Subclinical Effects and Control of Abomasal Worms in Dairy Cattle

Rupert Herd, *M.V.Sc., Ph.D.*
Department of Veterinary Pathobiology
The Ohio State University
Columbus, Ohio 43210

In the last decade considerable attention has been focussed on subclinical effects of internal parasites, particularly with respect to a possible reduction in milk yield in lactating cows. Subclinical effects have also been demonstrated in young animals and include depressed growth rate and interference with energy and nitrogen metabolism. These changes are largely related to infection with the abomasal worm *Ostertagia ostertagi,* which appears to be the most important pathogen in the U.S.A. and sometimes accounts for over 90% of the worm burden. In this presentation, the magnitude of the problem will be compared to young and mature dairy cattle and control strategies suggested.

Surveys in the U.S.A. indicate that dairy cows generally have low worm burdens (<3000) and low fecal egg counts (<10egp), although there is a high prevalence of infection (>80%).[1] The question arises whether such a small number of worms can depress milk yield. There have been only three experiments on the direct effect of worms on milk production and these gave variable results. [2-4] These experiments can all be critized for not simulating conditions of natural infection encountered on the farm. In one experiment,[2] cows given a large inoculum of 200,000 infective larvae produced 1-3 kg less milk/day, but in another experiment[3] an inoculum of 500,000 infective larvae had no effect on milk production. In the third experiment,[4] natural infection rates were simulated by giving cows 5000 infective larvae three times weekly for 9 weeks. This resulted in a suppression of milk yield of 2.2 kg/day, but the experimental cows had no previous exposure or acquired immunity to worms and were probably more susceptible than dairy cows that had been pastured as heifers.

Anthelmintic Trials in Adult Cows

Although there have been only three experimental infection studies, there have been at least 26 anthelmintic trials with naturally infected dairy cows on farms. Only 7 of these 26 trials showed a significant increase in milk production following deworming. These comprised 4 studies by Todd and co-workers in the U.S.A.[5-8] a study by McQueen *et al* in New Zealand,[9] Plumiers in the Netherlands[10] and Bliss *et al* in England.[11] Nineteen other studies in the U.S.A.,[12-21] Canada,[22] Britain,[23-25] Europe,[26-27] Australia[28,29] and New Zealand[30] involving cows both in confinement and at pasture, failed to show an oveall significant effect. This applied even when anthelmintics were administered at 2 or 4 week intervals for most of lactation.[29,30] In five of these trials, [12,15,18,23,24] untreated control cows showed increased milk yields of the order of 200-400 kg/lactation, comparable to increases claimed by Todd and co-workers for treated cows.

Recently, an anthelmintic trial of giant magnitude was done in Britain in an attempt to finally resolve the controversy.[31,32] The experiment started with 20,000 Friesian cows, but the final conclusions were based on data from 9271 completd lactations. Cows were allotted to 5 groups (untreated control, placebo control, levamisole, thiabendazole, fenbendazole) and treatment was given on the day of calving. There was a small and just significant treatment effect of 42 kg per lactation with a positive response in treated cows in 60% of herds. However the treatment effect relative to the placebo group was only 24 kg per lactation. Although a larger effect might have been expected in first calf heifers because of less immunity to worms, there was no response to treatment in this group. There was no relation between fecal egg counts of herds and response to treatment nor any difference in response between cattle calving in autumn or spring. It was concluded that there are no grounds for seeing treatment in the light of an insurance policy for the farmer. It was also calculated that the same economic benefit as that derived from a 42 kg milk increase could be obtained by (1) reducing dystocia by 0.3%, (2) shortening the average calving interval by 30 hours, or (3) increasing the calving weight of cows and heifers by 1.75 kg.[33]

Transmission of Parasites to Dairy Cows in Confinement

It is unlikely that treatment at calving could have more than a transient effect because cows at pasture would be immediately exposed to reinfection. In the case of cows in confinement, it is debatable whether they are exposed to appreciable parasitic infection. Todd and co-workers believe that cows in confinement are continually exposed to high levels of infective larvae (up to 702/3 sq. in.) inside barns, in loafing sheds and on dry lots.[8,34,35] In one paper,[35] they reported that over 97% of larvae (Farm 3)

were *Bunostomum spp.* This is a surprising result, in view of the fact that a Wisconsin survey showed only 1 of 54 dairy cows slaughtered to be infected with *Bunostomum spp.* and it harboured only 10 worms of this genus.[36] *Bunostomum* L_3 are small larvae [37] which fall within the length range of free-living rhabditids, but the Wisconsin workers made no mention of the presence of free-living nematodes in this study.[35]

In contrast to the Wisconsin results, studies of nematodes in mangers, stalls, calf pens and exercise lots at The Ohio State University dairy barn showed that over 99% of nematodes recovered (up to 59,600/100 cm^3) to be non-parasitic, free-living species of the family *Rhabditidae.*[38] The only parasitic species recovered was *Strongyloides spp.* which was found in moist bedding (7/100 cm^3) in calf pens. Studies on two commercial dairy farms in Ohio gave similar results. In addition, careful observations on 22 replacement heifers raised indoors in Ohio[38] and 6 worm-free control cows kept indoors in Maine[4] indicated that no infection took place while in confinement.

Although there is no evidence to justify mass treatment of dairy cows, there are some specific situations where prophylactic treatment may be justified. On some farms it is desirable to treat cows at the time of winter housing if they are transferred from contaminated pastures to a relatively parasite-free environment indoors. An anthelmintic effective against arrested larvae (e.g. ivermectin) should be used, because late autumn pastures may carry a high proportion of arrested-prone larvae. This treatment would help prevent Type II ostertagiasis the following spring. It is also advantageous to treat cows that have lost some immunity to worms and developed heavy worm burdens under conditions such as malnutrition, debilitating disease or use of immunosuppressive drugs, while exposed to a contaminated environment. On theoretical grounds, first calf heifers might be expected to benefit from treatment, but this was not substantiated by the big British Trial.

Dairy Replacement Heifers

It is now apparent that the advertising campaign to deworm adult cows was aimed at the wrong target and distracted attention from parasitism in dairy replacement heifers, the group that should be given priority in any parasite control program.[39] It is well established that heifers in their first year of grazing have little resistance to trichostrongylid worms, but that increasing immunity develops during their second and subsequent grazing seasons.[40-42] Consequently heifers in their first season at pasture are likely to carry heavy worm burdens, contaminate pasture with high egg output and be affected by both clinical and subclinical disease. In the U.S.A. this group is often neglected and the effects of parasitism frequently attributed to inadequate nutrition. When treatment is given, it is often given in the fall by which time pastures are heavily contaminated and it is too late to prevent production losses. Control procedures are commonly based on guesswork, tradition or fecal egg examinations and disregard reinfection, hypobiosis (arrested development) and selection for drug resistance. Few farm advisers understand the epidemiology of the worms or the prophylactic use of anthelmintics.

Worldwide studies have shown that anthelmintic treatments based on prophylactic treatments in the spring,[39,43-45] administration of sustained release devices at turnout,[46-48] treat and move in July, [49,50] or even repeated treatments in the face of reinfection [51-56] have all resulted in increased weight gains in calves or heifers in their first season at pasture. Subclinical effects are primarily related to reduced growth rates and the main economic loss occurs from delay in reaching optimal size for first breeding. The economic benefits from speeding up the time of first calving by proper worm control have been estimated at $20-$105/heifer. There is also evidence that heifers will produce more milk in their first lactation if they are raised with proper parasite control.[57] This effect is attributed to the increased weight gains in early life.

When considering the best approach for parasite control for replacement heifers in the U.S.A., it must be emphasized that different strategies will be needed for the north and south, since the epidemiology of helminths in the two regions is quite different. Although *Ostertagia ostertagi* is often the most prevalent and pathogenic cattle parasite in both the north and south, the pattens of pasture infectivity and hypobiosis are distinctly different.[58] In the north, Type I disease mainly occurs on heavily contaminated pastures in late summer and fall, hypobiosis occurs during the winter and Type II disease may occur in early spring as young adult worms emerge from the gastric glands. In the south, Type I disease occurs mainly in the spring, hypobiosis in the late spring and summer when conditions are unfavourable for larval transmission on pasture, and Type II disease may occur in late summer or early autumn. There is a need for much more epidemiologic data for the major cattle regions in the U.S.A. so that logical control programs can be tailored to the requirements of each region.

Strategies for Replacement Heifers

There are several strategies which can be utilized for worm control in dairy replacement heifers in northern regions:

1. Recent studies in Ohio have demonstrated the value of 2 spring treatments with levamisole[39] or ivermectin[59] 3 and 6 weeks after turnout. The rationale of this approach is that it kills overwintered worms picked up by the heifers, before the worms start shedding large numbers of eggs and contaminating pastures with second generation worms. By the time of the second treatment, most remaining overwintered larvae on pasture will have died off because of increasing temperatures. Treatment with either drug resulted in significant increases in weight gain by the end of

the grazing season (up to 40 kg) compared to controls exposed to pastures with similar levels of contamination with overwintered larvae. This strategy allows the farmer to keep his heifers on the same pasture, without requiring a move to clean pasture after treatment.

2. A new development in the prophylactic use of anthelmintics is the administration of a morantel tartrate sustained release bolus (MSRB) given to heifers at the time of turnout to spring pasture.[46-48] This bolus is available in Europe, but is not yet approved in the U.S.A. It has obvious labour-saving advantages which fit in with normal farm management. The intraruminal bolus is designed for continuous delivery of the drug for at least 60 days. In a large number of field trials in Britain and Europe MSRB-treated calves outperformed control calves in weight gains, and parasitologic data showed that disease prevention and improved performance were due to MRSB preventing the midsummer rise in pasture larval contamination. It is possible that controlled release devices select for resistant worms, but most control programs do this to some degree, and there is a need to compare the relative selection rates for different strategies.

3. A control strategy which has proved effective in Britain is the treat and move system.[49,50] The move to safe pastures is the most important component of the system and is done in July in Britain, just before the massive build up of second generation worms on pasture. Move without treatment gives almost as good results, whereas treatment without a move leads to serious parasitism. This system should be fairly effective in northern U.S.A. because conditions are somewhat similar to those in Britain, but it may not be popular with farmers because of difficulties in adapting the program to practical farm conditions. Since climatic conditions in northern U.S.A. favour a build up of infective larvae on pastures and heavy worm loads from midsummer until the end of the autumn grazing season, a July treatment of replacement heifers and move to clean pastues not grazed by cattle that year should prevent Type I disease later that year and Type II disease the following spring. The main purpose of dosing heifers at the time of the July move is to protect the new pasture from contamination.

The question arises whether the parasite control program should be continued during the second year of grazing, when replacement heifers have more acquired immunity to worms. Studies in Maine have shown that yearling heifers previously exposed to infection at pasture have high resistance to further infection when grazing heavily contaminated pastures in the second year of grazing.[42] In anthelmintic trials in England, growth responses were highly significant in calves given repeated anthelmintic treatment during their first season of grazing, but growth responses were only marginal following treatment in their second year of grazing.[52] Thus it appears that routine control programs are essential for heifers during their first grazing season, but less urgent thereafter. The decision to treat or not to treat in the second grazing year is probably best made on an individual farm basis. It would be essential to treat all heifers if first and second year heifers were grazed together on the same pasture.

The same principles of control may be applied to replacement heifers in the south, but the timing and other details are likely to be different because of the different epidemiology encountered. A number of studies are now in progress in southern states to test modern epidemiologic control strategies and it is hoped that concrete recommendations will result from these studies. At present there is no reason to recommend old approaches such as treatment at set intervals (e.g. every 3 months). These old systems have no relationship to the epidemiology of the parasite and the sequence of pasture contamination and infectivity. They disregard reinfection, hypobiosis or selection for drug resistance. It is not surprising that they do not reverse the adverse effects of parasitism.

Economic Gains from Parasite Control

The main economic loss due to worms occurs from reduced growth rates and delay in reaching optimal size for first breeding. It has been calculated from the Ohio data that well managed heifers on a prophylactic parasite control program gain 1.4 lb/day from birth to freshening and reach breeding size (750 lb for Holsteins) at about 15 months of age.[60] Assuming a rate of gain of 0.9 lb/day during the pasture season (5 months) for untreated heifers (or improperly timed treatment) and 1.4 lb/day during the rest of the year, heifers would not reach proper breeding size until 18 months. Dewormed heifers would be expected to

TABLE 1. Cost of Raising Dairy Replacements Calving at 26 Months of Age versus 29 Months of Age.

Costs	Age at Calving (months) (Dewormed) 26	(Untreated) 29
Variable costs[1]	$ 920.00	$ 980.00
Cost of additional[2] herd replacements needed	39.60	99.00
Worming medicine[3]	10.00	-----
Additional labor for worming	4.00	-----
Total variable costs	$ 973.60	$ 1079.00
Net return or savings ($1079.00 - 973.60) = 105.40/heifer		

[1] Feed, veterinarian and medicine, breeding, utilities, supplies, interest on equity capital from birth to calving.

[2] Cost per month for additional herd replacements when age at first calving exceeds 24 months ($19.80/heifer/month over 24 months).

[3] For computation purposes only. Costs will vary.

calve at about 26 months of age, assuming an average of 1.5 services per conception and an average heat detection efficiency of 50%. Untreated heifers would calve about 3 months later, at 29 months. Table 1[60] shows the costs and returns for these programs.

Looking at the situation on a national basis, there is a lot to be gained from prophylactic parasite control for dairy replacement heifers. If an estimated 5 million replacement heifers at pasture were given 2 treatments, the cost would be something of the order of $50 million in anthelmintics and labour. However, the net return to the dairy industry at $50-$100/heifer would be of the order of $200-$450 million. By contrast, the annual cost of treating 11 million adult dairy cows twice, as recommended by some drug companies, would exceed $100 million. But in this case the farmer would have no certainty of any economic benefit.

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Panel Discussion

Question: What about the reduction in the cell counts in cows over seven years of age?

Answer: Dr. Eberhart: There are very few cows in that group and the ones that make it that far are very resistant cows and they are there because of their resistance they have had very little mastitis.