# **"BOVINE PARASITISM"**

### **Recent Issues and Strategies for Parasite Control in Grazing Cattle**

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Bovine parasitism is a highly variable and complicated disease condition that exists with varying degrees in all grazing cattle throughout North America. Over the past twenty-five years, veterinarians and cattle producers have received a considerable amount of information about parasites of cattle, the damage they cause and methods for the control thereof. The following presentation is designed to summarize a major portion of this information and provide a review of the current knowledge of parasitism as it relates to cattle production. This discussion is intended to be a review in the understanding of what cattle parasites are, how they develop, survive and reproduce, and how they can best be controlled to prevent economic loss. From a producer standpoint, parasite control is the keystone for everything else they do in their operation. Parasite-free cattle overwinter better, milk better, raise bigger calves, breed better, find their vaccinations work better and most often have less disease problems than parasitized cattle. The most important aspect of deworming cattle is deworming at the right time and with the right product or products.

As a cautionary note, not all veterinary parasitologists agree on all aspects of the economic importance of bovine parasitism. There are applied parasitologists and there are "white ivory tower" parasitologists. Parasitologist that work in a laboratory usually have little knowledge or understanding about how, when and why cattle need deworming. There are many parasitologists that still believe and promote "spot worming" of cattle in the middle for the season after parasite contamination levels on the pastures are their highest points and cattle themselves are suffering from parasitisms. The major reason for this discrepancy is that determining the economics of parasitism in pasture cattle can only be tested under field conditions which is very time consuming, expensive, and the results are often directly affected by nutrition, climatic conditions and production standards. These studies must be conducted away from the laboratory and require a working knowledge of cattle and pasture management in order to be successful designed and conducted. Only a limited number of veterinary parasitologists throughout the world are truly qualified to be able to design and monitor field trials related to parasitism and beef production, and therefore, only a limited number of reliable studies exist have been properly conducted and reported in scientific journals over the past 25 years.

In cattle, gastrointestinal parasitism is frequently ascribed to as the summation of effects by several or many different species of parasitic nematodes which may be present in an animal at any given time. Seldom are cattle infected by a single species of parasites, in fact, most grazing animals will harbor five to eight different species of nematode parasites at the same time. These common gastro-intestinal nematode parasites of cattle are nearly all similar in morphology, pathology and nearly all undergo a "direct life cycle" where the animal becomes infected by ingesting an infective  $(L_3)$  staged parasitic larva which undergoes several molts until it matures to an egg laying adult living in the gastro-intestinal tract. The eggs are then passed out of the animal with the feces. Once out of the animal, the egg embryonates to a L1 larva, hatches from the egg and then develops into a L2 then an infective third staged  $(L_3)$  larva which moves with moisture away from the fecal pat on the existing vegetation waiting to be consumed by an unsuspecting animal.

<u>A direct life cycle</u> means that no intermediate host is required for normal egg to larvae to adult to egg development. Mature worms living in the gastro-intestinal tract or lungs lay eggs (lungworm eggs are coughed up and swallowed), which pass out in the feces. Infective larvae that develop from these eggs, move onto the herbage and are ingested by cattle while grazing. These ingested larvae develop to an egg-laying adult worm that starts the life cycle process all over again. Several generations of worms can develop within grazing animals during a single grazing season.

The major difference in economic importance between the various kinds of parasitic worms in cattle depends upon the species of parasites present within the animal, the type of damage each worm causes and numbers of parasites present in the animals at any given time. Cattle parasites are very specific to cattle with little or no cross transmission to humans, horses, or wildlife. The most common internal nematode parasites of cattle and they major damage they cause are as follows:

### ABOMASUM (4<sup>th</sup> stomach): (HOT Complex)

**Haemonchus (Barber Pole Worm):** A very important blood sucking parasite of cattle; both larval and adult stages live in the abomasum and have been identified as bloodsuckers. *Haemonchus* is the most prevalent and dangerous gastro-intestinal parasite of sheep and goats. If present in high numbers in young cattle, clinical parasitism will develop.

**Ostertagia:** Commonly called the <u>Brown Stomach Worm</u>. Larvae temporarily destroy gastric glands that produce hydrochloric acid and pepsinogen. This reduced acid production raises the pH of the abomasum reducing digestion which is very important in finishing cattle and high lactation dairy cattle. Adult worms damage the gastric mucosa. Damage is associated with a "Moroccan leather" appearance where high numbers of larvae are present. *Ostertagia* is the most prevalent gastro-intestinal parasite of cattle throughout North America.

**Trichostrongylus:** Lives off gastric fluids, and can cause necrosis of mucosa. Patches of necrotic tissue give a frosted appearance. Some hemorrhage occurs. *Trichostrongylus* does not usually appear in high numbers, however, when present in high numbers this worm can cause major damage thus the name "Bankrupt Worm." This term is especially common among sheep producers.

#### **SMALL INTESTINE:**

**Strongyloides:** These parasites are often called <u>Threadworms</u>. Larvae gain entrance orally but also through the skin; they migrate through the lungs and then to small intestine. This parasite an cause dermatitis when the larvae penetrate the skin and cause pneumonia when the larval stages pass through the lungs. Adults cause hemorrhage in small intestine. Most prevalent in young calves born in wet and

muddy calving areas or in calves raised in an area with infective bedding. Symptoms can be seen in calves as early as one month of age. It may take four to five months for the calves to recover if not treated correctly.

**Cooperia**: Adults disrupt digestive function in the intestinal mucosa. The second most prevalent gastro-intestinal worm found in cattle, especially prevalent on pastures in late fall. Two or three species of Cooperia may be found in the same animal. Pure infections have been shown to reduce gains in young cattle as much as 30 lbs over a single grazing season.

*Moniezia* (cattle tapeworms): This parasite develops in the soil mite, which is ingested by cattle and develops to an adult in approximately 6-8 weeks. The pathogenesis of this parasite has not been studies under field conditions. Numerous reports have been published showing intestinal blockage when numbers of worms found exceed 10-15 worms. Tapeworms in cattle can exceed six feet in length. They absorb nutrients through their cuticle. Tapeworm eggs are distinct and can easily be found in a fecal exam.

**Nematodirus:** One of the most pathogenic parasites in cattle. This parasite is most prevalent in young calves. *Nematodirus* can be found in as high as 60% of the young stock in areas of the Midwest and Great Plains and is most prevalent in young dairy calves. Larvae survive well in cold climates and can survive several years in the environment. Larval stages and adults can both be very pathogenic causing epithelial erosion and diarrhea. This parasite is seldom found in mature cattle because of an age resistance that builds up with animals exposed to this parasite at a younger age.

**Bunostomum:** Called the <u>Hookworm</u> of cattle. Larvae penetrate the skin and migrate through the parasite is commonly found in young dairy calves especially in calves housed on manure packs or on contaminated bedding.

#### Cecum:

**Trichuris:** Called the cattle <u>whipworm</u>. The head portion of adult whipworms deeply penetrate the intestinal and cecal wall. Mostly found in young calves. Can cause severe diarrhea and is often confused with symptoms of coccidiosis. Cattle are infected by ingesting embryonated eggs through direct fecal contamination. The whipworm egg can be viable for many years.

#### Large Intestine:

**Oesophagostomum:** Called the <u>Nodular Worm</u>. Larvae produce large nodules in the wall of the large intestine. Parasites are associated with anorexia, depressed weight gain and diarrhea. This parasite is commonly found in adult cows. Heavy infections found at slaughter usually leads to condemnation of the intestines.

#### Lungs:

**Dictyocaulus:** Called the cattle <u>Lungworm</u>. Adults traumatize the bronchioli causing excess mucosa secretions, bronchitis, and emphysema. Infected animals are predisposed to bacterial infections. Disease is often misdiagnosed as bacterial or viral infection. Clinical disease occurs most often in early spring or late fall and occurs mostly in young animals.

#### LIFE CYCLE OF GASTRO-INTESTINAL PARASITES:

It is essential to understand the life cycle and survival patterns of cattle parasites in order to develop successful control strategies. The life cycle for all gastro-intestinal worms is similar except for several deviations listed above. This animal/pasture/animal cycle is a direct life cycle. To simplify this cycle, it can be broken into three major periods; the developmental period during which the parasite develops from an egg to an infective larvae on pasture, the prepatent period which is the time interval from ingestion of infective larvae to the appearance of eggs in the feces, and the adult or patent period in which the parasite coexists with its host; reproducing for future generations.

**I.** <u>The developmental period</u> is the time in which the parasite spends outside the host and, therefore, is very dependent upon environmental conditions for survival and development. Eggs pass in the feces of the infected animal, hatch into larvae in the fecal pat and develop through the first and second larval stage to the third or infective stage larvae. Under favorable weather conditions, this larval development process can occur in two to three weeks. Under cool or adverse weather conditions, this development process can take as long as four to six months.

Moist, warm climatic conditions provide an optimal environment for eggs and larvae. Temperatures between 65° and 85° F are ideal, especially for *Ostertagia*. The infective larva has a protective covering or sheath that helps it survive unfavorable weather conditions, however, extreme dry conditions can rapidly destroy infective larvae, also cool or cold weather will retard larval development.

Infective larvae must be distributed on the pasture out of the fecal pat for transmission to be completed. This is greatly facilitated by rainfall and dew. Other mechanical means such as insects, birds, and cattle feet also help the distribution of larvae. The feet of cattle stepping on fecal pats become especially important when fecal pats become hard and crusty without rain and the larvae become trapped inside of the pat. Once rain breaks up this pat, parasitic gastroenteritis can follow, especially if large numbers of eggs/infective larvae are present in the manure pat and it has been dry for some time. Once the larvae escape the fecal pat, they move to and survive in the vegetative material surrounding the pat waiting to be ingested by grazing animals. Larvae will seldom move farther than several feet from the pat.

**II.** <u>The prepatent period</u> is the time from when infective larvae are ingested until they develop into mature egg-laying adult worms. Ingested infective *Ostertagia* larvae, for example, lose their protective covering and move to the abomasum where they penetrate and encyst themselves in the gastric glands. The encysted larvae then develop to the fourth stage in the gland in approximately four to five days growing in size very rapidly. By the end of the second week of infection, the larvae have usually molted in to a fifth stage or young adult worm. A few days following this molt, these worms then emerge, forcing their way out of the gland onto the surface of the gastric mucosa where they mature, mate and begin to reproduce. The prepatent period is influenced by the immune state of the infected animal. For young cattle the prepatent period is usually 3 weeks whereas for mature cattle it often takes four to five weeks before eggs appear in their feces following ingestion of infective larvae.

**III.** <u>The patent period</u> is the survival and reproduction time of adult worms in its host animal. Each female worm can lay thousands of eggs during her adult life, which may be as short as four weeks and as long as 12 months. Some parasites such as *Trichuris* have been reported to live in an animal as long as two years. Basically, the entire infection process is a dynamic process where larvae are constantly being ingested as old parasites

are dying off and passing out in the feces. Winter or dormant pastures changes this process when new larval ingestion slows down or stops; this is where inhibited larvae begin development and emerges, keeping the parasite life cycle going.

**Inhibition (arrested development):** Inhibition is a phenomenon reported to occur in most parasites of cattle. The inhibited or arrested state of developing larvae of *Ostertagia*, however, is the most notable. This inhibition state occurs primarily in yearling cattle with no reports in nursing calves and few reports in adult cattle. The speed with which larvae develop in the gastric glands and emerge into the abomasum as an early adult worm varies from 10 days to six months depending upon level of worm burden present and conditions of the gastro-intestinal tract at the time of larval ingestion.

During inhibition, infective exsheathed 3rd stage larvae move into the gastric glands and molt, but instead of continuing to develop, these early fourth stage larvae remain in a dormant state. During this period of inhibition, larval metabolism is thought to be minimal. This phenomenon of arrested development is also referred to as hypobiosis and can last for several weeks or even months. These larvae often emerge at a time of the year when parasitism isn't suspected such as during the winter.

The true cause for this inhibition is not fully known, however, it appears to be tied to total worm burden. When an animal becomes heavily infected, physiological conditions of the gastro-intestinal tract changes such that conditions such as high pH occur, for example, and conditions are no longer right for larval development. Development stops and the larvae become inhibited. As pasture conditions change such as during a pasture "brownout" caused by a hot dry summer or when cattle are removed from pasture during the winter; larval intake stops, no new larvae are being ingested, older worms die off and gastro-intestinal conditions begin to improve. Previously inhibited larvae begin developing, emerge and mature to adult egg laying worms.

The build-up of parasitic infections on pasture probably accounts for most of the variation found as to when inhibition is reported to take place in different parts of the country. In southern regions of the country, inhibition is reportedly most common in late spring or early summer. The reason for this is that favorable grazing conditions develop early in the year and parasite larval development can begin as early as late February or early March in very southern regions. Pasture contamination, therefore, can occur as soon as late April or early May with inhibition occurring in late May through August. Under northern conditions where cattle may only begin to graze in May and early June, pasture build-up doesn't occur until late August or early September with inhibition occurring from September onward, which coincides with the northern inhibition period.

The normal process is for these inhibited larvae to emerge slowly over a long period of time, however, if the inhibited larvae in a heavily infected animal emerge all at once; disease characterized as Type II ostertagiasis (as described below) occurs. Parasitic disease caused by *Ostertagia* is broken down into two types; Type I and Type II ostertagiasis.

**Type I disease** is parasitic gastro-enteritis caused by a heavy infection of *Ostertagia*. Type I disease most often develops in cattle grazing heavily infected pastures and is easily diagnosed since it occurs soon after infection. Type I disease is a very common syndrome in cattle exposed to heavy parasite infections on pastures while Type II disease is a fairly rare syndrome and is only found in non-treated, heavily infected, poorly managed cattle on a low plane of nutrition. Often, only several animals out of a group will show symptoms for Type II disease.

**Type II disease** may occur when most unexpected even several months after the animals have been removed from pasture and is caused by a large number of inhibited larvae emerging into the lumen of the gut at the same time. The mechanism, which causes this sudden emergence of larvae, is not fully understood but is usually associated with poor nutrition or other stress related conditions.

The inhibited phase is a survival mechanism that gastro-intestinal parasites have to help maintain a continued life process, enabling parasites to survive unfavorable pasture or weather conditions. It also helps parasite survival by preventing the parasites from overwhelming and killing the host. When an animal becomes heavily infected, if some of the infecting larvae stop development and remain inhibited, both the parasite and its host have a better chance for long-term survival. Some Parasitologist have compared this inhibition mechanism to the diapause's phenomenon in insects which works like a biological clock which programs the larvae to cease development until a more favorable environment is present.

**EPIDEMIOLOGY: Parasite Development within the Host and Egg to Larvae Survival on Pasture:** During the parasite life cycle, pastures are contamination by infective larvae, which develop from worm eggs passed in the feces of infected cattle. This is a very important part of the transmission cycle. Worm eggs must hatch and develop to infective larvae for this parasite cycle to continue. Worm eggs need favorable weather conditions to develop. Ideal conditions are plenty of moisture and warm temperatures. When these conditions are present worm eggs can develop into infective larvae in just a few days. If the temperatures are cool it will take longer for development to occur; it may take weeks or even months for the development to take place. If the temperatures are too cold the development process stops altogether.

Since a female worm can lay thousands of eggs in its lifetime, and the parasite's life cycle in a young calf can take as little as three weeks for infective larvae to develop to an egg laying adult parasite, one worm and her progeny can produce several million eggs over a summer grazing season. So, if only a few worms survive the winter or a dry period, the pastures can still become heavily contaminated in a short time.

Factors that affect the level of pasture contamination include:

- 1. The level of worm egg excretion.
- 2. The stocking rate or density of grazing animals.
- 3. The survival rate of different worm eggs.

- 4. The survival rate of infective larvae.
- 5. Pasture management variations.
- 6. Anthelmintic treatment.

Parasitic larval contamination on the pasture is a continual process. A normal pattern of pasture contamination is as follows: when spring grazing begins - cattle consume infective larvae which have survived the winter from the previous grazing season. Within three to four weeks after ingestion, these larvae grow into adult egg laying worms in the gastro-intestinal tract, further contaminating the pastures. Once contaminated in the spring the pastures then remain contaminated through the following spring.

Most parasitic infective larvae can survive long periods on pasture even under adverse weather conditions if sufficiently protected by the manure pat, forage cover, soil or even snow. Most parasitic larvae can survive for one year on pasture. This means that parasite eggs shed in the spring can survive on pasture until the following spring. One parasite species, *Nematodirus*, larvae have been reported to survive for several years under Canadian winter conditions. Parasitic larvae that survive the winter have a limited life however, and will only survive for several months into the spring. Parasites that get caught on pasture herbage when severely cold or dry conditions develop soon die without moisture, those larvae in the pat and soil, however, usually have sufficient moisture to survive even very dry or very cold conditions.

Research has shown that parasite-free tracer calves can pick-up several thousand infective larvae over a several week period at the beginning of spring in northern areas such as Wisconsin, Idaho, Montana and Canada. It appears that once favorable conditions return and grass growth resumes, larvae that survive from the previous season become available to the animals grazing these spring pastures.

In the springtime, the larvae that have survived the winter move away from the fecal pat and onto the herbage. These larvae, since a protective sheath surrounds them, cannot feed and, therefore, soon die if not consumed. This becomes an important part of the epidemiology, since resting the pasture from grazing animal during the first three months of the grazing period will render that pasture relatively parasite free. For gastro-intestinal parasites to survive from year to year under harsh winter conditions; cattle grazing the spring pastures must either be shedding worms eggs from adult worms that they have carried through the winter or from newly developed larvae they've consumed that have survived on pasture from the previous season which are now egg laying adults.

Worm egg development on the pastures follow a cyclic pattern with peak contamination rates occurring at various times of the years depending on the weather, the grazing pattern or pasture management used and the type of animal grazing these pastures. Egg development in the spring is slow while the weather is cool, but as the temperature becomes warmer, the time for egg development decreases. In this way, a large number of worm eggs can reach maturation almost simultaneously, resulting in a large increase in pasture contamination. This sudden increase in pasture contamination is called "spring or mid-summer rise."

When conditions that are unfavorable for parasite development return to the pastures such as extremely hot and dry or cold and snowy conditions, this high level of contamination begins to disappear and continues to decline until favorable conditions return. Despite the destruction of larvae already on the herbage by these adverse conditions, the reservoir of larvae in the fecal pat and soil usually remain unharmed.

**THE EFFECT OF NUTRITION ON PARASITISM:** The importance of nutrition in affecting the host's resistance to parasitic disease has been recognized a long time. The role of dietary protein in the resistance of animals to parasitic infection is that only when the crude protein intake falls below a critical level will the host's resistance to infection be diminished. Resistance to establishment of infections has also been shown to depend greatly upon the nutritional status of the host. Any variation in nutrition produces a variation of equal magnitude in resistance. The reverse is also true where the establishment of parasites can affect the nutrition of a host and thereby cause further individual variation depending upon the reaction given by the host.

The resistance of an animal to the parasites is different than the resistance of the animal to the effects caused by the parasites, for it is often possible to feed past an infection. Animals given a nutritionally balanced diet will seldom show a visual effect of parasitism (unless heavily infected) although feed conversion may be very poor due to the direct effect of the animal harboring parasites in its gastro-intestinal tract. A recent study conducted in Nevada demonstrated that the strategic deworming of nursing calves on creep feed showed less benefit in improved weight gain than deworming nursing calves which were not on creep feed. This study indicated that if feed cost is not important, creep feeding the calves equaled the benefit of strategic deworming, however, it makes more sense to deworm in addition to adding creep feed so as to not loose money feeding wormy calves.

Within different age groups, competition for food increases the chance of exposure to parasitism, both, in quest of food to fulfill their hunger (i.e., in eating down pastures or grazing closer to fecal pats where parasitic larvae may be concentrated), and in reduced resistance to the establishment of parasites because of poor nutrition. The effect of nutrition on the course of a parasitic infection falls broadly into two categories, the effect on the parasite and the effect on the defense mechanisms of the host.

Studies on performance and parasitism in beef herds showed that the level of protein in the diet rather than the forage consumption determined the parasite load. Calves fed on fescue pastures had heavier worm burdens than similar calves on clover pastures. It is possible, however, that a variety of factors (often times not taken into account during the assessment of the nutritive value of a pasture) may increase parasitism differentially by allowing some species to be available to the host in greater numbers than others. An example of this is the effect of forage height on the acquisition of larvae where some larvae may not migrate as high on the forage as another parasite.

**VARIATIONS DUE TO MULTIPLE SPECIES INTERACTION:** Interactions occur between parasite species which affect the establishment and virulence of various parasites. This phenomenon appears to be operative in most infections and further confounds the host-parasite interaction. In the presence of *Haemonchus* infections, a proportion of *Nematodirus* was inhibited at the fourth stage and the development of the remainder through the fifth stage was slowed. Because of the prevalence of this parasite-parasite interaction, it is suggested that a patent infection may play an important role in resistance to subsequent challenge infections and the development of inhibition.

**THE ROLE OF INHIBITION IN PARASITISM:** Originally, inhibition was defined as occurring when a population of adult worms was accompanied by a population of members of the same species that had been arrested in the early fourth stage of development even though considerable time might have lapsed since they were ingested as infective stages by the host animal. An important aspect of this interaction become evident when it was discovered that the inhibited larvae gained the capacity to complete their development after some, or all, of the adult worm population were removed. It seems that once sufficient numbers of adult parasites have died and the digestive tract begins to return to more normal conditions, inhibited larvae are triggered to resume development.

Massive inhibition of larval development in the host has been noted in many host-parasite systems: *Trichostrongylus retortaeformis* in rabbits, *Ostertagia circumcincta* in sheep, *Ostertagia ostertagi* in calves, *Cooperia oncophora* in calves, *Dictyocaulus viviparus* in cattle, *Haemonchus contortus* in sheep and *Nematodirus* spp. in sheep. The above observations have all reported the phenomenon of inhibited, arrested or retarded development of worms to be one of a seasonal occurrence and is usually found in the young animals carrying heavy worm burdens.

Inhibition periods begin in early fall but primarily are associated with late autumn grazing in temperate areas (Northern U.S.) and in late spring grazing in semi-tropical areas (Southern U.S.). The inhibition of development of parasites has been reported to start to occur during the last part of September in New England. By the last two weeks of October and early November, approximately 90 percent or more of the mean larval worm burdens in weaned calves were inhibited in the early fourth stage.

Arrestment of infective larvae in the fall of the year does not seem to produce disease conditions although massive build-up of L4 larvae can be present in early winter months. The occurrence of acute signs of parasitism is evident as a result of the emergence and further development of inhibited L4 larvae. Differences between individuals can be from the degree to which the build-up of L4 larvae occurs and the timing of emergence of these inhibited larvae. In one study, signs of Type II disease developed in animals approximately a month after they are stabled. As winter progressed other animals developed the disease symptoms.

The importance of inhibition in the relationship of the parasite to its host in temperate regions of the world lies chiefly in the ability of the parasite to survive the adverse winter or summer conditions by maintaining itself inside the host during these unfavorable times.

The termination and maturation of arrested larvae occurs spontaneously in the spring on completion of a physiological process analogous to diapause development. This process is especially favorable for parasites with free-living stages susceptible to winter conditions. The development of arrestment at the early fourth stage is probably a biological adaptation for overwintering and, as such, forms an integral part of the epidemiological pattern of this parasite in temperate regions. The status of the individual animal at the time immunological control of the parasite is needed is possibly of greatest importance. The emergence of inhibited larvae soon after parturition, therefore, reflects a temporary suppression of the host's immune capacity.

**DISEASE VARIATIONS DUE TO STRESS FACTORS:** Several investigators have put forward a hypothesis that for the first time clearly explains the role of lactation in this phenomenon. These authors found in a study of nematode parasite populations that lactating animals showed higher fecal egg counts and carried larger worm populations than did unmated females or females deprived of their offspring at birth. These authors also demonstrated that non-lactating animals acquired fewer parasites than did lactating animals when grazed together on the same infected pasture during parturition and lactation. It is possible, however, under these field conditions that the animals of different physiological states may not have had similar levels of larval intake.

Animals in late pregnancy behaved essentially in the same manner as the lactating animal, including a higher rate of establishment and development of worms when compared with non-reproductive animals. It has been reasoned that the maturation of inhibited larvae, increased fecundity of existing parasites, and increased susceptibility to fresh infection were all manifestations of temporary relaxation of immunological control, caused by endocrine changes associated with lactation and late pregnancy.

The effect of lactation and pregnancy on the temporary suppression of the immune system and subsequent alteration of the host-parasite system in cattle is an important part of the study on the nature of parasitism's. An animal calving early in winter may escape any ill effects of increased establishment of parasites because of a lack of available infective larvae; however, her reduced immunological status may precipitate the emergence of arrested larvae and thus cause stress to this animal in another way. The observed ill effects from parasitism in lactating cattle may not reach clinical proportions as often happens with lactating ewes. The major problem caused by parasitism in brood or dairy cows seems to be one of economic consequence from reduced milk flow rather than one of obvious disease syndromes. Of secondary importance is the survival and propagation of parasites within a herd which in turn may serve as a source of infection for the more susceptible young stock.

**DISEASE VARIATIONS DUE OF BEHAVIORAL FACTORS:** In studying the nature of parasitism in cattle, it is not sufficient to consider only those biological processes and host-parasite interactions that occur during the parasitic phase. The accessibility of the cattle to the infective stage of the parasites becomes important in field situations. The behavior of the cattle, the management of the cattle and the external environmental conditions all interrelate to determine the existence or non-existence of parasitism.

The behavior of each individual animal plays an important role in the acquisition of larvae by the animal. The grazing and feeding habits of the host may influence transmission. Cattle will not eat around fresh fecal pats, but will return to graze these same areas in four to five weeks later, which is sufficient time for the infective larvae to have developed.

It cannot be ignored that animals often behave differently in their individual feeding and grazing behavior. Animals are often observed eating dirt especially common after a long winter when cattle are first turned out on pasture. Some animals are more curious than others and, therefore, may consume more larvae than non-curious animals. This habit may vary from day to day, most probably depending upon the expression of the animal's mood. This individual behavioral difference is important, but in a practical sense, little can be done to alter animal behavior and prevent animals from exposing themselves to infective larvae.

**The control of parasitism in grazing cattle:** The control of gastro-intestinal parasites in cattle has evolved over the past 30 years from the use of dewormers simply as a way to purge an animal of its parasite burden to the strategic use of dewormers as a way to prevent the build-up of a parasitic infection on the pasture and subsequent infections in animals grazing these pastures over an entire grazing season. This evolution is having a major economic impact, both on the cattle industry as well as the animal health pharmaceutical industry.

Despite this evolution, there are still a number of cattle throughout the country that have never received a dewormer although the numbers of producers that don't treat their cattle for parasites are getting smaller every year. Also, many cattle which are routinely being dewormed are receiving either an inadequate number of treatments or are being treated at the wrong time of the year to be effective.

Some cattlemen and veterinarians still use a dewormer strictly as a therapeutic treatment to remove an existing worm burden based on observing clinical signs of a heavy parasitic infection as exhibited by the animal in terms of rough hair coat, etc. Others use dewormers in a "tactical deworming regime", treating the cattle at various times of the year when worm burdens are known to be the highest. Still others are deworming their cattle according to a preventative "strategic treatment regime" attempting to treat cattle at a strategic time of the year in order to prevent high levels of pasture contamination from developing and thus suppressing parasite development throughout an entire grazing season.

**THERAPEUTIC USE OF A DEWORMER**: Economically, the use of a dewormer simply as a purge dewormer to remove an existing worm burden often produces a poor return on investment depending on the condition of the animal when the dewormer is given. Most of the time when a dewormer is used therapeutically it is given too late and the parasitic outbreak has already cost the producer substantial economic loss. Also, many times, clinically infected cattle, seldom fully recover and unless the animals are removed from the source of infection, the animals will become re-infected immediately.

Spot treatment of certain "poor doing" cattle within a herd is a good example of the therapeutic use of a dewormer, since in this case, the producer is only treating those cattle that appear parasitized or are obviously not producing or growing as desired. Spot treatment has very little long term economic advantage since the cattle remain in the contaminated environment.

These "poor doing" animals are really "marker animals" that tell the producers that parasitism is an economic problem throughout the herd. So not only are non-treated animals contributing to the concept that parasitism is primarily a "production disease", but also that it is a "herd disease"; although some animals may show signs of the disease more than others. Treatment of a few of the worst looking animals does little to solve the overall problem and probably does nothing to prevent a re-occurrence of parasitism in the future.

**TACTICAL USE OF A DEWORMER:** Tactical treatment program involves the administration of several dewormings at various times throughout the year. These treatments are given either at a time just prior to the time when clinical signs usually appear or at a time when worm burdens are know to be high. If the dewormer used is highly efficacious, the program will normally produce an economic benefit to the producer. A tactical treatment program, however, seldom has any effect on pasture contamination and does little to prevent reinfection.

Under southern climatic conditions, a favorite tactical deworming program often recommended to producers is to deworm cattle in mid-summer when pasture conditions have deteriorated due to hot, dry weather. This treatment recommendation has two main problems; first of all, it is usually impractical for most producers to work their cattle in the middle of the summer when weather can be very hot and, secondly, this treatment usually only has temporary success since reinfection on the pastures re-occurs as soon as moisture returns allowing parasite transmission to occur.

**STRATEGIC USE OF A DEWORMER**: Strategic deworming involves the strategic timing of anthelmintic administration during the grazing season with the objective of providing maximum control for minimum treatment cost. This deworming schedule is designed to apply treatments in a preventive way to achieve maximum long term suppression of parasite development on pastures. The main objective of strategic deworming is to allow cattle to graze "parasite safe" pastures and not consume large numbers of infective parasitic larvae every time they consume pasture forage.

These strategic timed dewormings are carefully timed to eliminate parasite egg shedding during a critical, first three months of the grazing season (which is usually the first three months of the spring season). By eliminating parasite contamination early in the year, "parasite safe" pastures are maintained for the rest of the season.

Reducing potential pasture contamination early in a grazing season decreases the chances for development of adult worms, as well as, developing and inhibited larval stages, later in the grazing season. The timing of treatment to achieve pasture control varies according to the category of cattle, such as mature cows, yearling stockers, bred heifers, nursing calves, or weaned calves that might be involved in the designated treatment program. The reason for this is that the life cycles of internal parasites are influenced, among other things, by the age of an animal. Parasite development in adult cows, for example, takes several weeks longer than it does in yearling cattle or young calves.

The type of dewormer and formulation used is also important. Administering a dewormer in the feed, in the mineral, or in a block to cattle on pasture, for example, may be the only way that these animals can be treated if they're not being worked. The use of a dewormer that rapidly removes adult worms can be of benefit to further ensure that worm eggs are not shed for a long period following treatment. Also, dewormers that kill all stages of developing and inhibited parasites have more flexibility in a strategic deworming program than one that only kills mature parasites.

Timing becomes slightly more critical with dewormers which kill late developing larvae and adult parasites. These dewormers will work most efficiently if, for example, they are used to treat cattle several weeks after coming off fall pastures. This allows the parasites, acquired in the last few weeks of grazing, to mature sufficiently to be efficiently removed by the dewormers helping to maintain the animals free as possible from harmful parasites until the following spring when the infection process starts over again.

An important concept for strategic control is that when conditions are right for pasture growth, the conditions are also right for larval development and the clock starts ticking for when treatment needs to be applied. From an epidemiological standpoint, the amount of parasite transmission that takes place in the spring is very important in determining the contamination rate of a particular pasture for the rest of the grazing season. These parasite infections which are naturally available in the spring can come from two major sources;

1. If cattle are old enough to have previous grazing experience, these cattle will harbor a mature (or immature) worm burden unless they were dewormed. They will contaminate spring pastures as soon as conditions are right for egg development. A fall or winter treatment eliminates this contamination source.

2. The second source of infection comes from the overwintered larvae present on the pastures in the spring. Once consumed, three to four weeks pass before the larvae mature into egg-producing adults in young cattle. This is why a four-week interval between treatments is an important part of a strategic deworming program for young cattle. Just as the worms are beginning to mature, they are strategically removed before they can shed eggs.

The parasite cycle continues from year to year because worm eggs and infective larvae are able to survive winter conditions, becoming available to grazing cattle the following spring. All parasites have different abilities for winter survival on pasture. Also, each pasture is different in regards to soil type, herbage cover, condition of fecal pats, and type of prevailing winter conditions which all reflect on how the parasites can survive winters and infect cattle grazing spring pastures. Even on the same pastures, survival rates vary greatly from year to year.

At ten locations across North America when worm-free tracer calves were allowed to graze for the first three weeks of spring, worm development in these calves was found at every location with a mean worm count of 6,801 mixed gastro-intestinal parasites developing in each animal over this three-week period of time. The ten trial locations were located in Arkansas, Florida, Idaho, Indiana, Maine, Mississippi, Oregon, Wisconsin, and two locations in Quebec, Canada<sup>1</sup>.

These data indicated that the over-wintering of parasites occurred at all locations and that sufficient parasite infection developed to maintain parasite exposure to grazing cattle. It should be noted that these worms also have a safety valve in that they can survive the winter inside cattle either as mature worms or as inhibited larvae, acquired from the previous grazing season which mature and deposit worm eggs on spring pastures.

It has been shown that new worm eggs deposited on spring pastures develop into infective larvae at a rate comparable to improving spring weather conditions, reaching maximum contamination levels once warm weather prevails. Additionally, parasite eggs that are deposited on spring pastures have been shown to have the ability to survive an entire summer grazing season, lasting even until the following spring if sufficiently protected or not consumed by grazing cattle. Preventing this egg shedding is one of the primary goals of strategic deworming.

Treatments during the period of first extended grass growth in the spring is the key to interrupting the parasites life cycle and preventing a new generation of parasites from developing on the pasture. Under southern conditions where cattle are often left on pasture year around, these cattle should not be shedding eggs when spring grass growth begins whereas in the north, the cattle should be parasite-free prior to spring turnout.

Because of weather conditions and lack of transmission during winter months in most parts of the country, this first treatment can most efficiently given in late fall or early winter. If given in late fall, the producer is not feeding "worms" in their cattle all winter and at the start of the spring season, these animals should still be "worm-free" if an efficacious dewormer was used. Having animals "worm-free" at the beginning of the season is critical for the program to be successful because it is this treatment that removes any of the worms which may have overwintered within the animals.

With the parasites which overwintered within the animals removed, there is still a potential problem with the ingestion of parasites which have overwintered on the pastures. Once the pastures are contaminated, little can be done to remove these larvae off the pastures except through grazing. So, by letting the cattle work like vacuum cleaners picking up these larvae, the producers' task now is to remove these newly acquired worm burdens before they mature and begin laying eggs back on the pasture. The timing of the dewormer to kill developing phases of gastro-intestinal parasites is critical and must be given prior to the time that the newly consumed larvae reach adulthood.

The reason that the spring treatments can be successful is due to a special characteristic of the epidemiology of gastro-intestinal parasites. The larvae which have survived the winter from the previous grazing season move onto the vegetation and become available to grazing cattle. If these larvae are not consumed by cattle during the early part of the grazing season, they soon die. Developing L1 and L2 larval external stages of the parasites normally feed on bacteria and other organic debris present in the fecal pat. Once these larvae molt to the infective stage (L3) they can no longer feed because of a protective sheath which covers their mouth parts. These infective larvae, therefore, depend upon an internal food source. Overwintered larvae have already depleted some of their internal food source, and thus have limited food supply for the spring. Once favorable weather returns, the larvae become active and deplete their internal food supplies and die if not consumed.

This phenomenon has special application under a strategic deworming program since this natural die-off of parasitic larvae in the spring rids the pasture of parasite contamination by early summer if no new worm egg excretion and subsequent larval contamination of pastures is allowed. In several European countries including the Netherlands, producers take advantage of this phenomenon by keeping their young calves in stables until after they have taken a cut of hay or haylage from the pastures and then turn the calves out onto the after growth in early July. These pastures are now "parasite safe" pastures. Although resting pastures in the spring is impractical in North America, this die-off phenomena of parasitic larvae, forms the basis of the repeated spring treatments that constitutes "strategic control."

The reason for the repeated treatment in the spring is to duplicate the situation that occurs with the resting of pastures during the first three months in the spring. Since nearly all North American cattlemen need to use their spring pastures for grazing, and since these pastures are contaminated with overwintered larvae a treatment regime is necessary. This treatment interval must be carefully timed based on category of cattle to be treated and time of first extended grass growth in the spring (or time of turn-out) in order not to let worms mature and begin to shed eggs and re-contaminate the pasture.

The treatment interval is based on the average time it takes for ingested infected larvae to develop to maturity and begin laying eggs. Unfortunately, this time interval varies with cattle of different ages. With young calves and yearlings this time interval is approximately three to four weeks, hence the four week interval between treatments is an important part of strategic deworming. To achieve three months of control in the spring, these young or yearling cattle should be parasite-free at the start of the spring, and treated four weeks into the spring followed by a second deworming four weeks later. The three months or 12 weeks of control is achieved because there is a four week interval after the second deworming before egg shedding will occur.

In mature cattle (cow/calf operations), a treatment six weeks into the spring grazing season is strategic since the prepatent period for worm infections in adult cows take longer than in young cattle and approximately five to six weeks are required before worm eggs

appear in the feces. With cow/calf operations, the cows should be parasite -free prior to the beginning of the new grazing season in the spring. The "6-week" treatment should occur in mid-spring at which time any of the suckling calves that are 200 lb or larger should also be treated. It is very important that all of the calves over 200 lb be treated since they can be a very significant source of continued pasture contamination. The three months control is achieved because egg shedding will not occur until a six-week period following treatment making a total of 12-weeks or three months of no egg shedding.

The time needed to rid the pastures of overwintered larvae varies somewhat from area to area, but it appears that pastures are mostly free of overwintered larvae by the third month into the grazing season. The larvae seem to survive slightly longer under southern conditions than northern conditions for some unknown reason, however, four and eight weeks after the beginning of spring grazing in young stock and six weeks after the beginning of spring grazing in Cow/calf treatment programs have been demonstrated to work well throughout the country.

**THE ECONOMICS OF STRATEGIC DEWORMING PROGRAMS**: The economic advantage of strategically deworming stocker cattle and replacement heifers according to a treatment four and eight weeks into the grazing season is well established. Over a single grazing season, weight gains of between 20 and 100 lbs. per animal have been recorded. Average season improvement in weight gains of between 40 and 60 lbs. can routinely be expected by producers using a strategic deworming program on their youngstock<sup>2</sup>.

Studies conducted throughout the USA in cow/calf operations from Nevada to Florida have demonstrated that cows strategically dewormed in late fall and again six weeks into the spring grazing season have provided an extra 20-60 lbs. more weight at weaning. Furthermore, these trials have demonstrated that reproduction efficiency of the cows can be significantly improved by 5% to 22%.<sup>3</sup>

The economics of deworming strategically depends heavily upon the management of the cattle being treated. The better managed cattle tend to respond to treatment better than poorly managed cattle. It appears that the greater the performance that is demanded of animals, the more important it is to maintain these animals free of parasites.

**EDUCATING PRODUCERS ON STRATEGIC CONTROL**: Since the information on strategic deworming is relatively new to cattle producers and veterinarians alike, a lot of confusion still exists as to the details involved in strategic deworming. Also, many universities and veterinarians have yet to properly study and/or to endorse the use of strategically timed dewormings to control parasitism. In certain parts of the country, cattlemen are still receiving mixed signals on whether to and when to treat their cattle. Also, many animal scientists erroneously still believe that cattle should only be treated after they've been clinically (severely) affected or that mature cattle should never be treated because they are thought to be immune to parasitism.

Cattle management systems in the U.S.A. are extremely complex and, therefore, a deworming regime needs to be tailored to each operation based on category of cattle

raised, type of pasture and pasture management used, and geographical location of the operation. Questions need to be answered on nearly every operation as to which animals should be treated, when is the best time to treat, which dewormer is the best product to use, and what is the return on investment for the use of a deworming program.

Strategic deworming has been well tested throughout the world, however, its use in the field with commercial cattlemen is only beginning to find success. There is no easy method for the transfer of scientific information of this nature to the producer and, therefore, a significant joint effort is needed by animal scientists, veterinarians and the animal health industry itself to help develop and transfer this information on strategic deworming to the cattle producer.

# PART II DIAGNOSISTIC PROCEDURES:

The acceptance of the economic importance of cattle parasitisms by producers and their veterinarians in most parts of the U.S. has brought about a significant change in their attitude toward the control of parasitic infections. Part of this change has come about because of the benefits derived through the use of effective dewormers in strategic parasite control programs.

An example of increased awareness in cattle parasitism over the past 30 years can be seen by the change in estimated losses in agriculture due to parasitism as reported by the USDA during this period. In 1956, the USDA reported that internal parasites were causing an annual loss in all livestock of nearly \$40 million. By 1965, the USDA estimated this loss to be \$162 million while in 1972 the USDA estimated this loss to be \$400 million annually in cattle alone. This reported increase in estimated economic loss is not due to a sudden increase in parasitism, but rather, to an increased awareness of parasitism. This awareness has come about through field research on the use of new dewormers in efficient control programs that have demonstrated the potential efficiency of production once parasitism is under control.

Despite this increased interest in parasite control, considerable confusion exists on the incidence, prevalence, and economic importance of parasitism in grazing cattle in different parts of the country. Also, there appears to be a general misconception on the meaning of fecal worm egg counts and a lack of knowledge on the proper timing of anthelmintic treatment to achieve maximum economic benefit for a producer with minimum drug and labor expenditures.

First of all, current diagnostic techniques to determine economic levels of parasitism in cattle under field conditions are mostly inaccurate. This problem is due to the fact that the commonly used fecal examination techniques generally lack the necessary sensitivity to detect "normal" levels of parasitisms. Many of the fecal worm egg count techniques employed by veterinarians, diagnostic laboratories and research groups were originally

developed for use in sheep which have a low fecal mass and subsequently high worm egg counts compared to those found in cattle.

Some of the fecal tests routinely used today, for instance, require levels of at least 50 worm eggs per gram (epg) of feces before detection. On a pound of feces basis, this is equivalent to 20,000 worm eggs/lb. Since an adult cow can easily produce between 20 and 40 lb of manure daily, an animal with an egg count of 50 epg could be excreting more than a half million eggs per day before the parasitism could be routinely detected.

Researchers at the University of Florida demonstrated that controlling a parasitic infection in dairy heifers with a seasonal average of only 54 eggs/3g resulted in 23 lb more weight per calf at the end of the grazing season. Similarly, other studies conducted in Maine demonstrated average weight gain improvements of 30 lb and 17 lb in cattle with average worm egg counts of 66 eggs/3g and 13 eggs/3g respectively over a summer grazing season. High worm egg counts, therefore, are not a perquisite in determining economic levels of parasitism in grazing cattle.

The widespread prevalence of internal parasitisms has been apparent over the past two decades based on fecal worm egg counts surveys conducted all across North America. In full acceptance of the many uses, limitations and interpretations of egg counts, these nevertheless have led us to conclude that, when eggs are found, live worms are present within the cattle, that is, that active and important parasitisms are present.

Currently, the Modified Wisconsin Sugar Flotation Technique is reportedly the most sensitive fecal worm egg count technique for adult cattle. Comparing the Wisconsin Sugar Centrifugal Method, McMaster's and the Sodium Nitrate Flotation methods, researchers at the University of Wisconsin found of 275 dairy cows examined, 90% were positive for eggs by the sugar method, while only 18% were positive using sodium nitrate and 10% when using the McMaster's technique. It was these authors' conclusion that the widespread uses of the McMaster, the Sodium Nitrate flotation, and other similar flotation techniques have effectively prevented the recognition of economic parasitism in mature cattle for many years.

The meaning of fecal worm egg counts has also long been misunderstood. Many authors have unsuccessfully attempted to link egg counts with worm burdens. Since the first description of the epidemiology of parasitic gastro-enteritis in grazing cattle, fecal worm egg counts have taken on a new meaning. Producing and excreting eggs in the manure is the only way most cattle parasites have to maintain survival. It is this contamination of the animals environment, most notable that of the pastures, that the parasites must depend upon for continual recycling and survival.

Low, medium, or high counts are arbitrary levels, however, low counts are usually described as counts less than 10 eggs/3 gram, medium as 10 to 50 eggs/3 gram, while high counts are usually described as 50+ eggs/3 gram fecal sample. The type of parasite present will influence the number of eggs shed and the importance of a low to high count. A low count of *Nematodirus*, for example, may indicate that economic loss may be occurring

while a low tapeworm egg count, on the other hand, may indicate the presence of a single worm with little economic loss. The time of the year that fecal samples are taken for analysis is also a very important consideration in the interpretation of the egg counts.

1). Fecal worm egg counts taken in the winter in most parts of the country indicate whether or not worm burdens were controlled properly the previous fall. The winter feeding of cattle harboring parasites can be costly especially where winter calving is prevalent.

2). Fecal worm egg counts taken in the spring of the year indicate the level of worm eggs being shed on the pasture at the beginning of the summer grazing season. If parasite eggs are present, along with favorable environmental conditions, the number of eggs shed will determine the rate of reinfection that will exist on the pastures during the rest of the year. The lower the worm egg count is in the spring, the lower the level that parasite pasture contamination will be during the following summer and fall season.

3). Interpretation of fecal worm egg counts taken during the summer can be difficult because of the summer infection process. Low counts can be misleading because they do not indicate whether or not immature (or inhibited) stages are present. High counts can also be misleading if pasture conditions have deteriorated causing low fecal output which in turn can cause high worm egg counts. Very low and even negative counts can be helpful, on the other hand, because they often indicate that successful treatment has occurred.

4). Sampling in late fall usually produces better results. Egg counts taken in late fall indicate whether an accumulation of adult parasites has developed during the grazing process. Low counts usually indicate a low level of transmission has occurred while high counts usually indicate a heavy infection level has occurred, especially in young cattle with high counts.

**Treatment as to market**: Deworming cattle in late fall or early winter has two strategic advantages. The first advantage is that cattle enter the winter free of parasites and, therefore, are in better shape to handle the stresses of winter. The second advantage is that cattle which are treated in late fall (after the end of the grazing season or after a frost) or during the winter time do not become re-infected until the beginning of the new season especially if these cattle are treated with an anthelmintic efficacious against immature worms. It is important that animals with previous grazing experience are dewormed prior to the beginning of the spring grazing season so that they do not add to the contamination of the pastures at the start of this new grazing season.

In the South, cattle that received fall anthelmintic treatment in September or October prior to the end of the grazing season can become re-infected before winter and, therefore, the winter to spring contamination cycle continues despite the treatment. Fall treatment in the South should probably occur after the first part of November but before grass growth begins in the spring for best strategic, long term control. In the North, fall treatment is determined by when the cattle are brought in for the winter or after a hard frost has occurred if the cattle remain outside.

Worm egg counts taken in April, May and June have demonstrated that if cows are treated in late fall or early winter, they have negative or very low counts during the first two months of the new grazing season, following which these counts began to rise indicating reinfection from overwintered parasites on the pasture. A second treatment of the cow and her calf sometime in May to early June has been designed to prevent a secondary pasture recontamination pattern and provide parasite 'safe' grazing for an entire summer grazing season.

This information, along with other information available on prepatent periods of different worms in different age category of animals has helped to determine the most strategic times of the year for treatment. The fact that worms are slower to develop to maturity in adult cattle than in younger cattle has a profound effect are the way these treatment programs have been developed. A three weekly treatment program for stocker cattle may be too costly and to intense for adult cattle.

# PART III

## EXTERNAL PARASITES AND PESTS OF CATTLE

Under natural field conditions it is difficult to understand internal parasites and their control without some awareness of external parasites and their importance in cattle production cycles. Controlling parasitic gastro- enteritis in a animal covered with lice, for example, may not elicit the desired response without also controlling the lice infestation. Some of the important external pests and ectoparasites of cattle are as follows:

**Horn flies**: The <u>horn fly</u> is one of the most irritating fly for cattle. This fly feeds on the back, belly, neck, and at the base of the horns of cattle. Thousands of these flies may be found on a single animal. The fly remains on the animal and leaves it only to pass to another animal or to lay eggs when the animals defecates. Since each fly feeds several times daily by sucking blood, the afflicted animal experiences much annoyance and disturbance along with loss in vitality and poor performance. Horn fly control has been shown to result in significant increases in milk and beef production.

**Stable flies:** are most troublesome around barns and feedlots where it breeds in wet straw, manure, spoiled feed and other kinds of debris. The material must be moist otherwise it is unsuitable. Hatched larvae grow to maturity in 14 to 24 days with a life cycle of about 30 days. The flies are most abundant in summer and autumn and live about one month under natural conditions. Both males and females are blood suckers.

**House flies:** are found nearly everywhere and are important as a mechanical carrier of various infectious agents including virus, bacteria, and protozoa. The life cycle of this fly

is approximately eight days. The eggs, larvae, and pupae can resist a fair degree of cold when they lie protected and are responsible for the new crop of flies in spring.

The <u>face fly</u> is slightly larger than house flies. The eggs of the face fly are deposited in fresh manure where they develop with a life cycle similar to the house fly. They gather around the eyes and nostrils, feed on ocular and nasal discharges and cause annoyance and irritation. It is also the intermediate host of the eyeworm and transmits infectious bovine keratitis (pinkeye). Economic losses caused by this fly due the interruption of both growth and milk production can very high.

**WARBLES/CATTLE GRUBS**: The ox warble or grub is an important ectoparasites of cattle in nearly all parts of North America. It has long been present in every state although in many localities it is of little economic importance. The degree of infestation appears to vary from year to year even in highly infested areas. The highest infestations seem to be found in the Southwest, but some of the most severe outbreaks have been recorded in the Midwest originating principally in feeder cattle. Due to extensive treatment of cattle for grub control over the past 10 to 15 years, the prevalence and economic impact of this parasite has greatly declined in recent years.

<u>Life cycle</u>: The adult fly is commonly known as the heel fly and usually first appears in late spring. It deposits a cluster of eggs on the skin of cattle, fastening them to the hairs about the feet and legs, especially just above the heel. Although the flies neither bite nor sting, cattle show a natural dread of them and often run frantically from one end of the pasture to the other holding their tails high in the air, attempting to escape. Yearling cattle are more likely to be attacked than calves and calves more than adults. Since the heel fly does not fly over water, cattle often protect themselves by retreating into ponds and streams.

The larvae, which hatch in four to six days, immediately penetrate the skin at the base of the hairs to which they were attached. The larvae then proceed, by no well-defined route to the region of the neck, where they gather about the esophagus. During early winter in the Midwest and as early as October in the Southwest, the larvae leave the neck region and migrate to the back where they form the characteristic swellings beneath the skin. Here a hole is made through the hide, toward which the larva directs its posterior end. In this position the larva increases rapidly in size and molts at least two times. The pus and mucus caused by its presence constitutes the food material from which this growth is affected. Finally, with the arrival of spring, the grub now fully grown works its way through the hole in the skin where it falls to the ground and it pupates (previous to its transformation) into a mature fly when it begins the cycle over again.

**LICE**: Cattle are subject to three species of lice: long- and short-nosed blue sucking lice and the red biting louse. Inasmuch as the biting lice feed principally on the particles of hair and dead skin, they are not especially troublesome. The sucking lice, however, cause much annoyance and if numerous, sap large quantities of blood. Lice infested cattle spend much time rubbing themselves against posts, trees and so forth, in an effort to allay the

intense itching. Frequently, large patches of hair are worn off and the hide becomes hard and calloused. Lice are most numerous during the late winter and early spring. Treating cattle for lice is often difficult because of the cold weather and winter hair coat. Once treated, cattle should be retreated 15 to 16 days later in order to kill any lice which may have hatched since the first treatment.

**MANGE/SCABIES**: Mange of cattle is caused by small mites who attack the skin causing it to become thickened, covered with crusts, and devoid of hair. As a rule, the rump is the part of the body most likely to be affected, although the back and shoulders may also be included. Owing to the looseness of the scabs and the hardiness of the mites, scabies is quickly and easily spread from one animal to another by direct contact and by rubbing against fences, trees and the like. Scabies is a reportable disease and is currently under an eradication program. Cattle coming from scabies infected areas must be dipped or treated with Ivomec® before movement.

**TICKS**: At one time the "North American Tick" was widely distributed throughout the southern USA. A campaign started in 1906 resulted in its eradication. It is, however, introduced periodically into the USA by illegal movement of cattle from Mexico, where this tick is still prevalent. At one time, this tick caused very serious economic loss to the cattle industry through the transmission of Texas Tick Fever (*Babesia*). It is a one host tick and can occur on domestic and wild ungulates.

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