THE ENIGMA OF TICK PARALYSIS [1].

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Introduction

The object of this paper is to bring together observations on a series of tick paralysis experiments that have been conducted at the Livestock Insect Laboratory of the Dominion Department of Agriculture, Science Service; to compare them with the forms of tick paralysis that occur in other countries, and by these comparisons to clarify our impressions regarding the true nature of the disease. These experiements, often negative and at times apparently producing contradictory results, at least do emphasize the fascinating mystery of one of British Columbia's main livestock pests, the wood tick, *Dermacentor andersoni* Stiles.

Tick paralysis in British Columbia is a flaccid ascending motor paralysis that may be produced in livestock or humans by the feeding on them of one or more female ticks of the species *D. andersoni*. The symptoms do not occur until about the sixth day after the tick has attached, and progress from then on until the tick drops off replete, or is removed, after which there is usually a rapid recovery within half to two or three hours. Death may ensue if the respiratory center becomes paralysed before the tick leaves the host.

Tick paralysis also occurs in Australia, where man, sheep, dogs, pigs, cats and poultry have been reported by Ross (1935) as victims of *Ixodes holocyclus* Neumann, and in South Africa, where sheep have been recorded by Rensburg (1928) as having been paralysed by *Ixodes pilosus* Koch. Clark (1938) has also recorded paralysis in the same host, caused apparently by the African tick *Rhipicephalus evertsi* Neumann. In Yugoslavia, Mlinac and Oswald (1936) (1937) have described tick paralysis in ruminants, the vectors being *Hyalomma aegyptum* L. (Neumann), and *Boophilus calcaratus balanicus* (Minning). The symptoms produced by these foreign species are in a general way similar to those of the disease in this province. In America the disease is confined almost entirely to the ranching areas of British Columbia, and although the causative tick is just as abundant in southern Alberta as well as in Montana and other states to the south, tick paralysis is relatively scarce there.

Although considerable speculation on the etiology of American tick paralysis has been advanced, the fundamental cause of the symptoms still remains as much of a mystery as ever. It would seem that no theory attempting to explain tick paralysis has been put forward without arousing conflicting evidence.

Theories As to Cause of Paralysis

Briefly and in order of their probable validity, the main theories as to the cause of paralysis are here set forth.

1. *Paralysis of the nervous system by a toxin introduced by the feeding tick.*

This is the most generally accepted theory. It is substantiated by the fact that there is an almost immediate recovery following the removal of the tick, possibly due to the cessation of further injections of toxin. Also in support of this theory, assuming that Australian tick paralysis is similar to the British Columbia form, are the experiments conducted by Ross (1935). He found that the salivary glands of *Ixodes holocyclus*, ground and injected into mice, produced symptoms similar to those caused by the ticks feeding on dogs. The Australian disease, however, differs from that in Canada in that the paralysis appears to be more often fatal, and according to Ross (1934) may not even become ap-

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parent until *after* the causative ticks are removed. Opposed to the toxin theory are the facts that not all British Columbia ticks appear to be capable of producing paralysis, and that animals capable of being paralysed by a single tick under ideal conditions can tolerate several dozen fast feeding ticks with no ill effects—a number that surely would inject far more poison than the most virulent individual tick. Ross (1935) makes no mention of *holocyclus* varying in its power to produce paralysis, other than stating that dogs were less frequently paralysed by large numbers than by single females. This would probably be due to individual resistance in the animals, for it would appear from his infesting experiments with laboratory animals that the symptoms of paralysis become increasingly severe with heavier infestations.

2. *Infection of host by some tick-borne agent.*

This is suggested by the fact that only ticks in certain territories (British Columbia as compared with Montana for example) and of these, only certain individuals, apparently are capable of producing paralysis; and by the fact that a six day period, resembling an incubation period, elapses between the time when the tick attaches and the onset of paralysis. In the dozens of observations made at this laboratory, paralysis has never been observed in which this period is less than six days, or more than eight. In the case of Australian tick paralysis, this period may be shortened to four days. Rensburg (1928) in South Africa also observes the fact that only certain ticks are capable of producing paralysis. He states "one is inclined to accept the theory that an infected tick produces paralysis in an animal by slowly injecting into it a narcotic poison which was obtained by the tick in some way or other before it got on to that animal." Mlinac and Oswald (1937) in Yugoslavia, have succeeded in killing guinea pigs by injecting them with crushed and filtered eggs of the European paralysis-producing tick, *B. calcaratus balanicus*. These experiments, but for the fact that the egg emulsions did not contain pathogenic organisms, might have suggested the passage of an infective agent through the eggs of ticks. As it is, the results appear similar to the more recent findings in America where it has been discovered by Gregson (1941) and by Steinhaus (1942) that the eggs of *D. andersoni* contain elements that are toxic to guinea pigs. Opposing the infection theory is the rapid recovery upon the removal of the tick.

3. *Mechanical injury to host by tick feeding over nervous system.*

This theory is widely credited by ranchers who have observed that ticks usually congregate along the spines of paralysed animals. Unfed ticks being negatively geotropic naturally gravitate to this region, but it is well known as pointed out by Mail and Gregson (1938) that paralysis may ensue when the causative tick is attached to any part of the body.

Discussion

Discussing the first and most logical theory as to the cause of tick paralysis, it seems fairly evident that under certain conditions a female wood tick is able to inject a toxin into the blood stream of the host which will paralyse its motor nerves. It would also appear that this toxin is injected only immediately prior to the repletion and subsequent dropping of the tick, being perhaps some form of regurgitated fluid, or possibly a glandular secretion to aid in the release of the tick's mouth parts. Incidentally, it is not understood yet how a tick, so firmly attached to its host by its immovable hypostome teeth, is able to release itself at will when replete, or even before repletion as happens when the host dies and becomes cold. The paralysing toxin probably is readily destroyed by the host, which would account for the rapid abatement of paralysis when the tick is removed. It may even be possible that the host is sometimes sufficiently resistant to cope with

the toxin and thus escape paralysis entirely. This would obscure the fact that the tick might have been potentially capable of producing paralysis. While, as stated, paralysis usually is produced only by ticks on the verge of repletion, there are records of ticks only half or one third engorged having caused the symptoms. There is even a record of a lamb being paralysed when a close examination revealed no other ticks than a male.

Ross (1935) in Australia, killed mice by injecting them with crushed salivary glands of *I. holocyclus*. These experiments were repeated in the Kamloops laboratory with *D. andersoni* but the results were not the same. Even when several times as many glands as used by Ross were dissected from fast feeding ticks (frozen at the time of removal from host to prevent any possible deterioration of a labile toxin) and injected intravenously and subcutaneously into mice and lambs, and intraspinally into puppies, the results continued negative. Similarly abortive have been the attempts to produce paralysis in lambs by injecting into them the crushed bodies of ticks that have been known to have produced paralysis. The latter injections were made alongside other engorging females in case some causative virus was dependent upon the simultaneous presence of tick venom in the host animal for its survival.

That only certain ticks appear to be capable of producing paralysis is demonstrated by the fact that often a *series* of ticks, feeding at the same time, may not paralyse an animal. The theory of acquired immunity does not detract from this observation, since on several occasions the feeding of numbers of ticks on previously uninfested lambs, which obviously would have no such immunity, has produced no paralysis. Moreover, in British Columbia one attack of paralysis does not necessarily produce an acquired immunity to subsequent attacks. Ross (1935) tates that the Australian tick *I. holocyclus* may produce an acquired immunity, but that it is not inherited. Such a resistance has been produced experimentally in guinea pigs against early stages of *Dermacentor variabilis* by Trager (1939) and in fitches and guinea pigs against *I. texanus* and *D. andersoni* by Gregson (1942).

Whether an inherited immunity towards *D. andersoni* exists is debatable. It is the common belief of ranchers in British Columbia, as quoted by Mail (1942), that animals exposed for several years to attack by ticks do build up an immunity, so that later ticks attacking such animals do not feed so readily. Indirectly this may lessen the chances of paralysis by eliminating fast feeding ticks. A slowly built up and inherited immunity might explain why wild deer, mountain sheep and moose, though often heavily infested, never appear to be paralysed by ticks. Such an inherent resistance however, may not be wholly responsible for the slow feeding rate of certain ticks, as is shown by tests made on Vancouver Island sheep, untouched for generations by ticks, and on which a dozen of these parasites, in July, were unable to feed rapidly.

Individual host susceptibility to ticks has been noted on a few occasions. In one series of infestations fifteen of sixteen ticks engorged in seven days on one lamb, while the average feeding period on six other lambs was nine days, with fifty per cent of them dying before repletion. Ross (1935) has also noted a variation among hosts and states that certain individual dogs are more resistant to *holocyclus* than others.

Nevertheless, although the condition of the host may play a part in the varying rate of tick feeding, it is also a definite fact that a large degree of these variations are due to the tick. This has been demonstrated in experiments by Gregson (1937) where specimens of *andersoni* were observed to feed at different rates in close proximity, on the same host.

It would thus appear from the foregoing remarks that certain ticks, and only certain ticks, are capable of pro-

ducing paralysis; and that these are usually fast feeding individuals. The condition necessary for this rapid feeding seems to be physiological, and either inherent, or produced in the tick by external stimuli. Since in the unfed condition potential paralysis producing ticks cannot be distinguished from harmless ones, an attempt was made to approximate the percentage of virulent ticks in a locality where paralysis normally occurred. Fifty week-old lambs each were infested with a pair of ticks from the same vicinity. The experiment was a failure, in that none of the ticks fed fast and no paralysis resulted. Nevertheless, a week later, ticks of the same stock *all* fed rapidly and paralysed the lamb upon which they were caged. The lamb was from the same farm as the previously infested stock. The weather, slightly warmer at the time of the second infestation, could hardly account for this difference in feeding, as experiments have shown that varying external temperatures have no effect on the feeding rate of ticks on sheep. It was thought that perhaps very young lambs might have been more resistant to ticks and thus have accounted for the slow feeding rate on the fifty lambs, but similar tests a year later on ten ewes and their twin lambs showed the parasites to feed no more readily on adult sheep than on lambs.

Perhaps the greatest hope of progress towards an understanding of the primary cause of tick paralysis lies in the fact previously stated that this disease as a rule is produced only by fast feeding ticks. One observation may prove significant and afford a clue, and that is the progressive inability of ticks in British Columbia to feed readily with the approach of mid-summer and fall. This phenomenon which, as shown by Gregson (1937), appears to be due to an inability on the part of the tick to produce sufficient disturbance within the host tissues to liberate an adequate blood supply, will probably be found to be closely connected with some climatic condition. Gregson (1938) noted that when ticks were subjected to increasing doses of ultraviolet light, their feeding powers were stimulated. Smith and Cole (1941) conclude from their recent experiments that the length of day is an important factor in controlling the activity of hibernation of larvae and nymphs of *D. variabilis*, that long photoperiods are more favorable to activity than short ones, and gradually increasing photoperiods more favorable than gradually decreasing ones of even greater absolute length. Rowan (1929) has shown that the length of light-day has an important effect on the physiology of birds. Similar light experiments on tick hosts, in which sheep were subjected to decreasing photoperiods, have shown, however, that any effect produced in them does not influence the feeding rate of the tick. Nor, according to Carrick's (1940) experiments with hedgehogs, does there appear to be any relation between the ticks' feeding rate and the presence or absence of sex hormones in the blood of the host. This theory has been suggested by MacLeod (1932) and by Rowan and Gregson (1935).

Bruce (1925) states that it is claimed that more cases of paralysis develop when there is an extreme range between the maximum and minimum temperatures, and suggests that this might induce a healthier appetite in the gorging female. At the Rock Mountain laboratory of the U.S.P.H.S., it has been noticed also that ticks feed more readily when subjected to a series of cold temperatures prior to infesting.

Thus temperature as well as light—and perhaps even humidity and other conditions not yet understood—may enter into the picture of tick behaviour, and these even may not act as stimuli until they are varied from their usual intensity in such a way as to disturb the rhythm of the individual. Considering how nebulous is our conception of such possible forces produced by fluctuations or gradual changes of external conditions, and regarding the apparently contradictory

nature of the etiological evidence of tick paralysis, it will be seen that the fundamental mechanism of this disease is most intricate and perplexing.

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The Weevil Auletobius congruus (Walker) a Pest of Strawberries

In May, 1940, Mr. C. R. Barlow, Provincial District Field Inspector, drew my attention to serious damage being done to a strawberry patch at Salmon Arm, by a small dark weevil. Specimens of this insect were sent to Mr. W. J. Brown, of the Division of Systematic Entomology at Ottawa, who identified them as **Auletobius congruus** (Walker).

An examination of the patch on May 18 showed some damage over about four acres; on half an acre about 35% of the blossoms were destroyed. The weevils were seen to cut into the base of the blossoms, causing the flowers to wilt and die.

The owner of the patch had never seen this beetle on his strawberry plants before and it has not appeared since. Examination of the available literature shows no reference to this weevil as a strawberry pest, and its sudden appearance in injurious numbers in 1940 may be of interest. Professor G. J. Spencer has found adults feeding on the flowers of a native buttercup, **Ranunculus** sp., on the hills near Aspen Grove, B.C.—E. R. Buckell, Field Crop Insect Laboratory, Kamloops, B.C.

CULICOIDES GIGAS R. & H. AT VERNON, B.C. (Diptera:Ceratopogonidae). This species was recorded from the Kamloops district by Curtis (1941. Ent. Soc. Brit. Col., Proc. 37:19). At dusk on May 7, 1942, the flies were common at a small pond on the hill above Goose Lake, and adjacent to Gartrell's mine. They settled on the neck and arms, but did not bite. Specimens were identified by Mr. A. R. Brooks of the Division of Entomology at Ottawa.—Hugh B. Leech.