clusters of ten to 50 ticks and it is doubtful whether these animals could be paralysed by solitary specimens. However, not all animals with heavy infestations will become paralysed — suggesting again an individual resistance.

The data for lambs 10, 13, 14, 18, and 19 substantiate a variation in virulence among ticks. At the same time, there is an indication that a paralysed animal is so weakened that it may then be affected by ticks, which on a healthy animal, would not be considered virulent. This is evident in animals on the range which often remain prostrate for several hours until the last ticks are removed, and then make a rapid recovery. This could be the case in lamb 13.

Lamb 9 presented an interesting opportunity for a further experiment in that paralysis commenced when the two engorging ticks were only half-replete. The ticks were transferred to lamb 1, which, though having been subjected to ticks, had not yet been paralysed. The virulent ticks attached, and dropped within a day—but did not cause paralysis. Similar results occurred in a previous year when a partially fed tick from a paralysed child was induced to attach to a mouse, whereupon it mated and fed rapidly without harm to its second host. Again there are too many unknowns to establish a reason for this behaviour. It appears that a virulent tick can paralyse only the host upon which it commences to engorge.

In summary, it is assumed that tick paralysis is brought about in the host by a toxin secreted by the engorging tick. It appears that the production of tick paralysis in an animal depends on a combination of host susceptibility and tick virulence; and that where the host is relatively resistant, as in sheep and cattle, only a certain portion of rapidly feeding ticks produce the symptoms, whereas in humans, considered to be more susceptible, solitary, slow-feeding ticks are sufficient to cause paralysis. The conflicting evidence concerning the varying resistance of the host and the unknown virulence of the ticks makes it difficult to arrive at any conclusion.

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A REVIEW OF STUDIES ON THE SYSTEMIC CONTROL OF LIVESTOCK INSECT PARASITES[1]

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If, without causing harm to the animals, it were possible for a stockman to introduce into the diet of his livestock some substance that was toxic to bloodsucking arthropods feeding upon these animals, he would find a solution to one of the important problems of animal husbandry. Economic entomologists have recently devoted a great deal of effort toward the attainment of this ideal, and the investigations seem to indicate that it may be possible to achieve it.

However, the problem is not simple. Many difficult biochemical and toxicological factors must be studied and their roles elucidated before the problem can be solved. Because of these factors, the entire matter must be approached with caution. A full discussion of these aspects is beyond the scope of this paper because it is my intention only to review some of the more important studies that have been made in the past.

The first important contribution is that of Knipling (1938), who reported

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that, when cattle ingested phenothiazine, horn fly larvae did not develop in the manure subsequently eliminated by such animals. Bruce (1939) confirmed these findings and established the dosage levels required for maximum control. Creighton (1943) made an unsuccessful attempt to control chicken lice by adding sulphur to the concentrate portion of the poultry diet.

The recognition of the insecticidal properties of DDT and BHC instituted widespread investigations of the insecticidal properties of many organic chemicals, and the systemic aspect was not neglected. Linquest (1944) reported that oral ingestion by rabbits of either DDT or pyrethrum in massive dosages was toxic to bed bugs subsequently fed on the rabbits. de Meillon (1946) made a similar observation concerning the bed bug feeding on rabbits that had ingested the gamma isomer of BHC. This observation was confirmed by Wilson (1948), who administered it orally as a control for tsetse flies on cattle.

J. D. Gregson (personal communication) noted that ingested gamma isomer of BHC was toxic to ticks and lice feeding on horses, cattle, and chickens. This observation, together with the published data of deMeillon and Wilson, provided the basis for an extensive series of tests conducted at the Kamloops laboratory in the summer of 1949.

During that summer I carried out a test of the gamma isomer of BHC, using domestic hogs and their natural parasite, *Haematopinus suis* (Linn.), as experimental animals. Single dosages at the level of 40 milligrams per kilogram of animal body weight provided 100 per cent. control of the parasite for about eight to ten days. Daily dosages at the level of 20 milligrams per kilogram for four days were required to produce similar results. However, a series of toxicity tests, with guinea pigs as the test animals, showed that daily treatment at dosage levels of 20 milligrams per kilogram was a dangerous practice. It is perhaps invalid to extend experimental results determined with one animal species to other, dissimilar species; but since it is economically impossible to conduct satisfactory toxicity tests with animals such as cattle, horses, and hogs, it was decided that in this instance the extension was justifiable. Therefore these tests were considered to have indicated that this treatment was not a valid control method.

In the meantime other workers had conducted preliminary tests with many other chemicals. Knipling (1948) published the results of an extensive series of tests involving many different chemical compounds in an attempt to control the body louse, *Pediculus humanus corporis* Deg., feeding on rabbits. The most promising of the compounds tested was 2-pivalyl-1, 3-indandione. Babers (1949) published a favourable report of tests conducted with this chemical, using the same experimental animals.

This review of the systemic aspect of livestock parasite control is brief; however, it indicates that the problem is not being neglected and suggests a concrete basis for its solution.

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