest	*C. taurinus*			13
Red hartebeest	*Alcelaphus buselaphus*			9
Blesbuck	*Damaliscus dorcas phillipsi*	28	8,27,28,29[c]	
Duiker	*Cephalophus* sp.			6
Springbuck	*Antidorcas marsupialis*	9,30,37,41,43		
Impala	*Aepyceros melampus*			13[b]
Scimitar-horned oryx	*Oryx dammah*			9
African buffalo	*Syncerus caffer*			13,20
Greater kudu	*Tragelaphus strepsiceros*			13[b]
Bushbuck	*Tr. scriptus*	39[b]		5
Sitatunga	*Tr. spekei*	31[b]		
Eland	*Taurotragus oryx*	43	14	
Extra-African ruminants:				
Arabian camel	*Camelus dromedarius*	19[b]		
White tailed deer	*Odocoileus virginianus*	21		
Fallow deer	*Cervus dama*	16[b],42,43		
Timor deer	*C. timorensis*	33		
Bison	*Bison* sp.	12[b]		
Water buffalo	*Bubalus arnee*	4,9,22,26		
Barbary sheep	*Ammotragus lervia*	9,16[b],42,43		
Himalayan tahr	*Hemitragus jemlahicus*	43		
Nilgai	*Boselaphus tragocamelus*	43		
Blackbuck	*Antilope cervicapra*	16		
Mouflon	*Ovis orientalis*	9,16[b],42		
Wild rodents:				
Four-striped grass mouse	*Rhabdomys pumilio*		7,17[b]	24[a]
Vlei rat	*Otomys irroratus*			24[a]
Angoni vlei rat	*O. angoniensis*		17[b]	
Brush-furred rat	*Lophuromys* sp.			17
House rat	*Rattus rattus*			17
Multimammate mouse	*Praomys coucha*	24[a],38[a]		
Birds:				
Crowned guinea-fowl	*Numida meleagris*		3[c]	
Reptiles:				
Leopard tortoise	*Geochelone pardalis*		3[c]	
Other:				
Mongoose	(not specified)			36
Genet	*Genetta* sp.			36
Vervet monkey	*Cercopithecus aethiops*	32[b]		
Warthog	*Phacochoerus aethiopicus*			13

* References later proved incorrect, such as those describing heartwater in the horse, pig and dog as well as those such as in cats and primates which were found as expected to be refractory, have been excluded from this table.

[a] Mouse adapted strains.

[b] Inconclusive evidence.

[c] Proven using ticks as vectors.

†1–44: *Reference numbers*

1. Adelaar, T. F., 1955. Cited in Henning, M. W., 1956
2. Alexander, 1931
3. Bezuidenhout, J. D. & Olivier, J. A., unpublished data, 1986
4. Bezuidenhout, J. D. & Thirion, C., unpublished data, cited in Bezuidenhout, 1982
5. Curasson, G. M. C., 1943, cited in Camus & Barré, 1982
6. Curasson, G. M. C. & Delpy, L., 1928, cited in Uilenberg, 1983
7. Daubney, 1941
8. De Vos, 1973
9. Du Plessis, J. L., unpublished data, 1986
10. Du Plessis & Kümm, 1971
11. Du Toit, P. J., 1924, cited in Alexander, 1931
12. Enigk, K., 1942, cited in Camus & Barré, 1982
13. Gradwell *et al.*, 1976
14. Grosskopf, 1958
15. Haig, 1952
16. Hofmeyr, 1956
17. Hudson & Henderson, 1941
18. Hughes, 1953

19. Karrar, 1960
20. Keffen, 1985
21. Logan, Linda L., personal communication, 1986
22. Mammerickx, M., 1961, cited in Camus & Barré, 1982 and in Cockrill, 1974
23. Mason & Alexander, 1940
24. MacKenzie & McHardy, 1984
25. MacKenzie & Van Rooyen, 1981
26. Mohan, 1968
27. Neitz, 1933
28. Neitz, 1935
29. Neitz, 1937
30. Neitz, 1944
31. Okoh, Oyetunde & Ibu, 1986
32. Pelissier, A., Troquereau, P., Trinquier, E., 1950, cited in Uilenberg, 1983
33. Poudelet, M., Poudelet, E. & Barré, N., 1982, cited in Camus & Barré, 1982
34. Ramisse & Uilenberg, 1971
35. Robinson, E. M., 1927, cited in Alexander, 1931
36. Simpson & Hudson, 1942, cited in Camus & Barré, 1982
37. Spreull, 1922
38. Stewart, C. G., unpublished data, 1986
39. Webb, 1897–1898
40. Weiss, K. E., personal communications, 1986
41. Young, 1970
42. Young, E., cited in Gradwell *et al.*, 1976
43. Young & Basson, 1973
44. Annual Report, Institut d'Élevage et de Médecine Vétérinaire des Pays Tropicaux, 1968, cited in Camus & Barré, 1982.

The most recent and most interesting of these reports were by J. D. Bezuidenhout and J. A. Olivier (unpublished data, 1986) who showed that the crowned guinea fowl and the leopard tortoise can be subclinically infected by, and act as a source of infection for *Amblyomma* ticks. These findings are extremely important in the epidemiology of the disease. One of the major shortcomings of most of the reports is the large amount of blood that was subinoculated from the test host into an indicator host to imply carrier status. Such results do not necessarily mean that ticks, especially larvae and nymphs, would become infected by feeding on such hosts.

Clinical hosts are regarded as those animals which showed overt clinical signs similar to those shown by domestic ruminants. In most of the cases listed the outcome was fatal. The signs displayed by animals reported as being susceptible have been very poorly recorded. The signs seen and described are:

In ferrets, a febrile reaction with no other observable signs (K. E. Weiss, personal communication, 1986) while T. F. Adelaar (1955, cited in Henning, 1956), indicated that the virulence of *C. ruminantium* in ferrets was increased by passage in these animals. The incubation period varied from 6 to 13 days and Mason & Alexander (1940) described signs of dullness, nervousness and inco-ordinated movements.

The only clinical sign shown by 1 of 2 black wildebeest infected by J. L. du Plessis (unpublished data, 1986) was mild depression from day 13 after inoculation. It was found dead on day 19 while the other showed incoordination during the morning of day 20. It walked into a water trough, fell down and died. Brain smears stained with Giemsa confirmed that both animals had died of heartwater.

In the blesbuck no clinical signs other than sudden death were described (Neitz, 1937) but Neitz (1937) showed that *Cowdria* circulated in their blood from 28 to 62 days after infection with *A. hebraeum* nymphs and the animals died 21 to 70 days after infection.

The eland, as described by Young & Basson (1973), showed the same signs as those seen in domestic ruminants including: hyperaesthesia, circling, a stiff, unsteady, high-stepping gait followed by prostration, opisthotonus, muscle tremors and convulsions, blinking of the eyes, prolapse of the nictitating membranes, continuous grinding of the teeth as well as discharge of froth from nostrils and mouth. Clinical examination revealed a body temperature above 43 °C, a severe tachycardia (more than 400 beats/min) and polypnoea. The animal was not treated and died 1 h after the onset of clinical disease. Grosskopf (1958) described only a slight pyrexia after an incubation period of 12–17 days after vaccination.

J. D. Bezuidenhout and C. Thirion (1982, cited in Bezuidenhout, 1982) described the typical pyrexia, hyperaesthesia, hyperaemia and petechiation of the conjunctiva in Indian buffalo. They also stated that the buffalo became blind and died on day 25 after inoculation.

Young (cited in Gradwell, Van Niekerk & Joubert, 1976) reported typical clinical symptoms in mouflon, Barbary sheep and fallow deer.

The remaining reports listed the species of animals in which fatal cases of the disease occurred but no clinical signs were described. In general it appears that the signs shown by the majority of susceptible wild animals are similar to those shown by domestic ruminants suffering from the disease. This supposition is strengthened by the fact that *post mortem* lesions (where described) were also similar to those seen in domestic animals. The incubation period appeared to be somewhat longer in the case of wild animals than in domestic animals but once symptoms appeared the course of the disease was very short, which explains, perhaps, why clinical signs were not described.

Possible reasons for the conflicting reports

The apparently conflicting reports present a very complicated picture, and at first glance may lead to greater confusion rather than to the hoped-for better understanding of the disease.

Closer scrutiny, however, and consideration of the following factors help place a semblance of understanding on the information.

The strain of inoculum and route of administration

It is important to take the strain of *Cowdria* used and the route of administration into account. Not all strains are equally infective for all species of animals, as is seen with the Kümm strain which infects mice and sheep but not cattle (Du Plessis & Kümm, 1971). In fact, MacKenzie & Van Rooyen (1981) showed that the Kwanyanga strain lost its ability to infect mice after it had been passaged 15–19 times in sheep. Much of the experimental work on wild animals has been done with the Ball 3 strain (Haig, 1952) which has been passaged for the past 34 years in sheep, and may possibly have changed in pathogenicity for other hosts. The route of administration should also be considered in the light of the fact that the Kwanyanga strain is more infective to mice via the intravenous route (MacKenzie & Van Rooyen, 1981) while the Kümm strain is infective both by the intravenous and intraperitoneal routes.

Breed and age

As is the case with domestic animals, it can be accepted that the breed and age of the wild animal will play an important role in the resistance shown by the animal to the disease.

Conglutinin levels

Du Plessis (1985b) has shown that the conglutinin levels of game animals prior to infection plays the same

important role in influencing the susceptibility of individual wild animals as it does with domestic animals. This is illustrated by his results with black wildebeest.

Previous exposure

It is extremely important, though not always easy to ensure, that the animal in question has not been previously exposed to the disease and is therefore possibly immune.

FUTURE PROSPECTS

On the surface the study of heartwater in wild animals is of value in ascertaining what species are susceptible but its significance could go much deeper than this. Valuable epizootiological background and even indications of the possible pathogenesis could be forthcoming. Differences in susceptibility of different species could possibly be correlated with physiological differences between these species. It is recommended that future work in this field takes the above factors into account and perhaps moves away from showing concern solely with ruminants which show clinical symptoms and considers all animals which host immature and adult *Amblyomma* ticks, whether they show clinical disease or not. It is not necessary for an animal to be clinically affected for it to act as a reservoir of the causative agent.

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