Bovine Parasites of Particular Importance in the Northwest

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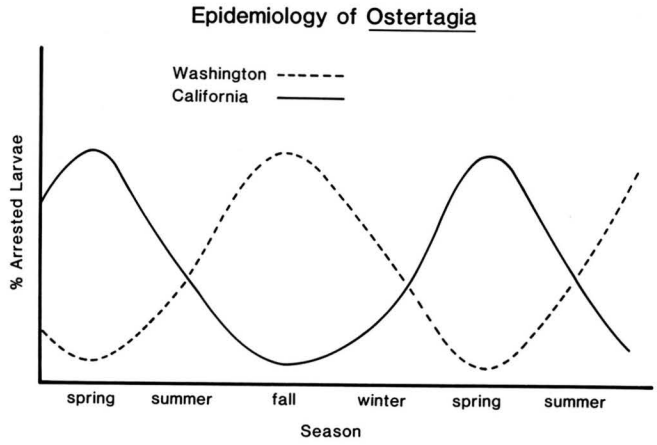
The problem of internal parasites in cattle is more unrpedictable in the Northwest than in geographic areas where clincial disease is the rule rather than the exception. The practitioner in this area is continually confronted with the question "will treatment provide an economic response?" The answer often is based on information obtained from research done in other regions and frequently may not apply to their conditions. Five examples, ostertagiasis, coccidiosis, fascioliasis, trichomoniasis, and cryptosporidiosis, of regionally important parasitisms will be discussed in this regard. All are often subclinical, all present diagnostic problems and all require different control measures. Each condition will be presented with a short introduction; a discussion of current diagnostic techniques; a review of available treatments; and a summary of how control might be best accomplished.

FIGURE 1. Seasonal occurrence of arrested stages of O. ostertagia in native Washington cattle.



Ostertagiasis

The subject of ostertagiasis has been covered by two other speakers[1,2] and my remarks will be limited to the features of the condition that apply to the Northwest.

First of all, *Ostertagia ostertagi* infections are found in most cattle kept on pasture for substantial periods of time[3]. They are usually subclinical and arrested stages ordinarily occur in the fall of the year (Figure 1). Clinical disease occurs, in my experience, when the animals are stressed by poor management practices or maintained on irrigated pastures. Numbers of adult and larval *Ostertagia* found in local cattle are generally much smaller than reported[1,2] in other geographic areas of the United States.

Economic benefits from worming pastured cattle have been difficult to demonstrate consistently using older anthelmintics (benzimidazoles and levamisole). This problem is illustrated in Tables 1-4 which represent a series of small field tests which were run with beef cattle in central Washington. Improved weight gains were found in yearling cattle and weaning weights of calves in 2 trials (Tables 1 and 2) and little or no response was found in 2 others (Table 3 and 4). All experiments were conducted under much the same conditions and most older cattle in these herds had trichostrongyloid egg counts of 50 to 250/gram prior to treatment. The only apparent variable was that the cattle or calves of cows kept on pastures with abundant forage seldom showed improved weight gains after worming. I believe that the effects of parasitisms were masked by good feed and management practices.

The situation in feed lot cattle also is unpredictable and depends on the source and parasite burdens of animals when received at the lots. Our investigations[4] of parasitism in these cattle indicate arrested *Ostertagia* larvae are present throughout the year and numbers of adult parasites vary from few to many. Tests[5] with local dairy cattle also have given inconclusive results regarding increased milk production related to anthelmintic treatment. The conclusion drawn from these observations is that no blanket recommendations can be made for worming. The decision to use anthelmintics must be made for each operation on an individual basis considering levels of parasitism and management methods.

Diagnosis of ostertagiasis is most accurately made when abomasums from necropsied animals are examined for adult and immature parasites. If this is not possible, egg counts may be of some value on a herd basis. We suggest evaluating egg counts from 10-12 individuals from suspect herds. Our experience has been that even when disease is caused by arrested larvae, at least 2 or 3 of the 10 to 12 samples will have relatively high (>150 EPG) counts.

Treatment of ostertagiasis is best accomplished using drugs which are effective against the arrested larval stages of the parasite as well as adult worms. Anthelmintics currently

TABLE 1. Spring Protective Treatment of Pastured Yearling Cattle with TBZ[1] — 1981 Ellensburg, WA.

Group	No. Calves	Mean Weight[2] gain (lbs.)	Result
Treated	20	225.6	26.3 lb.
Controls	17	199.3	advantage

[1]44 mg/kg thiabendazole July 2.
[2]lbs. gained July 2-Oct. 8.

TABLE 2. Weight Gains of Calves from Cows Treated with ABZ[1] — 1978 Ellensburg, WA.

Group	No. Calves	Mean[2] Adj. Wt.	Result
Treated	105	635	14 lb.
Control	28	621	advantage

[1]15 mg/kg albendazole on June 7.
[2]adjusted 205 day weaning weights in lbs.

TABLE 3. Spring Protective Treatment of Pastured Yearling Cattle with TBZ[1] — 1982 Ellensburg, WA.

Group	No. Calves	Mean Weight[2] Gain	Result
Treated	26	275	No
Controls	22	275	advantage

[1]44 mg/kg thiabendazole April 30 and July 30.
[2]lbs. gained April 17-Nov. 10.

TABLE 4. Weight Gains of Calves from Cows Treated with TBZ[1] — 1982 Ellensburg, WA.

		Mean Weights[2]		
Group	No. Calves	4/30	9/24	Result
Treated	13	190	598	No Economic
Control	14	199	601	advantage

[1]44 mg/kg TBZ April 30 and July 30.
[2]Weights in lbs.

available which have this activity are ivermectin[a], fenbendazole[b] and albendazole[c].[1,2,6] The former appears to have efficacy which approaches 100% and should eliminate most worms with a single treatment. The latter drugs are less effective and removal of arrested stages may require twice the dosage recommended for adult parasites.

Control of economic ostertagiasis in the Northwest depends upon individual management practices. Cattle harboring moderate numbers of parasites will benefit from anthelmintics given before placing them on irrigated pastures. Treatment is less important where pastures are not irrigated and should be concentrated on younger (<2 years) animals. In feedlot and dairy operations each situation should be considered separately. As a general rule, lots of cattle with more than 10% of fecal samples showing <75 EPG will benefit from treatment. With dairy cattle we recommend worming of replacement heifers routinely. Treatment of adult cows under most circumstances is difficult to justify.

The time of year may dictate the product used. With the older anthelmintics (thiabendazole[d] and levamisole[e]) treatment prior to placing cattle on pastures in spring should give best results. Ivermectin, on the other hand, may be used to advantage in the fall for grub control and to eliminate immature *Ostertagia* that would be carried through the winter in infected cattle. In either case, one treatment per year should be sufficient.

Coccidiosis

Most cattle are infected with *Eimeria* spp in the Northwest.[3] The magnitude of the problem is unrecognized, however, because many of the infections are subclinical and largely ignored by producers and veterinarians. When clinical disease occurs it can be dramatic and cause obvious economic losses in feedlots, young dairy calves and overwintering beef cattle. The subclinical form of the disease is also of economic importance and recent evidence[7] suggests prophylactic treatment may be worthwhile. The epidemiology of the clinical disease suggests beef cattle are not severely exposed until they are removed from pastures in the fall of the year and grouped in feedlots or overwintering quarters. Dairy operations, on the other hand, experience problems year around with younger calves. Maximum exposure occurs early in life and older animals apparently become sufficiently resistant so that they seldom show clinical signs.

Diagnosis of coccidiosis presents few problems when clinical signs are present. In these cases, gross lesions are obvious at necropsy and massive numbers of oocysts appear in fecal samples. Subclinical infections are another matter. Recent research[7] suggests that this is a common problem, however, and can occur when cattle are passing few oocysts in their feces. These cases generally benefit from feeding coccidiostats on a prophylactic basis.

Treatment of clinical coccidiosis is the least effective way of handling the problem. However, when it occurs, amprolium[f] fed at 20-25 mg/kg for 4 or 5 days is recommended.[8] Prophylactic treatment is more effective

[a] *Ivomec®, Merck and Co., Rahway, N.J.*
[b] *Safeguard®, American Hoechst Corp., Somerville, N.J.*
[c] *Valbazen®, Smith Kline Animal Health Products, West Chester, PA.*
[d] *Omnizole®, Merck and Co., Rahway, N.J.*
[e] *Tramisol®, American Cyanamid Co., Princeton, N.J.*
[f] *Corid®, Merck and Co., Rahway, N.J.*

and best accomplished by feeding decoquinate[g] or lasalocid[h] at low level (50 mg/kg of feed) through critical exposure periods.[7]

Control is the goal of any treatment program for coccidiosis. Recent studies[7] show that little protection is acquired while cattle are fed coccidiostats and this should be considered when formulating treatment regimens. In other words, once prophylactic treatment has been started it should not be discontinued until the high exposure, high risk period is over. In operations that have experienced clinical coccidiosis in the past, these periods can be anticipated. In operations where only subclinical disease occurs, thought should be given to initiating prophylactic treatment and, if started, when it can be discontinued.

Fascioliasis

Liver flukes, *Fasciola hepatica* and *Fascioloides magna,* are common parasites in the Northwest. [3,9] Historically, the endemic geographic areas for both parasites were rather well established but expanded use of irrigation has changed the prevalence of *F. hepatica* in recent years.[10] This factor, together with the movement of livestock, has resulted in cases appearing in unexpected places. Epidemiologic studies[11] suggest *F. hepatica* metacercaria do not overwinter well on pastures in the Northwest and this accounts for the fact that many infections are subclinical. The period of pasture transmission is relatively short and does not allow sufficient exposure to produce the severe infections observed in the southeastern and south central states. *Fascioloides magna* infections in cattle still occur in rather well defined geographic areas where the reservoir hosts (deer and elk) are abundant but generally are not considered an important problem in livestock.[9]

Diagnosis of *F. hepatica* is accomplished by demonstration of adult flukes in the bile ducts at necropsy or eggs in fecal samples using sedimentation techniques. Serologic tests also are reasonably effective but are not yet available commercially.[10] As with *Ostertagia,* one negative fecal does not rule out herd infection. We suggest doing at least six or eight examinations for each suspect herd. Finding *any* positives in a sample of this size is indicative of a potential problem. *Fascioloides magna* infections do not become patent or produce eggs in cattle so diagnosis can be made only at necropsy. Specific serologic tests are not available for *F. magna* and those used for *F. hepatica* are of limited value because cross reactions occur with these two closely related parasites.

Treatment of *F. hepatica* currently is accomplished with albendazole.[12] This drug was released on an emergency basis in 1980 and is less efficacious (± 80%) than flukicides available abroad or other products (triclabendazole,[i] clorsulon[j]) being developed for use in the U.S. However, treatment with albendazole still has merit and is recommended once/year for herds with *F. hepatica.* Treatment for *F. magna* in cattle is seldom recommended because of the difficulty in diagnosing the infection. When indicated, albendazole can be used on an emergency basis.[9]

Control of liver flukes currently is best accomplished by treatment of breeding cattle with albendazole (7.5 mg/kg) prior to placing them on pastures in the spring of the year. The poor survival of metacercaria during Northwest winters offers hope for eventually eliminating the problem with the advent of more efficacious anthelmintics.

Trichomoniasis

Infection of beef cattle with *Tritrichomonas foetus* has increased dramatically in the Northwest in the past ten years.[13] I suspect an increasing awareness of the problem and improved diagnostic ability of our laboratory has magnified the number of cases encountered, but even discounting the impact of these considerations, the prevalence is alarming. It appears that the parasite has become firmly established in range cattle and measures currently practiced to limit its spread are inadequate.

Diagnosis of bovine trichomoniasis is usually made after producers have experienced diminished calf crops and the condition is suspected (together with many others) as the cause.[14] In these cases, the *T. foetus* may be isolated from preputial washings or scrapings from suspect bulls or the stomach contents of aborted fetuses.[15] The procedures used to demonstrate the organisms are not complicated and have been mastered by many practitioners. However, they require careful collection, and examination of samples to demonstrate the organism. Should trichomoniasis be suspected, I recommend contacting a diagnostic laboratory to discuss these techniques and to confirm the diagnosis until some expertise and confidence have been gained in working with this organism.

Treatment, under ideal circumstances, would be accomplished by the use of artificial insemination with semen from bulls shown free of *T. foetus.* Where this is not possible, sexual rest of females for 90 days usually clears them of infection and treatment of bulls with dimetridazole[k], given orally at 50 mg/kg once daily for 5 days, or ipronidazole hydrochloride[l], given intramuscularly once at 30 g/adult bull, is usually satisfactory. However, caution should be observed when using these drugs because they are not entirely effective and the carrier state is very difficult to

[g] *Deccox®, Rhone Poulenc, Atlanta, GA.*
[h] *Bovatec®, Hoffman-LaRoche, Nutley, N.J.*

[i] *Triclabendazole, Ciba-Geigy, Greensboro, N.C.*
[j] *Clorsulon, Merck and Co., Rahway, N.J.*
[k] *Emtryl®, Salbury Laboratories, Charles City, Iowa.*
[l] *Ipropran®, Hoffman-LaRoche Inc., Nutley, N.J.*

detect. Once a herd is infected owners should be advised that it may take years to eliminate the problem.

Control is based on the type of management methods available to the producer. If artificial insemination can be used, it is the surest way of eliminating the disease. If the animals involved are range cattle and this is not possible, current practice is to cull any cows without calves prior to turning them out on pasture and use only bulls four years of age or younger (these bulls will be much less likely to be carriers than older animals).[14] [15] If a bull is treated to eliminate the infection, great care must be taken to insure treatment was successful. This is a costly procedure and only justified for extremely valuable animals. Unfortunately, pharmaceutical companies have not shown great interest in developing products to treat *T. foetus* and efforts at development of vaccines[16] have been unsuccessful.

Cryptosporidiosis

Cryptosporidium spp were first suggested[16] [17] as a factor in diarrhea of dairy calves in the early 1970s. Since then the condition has been noted[16] with increasing regularity in Idaho and Washington until now the diagnosis is considered routine. More research needs to be done before the epidemiology and pathogenicity of cryptosporidiosis are understood but, at this time, most practitioners who have encountered the condition agree that (1) it can contribute to disease under some circumstances; (2) no treatment is available; and (3) once it becomes established in a herd the problem continues indefinitely.

Diagnosis is accomplished by finding *Cryptosporidium* oocysts in fecal samples of affected calves. As with many parasitic diseases, several samples from more than one animal should be examined because the oocyst shedding period is relatively short (usually the second week of life of the calf) and samples taken before or after this time ordinarily will be negative. It also should be emphasized that the oocysts are very small ($<5\mu$) and are easily overlooked.[17] Several techniques[m] are available to stain the cysts and facilitate identification. However, we find routine sugar flotation is satisfactory once technicians have seen the parasite.

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[m]*Acid Fast Stain Kit, Volu-Sol Medical Industries Inc., Las Vegas, NV.*

Treatment and control measures have not been established. Since no drugs are available and the life history is poorly understood, recommendations are difficult to justify. Perhaps treatment of clinical signs accompanied by good sanitation to prevent consumption of large numbers of oocysts by susceptible calves is of some value. However, actual prevention of cryptosporidiosis will require the development of an effective chemotherapeutic agent or vaccine. Until that time the best approach is to consider cryptosporidiosis as a mild calfhood disease of the second week of life which contributes to concurrent enteric problems.

References

1. Williams, J.C. 1984. *Ostertagia ostertagi;* Clinical condition, recognition of disease states and approaches to control through anthelmintics and management. Proc. 17th An. Conf. AABP. 2. Corwin, R. 1984. Ostertagiosis in Missouri beef cattle--the scenario from calving to feedlot. Proc. 17th An Conf. AABP. 3. Malczewski, A., Wescott, R.B., Sprattling, B.M. and Gorham, J.R. 1975. Internal parasites of Washington Cattle. AJVR 36:1671-1675. 4. Wescott, R.B. 1984. Unpublished information. Dept. Vet. Micro/Path, WSU, Pullman, WA 99164. 5. Murdock, M. 1984. Unpublished information. Dept. Animal Science, WSU, Pullman, WA 99164. 6. Wescott, R.B., Farrell, C.J., Gallina, A.M., and Foreyt, W.J. 1979. Efficacy of albendazole for treatment of naturally acquired nenatode infections in Washington cattle. AJVR 40:369-371. 7. Conlogue, G., Foreyt, W.J., and Wescott, R.B. 1984. Bovine coccidiosis: protective effects of low level infection and coccidiostat treatments in calves. AJVR 45:863-866. 8. Slater, R.L., Hammond, D.M., and Miner, M.L. 1970. Eimeria bovis: development in calves treated with thiamine antagonist (amprolium) in the feed. Trans. Am. Microsc. Soc. 89:55-65. 9. Foreyt, W.J., and Hunter, R.L. 1980. Clinical Fascioloides magna infection in sheep in Oregon on pasture shared by Columbian white-tailed deer. AJVR 41:1531-1532. 10. Wescott, R.B., Farrell, C.J., and Shen, D.T. 1984. Diagnosis of naturally occurring Fasciola hepatica infections in cattle with an ELISA test. AJVR 45:178-179. 11. Hoover, R.C., Lincoln, S.D., Hall, R.E., and Wescott, R.B. 1984. Seasonal transmission of Fasciola hepatica to cattle in northwestern United States. JAVMA 184:695-698. 12. Foreyt, W.J., Wescott, R.B. and Armstrong, D.A. 1980. Field tests of three formulations of albendazole against Fasciola hepatica in cattle. VM/SAC 75:299-303. 13. Mickelsen, W.D. and Ward, A. 1984. Personal communication. WA Animal Disease Diagnostic Lab., Pullman WA 99164. 14. Clark, B.L., Dufty, J.H., Parsonson, I.M. 1984. The effect of Tritrichomonas foetus infection on calving rates in beef cattle. Aust. Vet. J. 60:71-74. 15. Abbitt, B. 1981. Trichomoniasis. Proceedings Soc. for Therio. Pages 31-37. 16. Anderson, B.C. 1982. Crytospridiosis: a review. JAVMA 180:1455-1457. 17. Anderson, B.C. 1981. Cryptosporidiosis in calves: epidemiologic questions, diagnosis and management. Proc. 14th An. Conf. AABP pp. 92-94.