area was measured at the time of pregnancy diagnosis, prebreeding, or four to five months after calving in studies not showing an influence of pelvic area on dystocia.[4,10] Studies that have shown pelvic area to have a significant influence on dystocia measured pelvic area within one month or less prior to calving.[6,7,8] Considering the fact that pelvic area at calving has low correlation to pelvic area measured at other time it can not be concluded from the former studies that pelvic area does not influence dystocia.

Yet, only 73% of cases of dystocia were accurately classified by pelvic area at calving and calf birth weight (Table 5). Often a holdout sample is used to validate a model developed with discriminant analysis techniques. When such an approach is used, the model is developed from a random sample of the initial data set. The model is then tested for accuracy of prediction in the rest of the data set. We did not use this approach because we had a limited number of animals (76) with which to develop our models. This may result in some upward bias in percent accurate classification. Therefore, factors other than calf birth weight and pelvic area which were not identified in this study may influence dystocia.

Development of strategies to control dystocia should focus on methods of reducing calf size and increasing pelvic area at calving. Calf size is significantly influenced by sire.[4] Measurement of pelvic area, however, at any time other than calving does not accurately represent pelvic area at parturition. The high degree of variation in pelvic growth and dilation prior to calving indicate factors inherent to the individual determine pelvic area at calving and dystocia. Research efforts should be focused on identification and control of these factors.

Conclusion

The ratio of pelvic area at the time of calving to calf birth weight is a major determinant of dystocia. The high degree of variation in pelvic growth and dilation, however, results in low correlation of pelvic area at calving to pelvic area measured prior to calving. Prediction of dystocia utilizing pelvic area measured prior to calving is difficult and not highly accurate.

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Variable Efficacy of Benzimidazole Anthelmintics Against Inhibited Larvae of *Ostertagia Ostertagi*

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Abstract

Variable efficacy of benzimidazole anthelmintics (albendazole, fenbendazole, and oxfendazole) against inhibited *Ostertagia ostertagi* larvae has been reported in the literature. Efficacies at manufacturer's recomended dosages for the three anthelmintics, respectively, were 18.6%-84.9%, 61.5%-97.5%, and 33.5%-93.6%. Respective efficacies for dosages lower than recommended were 30.8%-86.6%, 0.0%-97.5%, and 0.0%-85.8%. Respective efficacies for dosages higher than recommended were 84.9%, 92.0%-99.0%, and 78.8%-95.0%.

One of the hypotheses for variable efficacy is a difference in larval metabolic activity during the inhibition season (i.e. when metabolic activity is high efficacy is high and vice versa). This hypothesis was tested in a critical evaluation using oxfendazole. Forty-eight steer calves commenced grazing 10 acres of pasture in November. In the months of March,

May, July, and September, 12 animals were removed and placed on concrete. After 3 weeks, 6 animals were treated intraruminally with oxfendazole (4.5 mg/kg) and 6 were left as untreated controls. Seven days after treatment, the steers were slaughtered and gastrointestinal nematodes were enumerated and identified. Results of this hypotheses-test trial and some implications for beef practitioners will be discussed in this presentation.

It has been well established that gastrointestinal nematode parasitism can adversely affect cattle production. These affects are usually observed as poor weigh gains or weight loss, however, under heavy infection deaths can occur. The nematode causing the greatest damage is *Ostertagia ostertagi* which thrives when climatic conditions are cool and wet. A phenomenon known as developmental arrest or larval inhibition plays a major role in ensuring survival during periods of environmental stress on the free-living stages. When environmental conditions become favorable for development and survival of free-living stages outside the host, inhibited larvae in the animal mature and can provide potentially large adult populations to contaminate pastures. The onset of inhibition generally occurs just prior to the time when the weather conditions are unfavorable. In regions (cool temperate) of winter inhibition, the onset occurs during fall and in regions (warm temperate) of summer inhibition, onset occurs during spring. In the United States, the winter epidemiologic pattern prevails across the northern tier from Maine through the northcentral region (Michigan, Ohio, Minnesota) into the northwest, and the summer epidemiologic pattern prevails across the southern tier from North and South Carolina through the southcentral region (Arkansas, Louisiana, Texas) to California. It is not known at what point moving south or north where the transition from one pattern to the other occurs. In fact, reports have indicated that both types of inhibition can occur in the same transitional region. Therefore, it is difficult to recommend control programs to include inhibited *O. ostertagi* except in those regions where the inhibition pattern has been established.

The inhibition period can be broken into three phases. Phase 1 occurs as larvae acquired from pasture become inhibited in the host (September-November for winter inhibition, and February-April for summer inhibition). In phase 2, transmission is minimal and maximal numbers of early fourth stage larvae are inhibited because climatic conditions are unfavorable for development and survival of free-living larvae (December-February for winter inhibition, and May-July for summer inhibition). Phase 3 is when inhibited larvae emerge (March-April for winter inhibition, and August-September for summer inhibition).

Because inhibited larvae have been hypothesized to play a role in ensuring survival of the species by providing the major source of pasture contamination after seasons of adverse climatic conditions, it has been suggested that anthelmintics effective against them in mid-winter or mid-summer (i.e. phase 2) can help break the life cycle and provide cleaner pastures.

The benzimidazole anthelmintics albendazole, fenbendazole, and oxfendazole have been shown to be effective against *O. ostertagi* inhibited larvae although the efficacy has been variable at similar and different dosages (Table 1). There have been some reports of efficacy against inhibited larvae when anthelmintics were administered outside the recognized inhibition season where only 1-13% of the larval population was early fourth stage. Results of these reports are not included here. Efficacies at manufacturer's recommended dosages (reports from summer inhibition regions only) for albendazole (Valbazen®, 10 mg kg^{-1}), fenbendazole (Panacur®, 10 mg kg^{-1}), and oxfendazole (Synanthic®, 4.5 mg kg^{-1}), respectively, were 18.6%-84.9%, 61.5%-97.5%, and 33.5%-93.6%. Respective efficacies for dosages lower than recommended were 30.8%-86.6%, 0.0-97.5%, and 0.0-85.8%. Respective efficacies for dosages higher than recommended were 84.9%, 92.0%-99.0% and 78.8-95.0%. Reasons given for the variable efficacies include: 1) degree of depressed larval metabolism and consequent effect on anthelmintic uptake, 2) stimulus for inhibition (season and length of environmental exposure prior to treatment) 3) host/inhibited larvae immunity interactions, and 4) route of administration/reticular groove reflex association.

In order to test the hypothesis that degree of reduction of larval metabolism might have an effect on anthelmintic uptake (1 above), oxfendazole was administered to four groups of steers at four times during the inhibition period (Table 2). Forty-eight steer calves commenced grazing 10 acres of pasture in November, 1988. In each of the months of March, May, July, and September, 1989, 12 steers were removed from pasture and placed on concrete. After three weeks, six steers were treated intraruminally with oxfendazole (4.5 mg kg- 1) and six were left as untreated controls. Seven days after treatment the steers were slaughtered and nematodes were recovered, enumerated and identified. Oxfendazole had excellent efficacy (>98.1%) against adult nematodes, high efficacy against inhibited *O. ostertagi* in March (89.4%) and September (94.3%), low efficacy in May (41.5%), and moderate efficacy in July (68.5%). These results indicate that the efficacy of oxfendazole (and perhaps other benzimidazoles) correlated nicely with hypothesized larval metabolic activity, i.e. high when acquired larvae arrest in development and subsequently emerge (phases 1 and 3), and low during quiescence (phase 2).

Taking these and the other efficacy results of albendazole, fenbendazole, and oxfendazole into account, one might expect adequate control of inhibited larvae

Table 1. Efficacy of Albendazole, fenbendazole, and oxfendazole against inhibited larvae of *Ostertagia ostertagi*.

State/ Country	Inhibition Region	No./Percent Inhibited	Dosage (mg kg^{-1})	Efficacy
Albendazole				
Florida (Courtney, 1986)	Summer	4,033/81.8	7.5	62.0
Louisiana (Williams, 1977)	Summer	3,767/48.9	7.5	83.8
Louisiana (Williams, 1979b)	Summer	107,166/96.2	7.5	62.2
			15.0	84.9
Louisiana (Williams, 1981b)	Summer	51,500/76.8	7.5	30.8
			10.0	18.6
Louisiana (Williams, 1991a)	Summer	21,719/51.8	10.0	84.9
Netherlands (Borgsteede, 1979)	Winter	50,310/86.4	7.5	85.0
Washington (Westcott, 1979)	Winter	989/26.3	7.7-8.2	86.6
Fenbendazole				
Arkansas (Yaswinski, 1985)	Summer	4,483/57.5	5.0	70.0
Australia (Anderson, 1979)	Summer	58,607/60.3	2.5	9.7
			5.0	74.6
			10.0	61.5
			20.0	92.4
		99,374/60.6	7.5	86.5
Louisiana (Williams, 1979a)	Summer	107,166/96.0	10.0	97.0
			15.0	99.0
Louisiana (Williams, 1981a)	Summer	51,500/76.8	5.0	74.7
Louisiana (Williams, 1984)	Summer	167,931/90.6	7.5	55.0
			10.0	80.0
Louisiana (Williams, 1991a)	Summer	21,719/51.8	10.0	97.5
New Zealand (Elliott, 1977)	Summer	109,917/81.3	8.5-9.3	74.6
		44,813/77.3	8.1-9.6	35.9
Texas (Craig, 1978)	Summer	9,260/60.2	5.0	24.0
			7.5	72.0
England (Duncan, 1976)	Winter	88,240/95.3	7.5	97.5
England (Lancaster, 1977)	Winter	51,567/57.7	7.5	20.9
England (Duncan, 1978)	Winter	60,900/93.8	7.5	97.0
	Winter	72,240/95.5	7.5	89.3
England (Lancaster, 1981)	Winter-Yr1	72,733/69.9	7.5	74.9
	Winter-Yr2	6,000/44.0	7.5	0.0
		24,800/78.0	7.5	49.0
		20,284/65.0	7.5	61.0
		6,700/42.0	7.5	20.0
Oxfendazole				
Australia (Anderson, 1979)	Summer	58,607/60.3	0.625	67.2
			1.25	56.7
			2.5	85.3
			5.0	88.8
		99,374/60.6	2.5	85.3
Australia (Chalmers, 1978)	Summer	76,735/64.9	2.5	76.3
Louisiana (Miller, 1988)	Summer	22,619/85.0	2.25	0.0
			4.5	33.5
			6.75	90.2
Louisiana (Williams, 1991b)	Summer	23,976/59.6	4.5	93.6
Mississippi (Couvillion, 1989)	Summer	19,560/89.5	2.25	20.3
			4.5	58.9
			6.75	78.8
England (Armour, 1978)	Winter	10,433/76.5	2.5	85.8
			5.0	90.7
Oregon (Kistner, 1979)	Winter	197,931/97.5	2.5	82.0
			5.0	95.0

during phases 1 and 3 and less than adequate control during phase 2 of the inhibition period in summer inhibition regions when administered at the recommended dosage. Whether this same effect could be extrapolated to winter inhibition regions is not known. If less than the recommended dose is given, efficacy is extremely variable and adequate control cannot be expected. The fact remains that benzimidazoles are variable in efficacy against inhibited *O. ostertagi* and level of larval metabolic activity may be a factor. Therefore, to

Table 2. Efficacy of oxfendazole (4.5 mg kg^{-1}) against inhibited larvae of *Ostertagia ostertagi* when administered at different times during the inhibition season.

Month	Treatment Group[1]	No./Percent Inhibited	Efficacy
March	Control	59,931/58.2	
	Treated	6,327	89.4
May	Control	237,183/95.0	
	Treated	138,823	41.5
July	Control	339,325/92.3	
	Treated	106,963	68.5
September	Control	106,506/66.9	
	Treated	6,062	94.3

[1] n=6 for each treatment group

have the best chance at eliminating the maximum number of inhibited larvae, consideration should be given to seasonal timing and administration of the recommended dosage based on individual animal weights or the weight of the heaviest animal in the group.

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Clostridium perfringens C & D Vaccination in Young Beef Calves: How Protective Is It?

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Abstract

Enterotoxemia in young beef calves is a common clinical diagnosis of acute deaths and/or neonatal scours. Since it is difficult to obtain a definitive diagnosis due to the nature of the agent, clinical recommendations are often made to utilize available vaccines, antiserum, or both to prevent further losses. Due to scarce data available, this field study was undertaken to determine antibody response levels to various injection protocols.

Approximately 240 beef calves born unassisted to first-calf heifers (synchronized at breeding and due to calve within 3 days of each other) were divided into four groups. After each calf nursed colostrum naturally, it received either an appropriate dosage of 7-way Clostridial toxoid subcutaneously using the "tented" technique in the cervical region; 10 cc of Clostridial antitoxin in the same manner; both injections; or 10 cc of saline as a control. All dams were boostered with *Clostridium perfringens* Types C & D toxoid 2-4 weeks prior to the due date. All calves were boostered at branding (80-90 days-of-age) with 7-way Clostridial toxoid.

Blood samples were taken (pre-injection) within 48 hours of birth; at branding; and at weaning (6-7 months). Assays using ELISA techniques were performed to determine the antibody response levels for each treatment group.