IMPACT OF INTERNAL PARASITES ON BEEF CATTLE

Thomas M. Craig
Texas A&M University, College Station 77843

ABSTRACT

Internal parasitism is a pervasive constant that reduces returns in beef cattle production. Parasitism may influence production sufficiently so that data derived investigating performance response will be erroneous. The interaction of internal parasites of cattle with other facets of their lives makes it imperative that more parasite research be done on cattle to consider their impact. Results of trials designed to determine forage production at various stocking densities may not reflect the nutritive value of the forage, but instead the severity of parasite exposure. Relative resistance or susceptibility to certain parasites vary with the breed of livestock and the species of parasite. Extensive work to evaluate the ability of various breeds and sires to influence parasitic numbers has been conducted in sheep but not in cattle. Some parasite trials have ignored the effects of nutrition on the establishment and retention of parasites. This becomes especially important where multiple parasite exposure may occur. Susceptible cattle, even on an optimal diet, will become parasitized, but if reexposed to the same species of parasite, cattle on an optimal diet may be able to resist reinfection, whereas those on deficient diets will not.

(Key Words: Beef Cattle, Parasites, Pasture Management.)

Introduction

Internal parasitism in cattle can be divided into three categories with regard to the parasites' effect on the host: infection, economic and clinical. Infection is the presence of parasites within cattle, but because of the numbers and/or species composition of parasites there are no demonstrable adverse effects. Infection is universal and is manifested as a constant equilibrium between host and parasite. Economic parasitism is the level of infection that prevents the host from reaching its genetic potential in the production of meat, milk or other measurable criteria. Economic parasitism is widespread, seasonal and often affected by other factors including quality and abundance of feed, stocking rate, age, sex, breed or acquired resistance. Clinical parasitism occurs when an imbalance exists between host and parasite to the extent that overt disease is detected. This may be manifested as anemia, diarrhea, poor growth rate, anorexia or other departures for a physiologically normal state. Clinical parasitism usually can be anticipated by an experienced husbandman, and treatment or control measures can be implemented.

Of the three categories, economic parasitism is the most difficult to assess because of the many factors that may be involved. One common way of assessing economic parasitism is by the administration of an anthelmintic to cattle and then, after a predetermined period of time, comparing weight gains between treated and untreated control animals (Table 1). This is a valid method of determining anthelmintic effects, but it may not always assess the true effects of parasitism. If sufficient time elapses between treatment and evaluation, reinfection may have occurred such that both groups have similar numbers of parasites. Conversely, if the cattle are not reexposed to parasites (such as in a feedlot), natural attrition may decrease parasite numbers and the effects of compensatory growth may negate any differences occurring shortly after treatment (Nansen, 1987). The greatest problem encountered in pasture studies involving cattle is determining whether or not to graze the treated and untreated cattle in separate but "comparable" pastures or to allow treated cattle to graze pastures being


2 College of Vet. Med.

Received October 5, 1987.
Accepted January 28, 1988.
TABLE 1. REDUCED LIVEWEIGHT GAINS CAUSED BY ECONOMIC
HELMINTH INFECTIONS IN YOUNG CATTLE

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Observation period, mo</th>
<th>% Reduction in weight gain</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untreated vs 4 treatments</td>
<td>7</td>
<td>22</td>
<td>Cairns and Gallagher, as cited by Barger (1982)</td>
</tr>
<tr>
<td>Untreated vs 5 treatments</td>
<td>11</td>
<td>18</td>
<td>Brunsdon, as cited by Barger (1982)</td>
</tr>
<tr>
<td>Contaminated vs clean pasture</td>
<td>5</td>
<td>24</td>
<td>Helle and Tharaldsen, as cited by Barger (1982)</td>
</tr>
<tr>
<td>Metacercaria vs uninfected</td>
<td>6</td>
<td>29</td>
<td>Hope-Cawdery et al. (1977)</td>
</tr>
<tr>
<td>Untreated vs suppressive treatment</td>
<td>12</td>
<td>33</td>
<td>Morley et al., as cited by Barger (1982)</td>
</tr>
<tr>
<td>Metacercaria vs uninfected</td>
<td>7</td>
<td>14</td>
<td>Chick et al. (1980)</td>
</tr>
<tr>
<td>Untreated vs suppressive treatment</td>
<td>16</td>
<td>24</td>
<td>Hutchinson et al., as cited by Barger (1982)</td>
</tr>
<tr>
<td>Untreated vs 2 treatments</td>
<td>3.5</td>
<td>17</td>
<td>Ciordia et al. (1984)</td>
</tr>
<tr>
<td>Untreated vs 1 treatment</td>
<td>2.5</td>
<td>26</td>
<td>Ryan and Guerrero (1987)</td>
</tr>
<tr>
<td>Untreated vs monthly treatments</td>
<td>4</td>
<td>13</td>
<td>Isles et al. (1985)</td>
</tr>
<tr>
<td>Untreated vs 2 treatments</td>
<td>5</td>
<td>15</td>
<td>Craig et al. (1982)</td>
</tr>
<tr>
<td>Untreated vs 2 treatments</td>
<td>5</td>
<td>23</td>
<td>Herd et al. (1985)</td>
</tr>
<tr>
<td>Untreated vs sustained release</td>
<td>11</td>
<td>16</td>
<td>Tolling et al. (1981)</td>
</tr>
</tbody>
</table>

contaminated with parasites by the untreated controls. These are mutually exclusive factors, and no satisfactory solution has been proposed that will address both the nutritional differences between pastures and parasite exposure between groups of cattle.

Generally, young grazing cattle are the most adversely affected, carry heavier burdens of parasites, and are the primary source of contamination to the pasture (Dunn, 1978). Clinical parasitic disease may be seen in dairy replacement heifers or stocker calves that are intensively grazed, whereas single-suckled beef calves seldom exhibit clinical signs of parasitic infections. One reason for this difference is that most of the larval worms ingested by mother cows fail to establish because of her acquired resistance, so the contamination of the pasture is considerably reduced (Dunn, 1978; Overend et al., 1984; Soulsby, 1985; Nansen, 1987). In addition, the nursing calf has the advantage of high-quality nutrition provided by its dam. Therefore, control programs should be targeted either directly at calves or at the pastures they will occupy. In some instances, cows occupying the pasture prior to calving or during early lactation will contribute to pasture contamination due to a periparturient rise in egg count (Hammerberg and Lamm, 1980). However, most parasite larvae are present in the pasture due to shedding by young cattle (Eysker and Van Meus, 1982; Overend et al., 1984).

There are four important types of internal parasites of cattle: gastrointestinal nematodes, lungworms, liver flukes and coccidia. Other internal parasites such as tapeworms only rarely are important in the U.S., but they may cause condemnation or necessitate special handling of carcasses, as in the case of *Taenia saginata*, the human beef tapeworm.

Gastrointestinal Nematodes and Lungworms

Gastrointestinal nematodes and lungworms have similar environmental requirements. The bionomics of the free-living stages of parasites are important to the survival and transmission of parasitic nematodes. Development from the egg to the infective stage is temperature- and humidity-dependent (Dunn, 1978). Development of the embryo within the egg, hatching of the larvae, feeding and molting two times may occur in as little as a week during the summer, but this process may be delayed for a month or longer during the winter. During the developmental stages the larvae are very susceptible to
desiccation, and the longer the development time, the lower the survival. However, after larvae have reached the infective stage, they become relatively resistant to drying and cold and will survive as long as energy stored during the earlier larval stages remains available. Energy is expended in a temperature-dependent fashion. After the energy stores are depleted, the larvae die. Hence, larvae that were deposited as eggs in the autumn will survive the winter and be available to grazing cattle the following spring. If larvae do not infect a host, they are unlikely to survive the summer. Rotational grazing systems are unlikely to give sufficient rest to a pasture to allow a meaningful number of larvae to die before regrazing.

The adverse effects caused by the presence of the parasite vary depending on the species of parasites involved. In some cases the most severe damage is caused by the immature stages, rather than by the adult worm. Thus, cattle can exhibit clinical signs of parasitism while having few or no parasite eggs in their feces. Some nematode species do not cause economic loss unless they are present in tremendous numbers, whereas others in comparatively low numbers may cause disease.

The species of parasites present in a given area is determined by climate. Irrigation or other agricultural systems may change the microclimate of the parasitic larvae's environment. This may enable a parasite to survive in an area where otherwise it would die. As climate determines where a parasite is found, weather determines when the parasite will be transmitted. The free-living, ensheathed, infective larvae need moisture to leave the dung pad and ascend the herbage. Once they are free of the dung pad and on herbage, their sheath protects them from desiccation and, unless the environment becomes extremely dry, they survive. There is little evidence that larvae ascend and descend the herbage in a regular pattern. They move with moisture and may be trapped in leaf nodes or elsewhere on the plant, where they remain until ingested by the host.

Each species of parasite has its own niche within the host and requirements for survival outside of the host. Unless this is considered, control programs are unlikely to be effective against the primary pathogens. The most important species of internal parasite of cattle in temperate regions of the world is Ostertagia ostertagi. This extremely adaptable parasite has evolved in different regions of the world to enable it to survive the most adverse weather conditions. It avoids unfavorable weather conditions by undergoing arrested development (a phenomenon akin to diapause in insects; Armour, 1970). For instance, where hot, dry summer conditions are the most devastating period for survival of larvae, as in Texas and Louisiana, development of the parasite is arrested during this period (Craig, 1979; Williams et al., 1983). Conversely, in areas where the cattle are housed during the winter because of snow and lack of forage in the pasture (e.g., Maine), parasite development is arrested during the winter (Gibbs, 1979). Because the larvae within the cattle are metabolically inactive during arrested development, little if any damage is done to the abomasum of the host. Cattle may accumulate massive numbers of arrested larvae within their abomasum with no signs of disease. However, when larvae resume development (during the period of time usually most favorable to the parasite), clinical disease will be evident. While in the arrested state, the worms are not producing eggs, imbibing blood or engaged in other activities to make their presence known. Hence, cattle considered to be unparasitized in fact may host hundreds of thousands of larvae. The clinical disease thus may be seen in situations where the cattle could not have possible have become infected by larvae, such as in a feedlot. Even if numbers of parasites are insufficient to cause clinical disease, economic parasitism may influence production (Schillhorn van Veen and Melancon, 1984).

The damage caused by parasites varies considerably with the species of parasite present, the resistance and resilience of the host, and the quality and quantity of feed available (Goldberg, 1965; Chalmers, 1980; Holmes, 1985). The presence of some nematodes such as Ostertagia or Trichostrongylus will reduce feed intake by the host. Although the reason for this inappetance is not fully understood, feed intake may be reduced by up to 20% as compared to nonparasitized livestock (Symons, 1985; Holmes, 1987). There also are changes in gastrointestinal motility, digestion and absorption. The movement of ingesta may be either decreased or increased. Digestive enzymes such as pepsin and brush border enzymes may be depressed so that nutrients are not utilized (Randall and Gibbs, 1981; Hammerburg, 1986). Mechanical damage to the gastric mucosa reduces enzyme production and activa-
tion and may allow macromolecules to pass into the circulation or be lost through the intestinal tract (Murray, 1969).

The quality and quantity of meat and milk can be decreased in parasitized cattle due to loss of protein (blood, plasma) into the gastrointestinal tract and increased protein metabolism by the intestinal tract. Skeletal changes also can occur due to limited absorption of P caused by intestinal nematodes. Loss of K increases in parasitized calves, which can increase retention of body fluids (Holmes, 1987). Both carcass quantity and quality may be affected by internal parasitism, even following recovery and a feeding period (Enterocasso et al., 1986). Although calves usually are affected most often by internal parasites, clinical and economic disease also may occur in adult cattle (Selman et al., 1976).

Some nematodes, such as *Dictyocaulus viviparus*, the cattle lungworm, cause devastating disease when present in large numbers but appear to be of little significance when present in low to moderate numbers. The lungworm stimulates an early and strong protective immune response in calves. Thus, most single-suckled beef calves in endemic areas will have established a strong resistance to infection prior to weaning. Lungworms require more moisture for survival and dispersal on pasture than gastrointestinal parasites. If cattle from areas where lungworm is not found are moved into an endemic area and the weather conditions are favorable, parasitic bronchitis may occur; parasitic bronchitis can kill cattle.

**Liver Flukes**

Two species of liver fluke occur in cattle in the U.S., *Fasciola hepatica*, the common liver fluke, and *Fascioloides magna*, the giant deer fluke. Approximately 1.5 million livers are condemned annually in the U.S. due to these parasites (AAVP, 1983). Other losses in cattle due to the presence of live fluke are difficult to quantitate. Trials conducted under varying conditions indicate vastly different production effects of flukes (Chick et al., 1980; Dargie, 1987). The incidences of anemia, hypoalbuminemia and hyperglobulinemia are markedly increased with fluke infections. Anorexia and increased protein turnover contribute to poorer feed efficiency and slower weight gains in cattle with as few as 54 flukes (Hope-Cawdery et al., 1977). In addition to economic parasitism, liver damage caused by migrating flukes provides a substrate on which bacterial agents such as *Clostridium hemolyticum* thrive, leading to bacillary hemoglobinuria.

Flukes require two hosts in the life cycle, a snail and a final host. *Fasciola hepatica* may parasitize a number of hosts, but cattle, sheep and goats are the most important economically. The final hosts of *F. magna* normally are deer, in which the life cycle is completed. However, they often parasitize other ruminants. Only a few *F. magna* will kill small ruminants; they are economically important in cattle (Foreyt and Todd, 1972, 1976).

**Coccidia**

The life cycle of the coccidia, *Eimeria* spp., is considerably different from that of the helminths in that the free-living infective form of the coccidia is retained within a "cyst" that can occur only on vegetation if carried there by mechanical means. The cyst is quite resistant to the environment and may survive for a year or longer if protected from direct sunlight and desiccation (Hammond, 1973). The cyst is small (10 to 40 μm) and can be transported by wind or rain, but fecal contamination of feed or water is the primary means of spread. As with other parasites, the level of exposure will largely determine the extent of disease, if any. Coccidiosis stimulate a host immune response. Resistance seems to be of fairly short duration and can be overcome by stress (Niilo, 1970). Coccidiosis is primarily a disease seen in crowded conditions where fecal contamination of feed or water occur readily. It also is a disease of the young when exposed to a particular species of coccidia for the first time.

Pathogenic species of *Eimeria* damage the mucosa, especially of the cecum and colon, causing blood loss and diarrhea. Large areas of epithelium may be sloughed. Most calves will make an uneventful recovery in a few weeks following clinical signs; however, some calves become permanently stunted (Ernst and Benz, 1986). In less than clinical numbers, the economic losses due to coccidia are difficult to ascertain and probably are negligible in most animals.

Another condition, *Cryptosporidium*, has received considerable interest in recent years. This organism, associated with a watery diarrhea in young calves, is extremely small and often is overlooked as a pathogen. It is the primary cause of diarrhea in dairy calves that are exposed early in life. After the initial
infection, calves apparently are immune to re-infection. However, the organism is zoonotic and may cause diarrheal disease in humans (Fayer and Ungar, 1986).

**Literature Cited**


Murray, M. 1969. Structural changes in bovine ostertagiasis associated with increased permeability of the bowel wall to macromolecules. Gastroenterology 56:763.


