Onderstepoort J. vet Res., 53, 19–24 (1986)

STUDIES ON THE ABILITY OF DIFFERENT STRAINS OR POPULATIONS OF FEMALE *RHIPICEPHALUS EVERTSI EVERTSI* (ACARINA: IXODIDAE) TO PRODUCE PARALYSIS IN SHEEP

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ABSTRACT

GOTHE, R. & BEZUIDENHOUT, J. D., 1986. Studies on the ability of different strains or populations of female *Rhipicephalus evertsi evertsi* (Acarina: Ixodidae) to produce paralysis in sheep. *Onderstepoort Journal of Veterinary Research*, 53, 19–24 (1986)

Simultaneous infestation of 3–6 month-old Black-head sheep with 15 South African wild strains of *Rhipicephalus evertsi evertsi* males and females as well as a strain from Rwanda clearly showed that all strains are capable of inducing paralysis.

Assessment of the infestation-rate of engorging female ticks during the period that their mass ranged between 15 and 21 mg/kg sheep body mass indicated that toxicity is quantitatively identical and exhibits no intraspecific gradations. The period between the beginning of infestation to the manifestation of the first clinical symptoms is, however, strain dependent: 4 days for ticks from Warmbaths, and at least 5 days for all other strains.

INTRODUCTION

Very little has been published on *Rhipicephalus evertsi evertsi* paralysis, only a few cases in the southern part of the tick's distribution having been described. It is generally accepted that a toxin is involved in the aetiology of the disease and there are some experimental results to support this view (Gothe & Kunze, 1981, 1982; Nolte & Gothe, 1982; Schniewind, Gothe & Neu, 1983; Kraiss & Gothe, in press).

The first reference to this toxicosis is that by Lounsbury (1900). He quotes South African farmers who associated this tick with a temporary paralysis in sheep and goats. Du Toit (1917) casually mentions a possible connection between *R. e. evertsi* and human paralysis in South Africa. It is impossible to determine whether this tick was responsible for the cases of paralysis described by Anon. (1890), Ralph (1980) and Hellier (1892).

A more direct correlation between paralysis and *R. evertsi* was observed by Clark (1938), who proved for the first time that the removal of these ticks from affected animals resulted in their full clinical recovery within 2 days. Laboratory studies by Neitz & Jansen (1950), in which adult ticks were fed on sheep, provided further proof of the paralysis-inducing ability of this species of tick. Recovered animals develop an immunity which is apparently of short duration, as it was found that an animal may become paralysed again 60 days after recovery (Neitz, 1962).

Taking into account the ecological adaptability of *R. evertsi* (Hamel & Gothe, 1974) and its widespread distribution in the Afrotropical faunal region (Hoogstraal, 1956; Theiler, 1950, 1962), as well as its potential distribution (Anon., 1960; Bruce, 1962; Diamant & Strickland, 1967; Abu Yaman, 1978), one would expect a high and widespread incidence of *R. evertsi* paralysis.

However, the paralysis induced by *R. e. evertsi* has to date only been proved beyond doubt in the Republic of South Africa (Clark, 1938; Stampa, 1959; Neitz, 1962; 1964; Howell, 1969). A seasonal indidence has been noted in the Orange Free State (Stampa, 1959; Neitz, 1964), with sporadic outbreaks in the Transvaal, especially in the Highveld region, and in Natal (Neitz, 1964; Howell, 1969). In the Eastern Cape Province *Ixodes rubicundus* is regarded as the tick that is primarily responsible for paralysis but *R. e. evertsi* may also play a role (Neitz, Boughton & Walters, 1971). In Botswana and Zimbabwe it has been accepted, but without any definite proof, that *R. evertsi* can produce paralysis (Theiler, 1962). Notes which were thought to refer to the presence of this toxicosis in Zambia are in fact irrelevant (Gray, 1961). The impression that tick paralysis is limited to the Republic of South Africa was further supported by a general remark, after unconfirmed field and laboratory observations, that not all strains of *R. e. evertsi* possess a neurotoxin (Neitz, 1964).

Consideration of all the published data suggests that there may be differences in the paralysis-inducing ability of different strains of populations of *R. evertsi*. However, this has not been proved experimentally. It was therefore decided to test the toxicity of female ticks collected from different parts of South Africa for the first time under standardized experimental conditions, to establish whether or not there are any differences between strains or populations as far as their paralysis-inducing ability is concerned.

MATERIALS AND METHODS

For the exact quantification of the paralysis-inducing ability of wild strain populations of *R. e. evertsi* in South Africa, 3–6 months old, male and female Black-head sheep, which had been reared free of ticks, were used. Each sheep was infested with 40–60 male and the same number of female ticks. The experimental animals were kept in isolation cages with slatted floors to ensure the protection and recovery of the detached ticks.

Changes in the state of engorgement of female *R. evertsi evertsi* were quantitavely determined during the course of the infestation by taking measurements of the ticks at 24 h intervals. The criteria used were the body length, excluding the capitulum, the width at the 4th pair of legs and the greatest circumference of the ticks. These measurements were taken from the first day up to and including the 10th day after infestation. By referring to previous *in vitro* mass versus measurement data, the mass of each individual tick was calculated and recorded. The masses were used to determine the infestation rate necessary to induce a certain degree of paresis as well as to ascertain the mean total mass of all infesting female ticks, or of those in a paritucular repletion phase. Values were expressed as a quotient based on 1 kg body mass of the sheep. Clinically apparent degrees of paresis

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Received 16 August 1985—Editor

or paralysis were distinguished according to the method of Hamel & Gothe (1978). Gothe & Budelmann (1980) and Gothe & Lämmler (1982a). They are:

L I—slight degree of leg weakness, unco-ordinated gait.

L II—medium degree of leg weakness, moderate to high degree of body sway, buckling of the limbs, strong impairment of mobility.

L III—high degree of leg weakness, animal still attempts to rise to its feet but cannot stand. Crawling forward motions.

T.P.—total paralysis, animal permanently lying on its side, no further attempt to rise, head raising still possible, gnashing of the teeth.

T.P.(d)—total paralysis ending in death.

In this investigation, one population from Rwanda plus the following 15 wild strains of *R. evertsi evertsi* were used:

1. Duncan (North-western Cape Province)
2. Heidelberg (Transvaal–Highveld)
3. Pretoria (Transvaal–Highveld)
4. Warmbaths (Transvaal–Bushveld)
5. Boschpoort (Transvaal–Bushveld)
6. Bezuidenhout (Transvaal–Bushveld)
7. Mkuze (Natal–Zululand)
8. Boshof (Orange Free State)
9. Warrenton (Orange Free State)
10. Sweetwater (Orange Free State)
11. Jordaan (Transvaal–Highveld)
12. Tzaneen (Transvaal–Highveld)
13. Cullinan (Transvaal–Highveld)
14. Athlone (Transvaal–Highveld)
15. Nelspruit (Transvaal–Lowveld)

RESULTS

By simultaneously infesting 3–6-month-old Blackhead sheep with male and female *R. evertsi evertsi*, it was shown that all 15 South African strains as well as the Rwanda strain can induce paralysis (Table 1). A comparison of the number of female ticks in the repletion mass range of between 15–21 mg/kg sheep body mass (SBM) and the corresponding paresis or paralysis showed that there are no differences in toxicity between the strains. The time needed for the first clinical symptoms to manifest themselves different between strains, however. Only 4 days were required for the Warmbaths strain to produce clinical symptoms, compared with at least a 5-day period for all other strains.

TABLE 1 A comparison between the maximum degree of paresis and the infestation rate of females with an engorgement mass between 15 and 21 mg (A) as well as the total infestation (B) per kg sheep body mass of 15 wild strains of *Rhipicephalus e. evertsi* populations from South Africa and 1 from Rwanda

Population	Incubation period (days)	Maximum degree of paresis	(A) Infestation rate during engorgement between 15–21 mg/kg sheep body mass	(B) Total infestation/kg sheep body mass
Duncan	5	L III	1,85	2,01
Heidelberg	5	L I	0,22	0,92
Pretoria	5	L II	0,4	0,4
Warmbaths	4	L III	2,1	2,1
Warmbaths	4	TP (d)	3,3	3,3
Warmbaths	4	TP	1,4	1,43
Warmbaths	4	TP	2,27	2,46
Boschpoort	5	L III	1,95	2,05
Bezuidenhout	5	L I	0,23	0,42
Mkuze	5	TP (d)	1,5	1,5
Boshof	5	TP (d)	1,5	1,5
Warrenton	5	L I	0,22	0,29
Sweetwater	5	L II	0,23	0,94
Jordaan	7	L II	0,3	0,31
Tzaneen	6	L I	0,11	2,0
Cullinan	5	L II	0,53	2,14
Athlone	6	L I	0,3	2,1
Nelspruit	5	TP	1,4	1,6
Rwada	5	L II	0,31	1,3

TABLE 2 A comparison of 15 strains of *Rhipicephalus e. evertsi* from South Africa and 1 from Rwada regarding the mean mass of female ticks (mg) during their engorgement and the appearance of the first clinical symptoms of paresis

Population	Days after infestation									
	1	2	3	4	5	6	7	8	9	10
Duncan	8,2	9,2	10,2	11,8	18,7	184,6	400	700,2		
Heidelberg	8,5	9,2	10,2	13,4	20,6	80,4	320,2	635,7		
Pretoria	8,2	9,3	10,3	14,5	20,0	70,4	343,8	732,6		
Warmbaths	8,5	9,5	12,0	19,4	Ticks removed					
Warmbaths	8,0	10,0	12,2	14,7	44,5	179,9	618,1			
Warmbaths	7,6	9,5	12,9	18,5	39,2	Sheep died				
Warmbaths	7,3	10,3	14,3	17,1	58,6	Ticks removed				
Boschpoort	7,8	8,2	9,2	12,0	20,4	Ticks removed				
Bezuidenhout	8,2	9,0	11,1	14,8	17,5	28,2	123,6	412,8	702,6	
Mkuze	7,2	8,3	10,2	14,1	20,4	Ticks removed				
Boshof	7,2	8,2	11,2	13,8	20,5	Sheep died				
Warrenton	8,1	9,0	11,1	14,4	17,3	24,0	183,4	638,6		
Sweetwater	8,2	8,8	9,6	11,2	16,3	52,0	167,3	713,4		
Jordaan	8,0	8,2	9,4	10,4	12,4	19,6	40,0	70,6	232,6	715,4
Tzaneen	8,0	8,4	11,0	12,8	13,4	17,6	62,4	282,5	697,5	
Cullinan	7,8	8,2	10,4	12,6	16,8	58,7	115,5	234,1	657,1	
Athlone	9,0	10,1	11,0	11,5	12,8	20,8	36,0	127,7	638,4	
Nelspruit	8,2	8,4	9,6	12,8	18,6	58,6	213,4	344,4	712,2	
Rwada	8,0	10,1	10,8	12,8	19,5	40,8	74,4	329,5	677.4	

Whichever strain was used, the females initially engorged at the same rate, increasing continuously from 7,2–9 mg on the first day of infestation to 8,2–10,3 mg after the 2nd day of the engorgement period. After the 2nd day, however, the engorgement rate of the Warmbaths strain was substantially accelerated and the females reached 12,0–14,3 mg on the 3rd day, 14,7–19,4 mg on the 4th and 39,2–58,6 mg on the 5th day after infestation (Table 2). Initial paretic or paralytic symptoms appeared only after attainment of an engorgement mass of approximately 15 mg. This is indicated by the boxed figures in Table 2.

The induction, duration and intensity of the clinical manifestations are, correspondingly, not dependent on the period of infestation by female *R. evertsi evertsi,* but are determined only by the number of ticks with individual masses of 15–21 mg. The mean infestation rate of ticks in this mass range increased progressively for all tick strains from 0,11–0,3 females/kg sheep body mass, which produced a slight degree of leg weakness, to 0,23–0,53 ticks and 1,85–2,1 ticks for a medium and high degree of leg weakness, respectively, to 1,5–3,3 ticks for total paralysis (Table 1). Analogously, the average total tick mass in this engorgement range changed from only 2,1–3,4 mg/kg sheep body mass, for a slight degree of leg weakness to 2,5–9,8 mg and 29,6–38,7 mg for medium and high degree of leg weakness, respectively, to 21–60 mg for total paralysis.

Discussion

To clearly understand the involved clinical symptoms of tick paralysis one has to take into consideration both the biotic and the abiotic factors which may affect both the vertebrate host and the responsible tick species. These determine the severity of paralysis or paresis as well as its persistence, seasonal occurrence and geographical distribution. As far as the host is concerned, factors playing a role are its susceptibility, age, behaviour and the position of the reaction as well as its degree of immunity. In the case of the ticks, their population density, dynamics and pathogenicity, as these affect the degree of infestation, sexual activity, stage of development, site of attachment, and state of engorgement, are of prime importance (Gothe, 1981). As far as these biotic aspects with their changing and complex interactions are concerned only a few rather unsatisfactory investigations have been reported on single tick strains inducing paralysis. This applies especially to differences in the paralysis inducing-ability of different populations of tick species and thus also to *R. e. evertsi*.

The present study, based on 15 strains from South Africa and 1 from Rwanda, shows that, contrary to the opinion of Neitz (1964), there are no population or strain-dependent differences in the toxicity of *R. e. evertsi*. These results were obtained after comparing the onset of paresis or paralysis with certain phases of engorgement. The extent and intensity of clinical signs were almost identical when ticks in their toxic phase were compared. Gothe & Lämmler (1982a) found that, with the Nelspruit strain, engorging females become toxic only when they had reached a mass of 15–21 mg. Moreover, the clinical signs that develop are also related to the number of females of this size/kg body mass of sheep (Gothe & Budelmann, 1980).

This classification of ticks into groups of increasing toxicity, based on the work done on the Nelspruit strain by Gothe & Lämmler (1982 a), is also applicable to the other tick strains we have now tested. Clinical symptoms appeared only when female ticks had reached an engorgement mass of 15–21 mg. Furthermore, paresis and paralysis followed the same course as regards the time period and the intensity of reaction after the removal of all female ticks from affected sheep. This was studied in the case of females from the Mkuze, Boschpoort and Warmbaths strains which, after reaching this engorgement state, were removed in order to save the sheep.

Thus, regardless of the origin of the ticks, the intensity of symptoms always increased directly in proportion to the infestation rate of female ticks with an engorgement mass of 15–21 mg. Experiments with Tzaneen, Cullinan, Athole and Heidelberg strains also showed that toxicity is dependent on the restricted uniformity of the ticks present, which does not support the assumption of strain or population differences in the paralysis-inducing ability of this tick species. In spite of a high total infestation, only a low or medium degree of leg weakness was produced. However, taking into account the fact that only the ticks with an engorgement mass of 15–21 mg are toxic, it is always possible to equate infestation values/kg sheep body mass when the different strains are compared.

A comparison between *R. evertsi* and other paralysis-producing Ixodidae can be made only with certain reservations. This is mainly because *in vivo* studies have not been done, and also because published data are in some cases contradictory. Because of the regional distribution of *Dermacentor andersoni* paralysis, intraspecific differences in paralysis-inducing capacity is accepted (Parker, Philip, Davis & Cooley, 1937; Farquharson & Bly, 1939; Gregson, 1943, 1953, 1956, 1962, 1966, 1973; Abbott, 1944; Ransmeier, 1949; Jellison & Gregson, 1950; Brunetti, 1965; Loomis & Bushnell, 1968; Rich, 1971). These differences in paralysis-inducing ability have been ascribed to mutation and/or to specific ecological conditions (Mail & Gregson, 1938; Neitz, 1962; Wilkinson & Lawson, 1965; Gregson, 1973). Besides environmental influences and ecological interactions (Gregson, 1936), both strain differences in the settlement areas (Wilkinson, 1972) and changes in the salivary secretion (Gregson, 1957) were considered, or referred to in general, as reasons for the variation in pathogenicity of this species (Gregson, 1936; Stiller, 1946; Boffey & Paterson, 1973; Bennett, 1974; Mongan, 1979). An initial, short-lived increase in the pathogenicity of these ticks, which were supposed to have originated in the north-western part of this strain's distribution area, was also discussed (Gregson, 1962, 1973; Rich, 1971).

Dermacentor occidentalis is widely distributed in California but is supposedly responsible for paralysis in a limited area only (Brunetti, 1965; Loomis & Bushnell, 1968). *Dermacentor variabillis* is found in the western part of the USA, as far as the Rocky Mountains, but cases of paralysis caused by this species were almost all described from the south-eastern part of its distribution zone (Mulherin, 1940; Ransmeier, 1949; Gregson, 1962). In this case also, an intraspecific difference in paralysis-inducing capacity was postulated (Wells, 1935; Randolph, 1947; Gregson, 1952). Furthermore, a decline in the toxicity of this tick species in the northern part of its distribution area was discussed (Gregson, 1962, 1973).

In the case of *Ixodes rubicundus* it has been assumed that only certain populations have the ability to cause paralysis or paresis (Anon., 1923; Neitz, 1964). It was shown that some animals of the same species could tolerate up to 100 ticks (Van Rensburg, 1928; Neitz *et al.*, 1971) while others were paralysed by a single tick (Van Rensburg, 1928). However, the frequency of occurrence of these potent ticks was difficult to quantify because it varied with respect to locality and time (Stampa & Du Toit, 1958; Stampa, 1959).

For *Ixodes holocyclus* a similar intraspecific variability of the paralysis-inducing ability was suspected (Cleland, 1913; Dodd, 1921a,b; Ross, 1926; Roberts, 1941; McCarthy, 1958; Seddon & Albiston, 1968; Cooper, Cooper, Ilkew & Kelley, 1976). No differences in toxicity either between geographical strains or between laboratory-reared and wild collected ticks could be determined though (Doube, 1975; Doube & Kemp, 1975; Doube, Kemp & Bird, 1977; Stone, Doube, Binnington & Goodger, 1979).

With respect to *Ixodes gibbosus,* Hadani, Tsafrir & Shimshony (1971) considered a possible climatic influence on toxicity, and suggested that the existence of certain genetic strains cannot be excluded. According to Nelson (1973), individual specimens of *Ixodes pacificus* within a population can become capable of inducing paralysis. This ability, however, can be lost again after a short time, probably during 1–2 seasons.

For *Ixodes ricinus* a strain specificity is likewise postulated (Hoffman, 1973).

It may be concluded from the literature that the existence of genuine intraspecific differences in toxicity cannot be totally excluded. Substantial evidence for this form of biological plasticity in tick species does not, however, exist at present.

A comparative description of toxicity with a temporal and topographical relation to toxin synthesis is not possible for the other paralysis-inducing ixodid tick species (Gothe, 1984). This is either because corresponding experimental investigations are still lacking, or because of shortcomings and errors in methodology, e.g. where resistant or tolerant vertebrates have been used as toxin indicators. Furthermore, bearing in mind the direct proportional relationship between the size of inoculum and body mass, toxin doses that are too low have sometimes been administered. In other cases, potential paralysis tick material outside the toxin engorgement state have been prepared and used. It is also important that ticks should be allowed to attach freely in order to clinically prove the toxicity. It has been shown (Gothe, Gold & Bezuidenhout, 1986) that, when the infestation is concentrated on a small, restricted skin area, even slight disturbances in the mobility of highly sensitive vertebrate hosts cannot be induced, despite a normal engorgement process and high infestation rate which should be sufficient to cause lethal paralysis.

Despite the seasonal periodicity of paralysis caused by *R. e. evertsi,* neither quantitative strain- or population-dependent gradations of the paralysis-inducing ability nor the regional existence of non-toxic ticks in South Africa can be deduced from the present investigation. The seasonal peak in the appearance of this toxicosis can be explained by the ecological and biological regulatory factors in the development of this tick species. Adult *R. e. evertsi* appear seasonally, predominantly at the beginning of spring (MacLeod, Colbo, Madbouly & Mwanaumo, 1977; Londt, Horak & De Villiers, 1979). Sheep are thus simultaneously infested by both sexes. As a result of the extremely high pathogenicity of this form of infestation, the animals contract paralysis sooner and more severely (Gothe & Budelmann, 1980). As was noted above, a direct correlation exists between infestation rate and body mass, and thus with the age of the host (Hamel & Gothe, 1978; Gothe & Budelmann, 1980; Gothe & Lämmler, 1982a). Consequently this toxicosis becomes clinically manifest primarily in lambs, thus giving rise to the name "spring lamb paralysis" (Clark, 1938; Stampa, 1959; Howell, 1969). Later in the season the animals are gradually infested asynchronously by adults because the males remain attached longer (Londt & Van der Bijl, 1977). A resident population of sexually mature male ticks thus exists, which could result in a reduction of up to 50 % in the pathogenicity of female ticks (Gothe & Budelmann, 1980).

In addition, a temporary antitoxic immunity becomes effective and this may be constantly boosted through reinfestation. The immunity is thereby prolonged and becomes persistent over the entire tick season (Gothe & Lämmler 1982b). As a result of the combined action of reduced pathogenicity of female *R. e. evertsi,* because of the preinfestation by male ticks, and the antitoxic immunity, the natural incidence of this toxicosis is reduced. Frequent weak or clinically inapparent symptomatology develops and the actual toxicity of the female ticks is therefore masked.

Whether, or to what extent, other possible ecological factors or additional mechanisms inherent to ticks regulate the prevalence of *R. evertsi* paralysis is unclear. Similar influences on the natural frequency of this form of toxicosis for other paralysis-potent ixodid species should be considered.

ACKNOWLEDGEMENTS

We wish to thank Miss Heloise Heyne for her expert technical assistance, Mrs Sigrun Bezuidenhout and Prof. A. W. H. Neitz for their help in the translation of the original manuscript, Mr A. M. Spickett and Dr E. M. Nevill for criticizing it and Dr J. B. Walker for her editorial expertise.

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Printed by the Government Printer, Private Bag X85, Pretoria, 0001.