General Session III

Parasitology

Dr. Larry Smith, *presiding*

This session was supported by American Hoechst Corporation.

Ostertagia Ostertagi: Clinical Condition, Recognition of Disease States, and Approaches to Control through Anthelmintics and Management

J. C. Williams, *Ph.D.,*
K. S. Marbury, *M.S.,*
S. W. Scheide, *M.S. and*
D. G. Luther, *D.V.M., Ph.D.*
Department of Veterinary Science
Louisiana Agricultural Experiment Station
Louisiana State University Agricultural Center
Baton Rouge, Louisiana 70803

J. W. Knox, *M.S.,*
M. D. Kimball, *B.S.*
Red River Research Station
Louisiana Agricultural Experiment Station
Louisiana State University Agricultural Center
Bossier City, Louisiana 71113

T. G. Snider, III, *D.V.M., Ph.D.*
Department of Veterinary Pathology
School of Veterinary Medicine
Louisiana State University
Baton Rouge, Louisiana 70803

Summary

***Ostertagia ostertagi* is the most important parasites of cattle and occurs in all major cattle raising sections of the United States. Recognition, diagnosis, management and treatment for ostertagiasis are often misunderstood because of the complex epidemiology of the parasite in northern and southern regions of the country. It has become evident that the different disease phases of *O. ostertagi,* i.e., type I, pre-type II, and type II, as described from other parts of the world, do occur in this country. In northern temperate regions inhibition-prone larvae are acquired in autumn, persist through winter, with type II disease being a threat from late winter into spring. Larval inhibition occurs during spring in southern temperate regions such as Louisiana, the larvae remain in the inhibited state through summer and type II disease outbreaks may occur from late summer through autumn. Based on research in other parts of the world in which integrated forms of parasite control have been developed and implemented, there is an increasing interest in such control programs in this country. Appropriately-timed anthelmintic treatment, tied in with some alteration of pasture management which provides safe pasture or pasture of reduced infection potential for younger, susceptible cattle is based on parasite epidemiological data. Such data is available for some sections of the country. Some aspects of research into parasite epidemiology and integrated control conducted in Louisiana are discussed.**

Introduction

The abomasal nematode parasite of cattle, *Ostertagia ostertagi,* has drawn a considerable amount of attention over the last two or three decades. It has been described as the single most pathogenic and economically important parasite of cattle in temperate areas of the world. Since the entire United States is within the temperate zone and the parasite has been observed in all sections of the country, the full extent of its importance has yet to be determined. During the last 20 years, *O. ostertagi* has been the subject of

more literature than any other parasitic nematode of cattle. Most of this literature has originated from other parts of the world (Armour and Ogbourne, 1982). Outbreaks of parasitic disease in cattle have been associated with this worm in the United States, Australia, and Europe since the beginning of the century. Apart from a bit of information on incidence, pathogenicity, and problems of control, little is known of its epidemiology and effective prevention and control in many parts of the United States. Most of what we know of ostertagiasis has been derived from research in the United Kingdom. This and our lack of information in North America generally, have provided for a good deal of confusion and misinterpretation.

Ostertagiasis is widely known as a common disease entity in cattle throughout the country and is very likely a cause of major production deficiency in subclinical parasitism. There are really no firm or vague figures available concerning the true economic importance of the parasite, but the number of documented and undocumented reports of real or suspected problems is extensive. Interest demonstrated by pharmaceutical companies in development and marketing of new anthelmintic compounds, especially those with activity against the inhibited larval state of *O. ostertagi,* is a significant indicator of concern about the parasite. The parasite is quite common in the southcentral and southeastern states, particularly in those bordering the Gulf of Mexico. Clinical disease has been reported from New Brunswick, Canada, to California and many points in between. Whether merely suspected or detected at necropsy or clinically in the field, there are many points of misunderstanding about ostertagiasis that need to be clarified. Some of the questions or misunderstandings may include the following: what is type I disease, type II disease, and they do occur in any section of the country; can cattle of any age be affected or is it most common in particular age groups; how can ostertagiasis be diagnosed before and during clinical episodes; can ostertagiasis be a feedlot problem; if the disease is so important, how can it be prevented or properly controlled once an outbreak is diagnosed; does larval inhibition occur in all parts of the country, when does it occur, and are there anthelmintics effective against inhibited larvae? It is the purpose of this report to clarify, as far as possible, these and other questions regarding *O. ostertagi.*

The parasite invades and damages the mucosa of the abomasum and consequently impairs digestion. Although all internal parasites of cattle exert harmful effects, in varying degrees, *O. ostertagi,* in its widespread incidence, disease potential, and almost year-round availability to cattle in many regions, is most important. It is probably the most common parasite in cattle even though surveys have not been conducted recently. Although present in cattle of all ages, its greatest effect is in young, growing animals through the yearling stage.

Propagation of nearly all of the gastrointestinal nematodes of cattle, including *O. ostertagi,* begins when parasite eggs are shed in the feces. Depending upon prevailing weather conditions, primarily temperature and moisture levels, the eggs hatch and give rise to developing larval stages. The third or infective larval stage is reached in as few as 7 days during summer, but may require several weeks during winter. Survival of infective larvae on pasture is generally short in summer, but those developing in autumn may survive up to 7 or 8 months. Infection occurs when larvae are ingested with forage as the animal grazes. Since cattle seldom ingest grass near fecal pads, it is necessary for the larvae to become free of the feces and dispersed onto grass. Rainfall is the major factor in dispersal, but mechanical means such as scattering of feces by hooves and farm implements are also important. Infective larvae are able to migrate vertically on grass blades in a film of moisture.

Seasonal Variation in Development of Ostertagia Ostertagi in the Host

Depending upon regional climatic conditions, age class of animals, management systems, and other factors, development of the parasite in the host after ingestion may proceed normally or it may undergo a period of dormancy. After ingestion by cattle *O. ostertagi* third stage larvae enter gastric glands and molt to the fourth larval stage. This requires about 4 days. In the course of the time pattern of normal development, the parasite grows and molts to the early adult or fifth stage at 10 to 12 days after infection. At this point they emerge from the tissue, mature to adults on the mucosal surface and mating occurs. Total time required for development from third stage larvae to sexually mature adults is about 3 weeks. Major pathologic changes in the abomasum and clinical signs occur when the young adults emerge from the tissue to the lumen. This describes the normal development pattern of the parasite during part of the year. The parasite increases in size about 10-fold during development in the tissue. During early larval development in the gastric glands, the only visible lesion is a flat, whitened spot or plaque 1 to 2 mm in diameter. Surrounding tissue may be erythematous and lightly thickened in heavy infections. Major cellular and pathological changes occur as young adults emerge from the gastric glands. At this time the lesions are white, raised, umbilicated nodules that are pitted in the center. In heavy infections, the lesions are coalesced into a continuous mass. The gastric mucosa may be highly erythematous and greatly thickened, and has been described as morocco-leather-like in appearance. Clinical severity of infection depends on the number of worms involved and the time period over which emergence of young adults occurs.

The diarrhea, weight loss, poor appetite and other clinical signs observed coincide with the damage caused to the mucosa as young adult worms emerge from the tissue. The adult worms, once free on the mucosal surface, are not the major factor involved in pathogenesis.

Inhibition of Larval Development

At another time during the year the majority of larvae ingested on pasture undergo the first parasitic molt to the early fourth stage in the gastric glands, but develop no further for a variable and often protracted period of time. The larvae at this stage are slightly over 1 mm long and are largely undifferentiated. Histologically, they can be seen deep in the abomasal mucosa at the base of glands. It has been theorized that larval metabolism is depressed during the inhibited state. There is little or no host response to the larvae and they are not susceptible to the effects of drugs such as thiabendazole, levamisole, or morantel tartrate.

Cause of Larval Inhibition

The occurrence of larval inhibition of *O. ostertagi* and its relationship to epidemiology and disease consequences have been studied primarily in the United Kingdom (Glasgow, Scotland and Weybridge, England), southern Australia, and New Zealand. The effect of environmental conditions on infective larvae on pasture, at certain times of the year, is currently understood as the primary factor which induces larval inhibition. In northern temperate climates, larvae acquired from pasture during late autumn and early winter became inhibited. The larvae remain in the inhibited state for periods ranging from weeks up to 3 to 5 months. In some southern temperate or similar climates, larvae acquired from pasture during late winter and spring become inhibited. Duration of the inhibited state under both conditions is similar. The terms larval hypobiosis, inhibited, arrested, and retarded development are used by various authors to describe the same phenomenon. Inhibition of this nematode has been likened to the state of dormancy or diapause, is which insect populations survive through periods of adverse climate.

It has been hypothesized that in northern temperate climates, inhibition serves to carry infection through winter, a climatic period adverse to development and survival of eggs and larval stages. Infective larvae can survive through even severe winter weather on pasture, but development of eggs and earlier larval stages may cease for extended periods (Armour, 1970; 1974). In parts of Australia, where inhibition-prone larvae are acquired during spring, it is postulated that inhibition acts to carry infection through hot and dry summer conditions (Hotson, 1967). Although environmental factors are considered of primary importance in induction of larval inhibition, the actual effects on larvae are undefined. Changes in day length and temperature have been considered and larval inhibition in northern temperate *O. ostertagi* has been experimentally induced by exposing infective larvae to chilling at 4°C for several weeks. Experimental conditions required to induce inhibition in southern temperate *O. ostertagi* have not been discovered. It is considered that mechanisms responsible for inhibition are multifactorial. Other factors considered are host immunity, host-mediated factors such as endocrine changes, management practices, and density-dependence of the worm population (that is, inhibition occurring as a result of a crowding effect following infection with large numbers of larvae).

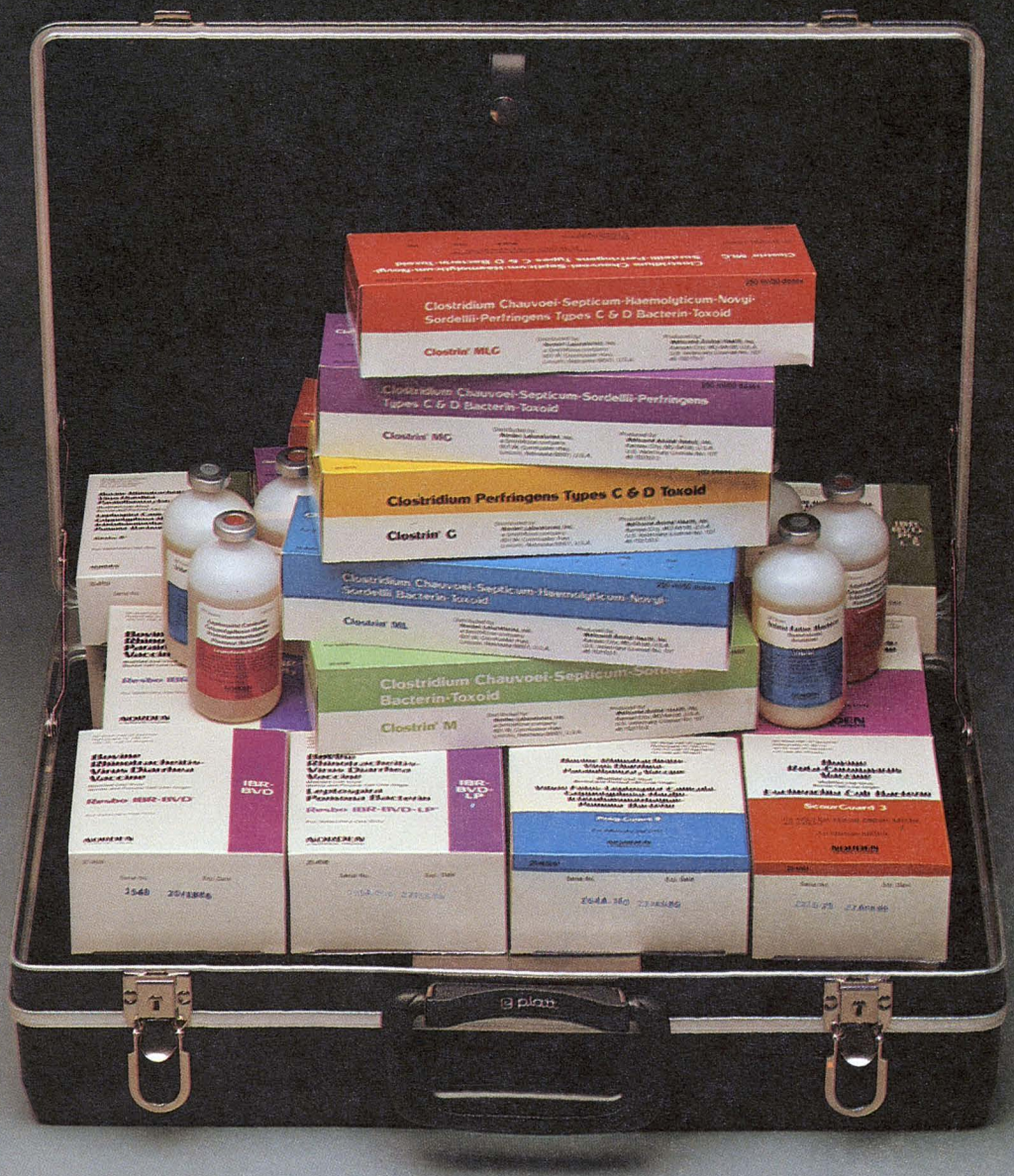
Duration of Larval Inhibition

As indicated earlier, the duration of larval inhibition is variable and may persist for weeks or months. Experimental evidence suggests that the duration largely parallels the period of climatic conditions adverse to development and survival of free-living stages. However, just as factors responsible for inhibition are considered to be multiple, the same is likely to be true for resumption of development or maturation of worms previously inhibited in development. Besides effect of environment, other factors that may play some role are parturition and lactation, number of adult worms present, anthelmintic removal of adult worms, host endocrine signals, breakdown of host resistance, and deficient nutrition (Michel et. al., 1976). Resumption of development can occur as follows: (1) a few worms mature each day from the pool of inhibited larvae, with the pool being gradually depleted; (2) inhibited larvae may resume development earlier, as in younger or weaker cattle; (3) larvae may resume development en masse at the end of the period of climatic adversity (Michel et. al., 1976). The intensity or severity of clinical effects on the host that occur when larvae resume development is variable also, depending on whether large numbers mature all at once, in waves, or a few each day over a long period of time. There may be only mild clinical effects or only a small setback in production.

Clinical Syndromes of Ostertagiasis

Parasitologists in Scotland in 1965 classified the disease bovine ostertagiasis into three phases, two of which are clinically apparent (Anderson, et. al., 1965). Both are characterized by diarrhea, weight loss, and growth failure. This classification, as initially described, was for conditions of a northern temperate climate as exists in northwestern Europe. Subsequently, the disease types were discovered to apply in parts of south Australia, but at a different season of the year than in Europe (Anderson, N., 1972, Hotson, I.K., 1967). A seasonal occurrence of these disease types in Louisiana has been found similar to what occurs in south Australia (Williams, et al., 1983).

Type I Ostertagiasis - This is the clinical entity resulting from rapid acquisition of large numbers of larvae that complete their development to the adult stage in the normal pattern of 3 to 4 weeks. Cases in beef cattle may occur from the time of weaning up to 18 months of age during winter and spring in southern temperate environments. In northern temperate environments, such infections are observed from about July



Now...clostridials from the same person who provides you with ScourGuard 3,® TSV-2,® Vibrin,® Leptoferm-5,® BovEye,™ BRSV,™ PregGuard-9® and Resbo® vaccines!

No other biological supplier has equalled the completeness of the cattle line available from your Norden representative.

And now — the Norden line becomes even more comprehensive with the addition of 'Clostrin' vaccines to prevent all eight major types of clostridial infections.

This expansion makes it even easier to consolidate your bio needs. To enjoy the convenience and time-saving advantages of doing business with a full-line supplier. To know that all the products you use are backed by the knowledgeable help of an up-to-the-minute Norden sales representative.

Let us know how we can serve you.

GOOD HEALTH
BASIC TO PROFITS

'Clostrin' clostridials

Distributed by Norden Laboratories, Lincoln, NE
Manufactured by Wellcome Animal Health, Kansas City, MO

'Clostrin M'
Cl. chauvoei-septicum and *sordellii*

'Clostrin G'
Cl. perfringens Types B, C and D

'Clostrin MG'
Cl. chauvoei-septicum-sordellii-perfringens Types B, C and D

'Clostrin ML'
Cl. chauvoei-septicum-sordellii-novyi and *haemolyticum*

'Clostrin MLG'
Cl. chauvoei-septicum-sordellii-novyi-perfringens Types B, C, D and *haemolyticum*

to the end of autumn grazing in October. Most animals in a herd may be affected, but mortality is low if treatment is administered within a few days from the onset of clinical signs. Nearly any broad spectrum anthelmintic is effective at this time. A major feature of type I disease is the finding at necropsy of large numbers of worms, most of which are adults. It is possible and highly probable that the same calves could be affected by type I infections during late winter-spring, acquire large burdens of inhibition-prone larvae during spring and then be affected by type II disease in late summer-autumn (southern temperate). Similarly, in northern temperate regions, young cattle near time of weaning could be affected by type I disease in autumn, acquire large burdens of inhibition-prone larvae in late autumn and be affected later by type II disease in late winter-spring.

Pre-type II Ostertagiasis - In this state the *O. ostertagi* population may number in thousands or hundreds of thousands of worms and the majority are inhibited early fourth stage larvae. No clinical effects are generally apparent although mild clinical signs and poor growth may be evident, depending on numbers of adult worms present. As indicated earlier, inhibited larvae are not affected by the longer established anthelmintics such as thiabendazole, levamisole, or morantel tartrate. Large accumulations of inhibited larvae in the host may result from a rather short period of acquisition over a few weeks or over 2 to 3 months. Acquisition occurs during autumn in northern temperate climates and during spring in southern temperate climates. The population of inhibited larvae basically remains static during the pre-type II phase, but small numbers of larvae mature continuously to replace adult worms lost by natural attrition. The replacement of adult worms from the pool of inhibited larvae has been described as a gradual, but continuous process (Michel, et. al., 1976) When large numbers of inhibited larvae mature simultaneously, however, the most serious of the clinical entities becomes apparent.

Type II Ostertagiasis - This form may occur in winter, but is more common in spring in northern temperate climates. In southern temperate climates such as Louisiana, it may occur anytime from late summer through autumn. In the latter situation, the occurrence is often during a drier part of the year, at a time when parasitic gastroenteritis is generally unsuspected. In both cases it occurs at a time removed from the acquisition of infection. Inappetance and marked loss of weight are usual signs and severe diarrhea is present. Submandibular edema may be a feature. Type II disease appears to be most common in yearling steers and two-year old heifers in late gestation may be particularly susceptible. Cases among older cattle, particularly bulls, are not uncommon. It is most unlikely that spring or fall born calves could be affected by type II disease at or shortly prior to weaning. Prevalence of severe clinical disease is usually low in the type II phase, and often only a few animals in the herd are affected. There has been some discussion that only the smaller and weaker animals of a herd are affected whether the type II disease is a cause or a result of their condition has not been demonstrated. Mortality of severely affected animals may be as high as 20% despite repeated treatment with the long-established anthelmintics (thiabendazole, levamisole, morantel tartrate) at short intervals. Such treatment only removes adult worms and may result in maturation of another wave of larvae. The poor prognosis in type II disease may be associated with repeated assaults on the gastric mucosa by maturation of inhibited larvae often following removal of adult worms. Detailed parasitologic, pathologic, pathophysiologic, and clinical aspects of ostertagiasis in the different phases have been previously described (Anderson, et. al., 1965; Armour, J., 1970; Armour, J., et. al., 1974).

Anthelmintics Effective Against Inhibited Larvae

Within the last year or two new anthelmintics have come on the market which have good to excellent activity against inhibited larvae. High levels of efficacy for the benzimidazole compounds albendazole, fenbendazole, and oxfendazole and for ivermectin have been established largely in experimental controlled-critical tests. Albendazole is available only on a restricted basis in several states for use against *Fasciola hepatica* and oxfendazole has not yet been cleared for use in cattle. To our knowledge there are no documented or published reports available in the United States where these compounds have been used under commercial grazing conditions to remove accumulations of inhibited larvae or to acutally control an outbreak of type II disease. However, based on efficacy data in general, fenbendazole and ivermectin should be highly efficient in removing inhibited larvae during the pre-type II phase and in minimizing losses during actual type II disease outbreaks, particularly if the condition can be detected at its onset in a herd. In experimental trials, ivermectin has been demonstrated to have a consistent high level of efficacy against inhibited larvae. Fenbendazole has been investigated more extensively and comprehensively by more investigators than all of the newer anthelmintic compounds. While generally showing good to excellent activity against inhibited larvae at the approved dosage of 5 mg/kg, fenbendazole may have some variability of efficacy against the inhibited forms. The variability has been demonstrated with all benzimidazole compounds and its cause is largely unknown. The variability is essentially an incomplete removal of inhibited larvae from individual treated cattle within a group. It should be emphasized that even with the availability of the newer broad spectrum anthelmintics, fenbendazole and ivermectin, prevention of ostertagiasis and other gastrointestinal nematodes can be efficiently achieved by strategically-timed administration of the older compounds. Pasture management alterations make such control even more efficient. This topic will be discussed in more detail in a later section.

Epidemiology of Ostertagiasis in North America

Little is known of the incidence of *O. ostertagi* disease phases in North America, although larval inhibition was recognized some time ago (Porter and Cauthen, 1946; Threlkeld, 1958; Vegors, 1958; Bailey and Herlich, 1953). It is tempting to think that they would be similar to those occurring in other parts of the world, but there is likely to be considerable variation based on a broad range of climatic conditions and differing systems of management. Do the disease types occur in North America and under what conditions? Are there variations of disease types from one climatic or geographic zone to another? At present the answers are only being developed.

Investigations conducted over the last 10 years or so indicate that regional variations in larval inhibition and disease types do occur. Larval inhibition during autumn and resulting type II disease during winter and spring have been documented in New Brunswick, Canada (Smith and Perrault, 1972). Observations of larval inhibition during autumn in Washington (Malezewski, et. al., 1975), Oregon (Kistner, et. al., 1979) and Maine (Gibbs, 1979) have also been reported. On the other hand, larval inhibition has been found to occur during spring in Georgia (Ciordia, et. al., 1971), northern California (Baker, et. al., 1981), Texas (Suderman, 1979), and Louisiana (Williams and Knox, 1976; Williams, 1981; Williams, et. al., 1981, 1983a, 1983b, 1984). In both Texas and Louisiana, the common occurrence of clinical parasitism in cattle during late summer and autumn may often be associated with maturation of worms inhibited since the previous spring. Although there are many gaps, published data and unpublished reports suggest that epidemiological patterns and occurrence of disease types of *O. ostertagi* are as shown in Figure 1. This would apply primarily to extreme northern or southern temperate regions of the United States. At present it can only be speculated as to what occurs under the varied climatic, physiographic, and management circumstances in different parts of the country. The prevalence of larval inhibition occurring in either spring or autumn is further illustrated in Figure 2. Investigation in Missouri indicated a late spring-early summer acquisition period of inhibition-prone larvae (Brauer, 1983), while autumn inhibition was strongly suggested by observations in Ohio (Herd, 1980) and Kentucky (Lyons, et. al., 1981). It is difficult to draw sharp lines delineating what *O. ostertagi* epidemiological patterns are of the northern temperate type in the absence of epidemiological observations or documented case histories of disease problems. Aside from some of the arid rangelands of the western states where the prevalence of *O. ostertagi* and other gastrointestinal nematodes is low, *O. ostertagi* should exhibit a spring or autumn inhibition in most parts of the country depending upon intensity of summer or winter climate, respectively.

In experimental field studies in Louisiana over the last several years, it has been shown repeatedly that larval

FIGURE 1. Occurence of Ostertagia ostertagi disease types in North America.

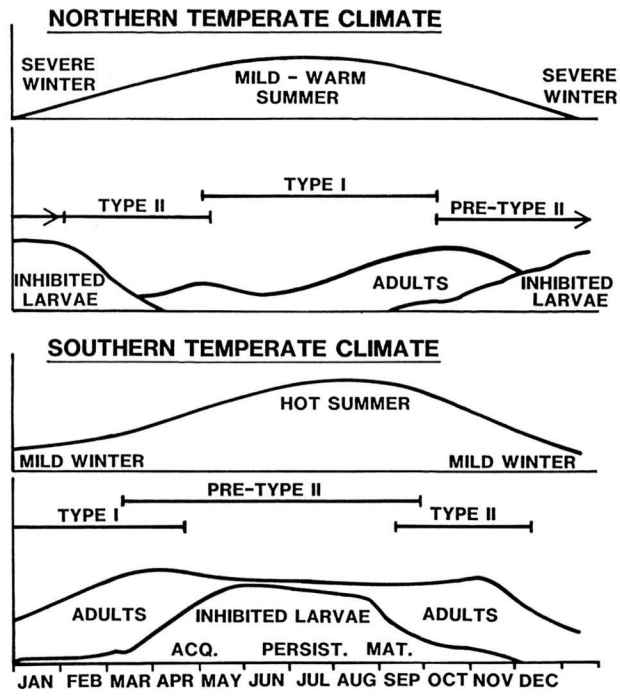
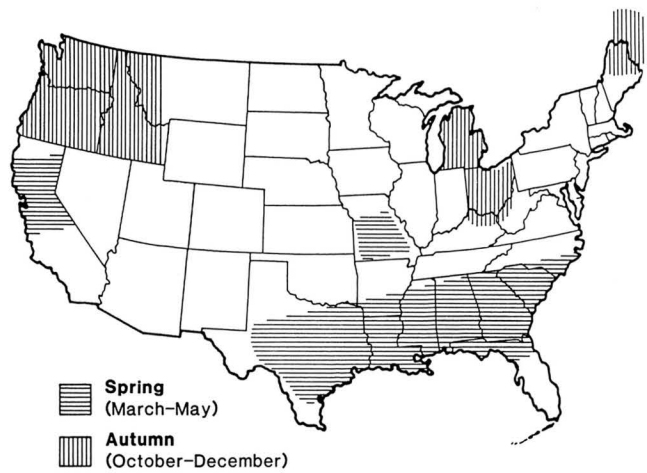
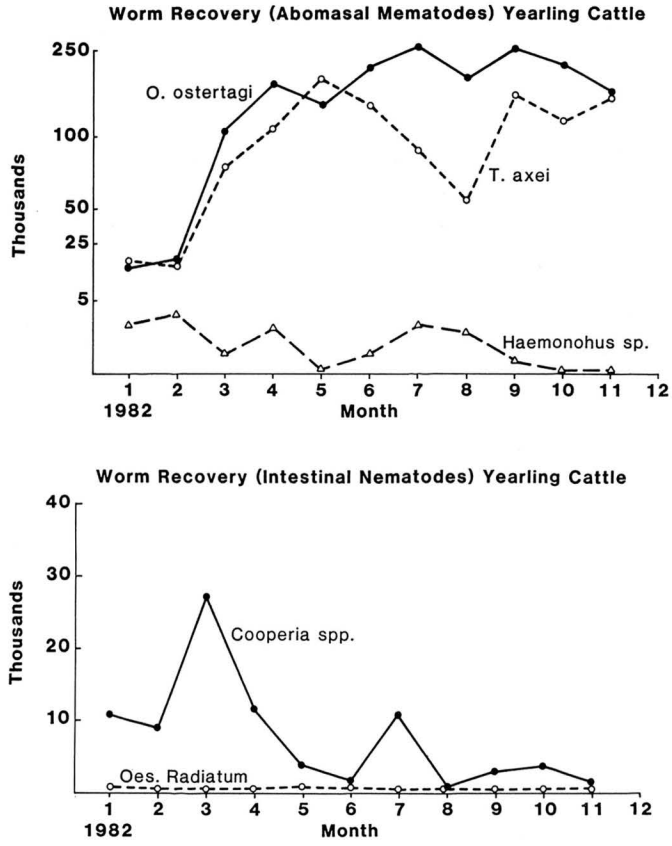


FIGURE 2. Prevalence of larval inhibition of Ostertagia ostertagi in the United States.



inhibition occurs during spring (Williams, et. al., 1983). The prevalence of different genera of nematodes in yearling beef cattle and in experimental tracer calves during 1982 is shown in Figure 3. The predominance of *O. ostertagi* particularly, and *T. axei* in both beef cattle and tracers is obvious. Worm counts from tracer calves (Figure 4) reflect the seasonal prevalence of the different genera, i.e., the winter-spring dominance of *O. ostertagi* and *T. axei* and the worm season prevalence of *Haemonchus, Cooperia,* and *Oesophagostomum.* A breakdown of the *O. ostertagi* population composition in yearling beef cattle and tracer

FIGURE 3. Recovery of nematodes from the abomasum and intestines of yearling beef cattle killed monthly in 1982.



calves in 1982 is shown in Figure 5. Small numbers of inhibition-prone larvae may be acquired in autumn and winter, but peak acquisition occurs from March through May. Adult worms are predominant in cattle from late autumn through winter and type I disease may occur from winter to early spring. Inhibited larvae acquired in spring persist in large numbers, usually until August or September. In some years maturation may begin as early as June. Few larvae are acquired from pasture during summer and worm populations in cattle at this time consist primarily of *O. ostertagi* inhibited larvae and adults of other worm genera. Maturation of inhibited larvae during late summer and early autumn is marked by increased numbers of adult and developing worms and decreased numbers of inhibited larvae. While general signs of parasitism and loss of productivity may be observed in the herd as a whole, some individual cattle will be severely affected and death can occur. Such conditions can persist to December in spite of treatment and nutritional supplement. Based on all clinical and parasitological parameters, type II ostertagiasis may occur in Louisiana from late summer through autumn. Current evidence suggests that the size of the inhibited larval population in cattle and intensity of the resulting type II disease are greater in years characterized by extremely dry and hot summer weather.

Problems of Diagnosis and Interpretation of Ostertagiasis

Not only in North America, but in parts of the world where epidemiology of ostertagiasis is well-known, diagnosis and distinguishing the clinical phases of disease is difficult either before or after death of an animal and consequently the application of appropriate treatment or management is problematic. The difficulties stem from lack of epidemiologic data, lack of documented case histories based on precise pathological, parasitological, and clinical investigation; and also from the fact that in the young animal, transition from type I through pre-type II to type II disease appears to be gradual and indistinct. It is essential at necropsy to examine abomasal washings and scrapings of the mucosa for the presence of inhibited larvae.

It should be understood that although the clinical aspects of the disease phases may differ - for example worm susceptibility to most common anthelmintics, shorter clinical course, and occurrence in younger cattle for type I disease in contrast to worm insusceptibility to the older anthelmintics, more protracted clinical course, poorer prognosis, and occurrence in yearling and older cattle for type II disease - the general pathogenesis and resulting pathology are similar in both forms. The difference is that type I disease results from recently acquired infection;

FIGURE 4. Recovery of nematodes from the abomasum and intestines of tracer calves grazed at 1-month intervals with yearling beef cattle in 1982.

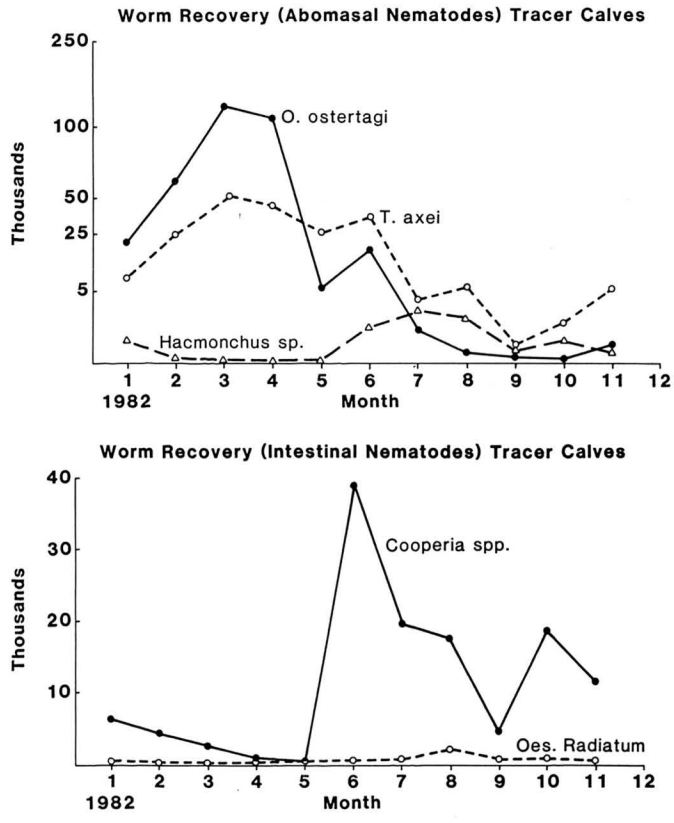
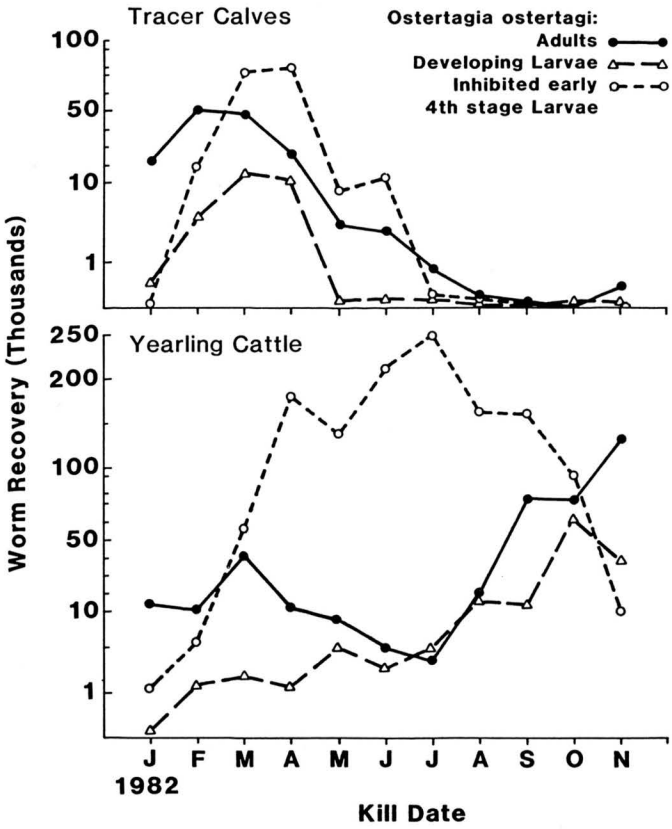


FIGURE 5. Population characteristics of Ostertagia ostertagi in yearling beef cattle and tracer calves killed monthly in 1982.



whereas type II disease results from infection acquired weeks or months earlier.

Fecal egg counts are of little value for cases of ostertagiasis and plasma pepsinogen determination is useful only in confirming diagnosis of acute clinical disease. It is necessary to consider all facets of diagnosis, including all possible predisposing factors to ostertagiasis and differentiation of parasitism from other diseases. Predisposing factors are most important and include such considerations as: location (northern or southern temperate type climate) where infection occurred, time of year or season, age class of animals, previous grazing history and management, and responsiveness to the older class of anthelmintics.

Detection of *O. ostertagi* in the pre-type II or dormant phase may pose problems in the feedlot. If cattle are treated with an effective anthelmintic at the time of arrival, parasitism as a problem may be largely ruled out. Transmission of infection under feedlot conditions is virtually nil and residual worms following treatment should be relatively short-lived. However, if cattle should come into the lot with large burdens of inhibited larvae (following spring grazing in the south or autumn grazing in the north), and were not treated or treated with an anthelmintic ineffective against inhibited larvae, then the possibility of severe clinical disease, deaths, and definite loss of productivity is a strong likelihood. It has been suspected for some time as our knowledge of ostertagiasis has increased, that certain cases of clinical disease or feed efficiency problems were of parasitic origin and possibly ostertagiasis. Such an acutal outbreak was recently described in a cattle feeder magazine (Brauer, 1984). In this case, cattle which has been backgrounded in Southwest Missouri during spring of 1983 arrived at an Oklahoma feedlot in July. Within a few days of arrival, some cattle were showing suspected signs of parasitism despite arrival treatment with an older class of anthelmintic. By 60 to 100 days large numbers of cattle were affected and some deaths occurred despite several treatments with older anthelmintics. Necropsy analysis and tracing the history of these cattle confirmed the occurrence of type II ostertagiasis. Eventual use of an anthelmintic effective against inhibitied larvae aided in resolving the disease outbreak.

Prevention and Control of *Ostertagia Ostertagi* and Other Gastrointestinal Nematodes

Any thought of eradication nematode parasites in cattle is impractical. It is imperative, however, that efforts be directed at prevention and control through the most efficient methods available. A general aim of control measures should be to keep parasite populations at levels that are not detrimental to economic production, essentially keeping the equilibrium between host and parasites shifted in the direction of the host (Brunsdon, 1980).

Until recent years the primary means of controlling nematode parasitism was by the protective use of anthelmintics. With few exceptions little attention was given to the nature or source of worm infections or to differential problems such as nutritional deficiency or other diseases. It is essential, however, in outbreaks of parasitic disease that anthelmintic treatment be given promptly to reduce liveweight losses and the chance of deaths. All animals in an affected herd should be treated, even those not showing signs of disease (Anderson, et. al., 1983). Such action does little, however, for the long term effects of parasitism through rapid reinfection after treatment. A single anthelmintic dose will kill worms in the animal over a period of a few days and this effectively relieves the clinical condition of the animal and prevents egg-shedding onto pasture for 2 to 3 weeks. On contaminated pasture cattle are readily reinfected a short time after treatment and adult worms may again be present in 2 to 4 weeks. Under these circumstances it is necessary that some form of preventive control be used to avoid the occurrence of disease outbreaks and reduced productivity associated with non-apparent parasitism.

On a worldwide basis and also in the United States, there is a developing and increasing interest in preventive systems of parasite control. Such systems are based on knowledge of the epidemiology of *O. ostertagi* and other nematodes and consist of (1) strategically-timed anthelmintic treatment alone or (2) integrated with some form of pasture

management alteration which can provide young, susceptible cattle with safe pasture or pasture of reduced levels of contamination. Use of anthelmintics alone relies on frequent dosing to totally suppress infection and pasture contamination, while integration with pasture management (safe pasture) can extend the effectiveness of treatment and reduce frequency of treatment (Anderson, et. al., 1983). Both in Europe, with summer-autumn parasitism peaks (Michel, 1967; Michel and Lancaster, 1970) and in south Australia with winter-spring peaks of parasitism (Anderson, 1980; Kroker, 1980; Smeal, 1982) use of such control systems has generally yielded significantly improved productivity over older control methods. The success of such control in minimizing subsequent pasture contamination is highly dependent on negligible reinfection after treatment and use of high efficacy anthelmintics.

Under conditions in the United States provision of safe pasture is most feasible during summer to early autumn in the south because climatic conditions during this time are adverse to survival of parasite eggs and larvae. The same very likely hold true for many other parts of the country. Use of certain pasters for harvesting hay over several weeks or use for grazing by dry cows or other older stock can effectively reduce levels of contamination. Provision of safe pasture during autumn through spring is less feasible, but the cultivated winter annual pasture or harvested grain fields of the plains states are prime examples of safe pasture. Yearly alterations of land for pasture and row crop production would offer another option. The simple expediency of "resting" or keeping certain pastures free of animals for shorter intervals during summer or longer intervals during autumn and winter may be considered, but land utilization priorities would often exclude this option. Cattle of any age going on to such pasture should always be treated prior to entry in order to keep contamination reduced. The capability of safe pasture provision for young, susceptible stock is a management tool that will vary according to resources available to individual producers.

There have at present been few experimental efforts at integrated parasite control in beef or dairy cattle in the United States. Exceptions to this are the recent work by Herd and Heider (1980) and Craig, et. al., (1982). However, a close examination of serious-minded cattle operations indicates that integrated control is in use, but not necessarily planned with parasite control in mind or based on the epidemiology of parasites in relation to pasture management or climatic factors. It is the result of individual common sense and managerial skill. It can be observed in the appropriately timed use of anthelmintics, in use of harvested row-crop land for weaned calves or stocker cattle, and in the opportune use of older cattle to graze certain pasture between different groups of young. Such practices as these and others can become more structured and widespread, providing that epidemiological data is generated for different regions.

Experimental Control Studies in Louisiana

As indicated earlier, epidemiological investigation of *O. ostertagi* and other nematodes, was conducted in Louisiana over several years. Data obtained from these observations was applied in the design of experiments on integrated control in cattle from weaning through the stocker phase of development.

The first grazing experiment was conducted between November 1982 and August 1983. The purpose was to test the efficiency of controlling nematode parasites and enhancing productivity by (1) use of safe pasture and strategically-timed anthelmintic, (2) use of the same treatment alone, with cattle on contaminated pasture, in comparison with (3) cattle on contaminated pasture in which little control effort was exerted. The anthelmintic treatment schedule and pastures used were as follows:

Group	November 11 and 30, 1982	Pasture	February 2, 1983	April 27, 1983
1 n = 15 5.1/ha	thiabendazole Paste 110mg/kg	Safe	tramisol injectable 6mg/kg	tramisol injectable 6mg/kg
2 n = 15 5.1/ha	thiabendazole Paste 110mg/kg	contaminated	tramisol injectable 6mg/kg	tramisol injectable 6mg/kg
3 n = 15 5.1/ha	thiabendazole Paste 66mg/kg November 30 only	contaminated	No treatment	No treatment

Fifty-two head of newly weaned beef steers (predominantly Angus x Hereford crosses) were divided into 3 groups based on an equal distribution of body weights and assigned to their respective group pastures on November 30. All experimental pastures were planted in ryegrass for winter-spring grazing and Coastal Bermudagrass was the predominant warm season forage. Initial treatment in November was to remove the large worm burdens characteristically present at time of weaning. Treatment on February 1 was primarily intended to remove adult *O. ostertagi* at a time when this population begins to contaminate pasture with eggs that will give rise to inhibition-prone larvae during spring. The April 27 treatment was given to eliminate adult worm burdens prior to the onset of hot and dry weather which would help to maintain low levels of pasture contamination during late spring and summer grazing. Pastures designated as safe (Group 1 cattle) were derived by alternately using the land for hay harvest, grazing by dry cows, and some resting between July and November. Contaminated pastures were grazed by infected yearling stock up until a few weeks before the experiment began. Various epidemiological and parasitological parameters were observed, but only fecal egg counts and cattle weights as presented here.

Group average fecal egg counts were shown in Figure 6. The high egg count noted for Group 3 cattle on November 30, was representative of initial pre-treatment egg counts of all cattle in the experiment. Group 3 cattle were not treated until November 30, and then only with a reduced dosage of thiabendazole. Counts for both Group 1 and Group 2 increased considerably prior to treatment again on February 1, and January 27, respectively. Counts for Group 3 cattle remained high from winter into spring, but decreased spontaneously (without treatment) in late spring. Such a decrease in counts in yearling cattle is a common occurrence and is possibly due to a host immune response to some genera such as *Cooperia* spp. and to the fact that most *O. ostertagi* ingested during spring become inhibited in development and remain dormant all summer. Counts for Group 1 cattle on safe pasture remained low for the duration of the experiment after the February 1 treatment, while counts for Group 2 cattle increased modestly during spring and began a gradual increase after April 27. Although spontaneously reduced during spring, counts for Group 3 cattle remained highest during summer.

Average daily gains and cumulative liveweight changes are shown in Figures 7 and 8. Cattle of all groups either lost weight or made very poor gains during December and January. The ryegrass stand was well-established, but weather conditions were consistently cold and wet; hay supplementation was necessary. With exception of cattle in Group 3, substantial gains were observed in February and continuing through May. Cattle in Group 1 made their

FIGURE 6. Group mean fecal egg counts of yearling beef cattle, 1982-83.

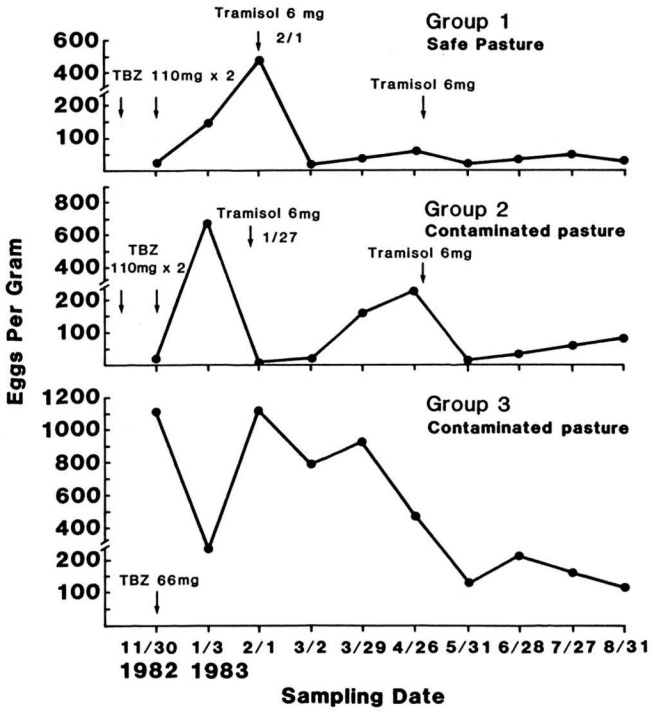


FIGURE 7. Group mean average daily gains of yearling beef cattle, 1982-83.

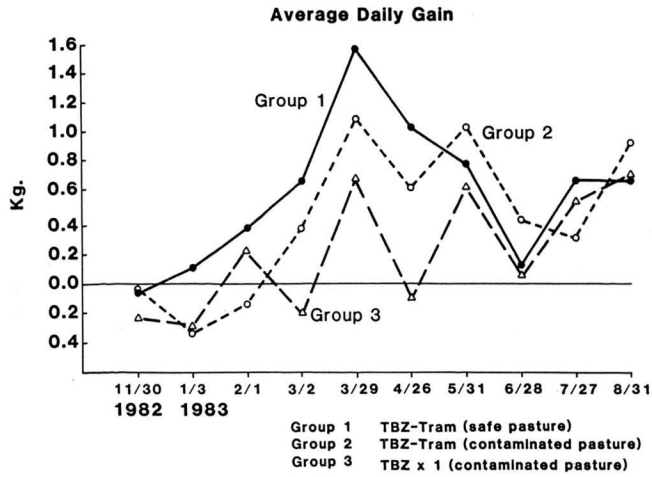
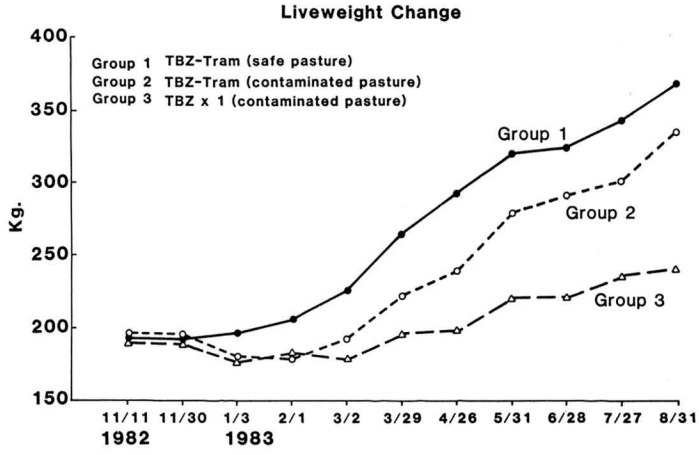


FIGURE 8. Group mean cumulative liveweight changes of yearling beef cattle, 1982-83.



greatest rate of gain (1.6 kg A.D.G.) during March. Although gains were generally very good for Group 2 cattle in the period, cattle of Group 3 had erratic gains and losses. While February through May is usually a period of active forage growth and increased nutritional quality, it is also the optimal period of the year for larval development and survival and transmission of infection. Gains made by Group 1 cattle on safe pasture in this period set it apart from the other two groups, which were at heavy risk of infection. Weight loss by all groups during June was attributed to hot weather and forage scarcity. After June no group had a gain advantage over the other. Final average weights for cattle of each group recorded in September, after 9 months on forage only were: Group 1 - 368 kg, total gain of 178 kg; Group 2 - 337 kg, total gain of 147 kg; Group 3 - 262 kg, total gain of 72 kg. Slaughter of tracer calves in December and April and representative yearling cattle in September indicated that *O. ostertagi* and other nematodes were effectively controlled on

safe pasture. In contrast cattle of Group 2 and Group 3 and tracer calves grazed with them had large numbers of *O. ostertagi* (particularly in Group 2 cattle) and largest numbers of *T. axei* were found in association with Group 3 pastures.

A similar control experiment was conducted between November 1983 and August 1984. Objectives of this experiment were to determine comparatively the effects of two anthelmintics (fenbendazole, Safeguard®, American Hoechst Corporation and morantel tartrate, Rumatel®, Pfizer Agricultural Division) on control of nematode parasites and productivity of cattle on contaminated pastures. The anthelmintic treatment schedule and pasture use is as follows:

Group	November 15 and 30, 1983	Pasture	February 1, 1984	April 25, 1984
1 n = 17 5.3/ha	fenbendazole 10% suspension 5mg/kg	contaminated	fenbendazole 10% suspension 5mg/kg	fenbendazole 10% suspension 5mg/kg
2 n = 17 5.3/ha	morantel bolus 9.7mg/kg	contaminated	morantel bolus 9.7mg/kg	morantel bolus 9.7mg/kg
3 n = 18 5.3/ha	fenbendazole 10% suspension 5mg/kg	Safe	fenbendazole 10% suspension 5mg/kg	fenbendazole 10% suspension 5mg/kg

Fecal egg counts for all groups were uniformly high prior to treatment in November; all were reduced to zero or near zero by the two initial treatments (Figure 9). Group 2 counts (morantel) rebounded to highest levels prior to the February 1 treatment. Both groups of cattle on contaminated pasture had higher counts than Group 1 cattle (safe pasture) in March. The April 27 treatment again reduced all counts to low levels, but those of cattle on contaminated pasture increased more extensively during the rest of the experiment.

Average daily gains and cumulative liveweight changes are shown in Figures 10 and 11. Record cold weather occurred during December and January and very likely was responsible for the consistent weight loss in all groups through March. Both short (ryegrass) and long term (Coastal Bermudagrass) effects on forage productivity were evident. All groups gained substantially through late May, but lost weight during June because of heat and forage scarcity. All groups performed well during the last 2 months, but Group 2 cattle lagged behind the two groups treated with fenbendazole. In contrast to results in 1983, cattle grazed on safe pasture in spring 1984 did not show a gain advantage over the other groups except in May, but did show a considerable advantage over the other groups during the last 2 months. Group 3 cattle (fenbendazole-safe pasture) finished the 9 month grazing period at an average weight of 311 kg, total gain of 117 kg; Group 1 cattle (fenbendazole - contaminated pasture) finished at 294 kg, total gain of 100 kg; Group 2 cattle (morantel - contaminated pasture) finished at 261 kg, total gain of 67 kg. Worm recovery from

FIGURE 9. Group mean fecal egg counts of yearling beef cattle, 1983-84.

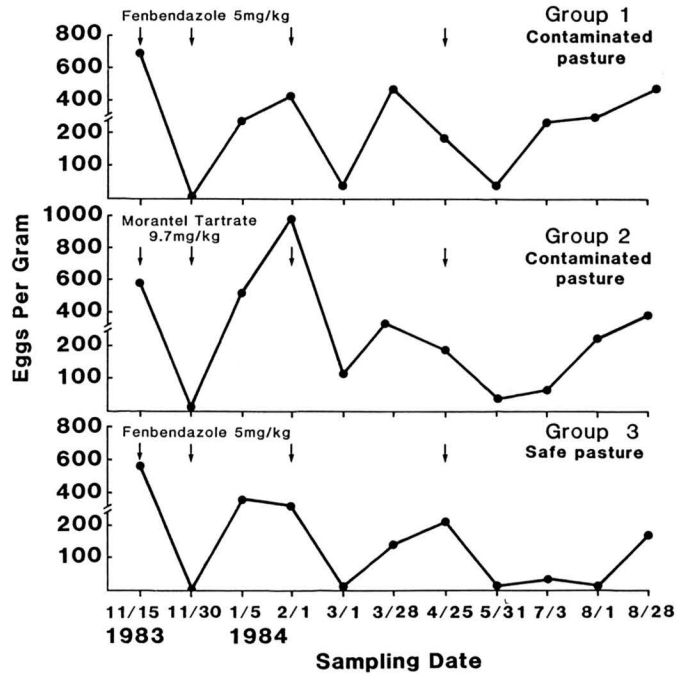
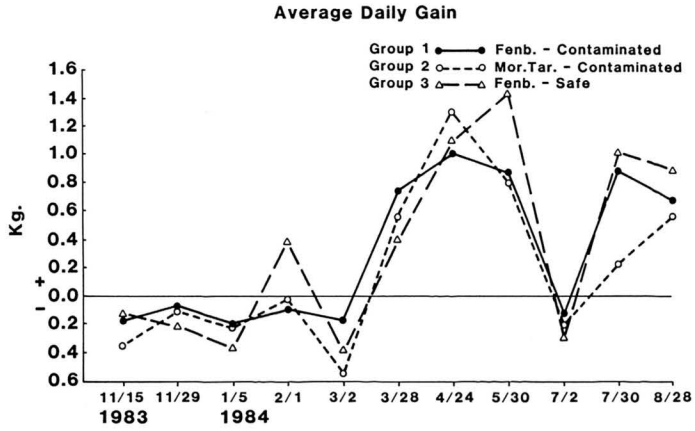


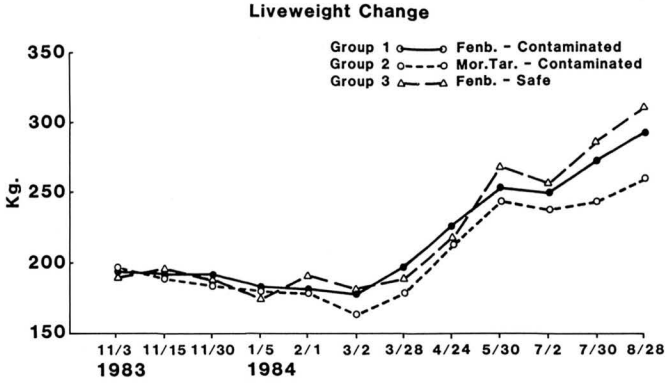
FIGURE 10. Group mean average daily gains of yearling beef cattle, 1983-84.



tracer calves in December and April indicated much reduced worm burdens in those on safe pasture in comparison to those on contaminated pastures. Numbers of *O. ostertagi* were greatest in tracers grazed with Group 1 cattle (fenbendazole - contaminated pasture) in December, but greatest numbers were found in tracers grazed with Group 2 cattle (morantel - contaminated pasture) in April.

Observed differences in levels of productivity and effects of parasitism in the two experiments have demonstrated the advantage to be gained in provision of pasture with reduced levels of parasite contamination through alterations in animal and pasture management. In the 1982-83 experiment final average weights of cattle on safe pasture exceeded those

FIGURE 11. Group mean cumulative liveweight changes of yearling beef cattle, 1983-84.



of the other 2 groups by 31 and 106 kg, respectively. In 1983-84, weight of cattle on safe pasture exceeded the others by 17 and 50 kg, respectively. It is understood that any forms of alteration in existing systems of management may not always be convenient or practical, but where possible, advantages in productivity could be great.

References

1. Anderson, N., Armour, J., Jarrett, W.R.H., Jennings, F.W., Ritchie, J.S.D., and Urquhart, G.M.: A field study of parasitic gastritis in cattle. Vet. Rec. 77, 1196-1204 (1965). 2. Anderson, N: Ostertagiasis in beef cattle. Vict. Vet. Proc. 30, 36-38 (1972). 3. Anderson, N: The epidemiology and control of bovine ostertagiasis in a winter rainfall region of southeastern Australia. Proc. Ostertagia Symp. 80, Roseworthy College, Australia, pp. 63-69, (1980). 4. Anderson, N., Donald, A.D., and Waller, P.J.: Epidemiology and control of parasitic gastroenteritis of cattle in the temperate climate zone. In: The epidemiology and control of gastrointestinal parasites of cattle in Australia (Ed. by N. Anderson and P.J. Waller), CSIRO, Div. Anim. Health, Melbourne, Australia, pp. 47-62, (1983). 5. Armour, J.: Bovine ostertagiasis: a review. Vet Rec. 86, 184-190 (1970). 6. Armour, J.: Parasitic gastroenteritis in cattle. Vet Rec. 95, 391-395 (1974). 7. Armour, J., Jennings, F. W., Murray, M., and Selman, I.S.: Bovine ostertagiasis. In: Helminth parasites of cattle, sheep, and horses in Europe. (Ed. by G.M. Urquhart and J. Armour), R. Maclehose, University Press, Glasgow, pp. 11-23 (1974). 8. Armour, J. and Ogbourne, C.P.: Bovine ostertagiasis: a review and annotated bibliography. Misc. pub. no. 7, Commonwealth Institute of Parasitology, Farnham Royal, Bucks, England, 93 p. (1982). 9. Bailey, W.S. and Herlich, H: An enzootic of parasitic gastritis in mature cattle. Auburn Vet. 9, 3-8 (1953). 10. Baker, N.F., Fisk, R.A., Bushnell, R.B., and Oliver, M.N.: Seasonal occurrence of infective nematode larvae on irrigated pasture grazed by cattle in California. Am. J. Vet. Res. 42, 1188-1191 (1981). 11. Brauer, M.A.: Epidemiology and bionomics of *Ostertagia ostertagi* in beef cattle in Missouri. Ph.D. thesis, University of Missouri - Columbia, 161 pp. (1983). 12. Brauer, M.A.: Whodunnit: tracking sleepy ugly (brown). Calf News, 22, 22-24 (1984). 13. Brunsdon, R.V.: Principles of helminth control. Vet. Parasitol. 6, 185-215 (1980). 14. Ciordia, H., Neville, W.E., Baird, D.M. and McCampbell, H.C.: Internal parasitism of beef cattle on winter pastures: level of parasitism as affected by stocking rates. Am. J. Vet. Res. 32, 1353-1358 (1971). 15. Craig, T.M., Spitzer, J.C., Leps, R., and Fulton, R.K.: The effect of anthelmintic treatment on growth of beef heifers on temporary winter pasture. Southwest. Vet. 34, 1-4 (1982). 16. Gibbs, H.C.: Relative importance of winter survival of larval nematodes in pasture and infected animal carriers in a study of parasitic gastritis in calves. Am. J. Vet. Res. 40, 227-231 (1979). 17. Herd, R.P: Animal health and public health aspects of bovine parasitism. J. Am. Vet. Med. Assoc. 176, 737-743 (1980). 18. Herd, R.P. and Heider, L.E.: Control of internal parasites in dairy replacement heifers by two treatments in the spring. J. Am. Vet. Med. Assoc. 177, 51-54 (1980). 19. Hotson, I.K.: Ostertagiasis in cattle. Aust. Vet. J. 43, 383-387, (1967). 20. Kistner, T.P., Wyse, D., and Averkin, E.: Efficacy of oxfendazole against inhibited *Ostertagia ostertagi* in naturally infected cattle. Aust. Vet. J. 55, 232-235 (1979). 21. Kroker, G.A.: Demonstration of a strategic drenching program for cattle at Ballorat. Proc. Ostertagia Symp. 80, Roseworthy College, Australia, pp. 101-105, (1980). 22. Lyons, E.T., Tolliver, S.C., Drudge, J.H., Henken, R.W., and Button, F.S.: Efficacy of levamisole against abomasal nematodes and lungworms in dairy calves: preliminary tests indicating a reduced activity for *Ostertagia ostertagi*. Am. J. Vet. Res. 42, 1228-1230 (1981). 23. Malczewski, A., Westcott, R.B., Spratling, B.M., and Gorham, J.R.: Internal parasites of Washington cattle. Am. J. Vet. Res. 36, 1671-1675 (1975). 24. Michel, J.F.: The epidemiology and control of parasitic gastroenteritis in calves. Proc. 4th Int. Meet. World Assoc. Buiatrics, Geneva, pp. 272-287 (1967). 25. Michel, J.F. and Lancaster, M.B.: Experiments on the control of parasitic gastroenteritis in calves. J. Helminth. 44, 107-140 (1970). 26. Michel, J.F., Lancaster, M.B., and Hong, C.: Observations on the resumed development of arrested *Ostertagia ostertagi* in naturally infected yearling cattle. J. Comp. Path. 86, 73-80 (1976). 27. Porter, D.A. and Cauthen, G.E.: Experimental infection of calves with the small stomach worm, *Ostertagia ostertagi*. J. Parasitol. 32, 14-15 (1946). 28. Smeal, M.G.: Bases and approaches for control of nematodes in cattle. In: Biology and control of endoparasites. (Ed. by L.E.A. Symons, A.D. Donald, and J.K. Dineen), Academic Press, New York, pp. 187-201, (1982). 29. Smith, H.J. and Perrault, J.P.: a type II ostertagiasis outbreak in cattle in New Brunswick. Can. Vet. J. 13, 114-117 (1972). 30. Suderman, M.T.: Seasonal hypobiosis of *Ostertagia ostertagi* (Stiles, 1982) Ransem, 1907, in catttle in Brazos County, Texas. M.S. thesis, Texas A&M University, College Station, 96 pp. (1979). 31. Threlkeld, W.L.: The histotropic phase and other biological aspects of *Ostertagia ostertagi*. J. Parasitol. 44, 342-343 (1958). 32. Vegors, H.H.: Observations on inhibited development of cattle nematodes. Proc. Helm. Soc. Washington 25, 86-90 (1958). 33. Williams, J.C. and Knox, J.W.: Effect of nematode parasite infection on the performance of stocker cattle at high stocking rates on Coastal Bermudagrass pastures. Am. J. Vet. Res. 37, 453-464 (1976). 34. Williams, J.C.: Bovine ostertagiasis: problems caused by the medium stomach worm. Anim. Nutr. and Hlth. 36, 6-9 (1981). 35. Williams, J.C.: Epidemiology and control of gastrointestinal nematode parasites of cattle. University of Arkansas Agricultural Experiment Station, Special Report 117, pp. 48-75 (1984). 36. Williams, J.C., Knox, J.W., Baumann, B.A., Snider, T.G., Kimball, M.G., and Hoerner, T.J.: Efficacy of ivermectin against inhibited larvae of *Ostertagia ostertagi*. Am. J. Vet. Res. 42, 2077-2080 (1981). 37. Williams, J.C., Knox, J.W., Baumann, B.A., Snider, T.G., Kimball, M.D. and Hoerner, T.J.: Seasonal changes in gastrointestinal nematode populations in yearling cattle in Louisiana with emphasis on prevalence of inhibition in *Ostertagia ostertagi*. Int. J. Parasitol. 13, 133-143 (1983a). 38. Williams, J.C., Knox, J.W., Sheehan, D.S., and Fuselier, R.H.: Experimental and natural infection of calves with *Bunostomum phlebotomum*. Vet. Parasitol. 13, 225-237 (1983b).