# New Concepts in Helminth Control in Cattle

**Professor J. Armour** 

Dept. of Veterinary Parasitology University of Glasgow Scotland

#### Introduction

Traditionally, attempts to control the helminths of cattle have been applied in a haphazard fashion and usually involved the routine application of anthelmintics on a convenience basis. For example, in temperate climates where animals are housed during winter, anthelmintics would be given at turnout to spring grazing, at housing in the autumn and perhaps in the middle of the summer grazing period or when clinical signs appeared. In the sub tropics or tropics where cattle graze the year round and under more extensive conditions, treatments were applied when the animals were routinely gathered for other purposes such as vaccination, spraying or dipping to control ectoparasites, or when clinical signs of helminthiasis became apparent.

Although these convenience treatments have sufficed on many farms, they have failed in others and it has become evident that the prevention of helminthiasis, rather than the treatment of animals once infection has occurred, has considerable production benefits.

## The Helminths and Their Life Cycles

Before discussing the ways in which helminthiasis can be prevented, I would like to emphasize the most important helminths of cattle on a worldwide basis and outline their life cycles. In temperate areas they are the gastrointestinal nematodes Ostertagia ostertagi and Cooperia oncophora, the lungworm Dictvocaulus viviparus and the liver trematode Easciola hepatica. In parts of the sub tropics these parasites are also important as are two other gastrointestinal nematodes Trichostrongylus axei and Haemonchus contortus while two other Cooperia spp., C. punctata and C. pectinata are also prevalent. Less frequently, a nematode of the large bowel, Oesophagostomum radiatum assumes importance. In the tropics, the scene is dominated by H. contortus, C. pectinata and C. punctata and the larger liver fluke E. gigantica and on a more local basis the paramphistomes and schistosomes. There are other helminths, such as the cestodes and filarioids which I have not mentioned but I believe that the species emphasized above are those of major global importance in cattle.

The nematodes of the lungs and alimentary tract have direct life cycles, involving a free-living phase in which the faeces act as a reservoir of larval infection and from which larvae move onto the pasture where they are ingested by the grazing animal. The infective stages within the faeces can live for periods of up to several months. Development to adult parasites within the host usually takes 3-4 weeks but is sometimes delayed at the 4th and 5th larval stages for prolonged periods, a phenomenon known as hypobiosis or arrested larval development.

In contrast, the important trematode parasites such as the liver fluke, paramphistomes and schistosomes have indirect life cycles and require an intermediate snail host to complete their life cycle. As a result the cycles are much longer and very much linked to the ecology of the snail vectors. Infection is by ingestion of the infective stages released by the snail vectors and encysted on grass.

## **Control of Helminthiasis**

As mentioned previously, the trend over the past two decades has been away from convenience therapy of the infected animal towards preventing infection. It was hoped that the development some 25 years ago of an irradiated larval vaccine for the control of lungworm in cattle (1, 2) would be the forerunner of similar vaccines to prevent bovine helminthiasis. Although the lungworm vaccine Dictol (RI), has been very successful in preventing parasitic bronchitis in Britain and parts of Europe, attempts to immunize against the gut nematodes and the liver fluke have either failed (3) or not been sufficiently protective to merit field usage (4). There are some interesting ongoing developments in this area which centre on the search for the essential protective antigens and their synthetic production using modern biotechnology, but in terms of field use they are still at the embryonic stage.

So, the approach to control is very much based on the epidemiological knowledge which has been acquired over the past two decades and involves the integration of control by pasture management and the use of the new broad spectrum anthelmintics, combined with knowledge of the host specificity of helminth species and host immunity to these species.

## The Epidemiological Basis of Control

Just as treatment of helminthiases have been haphazard, some of the assumptions on the epidemiology of these diseases have been equally awry. It is now known that development of the free-living stages (or the vectors) does not necessarily proceed at all seasons of the year or at best proceeds more slowly in some seasons. This results in a fluctuation in the seasonal availability of new infective stages. Since the survival of these infective stages is not finite and in many areas, for the majority, does not exceed a few months, there is a seasonal fluctuation in the numbers of infective stages on the pastures.

In climates with distinct summer and winter seasons, or dry and wet seasons, these seasonal fluctuations are more clear cut and periods when contamination of the pasture with eggs or larvae are likely to yield infective stages can be defined. For example, in Westen Europe the seasonal pattern of Ostertagia and Cooperia infective larvae may be summarized as follows: in the spring, infective larvae which survive the winter, are ingested by grazing animals. When these infections mature in about 3-4 weeks, eggs are deposited in the faeces and the cycle of free-living developments commences. The temperatures are such that it requires until July or August for the eggs to become infective larvae and migrate onto the pastures. Unless the early winter is particularly mild, development ceases by that time and there is no further development until contamination occurs again the following spring. So, the peak of pasture infections is reached in August/September and these decline steadily to reach low levels in the spring (5, 6). From a control point of view it is important to realize, that it is the contamination of pastures in the spring and early summer, which produces the heavy and sometimes lethal populations of infective larvae in the second half of the summer and autumn.

A rather similar fluctuation occurs with the metacercariae of *E. hepatica;* thus Lymnaea snails which recommence breeding in the spring become infected with miracidia from overwintered pasture infections of Easciola eggs or from infected carrier animals; development in the snail proceeds during the summer and provided there is adequate moisture, new populations of cercariae are produced from the snail to give metacercariae on the pasture from late August onwards (7, 8).

The timing of events in other continents differs but the principles are the same. For example, in Texas, U.S.A., trichostrongyle infections are acquired principally from November through May with the period up to February being the most important for contamination (9). In the Transvaal in South Africa, *H. contortus* infections in cattle are acquired mainly from May to August with the important period of contamination being from December to May (10). In New South Wales, Australia, transmission of infection with *O. ostertagi* is principally in the spring (September-October) from contamination of pastures in autumn and winter.

The contamination of pastures and therefore the epidemiology of bovine helminthiasis may also be affected by the numbers of susceptible cattle in the herd. Thus, the number of susceptibles are generally lower in beef herds where the unweaned young graze for several months alongside their immune dams; in contrast, in dairy herds, early weaned calves are segregated and constitute a highly susceptible group. The degree of immunity in older cattle will of course vary according to the previous level of exposure and the helminth species involved. In general, the effect of age is minimal and resistance is usually acquired rather than inate. Contamination will also be reduced where susceptible cattle graze alongside other hosts, such as sheep, provided there is minimal cross infection of the helminth species between the different hosts. Use is made of both the immune and alternate hosts in grazing control systems for helminthiasis and these are discussed below.

## **Specific Control Measures**

These can be conveniently divided into evasive strategies and prophylactic strategies (12). In the evasive strategies, pastures are rotated in a manner to avoid animals at risk grazing on pastures when infection levels are high; in prophylactic strategies, animals are treated with anthelmintics so as to limit contamination of pastures at a time when conditions for development to the infective stages are optimal.

#### **Evasive Strategies**

The most simple evasive strategy is to avoid any contact with infection, by housing cattle throughout their lives, the so-called zero grazing and this is done in various parts of the Northern Hemisphere. In the same areas, for example, in the Netherlands, where cattle are housed in the winter, another strategy is to delay the turnout in spring until most of the surviving overwintered infections on pasture have succumbed; at turnout all stock which grazed in previous seasons are also given an anthelmintic treatment (13).

Another evasive strategy is to move susceptible stock to safe pastures prior to the period when new larval populations build up on the original pastures. To be safe for the incoming cattle the recipient pasture should not have been grazed during the previous four months. In Europe, this is achieved by spelling pastures in the spring and early summer and cutting the grass for silage or hay; by the time the cattle are moved onto the pasture in mid summer, mortality of most of the overwintered infection will have occurred and with no fresh contamination in the spring the pastures will be safe; to ensure that they remain safe an anthelmintic treatment is recommended at the time of moving to remove any residual infection. In Australia (14), marked reductions were recorded in the worm burdens of young cattle grazed during autumn and winter on contaminated pasture and then doses with an anthelmintic and moved to prepared safe pastures, which had been rested for the previous five months, compared to the burdens on cattle which remained on contaminated pastures (14). In southern parts of the U.S.A., similar results were obtained by treating young cattle and moving them to safe pastures in early spring prior to the increase in pasture larval infections in March and April (15).

The above system is particularly good for the control of parasitic gastroenteritis and in some countries lungworm, but will not prevent trematode infections which can survive for a prolonged time in the snail vectors and so appropriate treatments may have to be introduced.

Since significant cross-infection of nematodes between cattle and sheep are limited to two main nematode species, H. contortus and T. axei, infections of most nematodes can also be prevented by rotating pastures grazed by cattle and sheep. The period between rotation will vary according to climate and region. In Europe where larvae survive rather longer on pastures an annual rotation is recommended; in contrast, in parts of Australia and South Africa where larval survival is shorter, rotation every 4-6 months is prescribed (16). Again, each rotation should be accompanied by an anthelmintic treatment to minimize carry over of infection. Good results have also been achieved by mixing the grazing of cattle and sheep, taking into account the equivalent stocking units of each species. The resultant dilution of stocking rate by each species limits contamination and the availability of infection.

In these systems the presence of H. contortus and T. axei presents a problem but field information on the degree to which sheep and cattle strains will cross-infect the other host species is very limited and it may be that relatively short exposure periods of six months to one year are not sufficient for major adaptations to the other host to develop. No such problem exists for the liver fluke parasite and the best evasive strategy for this parasite, is to avoid grazing areas of snail colonization when metacercarial levels are known to increase.

The final evasive strategy which is employed is particularly applicable to parasitic gastroenteritis control on dairy farms and utilizes a rotation in which the more susceptible young stock graze ahead of heifers or dry cowsthe so called leader/follower system (17). It is based on the premise that adult animals are immune and while this is true when the cattle have been successfully exposed as yearlings, it should be remembered that parasite naive adults are still susceptible. The system also requires careful management to ensure that the susceptible replacement calves do not remain for too long on a paddock but are allowed to graze only the leafy upper eschelons of the herbage where larval numbers are generally low, before moving on; the immune adults then move in and graze the paddock more extensively but are theoretically capable of resisting the nematode infections present and so only minimal contamination of the pasture results.

## **Prophylactic Strategies**

These are based on the anthelmintic treatment of cattle to limit the contamination of pastures at times which are optimal for the development of free-living stages. They are most useful in situation where the organized movement of livestock is not practical.

In the Northern Hemisphere, where the annual midsummer increase of nematode larval populations originates principally from eggs deposited by infected animals in spring and early summer, good control has been achieved by treating young cattle at these times. Using fenbendazole at three and six weeks after turnout in spring the subsequent pasture levels of Ostertagia and Cooperia larvae were reduced by over 90% (18); it is recommended that a regime involving treatment with fenbendazole at 3, 7 and 11 weeks after turnout will give excellent control. With the introduction of the anthelmintic, ivermectin which has a 10-14 day residual effect and prevents the establishment of larvae during this period, the interval between treatments could be extended to 5 weeks and two treatments in the spring should suffice for the control of the common gastrointestinal nematode infections in these areas.

A sustained release rumen bolus containing the anthelmintic, morantel tartrate, Paratect ( $R_2$ ), is also available for use on cattle and this has been shown to significantly reduce contamination over a 90 day period in many different countries (19).

In Europe this is administered at spring turnout in April or May and there is virtually no contamination of pastures with eggs until July by which time most of the overwintered larvae population on the pasture will have succumbed and so reinfection is at a low level. In warmer countries such as Brazil or Argentina where there is a longer period of important contamination it is suggested that two successive boli are used i.e. to cover a 180 day period.

To date, the above prophylactic strategies have not adequately controlled bovine lungworm and liver fluke in cattle, although ovine fascioliasis has been prevented by a series of epidemiologically based prophylactic treatments in West Scotland (20).

Where anthelmintics are used it is important to administer the new highly effective broad-spectrum compounds. Otherwise, the evasive or prophylactic strategies outlined above will fail. In terms of overall efficacy against nematodes, ivermectin appears to be very efficient and to date has given consistently good results; the residual effect of this drug for 10-14 days is an added bonus. The modern benzimidazoles or probenzinidazoles, fenbendazole, oxfendazole, albendazole and febantel are all highly effective against a range of stages of the important nematodes while the widely used levamisole, although also efficient against most adult and developing nematodes, fails against hypobiotic Ostertagia larvae.

For liver fluke in cattle, the efficacy of drugs such as nitroxynil and rafoxanide is very good against adult and later larval stages but not the young larval forms. A new drug, triclabendazole looks very promising and has been shown to remove the early larval stages in the liver and this could mean that the interval between prophylactic treatments could be extended to about 10 weeks and if used in evasive strategy repeat therapy would not be required. Are there any possible problems with the implementation of these integrated control methods for helminthiasis? I believe there are at least three. The first, is the possible selection of anthelmintic resistant strains of the parasite. Where animals are treated and then moved to safe pasture, the majority of the free-living stages which develop on the new pasture will be the progeny of worms which survived the treatment. Selection for resistance is therefore more likely than where animals are returned to dirty pastures with many free-living stages present. This is more probably in sheep than in cattle, since the survival of the faecal mass containing a reservoir of existing free-living stages is more prolonged in cattle.

The second, is the possible selection of helminth strains with a greater longevity in their free-living phase. Thus, where cattle pastures are rotated in such a fashion that the animals do not return for a lengthy period e.g. one or two years, then the larvae ingested and cycled, will be those with the greatest potential for free-living survival.

Finally, over zealous application of control measures may lead to an overall lowering of herd immunity and a shift in the population age-group most affected by helminthiasis, to the heifers or cows. This has already been recorded on some farms. Nevertheless, despite these possible strictures there is no doubt that infection of cattle with helminths can now be prevented and production significantly improved.

#### References

1. Jarrett, W.H.F., F.W. Jennings, W.I.M. McIntyre, W. Mulligan and G.M. Urguhart: 1955 Vet. Rec., 69:1329. 2. Poynter, D., B.V. Jones, A.M.R. Nelson, R. Peacock, J. Robinson, P.H. Silverman, and R.J. Terry: 1960 Vet. Rec., 72:1078. 3. Burger, H.J., J. Eckert, H.J. Chevalier, M.S.A. Rachman and G. Kongsmann: 1968 Vet. Med Nachr., 4:312. 4. Nansen, P.: 1975 Res. Vet. Sci., 19:278. 5. Michel, J.F.: 1969 J. Helminthol., 43:111. 6. Anderson, N., J. Armour, F.W. Jennings, J.S.D. Ritchie and G.M. Urguhart: 1969 Res. Vet. Sci., 10:18. 7. Ollershaw, C.B. and W.T. Rowlands: 1959 Vet. Rec., 71:591. 8. Armour, J., G.M. Urquhart, F.W. Jennings and J.F.S. Reid: 1970 Vet. Rec., 86:274. 9. Craig, T.M.: 1979 J. Am. Vet. Med. Assoc., 174:844. 10. Horak, T.G. and J.P. Louw: 1978 Onderst. J. Vet. Res., 45:23. 11. Smeal, M.G., G.G. Robinson and G.C. Fraser; 1980 Aust. Vet. J., 56:74. 12. Michel, J.F.: 1982 Biology and Control of Endoparasites. Academic Press, Australia. Editor L.E.A. Symons. p. 113-131. 13. Borgsteede, F.H.M.: 1977 The epidemiology of gastrointestinal helminth infections in young cattle in the Netherlands. PhD. Thesis, University of Utrecht. 14. Smeal, M.G., P.J. Nicholls, C.C. Robinson, J.K. Bowler, R.T. Webb and K.H. Walker: 1981 Aust. J. Agric. Res., 32:825. 15. Williams, J.C., J.W. Knox, B.A. Baumann, T.G. Snider, M.D. Kimball, and T.J. Hoerner: 1983 Inter. J. Parasitol., 13:133. 16. Southcott, W.H. and I.A. Barger: 1975 Inter. J. Parasitol., 5:45 17. Leaver, J.D.: 1970 J. Agric. Sci., Cambridge. 75:265. 18. Armour J.: 1978. In Intensive Grassland Use and Livestock Health. Proc. Brit. Grass. Soc. and Brit. Vet. Assoc. Conf., Berkshire College of Agric., p. 81. 19. Jones, R.M.: 1981 Vet. Parasitol., 8:237. 20. Whitelaw, A. and A.R. Fawcett: 1977 Vet. Rec., 100:443. 21. Armour, J. and J. Bogan: 1982 Brit. Vet. J., 138:371.

Paper presented at the XIIIth World Congress on Cattle Diseases, Durban, S. Africa, Sept. 17-21, 1984.

