# OBSERVATIONS ON AN OUTBREAK OF CLINICAL PARASITISM IN EWES DURING THE WINTER MONTHS

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LATE IN THE WINTER of 1962 a number of deaths attributable to parasitism, mainly by Haemonchus contortus, occurred in a flock of breeding ewes at the branch laboratory, Macdonald College, Quebec. These deaths took place over a relatively short period between March 26 and April 6. The first three animals that died were given only a cursory examination but at that time it was noticed that they appeared anemic. Subsequently, when two more ewes died, a careful necropsy was performed. It was then observed that these animals had been anemic at the time of death and examination of the gastrointestinal tract revealed large numbers of nematodes, principally *H. contortus*, in the abomasum. The number of worms present in the abomasums of these two ewes are shown in Table I. A number of the H. contortus were immature forms, mainly fourth stage  $(L_4)$  and early fifth stage  $(L_5)$ . At this time the other ewes in the flock were checked for parasitism and blood samples were taken from some of them. The results of the haemoglobin examination on two ewes showing evidence of anemia and high faecal egg counts are compared with corresponding values from a ewe, which apparently was lessheavily parasitized in Table II.

TABLE I

HELMINTHS RECOVERED FROM THE ABOMASUMS OF TWO EWES THAT DIED OF HELMINTHIASIS

Ewe	Stage of worm	Haemonchus	Ostertagia	T. axei	Unidentified larvae
1	adults	$15590 \\ 3520$	4010	4250	3070
2	adults larvae	6780 1280	1280	1290	1920

TABLE II

Results of Strongyle Ova Counts and Haemoglobin Determinations on Two Heavily Parasitized Ewes and One Lightly Parasitized Ewe for Comparison

Ewe	Egg count/g of faeces	Haemoglobin/mg $\frac{7}{c}$
3*	10600	3.3
4*	5000	5.8
5	300	8.9

\*Died in spite of treatment with phenothiazine.

The heavily parasitized ewes were subsequently treated with phenothiazine but they were in the terminal stages of the disease and did not recover. A total of seven ewes died, six of which were close to the termination of their pregnancy or had just lambed.

This outbreak appeared to be typical of the type described by other workers as

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due to the spring-rise phenomenon which seems to be of world-wide occurrence in sheep. The spring-rise phenomenon is manifested by a sudden increase in the magnitude of the faecal egg counts and is often associated with an outbreak of clinical parasitism. The phenomenon was first reported by Taylor (21) in England. Field *et al.* (7) have presented an excellent discussion of the topic and reported its occurrence in housed sheep that had been denied access to infective material for three to five months prior to the outbreak. Death from haemonchosis was seen in one of their sheep.

A number of theories have been advanced to explain this phenomenon. Some workers considered it to be due to an increase in fecundity of adult worms present in the sheep (4, 9, 21). Some have suggested that it can simply be accounted for by variation in the number of worms in individual sheep (10), or that it is due to adults derived from recently acquired larvae (11). Crofton (2) postulated a period of declining immunity during which there was a slow build-up of larvae in the gut. A development en masse then occurred after the immune status fell below a certain critical level and the worms reached patency simultaneously, resulting in a marked and sudden rise in faecal egg-count levels. The most acceptable view appears to be the one which maintains that the spring-rise is due to a sudden development of adults from larvae present as quiescent histotrophic stages in the tissue of the gut. A number of workers (1, 8, 16, 23) have shown that such stages do occur among parasitic nematodes. In the case of H. contortus there is no definite evidence that there is a persistent histotrophic stage. In fact Stoll (18) reported that the histotrophic stage of this worm was brief and rela-tively invariable in duration. However, Silverman and Patterson (15) have reported that retarded development can occur in resistant sheep and Field et al. (7) state that in view of their findings, *H. contortus* does have a persistent histotrophic stage.

Ancilliary observations made during the present outbreak support this contention. Lambs, raised parasite-free, were housed continuously with the ewes during the winter months as a check on the acquisition of the infective stages of nematodes during this period. No *H. contortus* were recovered from any of these lambs either previous to or during the outbreak of haemonchosis in the ewes; an indication that larvae were not available for infection at this time. This was expected since the highest temperature recorded during this period at the surface of the litter was 44° F., a temperature that is too low for the development of *H. contortus* larvae (5). It therefore appears that the worms must have been acquired by the ewes before they were stabled and persisted as adults or quiescent larvae.

Even though consecutive egg counts were not made to demonstrate a rise in count during the stabling period, it is unlikely that the ewes could have survived for long with patent infections of adult worms of the magnitude that were observed as it was some three to four months between stabling time and the onset of death. In the case of H. placei in cattle, Roberts (12) has demonstrated resistance to infection in calves raised worm-free and subsequently exposed to single, spaced and daily doses of larvae. On slaughter, several weeks after challenge, most animals showed only a few adult worms, but many fourth- and early fifth-stage larvae. Subsequently, Roberts and Keith (13) reported that while

it was possible to superimpose infections on existing infections, development of these secondarily acquired larvae did not extend beyond the fourth and early fifth stage. These forms could, however, persist for a long time in the host. An analagous situation probably existed in the case of H. contortus in the present instance. All of the ewes had participated in an experiment the previous summer in which they had been exposed to severe challenge from H. contortus. Ample opportunity was present, therefore, for them to have acquired heavy infections with these parasites. Probably, as a result of the heavy continuous exposure, a strong immunity prevailed resulting in the inhibition of development of numbers of larvae.

At this point, it is interesting to speculate about the mechanism underlying the spring-rise phenomenon. Soulsby (19) has shown that it is preceded by a fall in antibody titre and Stewart (17) has demonstrated an inverse correlation between a rise in egg count and serum antibody titre. A number of workers agree that the inhibition of larval development appears to be a manifestation of host resistance (1, 14, 19). Some stimulus must be necessary to maintain a sufficiently high level of immunity to suppress development of these larvae. In the case of Trichonema spp. in horses, Gibson (8) has shown that the presence of adult worms probably serves this purpose. Roberts (12) has also shown that the  $L_4$  and  $L_5$  stages of H. placei, after a period of delayed development in the presence of adult worms, will resume development when placed in an animal not previously exposed to infection. Further, Roberts and Keith (13) demonstrated that removal of adult worms with phenothiazine permitted the phenothiazine resistant  $L_4$  and  $L_5$  stages to complete development. Dineen (6) has recently shown that certain antigens present in larval H. contortus are also common to the adults and states that this further supports the contention that control of larval development could be exercised by the adult worms. The spring-rise phenomenon might, therefore, be due to the gradual spontaneous loss of adult worms during the winter months with a consequent decrease in the antigenic stimulus provided by these adults. It can then be postulated that, once the antibody levels fall below a certain critical point, the quiescent larvae could complete development and patent infections would result.

It has been shown by Silverman and Patterson (15), however, that the greatest antigenic stimulus is provided in sheep by the  $L_4$  and  $L_5$  larval stages of *H. contortus* rather than by the adults. It has also been shown that the moulting fluid produced by the third stage ( $L_3$ ) larva is highly antigenic (20). As an alternative hypothesis, the stimulus necessary for the maintenance of a high state of immunity might have been provided by the constant exposure of the ewes to infective  $L_3$ larvae on herbage during the summer with consequent invasion, moulting and partial development in the gut. Removal of this stimulus with the onset of winter could have resulted in a gradual loss of immunity, until a critical level was reached below which the development of histotrophic larvae could resume. The added stress of parturition would probably help to precipitate this condition. Both Crofton (3) and Field *et al.* (7) observed that spring-rise was more pronounced in breeding ewes than in other sheep and that it appeared to be enhanced by a stress factor such as parturition. In the present instance six of the seven ewes had recently lambed or were on the verge of lambing.

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These findings constitute supporting evidence of the importance of histotrophic stages in the epidemiology of parasitic gastroenteritis of sheep.

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