Introduction to Large Animal Parasitology Procedures for Veterinary Technicians

CHAPTER 1

What is Parasitism? Parasitism is defined as the condition of life that is both normal and necessary such that one organism can live on or within another host organism and that it nourishes itself at the expense of this host organism. The host organism is always a different species than the parasite and is almost always much larger than the parasitizing species. Since it is not in the best interest for a parasite to rapidly destroy its host as a predator does its prey, the parasite simply inflicts some degree of injury, negatively affecting the welfare of its host while gaining the benefit of life. The characteristic of this parasite–host ecosystem is that parasites contribute nothing beneficial to this system, but rather always cause harm to the host, which results from the activities of the parasites in their effort to survive and reproduce. Parasites often attach themselves to the gastric wall and absorb blood and nutrients (see Figure 1.1).

The survival of the parasite depends upon its ability to withstand the efforts of the host to destroy it. How harmful this relationship is dependent upon the species of parasites involved, the location of the parasite in the host, the number of parasites present, the virulence of the parasites,

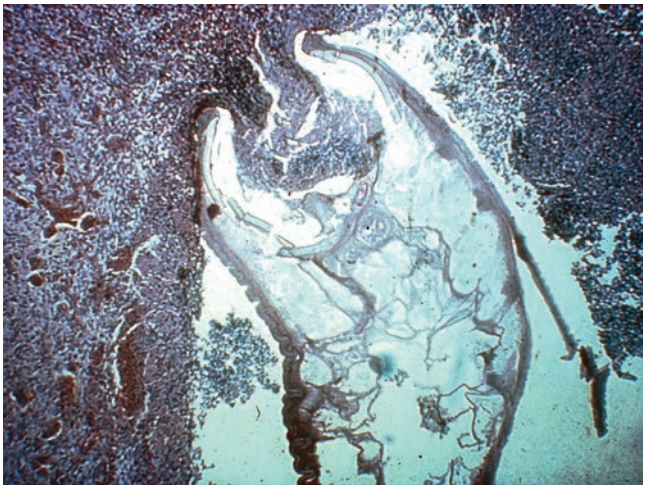

FIGURE 1.1 Strongyle nematode parasite attached to the gut wall of a horse [1].

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the age of the host, the genetic susceptibility of the hosts to the parasites, and the previous experiences of the host with these invading parasites. Seasonality and the stage of lactation or gestation for the host animal can also influence the amount of damage caused by parasites.

Parasites have learned to adapt to the conditions of their host. Cattle in southwest Texas grazing arid range country may have less parasite exposure than cattle grazing lush pastures in Wisconsin. Twenty-five goats raised on 75 acres of land will have significantly less "parasite pressure" or parasite exposure than the same 25 goats grazing 1 acre of land. When cattle are raised under semiarid conditions, parasite pressure can be low; however, damage to the animal may be high if nutrition is inadequate (see Figure 1.2).

A few years back, I conducted a "parasite evaluation clinic" with a veterinary clinic near the town of Houston in northern British Columbia, 674 mi north of Vancouver (see Figures 1.3 and 1.4). The very first set of cattle fecal samples we checked during the clinic were loaded with *Nematodirus* eggs (see Figure 1.5). The owner said they had lost three calves over the past few days. Clinical parasitism, therefore, can occur almost anywhere animals are raised. Parasitism and production losses due to parasitism are not based solely on what part of the country the animals are located in, but rather they are based more on grazing pressure or stocking rates, climatic conditions during the season, whether or not water irrigation is used, whether there are any ponds or water source on the pasture, what the parasite contamination levels were at the beginning of the season, whether or not the animals placed on a pasture were currently carrying a heavy worm burden, where any deworming treatments used during the season, if used when were the deworming treatments administered, and what deworming products were used.

By definition, parasites always cause harm to their host while obtaining their livelihood. Furthermore, this host–parasite relationship exists because of a complex relationship whereby the parasites can contaminate the environment of their host with offspring (either eggs or larvae) to continue

FIGURE 1.2 Cattle raised under semiarid conditions with low parasite pressure.
Source: United States Department of Agriculture/Wikimedia Commons/Public Domain.

FIGURE 1.3 Location of the "parasite evaluation clinic" near Houston, British Columbia.

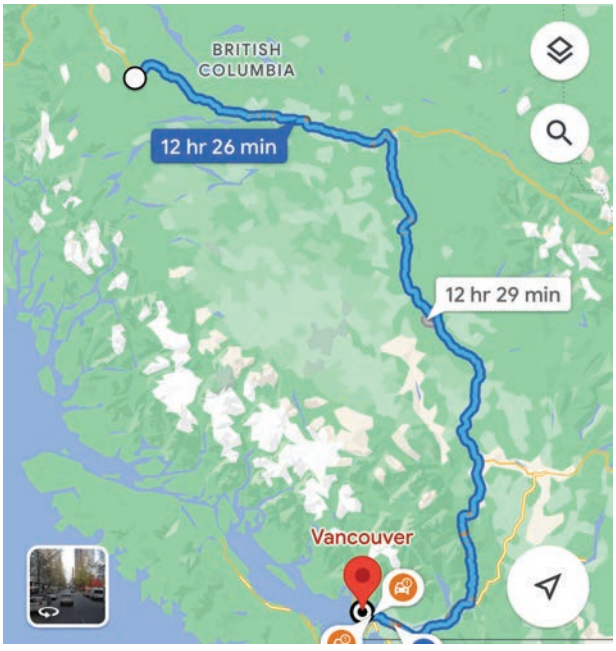

FIGURE 1.4 Distance north of Canadian border where clinical parasitism was detected (647 mi north of Vancouver).

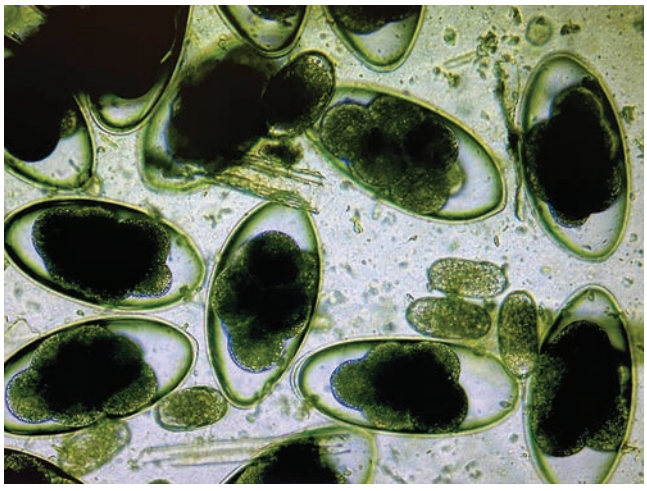

FIGURE 1.5 Microscopic picture of *Nematodirus* eggs from clinically infected calves examined in Northern British Columbia.

their relationship and to maintain their species. Whenever a parasite cannot complete their complete life cycle, they die off and disappear. A great example would be when an American traveler becomes infected through food contamination with a gastrointestinal parasite(s) when traveling abroad (especially in a country with poor hygienic conditions) but when they return home, the parasite cannot propagate because sanitary conditions in the United States are not conducive for reinfection to take place. Once this parasite is removed from the traveler (or it dies off), recontamination will not occur. Another example with domestic animals could be found when replacement dairy heifers are moved off pasture into a totally confined dairy facility where they calve and produce milk, but are now confined on concrete floors with no access to dirt lots or grass pasture areas (see Figure 1.6).

FIGURE 1.6 Young dairy cattle raised in confinement with no access to pasture.

Since these heifers are now no longer exposed to parasitic larval contamination found on pastures, their existing infections will eventually die off and since they are not replaced, they become parasite-free.

The universal existence of internal parasitism in domestic and wild animals requires the occurrence of encounters whereby the parasites can successfully establish host–parasite systems. The host–parasite relationship involves the accessibility of the host to the arrival and continued presence of the parasites. This parasitic relationship is called parasitism and the study of this relationship in domestic livestock and wild animals which are of interest to animal health is called "Veterinary Parasitology."

Veterinary parasitologists are exposed to many life-cycle variations of parasitism in animals. Cattle tapeworm (*Moniezia*) eggs, for example, simply pass through an intermediate host (Oribatid mites) while developing into an infective-stage embryophore, whereas cattle liver flukes undergo a complete development process while passing through an immediate snail host (*Lymnaea* snail), then back on vegetation to complete the life cycle once eaten by cattle. With liver flukes, they can also infect an alternate host (deer). These life-cycle variations are covered in detail in many books and research journals on veterinary parasitology.

Basically, with minor exceptions, most of the parasites of economic significance in domestic livestock and hoofed wildlife follow a simple direct life cycle. The cycle begins with the adult female and male parasites residing in the animals where they mate, following which the female worm then lays fertile eggs which pass out of the animal with the feces. Since these infections are unseen, this is the only point in the life cycle when a worm egg test can be conducted to detect the presence or absence of a mature infection. The standard, most efficacious, and simplest fecal worm egg count test I recommend is "The Modified Wisconsin Sugar Flotation Method" which will be covered in detail in the following chapter [2].

The typical life cycle of gastrointestinal parasites starts when each passed egg develops into a first-stage larva that hatches in the manure, provided that conducive (warm and moist) conditions persist. This first-stage larva then develops into a second-stage larva and then finally into an infective third stage, also called a L_3 larva. The infective L_3 larva is mobile and randomly moves away from the fecal matter onto vegetation. These infective larvae are subsequently ingested by the animal grazing this area and become re-established within the host where this life cycle continues (see Figure 1.7). Minor variations occur with nearly every parasite.

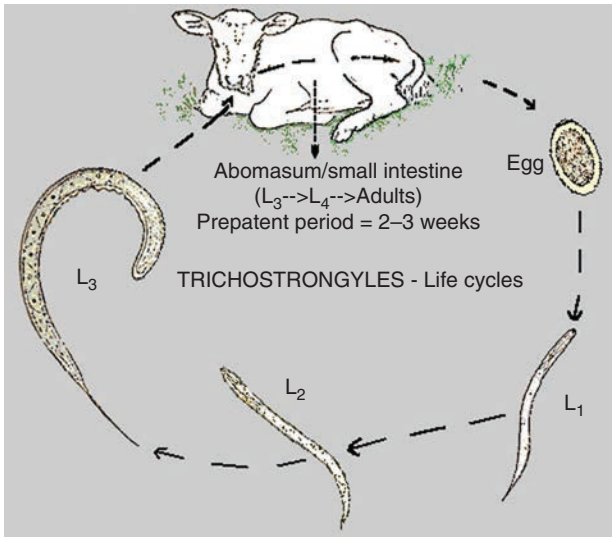

FIGURE 1.7 Typical life cycle of gastrointestinal parasites in grazing animals.

It is an essential feature of parasitic nematodes (roundworms), cestodes (tapeworms), and trematodes (flukes) that they can reproduce within a host, but then each new generation must undergo some development in the outside world before re-entering a host and adopting a parasitic way of life. The reproductive forms of these parasitisms are mostly ova (eggs), larvae, cysts, oocysts, or trophozoites, which pass out in the feces and redistributed into the environment of the host animal. This recontamination of the environment surrounding the host animals is paramount to the overall survival of the parasite because it ensures that future infections and/or re-infections occur, so the propagation of parasitic life continues. Internal parasites, therefore, have two basic goals in life: one is to live off their host animals that they invade, and the other is to reproduce back into the environment of host animal to ensure continuation of their species.

To the average person, the thought of having parasitic worms living in their own stomach, intestinal tract, or lungs is almost too much to bear. Yet, nearly all animals are exposed to parasitism sometime in their life, especially those parasitisms that invade and live in the gastrointestinal tract. Many animals become heavily infected on an annual basis depending upon environmental contamination levels and whether any means are taken to reduce their exposure level. It is not uncommon for horses, sheep, or goats to have fecal worm egg counts well over 1000 eggs/sample during the summer grazing season. This high level of worm egg shedding from a 3 g fecal sample can mean that a horse, for example, would be shedding 150,000 eggs per pound ($3\,g \times 150 = 454\,g$ or 1.0 lb, so multiply 150×1000 eggs) of manure or as many as 9 million eggs per horse per day should this animal be producing approximately 60 pounds of manure per day. As one can imagine, in just a short time, the pasture would become heavily contaminated by just one horse such that an animal grazing this pasture later in the year could be ingesting hundreds and even thousands of infective larvae every day. These larvae follow moisture trails moving up on the vegetation with the morning dew or with moisture from rainfall (see Figure 1.8).

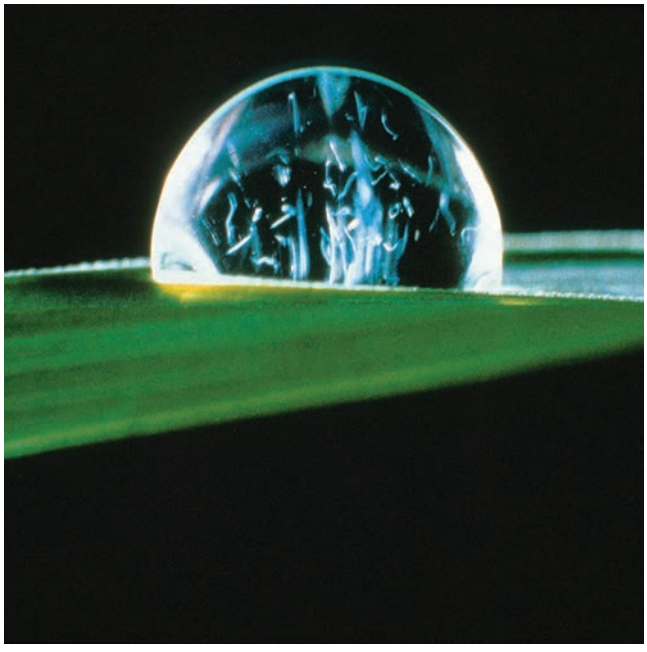

FIGURE 1.8 Famous picture of a dew drop with infective larvae on a blade of grass.

I believe there is a role humans must play to help prevent animals from suffering due to parasitic infections. I often see animals standing in fields all around the country looking heavily parasitized. I feel like it is our role as humans to help spread the word to owners and caretakers that these animals should not have to suffer. "Ivory tower" parasitologists often tell me, we cannot do anything because it will cause parasite resistance! My argument is that the correct treatment given at the correct time will help clean up the environmental contamination and thereby prevent parasite resistance. Allowing parasitism to reach very high levels makes the task of the dewormer much hard and provides a greater chance for leaving exposed parasites without killing 100% of their parasitic burdens. A dewormer with a 98% efficacy will leave 20 worms out of 1000 or 20,000 worms out of a million. So for a dewormer, it is also better treat when parasite burdens are low and prevent clinical parasitism, then wait until worm burdens have become extremely high. Remember, once the worm burdens are high in the animals, they are also high in the environment; so, treating heavily infected animals and then returning them to their heavily contaminated environment is a foolish waste of time and effort plus it greatly increases the chance for parasite resistance to occur. The correct deworming strategy, therefore, is to not only treat the animals but also to time the deworming to clean up the pasture environment, reducing the overall exposure to parasitism. This concept will be explained in detail later in this chapter.

Parasitism can be broken into two main stages or clinical conditions: The first stage is called "subclinical parasitism" while the second stage is called "clinical parasitism." Subclinical parasitism is often compared to an iceberg where the major portion of the iceberg is unseen being below the surface of the water (see Figure 1.9).

Therefore, with subclinical parasitism, the damage caused may not be readily visible to the owner or producer. Parasitism is most often first visibly evident in livestock through observing rough hair coats or animals showing "pasty rear ends" (see Figure 1.10). The largest part of the iceberg lies unnoticed under the sea and thus the greatest damage from subclinical parasite infections often occurs without being noticed by the owners, producers, or their veterinarians such as a drop in dry matter intake, reduced feed efficiency, lower weight gains, reduced milk production, reduced breeding efficiency, reduced body condition scores, and reduced immune function [3–8]. Subclinical infections may lead to "hard to observe" changes in behavior such as animals showing

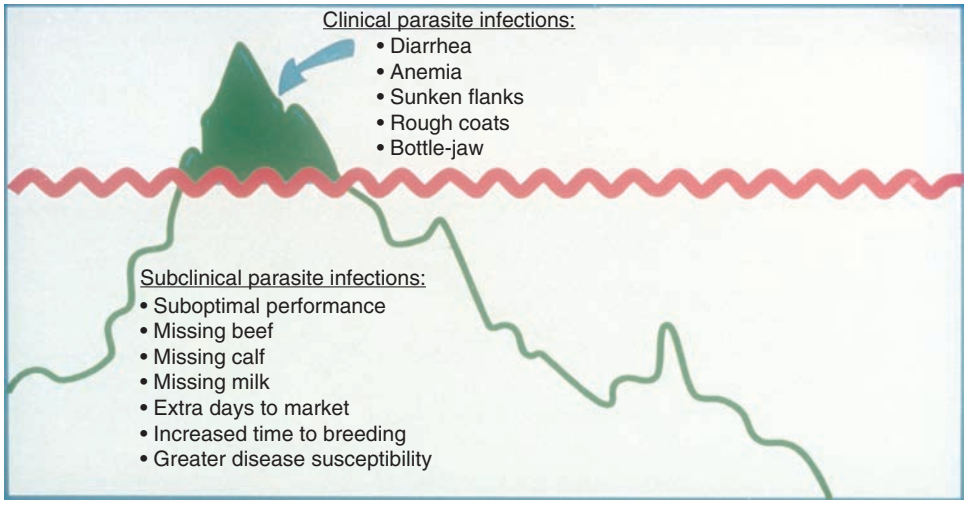

FIGURE 1.9 Subclinical parasitism is like an iceberg where the portion under the sea goes unnoticed.

FIGURE 1.10 Three Holstein dairy calves with clinical signs of *Nematodirus* infection.

reduced grazing time with longer periods of standing or lying around. Based on years of research conducted throughout the world, there is no question that gastrointestinal parasitisms even at subclinical levels are a major deterrent to efficient production. Parasitism commonly found in livestock follows an age pattern depending upon housing location and access to pasture (see Figure 1.11).

Clinical parasitism is simply the stage where physical effects of the infection are visible evidence in the physical condition of the animals infected. Sometimes the evidence is as simple as checking mucous membranes such as eyelids for sign of anemia (a pale membrane caused by the loss of red bloods cells), which is a common practice used with sheep and goat owners to detect heavy *Haemonchus* (barber's pole worm) infections. Even baby calves can become clinically infected with threadworms (Strongyloides) where larvae can pass in the mothers' milk as well as a direct infection acquired from the bedding (see Figure 1.12).

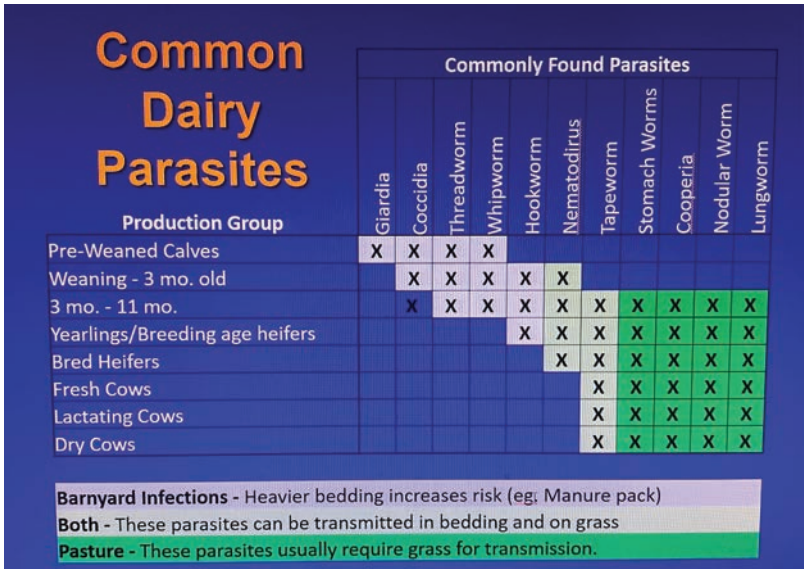

FIGURE 1.11 Parasites commonly found by various age groups.

FIGURE 1.12 Picture of baby calves showing clinical parasitism due to a heavy threadworm (*Strongyloides*) infection.

Since gastrointestinal parasites live unseen within an animal, they can go unnoticed until at which time they can build up to high and often dangerous levels before visible signs become evident. It has been stated that clinical parasitism occurs when insufficiently resistant animals are exposed to an excessive rate of infection. This situation may arise when susceptible animals graze a heavily contaminated pasture (see Figure 1.13). Once it has been decided that treatment is necessary, we find little success in treating heavily infected animals and then returning them back to the

FIGURE 1.13 Idaho yearling bulls with *Nematodirus* infections showing rough hair coats.

same contaminated environment. The contaminated pasture this animal or animals are grazing should be vacated at this time, with a better deworming strategy to be developed for the following year. Ignoring the presence of parasites means animals are unnecessarily being exposed to pain and suffering plus resulting in production losses for the owner.

Even when parasite infections reach a level when clinical signs are present, positive confirmation is best completed using a fecal exam. Furthermore, if one waits until clinical signs are present before treating, considerable damage may already have occurred in the animals. Although sometimes it is possible to "feed past a parasitism" with considerable feed loss, but remember "the parasites" always get their "bite first." There is a definitive clinical process of an infection in cattle as follows:

1. The first signs of parasitism are usually the development of a rough and scruffy hair coat. Some young animals will also develop a "pot belly" along with the rough hair coat (see Figure 1.14).
2. Lack of weight gain or loss of weight during the grazing season, especially in younger animals.
3. Reduced reproduction efficiency in animals of breeding age.
4. Reduced milk production in lactating animals.
5. Reduced digestion and poor fetal development.
6. Reduced immune function. Coccidia outbreaks become common here.
7. Body and survival maintenance affected, which will soon lead to death if not treated.

As worm burdens build and clinical parasitism develops, immediate treatment may be needed to save the life of the animal. Being able to detect, treat, and/or prevent parasitic problems before clinical signs appear should be the goal of every veterinary hospital or clinic in the world. This is exactly where a quick and accurate fecal exam can play a very important role in preventing production losses or in alleviating unnecessary pain for the animals in question. A fecal worm egg counts using the Wisconsin Sugar Flotation Method is the best way to determine the presence of subclinical parasitism (see Figure 1.15) (this method will be described in detail in Chapter 2).

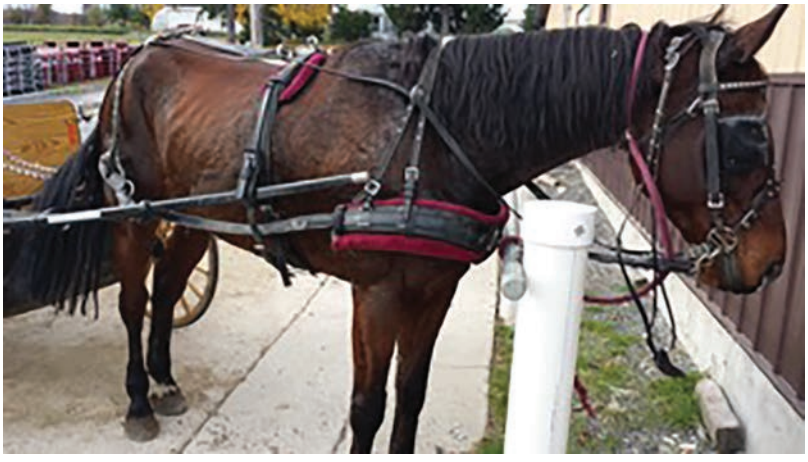

FIGURE 1.14 Working horse showing signs of clinical parasitism with an extremely high fecal worm egg count.

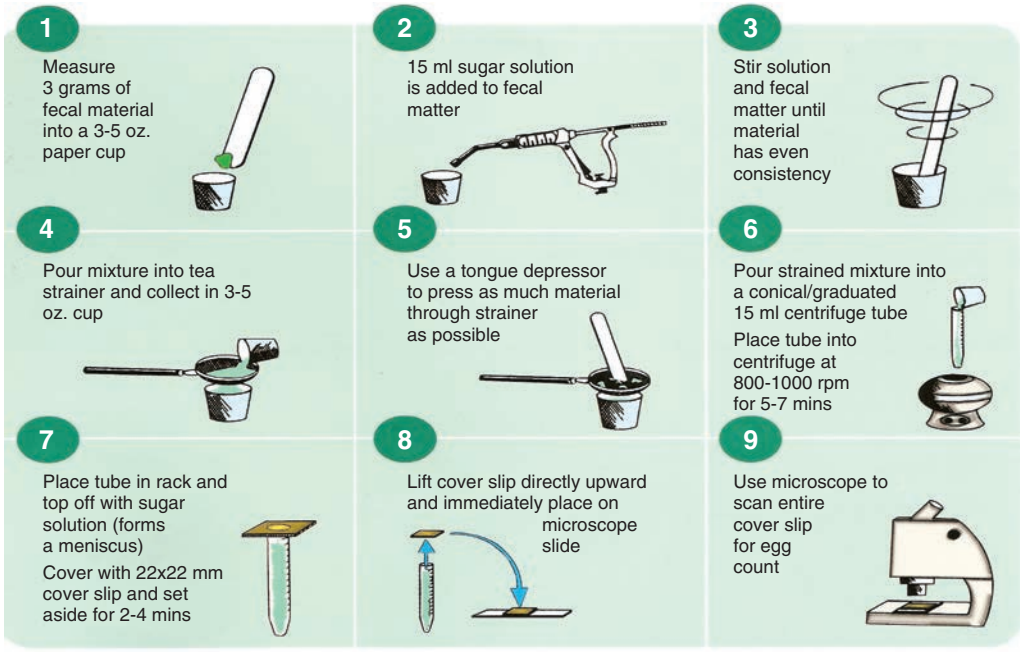

FIGURE 1.15 Modified Wisconsin Sugar Flotation Method.

The main question of this discussion is to ask veterinarians and their staff throughout the world why they do not use fecal exams as a routine method to monitor parasitism in livestock and what can we do to help change this. Treating without knowing whether an animal really needs treatment, treating animals at the wrong time of the year, and not knowing whether the treatment given actually works are all reasons the practicing veterinarian needs to be able to provide accurate fecal worm egg counts to all their producers. These fecal worm egg counts should be done in order to monitor fecal worm egg output with all livestock on a routine basis. We also need to convince owners on the value of fecal exams to monitor their animals.

Paying a Veterinary Service for a fecal worm egg counts on animals to know when to treat could save producers thousands of dollars for unnecessary treatment, incorrect treatment, using the wrong products, and not knowing whether the products they used were effective. Furthermore, there is now a number of FDA-approved deworming products on the US market that have become "parasite resistance." Being able to monitor these products is extremely important. Remember, products that fail to control parasites may be as bad for the animals as a failure to treat altogether causing unnecessary suffering in the animals and production losses for the owners (see Figure 1.16).

Fecal exams using the correct method can provide the science that is necessary to ensure treatment is given correctly, is given at the right time, and is effective. At the present time, very little is being done on a routine basis to monitor deworming treatments in domestic livestock. Thousands, even millions, of treatments are given annually to cattle, sheep, goats, horses, swine, and poultry, but very little is done to actually monitor infection levels or even determine whether or not treatments given are working correctly and providing the protection they were approved to do by the FDA regulatory authorities.

Over the past 38 years, I have dedicated considerable amount of my consulting time for conducting parasite evaluation clinics using the Modified Wisconsin Sugar Flotation Technique all

Trial Summary for Fecal Egg Count Reduction Tests Reported on the Merck National Data Base Conducted with FDA Approved Macrocyclic Lactone Products.

			Egg Counts/3 g*		
Products	**No. of Trials**	**No. of Samples**	**Pre-Rx**	**Post-Rx**	**Percent Efficacy (%)**
Injections					
Ivomec® Inj.	25	1352	70.1	37.1	47.0
Ivomec® Plus	17	823	102.6	55.7	45.7
Dectomax® Inj.	44	1791	64.1	15.4	76.0
Cydectin Inj.	12	614	36.9	5.3	85.7
Ivermectin Inj.	13	630	90.0	45.6	48.3
Ivermectin Plus	5	193	97.5	48.6	50.1
Inj. summary:	116	5403	76.8	34.6	54.9
Pour-ons					
Ivomec® PO	21	823	61.8	27.0	56.3
Ivermectin PO	81	3378	62.6	29.2	53.4
Dectomax® PO	23	941	67.9	23.7	65.1
Cydectin® PO	25	1044	60.9	14.5	76.1
Eprinex®	5	224	38.1	25.8	32.2
PO summary	155	6410	58.3	24.0	58.8
Overall summary:	271	11,813	68.4	29.8	56.4

* All samples taken at Rx and again 2 wks post-Rx. (Ivomec®, Eprinex® – Boehringer Ingelheim), (Dectomax® – Zoetis) (Cydectin® – Elanco).

FIGURE 1.16 Fecal worm egg reduction studies on commonly used endecticide deworming products.

across North America, working with the veterinary pharmaceutical industry, veterinary clinics, feed companies, feed stores, and dealer stores. Typically, I would fly into a designated location early in the week carrying a microscope equipped with a camera to conduct clinics at different chosen locations in a particular part of the country. The clinics would be set up by a pharmaceutical territory rep that would contact the desired clinic location, work with their clients by sending out invitations for the "parasite evaluation clinic." Producers would be invited to bring samples from their animals to be processed at the clinic location for a one-day free evaluation (see Figures 1.17–1.19). Our goal for each clinic is to have from 100 to 300 samples per clinic from invites sent to producers for the designated location. The centrifuge plus supplies needed to conduct the clinic would be supplied by the pharmaceutical rep.

The labs are usually set up in the morning and sample analysis would be conducted through late afternoon followed by a producer dinner meeting in the evening, where the results are shared with each individual producer and a presentation on strategic timed deworming would be given to the invited producers. Fecal samples are collected a few days ahead of the clinics (see Figures 1.20 and 1.21) and the labs are set where samples will be run throughout the day (see Figures 1.22–1.25) while training students and conducting producer meetings explaining treatment options (see Figures 1.25–1.35). For many of these clinics, individual treatment history and lab results would be entered into a national database for further evaluation, summarization, and publication. Whenever we were working with a veterinary clinic, the clinic would usually retain the results for their clients

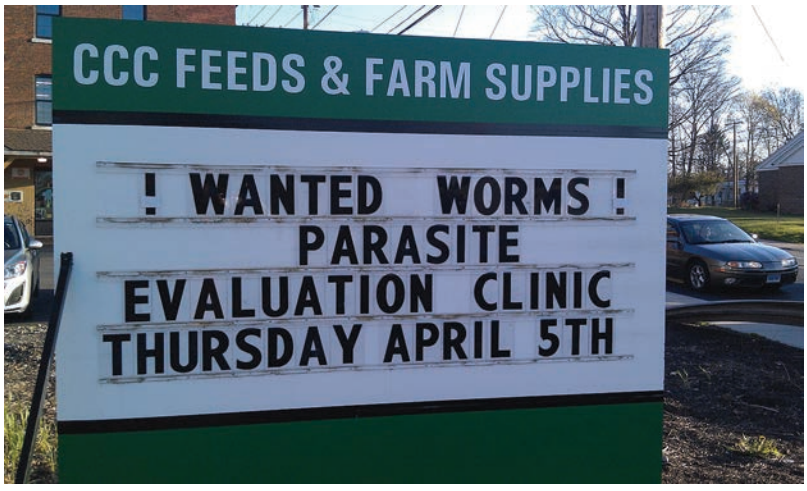

FIGURE 1.17 Sign inviting clients to bring fecal samples to "parasite evaluation clinic" (CCC feeds).

FIGURE 1.18 Sign advertizing parasite clinic with Bradberry's Best.

FIGURE 1.19 Sign telling animal owners to bring samples to our clinic and "get the scoop on your animal's poop."

FIGURE 1.20 Dr. Bliss on K Bar ranch in Oregon heading out to get fecals.

for future treatment evaluations. Any retesting or further treatment evaluations could then be sent to our lab in Wisconsin for further evaluations.

Because parasitism is a complicated phenomenon, it is often hard to separate diagnosis from treatment in terms of to how to treat, when to treat, and what products should be used. A further question should be asked as to whether treatment can be given in such a way as to prevent

FIGURE 1.21 Picking up fecal samples in Tepatitlan, Mexico.

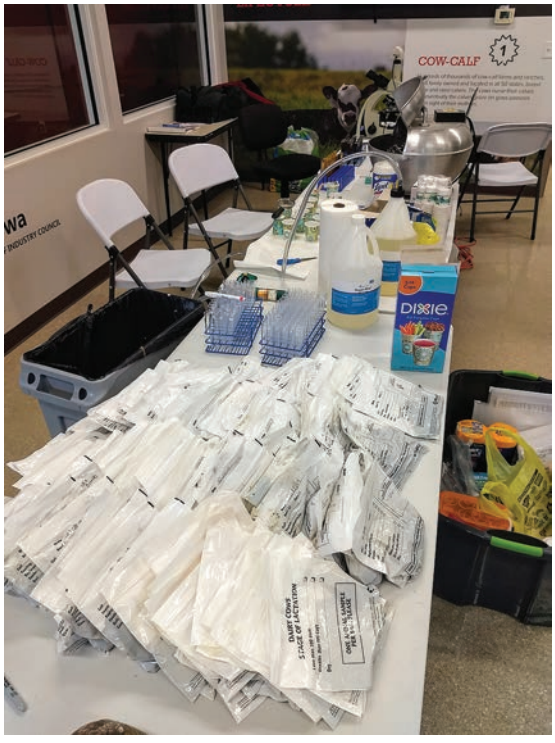

FIGURE 1.22 Lab setup for "parasite evaluation clinic" at Northeastern Iowa Community College in Calmar, Iowa.

FIGURE 1.23 Parasite evaluation at feed mill in Nebraska.

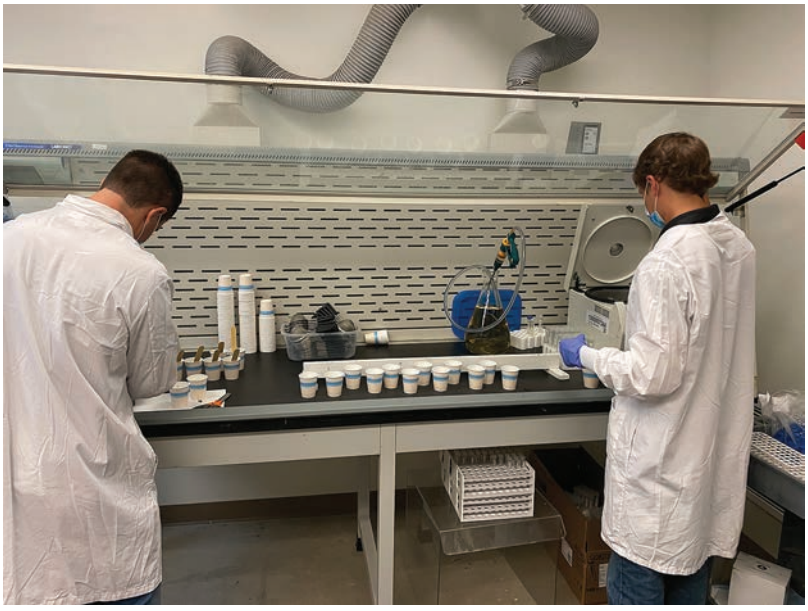

FIGURE 1.24 Lab setup conducting a large number of samples at the same time.

FIGURE 1.25 Shipping clinic supplies to Kona, Hawaii, for conduction of "parasite evaluation clinics."

FIGURE 1.26 Producer meeting on a ranch in the Puako, Hawaii.

FIGURE 1.27 Training vet tech students at California Polytechnic State University.

FIGURE 1.28 Parasite presentation to ag students at California Polytechnic University.

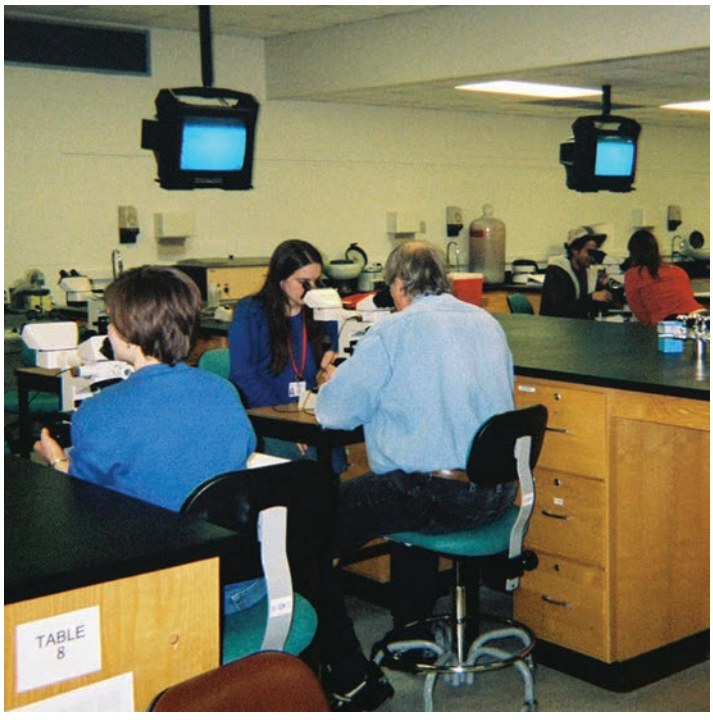

FIGURE 1.29 Scope training with vet students at University of Wisconsin School of Veterinary Medicine.

FIGURE 1.30 Scope training with vet students at University of Minnesota Vet School, St. Pail, Minnesota.

FIGURE 1.31 Large producer meeting in Saskatchewan.

FIGURE 1.32 Producer meeting in Texas.

future infections. The most common question from producers and animal owners about parasite treatment in an animal or group of animals is how high does the worm burden need to be before treatment should be recommended? Again this question is hard for most veterinarians to answer without knowing all the facts, like which types of parasites were found, what time of the year were the samples conducted, what is the age of the infected animals, are the animals pregnant, how long since the last treatment was given, what product was last used, and are the animals living in a contaminated environment?

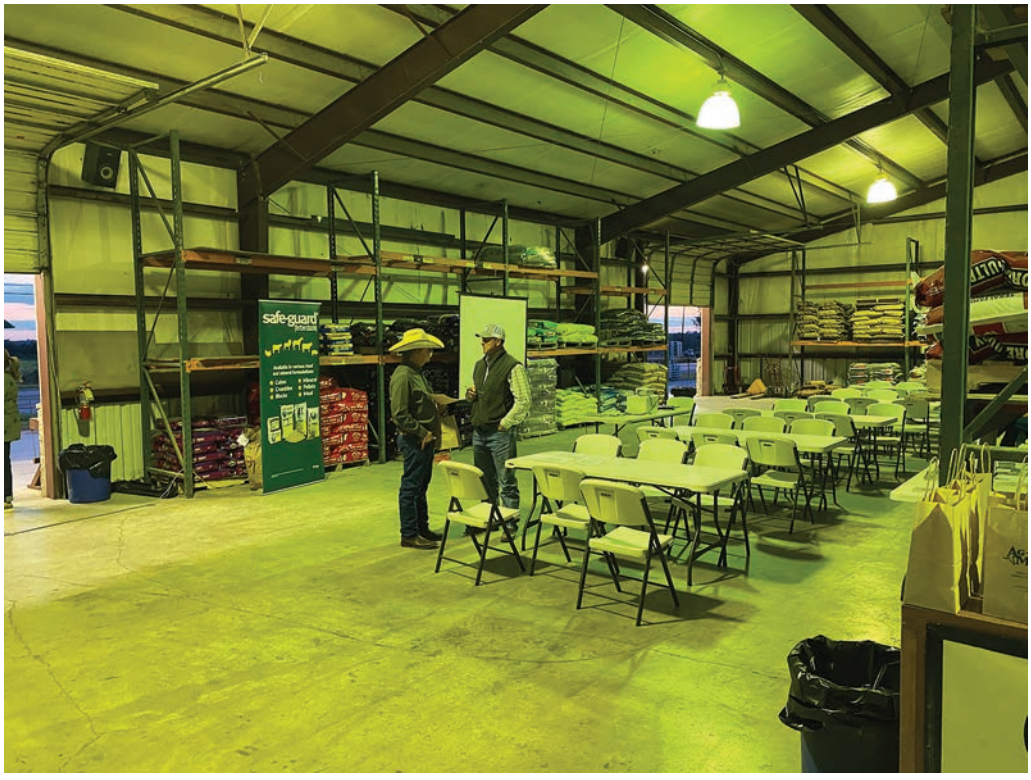

FIGURE 1.33 Setting up a producer meeting in Oklahoma.

FIGURE 1.34 Camera on scope showing a parasite egg.

FIGURE 1.35 Dr. Bliss talking to producers and showing them their fecal results.

GASTROINTESTINAL PARASITISM CAN BE CATEGORIZED INTO FOUR DISTINCT PERIODS OF DEVELOPMENT

EARLY LARVAE DEVELOPMENT AND TRANSLATION PERIOD

It is the period of time the parasite spends outside the host. During the development phase, the parasite is dependent upon environmental conditions for its survival as it develops into the infective L_3 phase. Adult male and female parasites live in the gastrointestinal tract, mate, and lay eggs which pass out in the manure. After eggs pass in the feces, a L_1 larva quickly develops inside the egg, provided the outside temperatures are sufficiently warm. This L_1 larva will hatch in just a few hours after passing while the manure pat is still warm, and then after several days, it will begin to molt into a L_2 larva. Provided environment conditions are good, it will then continue development into a L_3 infective-stage larva in a couple of weeks. The first and second larva stages remain in the fecal pat and feed on bacteria and debris in the manure pat while the L_3 larva is very mobile and will follow moisture trail away from the manure and onto the nearby vegetation. The L_3 larvae retain their outer sheath when they molt from a L_2 to and L_3 larvae which provides the L_3 larvae additional protection form environmental conditions (see Figures 1.36 and 1.37). When viewing live L_3 larvae under the microscope, they are in constant motion.

If temperatures go below freezing for a long period of time, the eggs, and L_1 and L_2 larvae are killed. Because the third-stage larva (L_3) retains its outer sheath, it now becomes a protective

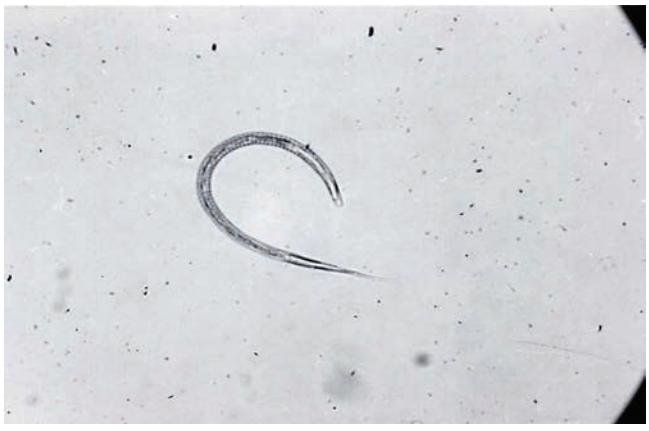

FIGURE 1.36 L_3 infective larva showing outer protective sheath.

FIGURE 1.37 L_3 infective larva found in stool samples.

covering that helps this larva survive unfavorable weather conditions. It can also move deeper into fecal pat or into the soil to find protection. Only the L_3 larvae are able to survive extremely cold temperatures. These L_3 larvae that survive the winter are immediately infective once temperatures warm up and grazing begins [9]. Because of this, some winter-time transmission can take place on warm winter days if the temperature stays above 65–70° for several days.

Moist, warm climatic conditions provide an optimal environment for eggs and larvae. When culturing larvae from manure under controlled laboratory conditions, L_3 larvae recovery is usually successful between 10 and 14 days after eggs are placed in the culture. Under natural field conditions, however, embryonation from newly excreted eggs to L_3 larvae development in the manure will take considerably longer time depending upon moisture and temperature at the ground level. Air temperature versus soil temperatures or temperatures in the manure pat will differ greatly, but temperatures between 65 and 85 °F seem to be ideal, for larval development. Some development will occur at lower temperatures; however, development stops after temperatures fall below 50°.

Under favorable summer weather conditions, this larval development process can occur in a couple of weeks. But under cool spring temperatures or during adverse weather conditions, this development process can take as long as three to four months. What happens in a normal year is eggs that are shed in early spring may take several months to become infective larvae, whereas when summer approaches, eggs will turn into larvae more quickly. This means many eggs are maturing at the same time and thus larvae contamination of pasture will increase rapidly once summer arrives. This maturation of infective larvae all at the same time provides a midsummer rise in pasture contamination and clinical disease will occur soon after, especially with sheep and goats. For cattle, this jump in contamination often occurs just as spring calves are large enough to begin grazing (see Figure 1.38).

Since an infective L_3 larva can no longer feed because its mouth parts are covered by the protective outer sheath, it has to been eaten in a timely fashion or it will die off naturally. Numerous studies show that once the L_3 larva survives the winter and moves onto the spring grass, it must be eaten by the host animal during the first 60–90 days of the season or it will eventually disappear off the vegetation [9–11]. If, for example, no animals are present on the spring pasture until late June or July, this pasture becomes a mostly parasite-free pasture.

On the other hand, extreme dry conditions can destroy L_3 infective larvae. Also, cool or cold weather may not be totally lethal but will retard larval development. Direct sunlight also can kill infective larvae. This is why eggs and larvae do not survive well under feedlot conditions or in concrete barn lots where there is no vegetation for the larvae to hide under in order to escape high temperatures or direct UV sunlight. Larvae that survive the winter from one season to another are protected in the manure pat as a L_3 infective larva. Spring rains break these pats apart and as temperatures begin to warm up, the surviving larvae move onto the vegetation as spring green-up begins. When conditions are right for spring grass growth, conditions are as right for parasite development and translation onto the vegetation for the infection process to start again.

The translation phase is the time after the larvae become infective. Infective larvae are very active moving away the fecal pat onto the vegetation in order 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 on the pastures. 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 soon after, especially if it has been dry for a long period of time and large numbers of infective larvae can move onto the vegetation at the same time.

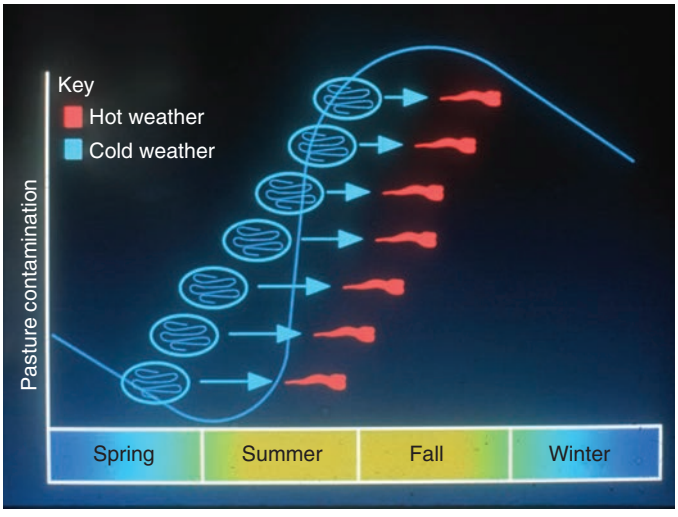

FIGURE 1.38 Graph showing seasonal development of gastrointestinal parasites on pasture.

Cattle going onto spring pastures shedding a high level of parasitic worm eggs will immediately begin contamination of the pasture (see Figure 1.39). Once the larvae escape the fecal pat, they move to and survive on the vegetative material surrounding the pat, contaminating the pastures just like weed seeds (see Figure 1.40) waiting to be ingested by grazing animals. The best time for transmission is usually in the morning when there is dew or a light rain providing the necessary

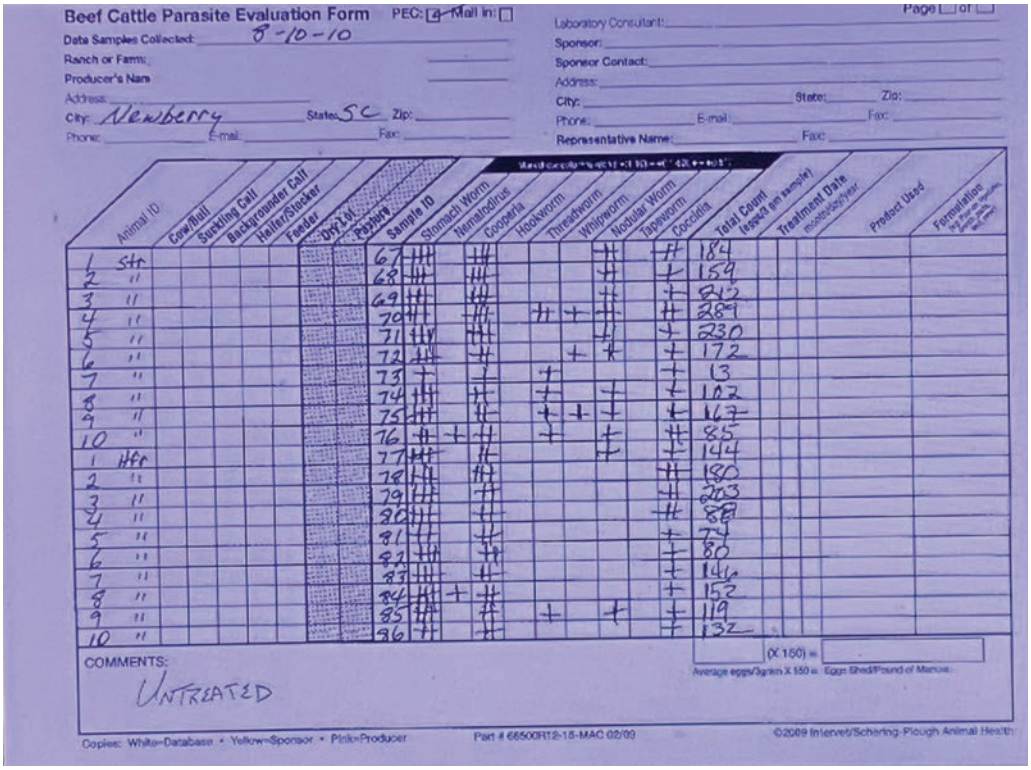

FIGURE 1.39 Fecal worm egg counts from heavily infected beef calves.

FIGURE 1.40 Worm eggs are like weed seeds recontaminating the pastures.

moisture to allow the parasitic larvae to move up on the vegetation in order to reach the animals when they are grazing. When the sun comes out and the moisture dries off, the larvae move back to the base of the vegetation. There is constant movement by the larvae in order to be consumed by the grazing animals. The survival of gastrointestinal parasites, therefore, depends on an encounter by the infective larvae with the host animal.

Worm eggs that are shed on the pasture in the spring of the first year have the propensity to survive until the spring of the following year. Larval contamination of spring pastures, therefore, comes from two sources: the first source is from the larvae that survive the winter from the previous season. The second source of larvae contamination is from the worm eggs that are shed on spring pastures after the grass has begun to grow and that have developed to infective L_3 larvae over the spring into early summer.

Due to temperature, the worm eggs that go down in early spring will take longer to develop into third-stage infective larvae than those eggs that are excreted later when temperatures are warmer. Hence, eggs shed in early spring will take longer to develop into infective larvae than eggs shed in midsummer when temperatures are the warmest (see Figure 1.41). If temperatures are perfect for development and moisture is lacking, egg development will stop. The inside of the fecal pat will stay moist much longer than outside conditions when the grass turns brown. If you step on a cow patty in the hot summer and it is still moist inside, larvae development will continue but translation to the grass may not occur until the rains come and break the manure pat open and allow the larvae to move onto the vegetation.

Eggs shed in late fall will stop development when temperatures reach freezing conditions. Eggs that make full development into infective L_3 larvae in the fall have a 15–20% chance to make it through the winter. Remember, since these larvae are infective as soon as conditions are right for grass growth, conditions are right for larvae movement onto the vegetation. Animals grazing these spring pastures can therefore become infected immediately. These larvae must be eaten or they die off since they can no longer feed on debris and bacteria in the manure. Data indicate that the larvae that survive the winter will disappear off the pasture within the first 60–90 days after first grass growth, if they are not eaten.

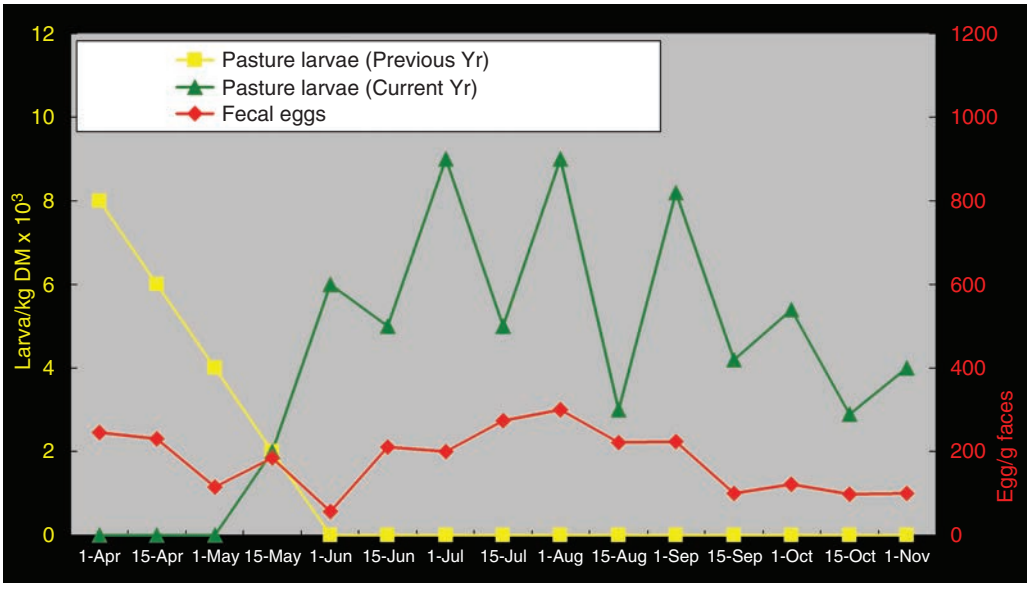

FIGURE 1.41 Seasonal epidemiological patterns of fecal worm egg counts and pasture larval development.

A comprehensive field study on the epidemiology of parasite gastroenteritis was conducted at 10 separate geographical locations across the United States and Canada [9]. The trial locations were Gainesville, Florida, Orono, Maine, West Lafayette, Indiana, Viroqua, Wisconsin, Oxford, Mississippi, Griffin, Georgia, Fayetteville, Arkansas, Moscow, Idaho, Eugene, Oregon, and Richmond, Quebec, Canada. The study demonstrated that the number of larvae present on the fall pastures (at the end of the grazing season) was five times higher than the numbers of larvae found on the spring pasture at the beginning of the grazing season. The number of worms recovered from tracer calves that grazed for two weeks prior to the end of the season from 10 separate locations across North America averaged 34,091 gastrointestinal nematodes of mixed infections. The number worms recovered the following spring on the same pastures from tracer calves that grazed for the first two weeks at the beginning of the spring season was 5731 parasites indicating that less than 17% of the parasite found on the pastures in the fall survived until the following spring season. These data indicated that approximately 83% reductions in the numbers of worms that were available to animals in the spring compared to those found in the calves that grazed for the same amount of time at the end of the previous grazing season. This reduction demonstrated that approximately 17% of the infective larvae on the pasture in late fall were able survive through the winter to be present on the spring pastures.

The second part of this comprehensive field study demonstrated that if the larvae picked during the first 90 days of the grazing were eliminated, the cumulative worm burdens in the cattle grazing these pasture for the entire summer grazing season were reduced by an average of 91% reduction. The cumulative worm burden from non-treated cattle at all 10 locations was 61,040 parasites per animal. The cumulative worm burden from cattle treated to prevent recontamination of the pastures was 10,963 parasites per animal for an 82% reduction in total worm burden per treated animal for the entire grazing season.

If the cattle are worm-free during the winter months, this is the first step to pasture control since they are not shedding eggs on the spring pasture. The first step in controlling parasites for an entire grazing season is to first stop reseeding the pasture with worm eggs during the early part of the season which reduces the number of worm eggs seeding the pasture in the spring, thus reducing the parasite challenge for the entire season (see Figure 1.42). The second step is to allow the cattle to graze the spring pasture and work like a vacuum cleaner picking up the infective larvae that survived the winter. As these larvae begin to mature in the cattle's gastrointestinal tract, the goal is to deworm the cattle strategically to kill the developing parasites before they have a chance to lay eggs and recontaminate the pasture. The prepatent period (period of time between the ingestion of infective larvae until an egg-laying adult worm is present for adult cows) is approximately 40–45 days for mature cows and bulls while for yearling cattle it is approximately 25–30 days [3].

When adult cows grazing the spring pasture are treated between 40 and 45 days or approximately 6 weeks into the grazing season to remove the larvae they have consumed during the first 6-week period, the new larvae they consume following treatment will take another 6 weeks before mature infections are actively laying eggs back onto the pastures. Thus, the mid-spring treatment will terminate the infections accumulated during the first six weeks of grazing, and then it will be another five to six weeks before eggs shedding begins again. This means that there is no major shedding of worm eggs for a total of 12 weeks or for the first 3 months of the grazing season. This means, since no new worm eggs have been shed on the pasture and providing the larvae from the previous season can either die off or been consumed by the animals grazing this pasture and no new worm eggs contamination has occurred, these pasture are now relatively parasite-free. In the northern more temperate areas of the United States, the holidays of Thanksgiving for the late fall deworming usually works and in the spring, we suggest more deworming should be completed before 4 July (see Figures 1.43 and 1.44). In the southern parts of the United States where spring

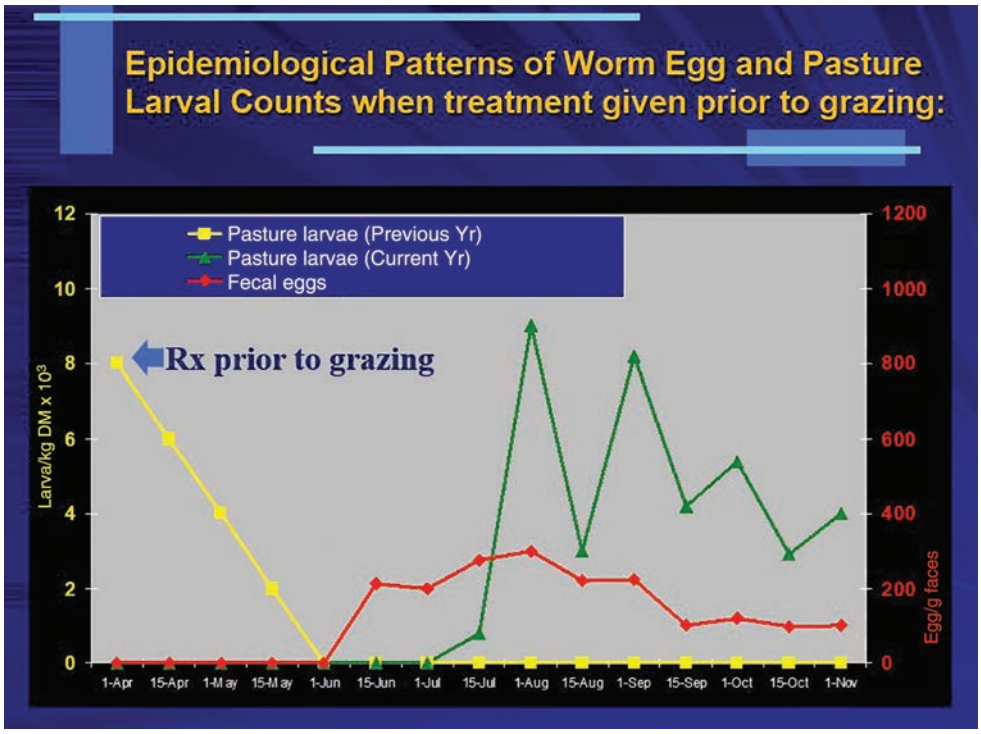

FIGURE 1.42 Epidemiological patterns of worm egg and pasture larval counts follow treatment given prior to grazing.

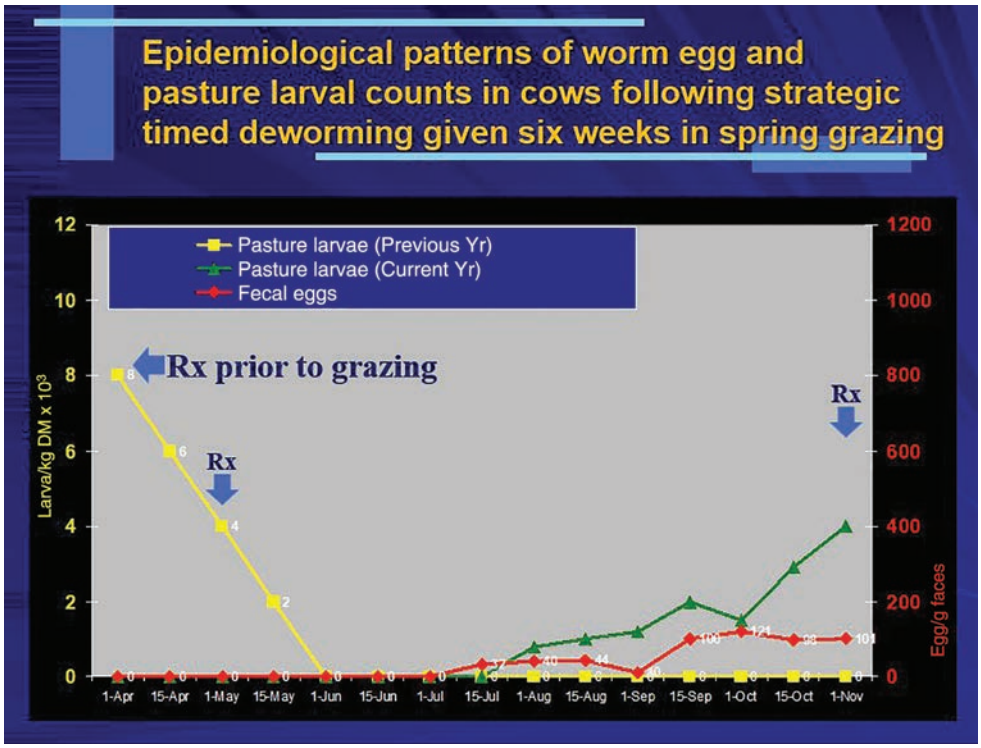

FIGURE 1.43 Epidemiological patterns of worm egg and pasture larval counts in cows following strategic timed deworming given six weeks into the spring grazing season.

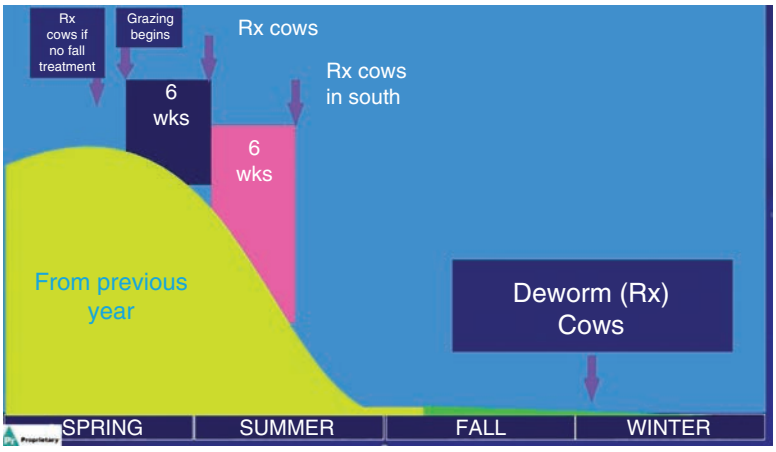

FIGURE 1.44 Graph showing fall and spring deworming effect on pasture contamination in brood cows.

begins and grass growth begins around 1 March, we use tax time (15 April), Memorial Day (late May), and Thanksgiving Day (late November) as the key suggested (approximate) treatment times.

The idea of seasonal control of parasites on pastures is to time the treatment to coincide with larval development and to treat just prior to when the maturing worms would begin laying eggs back on the spring pasture. This strategy prevents recontamination of the spring pasture since the cows are treated before their worm burdens have a chance to shed eggs. During this period, the larvae from the previous season are dying off naturally or are being eaten by the grazing cattle, which are then treated before they can shed eggs on the pasture. This is called strategic timed deworming to clean up the pastures and prevent parasite contamination from infective larvae.

In summary, fecal worm egg counts demonstrate that the egg shedding for cows on a parasite-contaminated pasture occurs approximately six weeks after ingestion of L_3 larvae. The mid-spring treatment is timed to treat the animals six weeks into the spring just before worm egg shedding should occur and then this treatment will provide a 6-week protection period following treatment for a total of 12 weeks (84 days) with no egg shedding [9–11]. This single mid-spring treatment in cows will therefore provide season-long control because if the parasites in the spring are killed and no new parasite eggs are shed on these pastures, the pasture will remain relatively free of infective larvae for the rest of the season. The goal is to reduce the level of parasite contamination on the pasture below the level that will interfere with efficient production. The data demonstrated an 80% reduction in parasite contamination for an entire grazing can be achieved. In the Deep South, a second treatment six weeks following the first treatment may be necessary to achieve the same result because of a longer growing season [12, 13].

For yearling cattle, replacement heifers, and stockers, since the prepatent period is shorter (approx. 25–30 days) compared with mature cattle, a second mid-spring treatment is recommended at a 4-week interval called a 0–4–8 week treatment period is recommended (see Figure 1.45). Treatment is given at Day 0 only if the cattle in question are determined to be parasitized to start the grazing period. These younger animals will begin shedding worm eggs approximately 25–30 days after exposure and, therefore, two treatments are recommended given four weeks apart beginning 4 weeks after grazing begins. The second treatment (given four weeks after the first treatment), means these cattle will not be shedding parasitic worm eggs for another 4 weeks for a combination of 12 weeks with no eggs shed during for the first 12 weeks of the grazing period. This recommended interval for small ruminants is only 3 weeks (see Chapter 7).

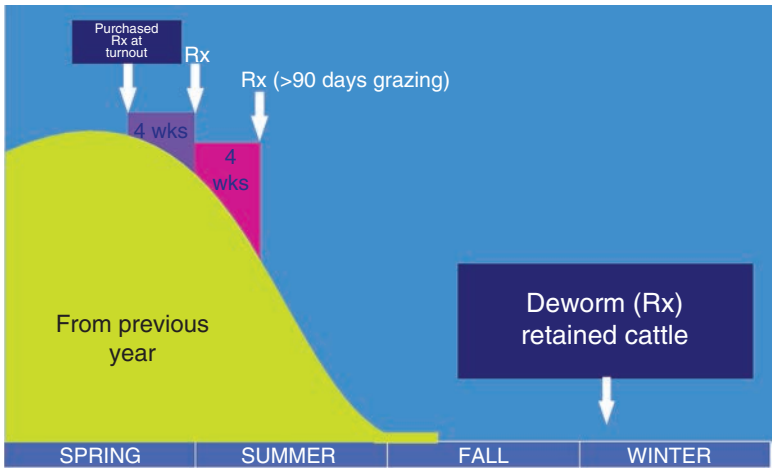

FIGURE 1.45 Graph showing strategic timed deworming in yearling cattle (two spring treatments given four weeks apart).

THE INGESTION AND INFECTIVE PREPATENT PERIOD

It is the time from when infective larvae are ingested until they have developed into mature egg-laying adult worms. Ingested infective *Ostertagia* larvae, for example, lose their protective covering in the first stomach (rumen) and then move down to the abomasum where they penetrate and encyst themselves in an existing gastric gland. The L_3 larva moves into the gland and then begins development into the fourth stage (L_4) in the gland in approximately four to five days. A mucus plug covers the opening of the gland and the larva begins to destroy the gland by stopping stomach acid (HCL) production. Usually by the end of the second week of infection, the larva has molted into an early fifth-stage larva (L_5), also categorized as a young or early adult worm (see Figure 1.46).

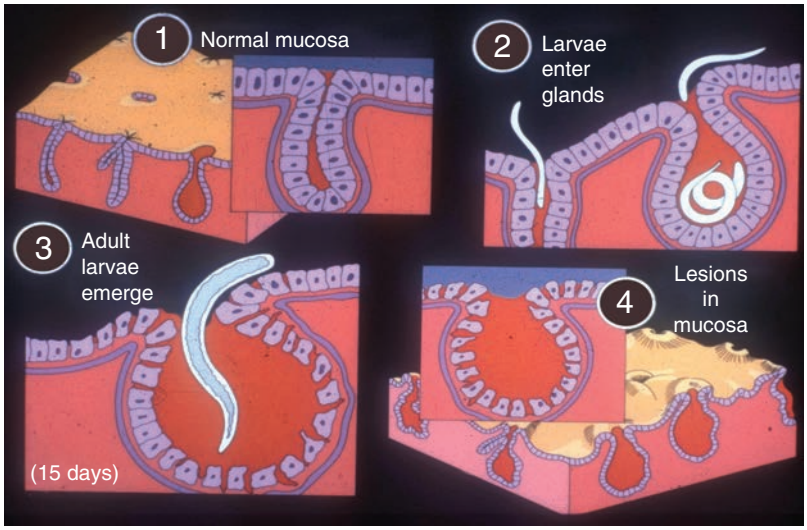

FIGURE 1.46 Exsheath L_3 larvae penetrating gastric glands and developing to a late L_4-stage larvae when it remerges on to the surface of the abomasum.

If conditions are right in the gut track, 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 attach to the wall of the abomasum, mature, mate, and begin to reproduce eggs which pass down the gastrointestinal tract into the environment where the development process begins again. This prepatent period is influenced by the immune state of the infected animal and the pH level of the abomasal fluid. For young cattle, the prepatent period is usually three weeks, whereas for mature cattle, it often takes four to five weeks before an egg-laying adult worm develops and eggs appear in their feces following ingestion of infective larvae.

THE INHIBITION OR ARRESTED DEVELOPMENT PHASE

It is the period when arrested development or the inhibition of larval development can occur. During this period of time which occurs soon after infection, the developing larva stops the maturation process and becomes dormant for an extended period of time. The reemergence of these larvae usually occurs during a period when parasitism is not expected to be a problem just after prolonged drought period or during the middle of a winter season. For the northern half of the United States and Canada, the best time to check fecals for parasite eggs in horses and small ruminants is in late March or April just prior to the beginning of the grazing season. It is not uncommon to find very high eggs at this time of the year despite having been treated in late fall. The reason for this is that heavily infected animals coming into the fall and early winter period are carrying heavy worm burdens and often high levels of inhibited larvae in the abomasal glands. As the animals go into the winter period, they are no longer ingesting infective larvae and as the old worms die off, the condition of the gut improves thus communicating to the inhibited larvae that conditions are conducive for redevelopment. Of course, as the inhibited larvae begin to maturate, it is just the right time to begin seeding the spring pasture with a new crop of parasite eggs (see Figure 1.47).

Inhibition is a phenomenon that reportedly can occur in most parasites but is most common with the Brown Stomach Worm (*Ostertagia*) in cattle, the Small Strongyles (Cyathostomins) in equine, and the barber's pole worm (*Haemonchus*) in small ruminants. Inhibition appears to be a somewhat continual process that only occurs starting when worm burdens are at their highest point. There are numerous reports, however, when animals are reportedly dying from parasitism in March when it seems least highly unlikely since the pastures are dormant. The reason is that

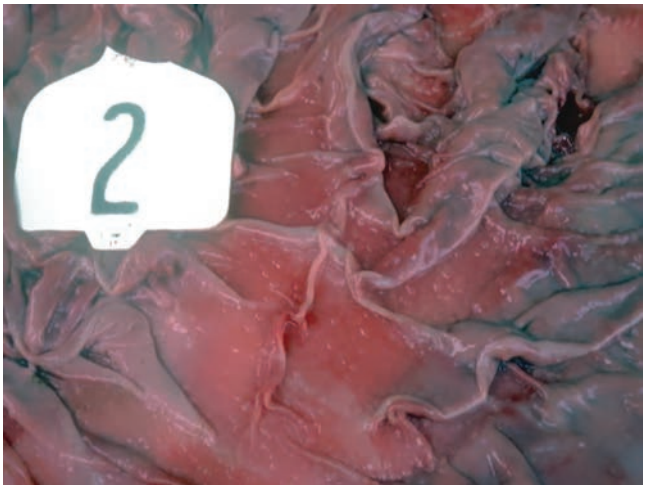

FIGURE 1.47 Inhibited *Ostertagia* larvae in the abomasum observed at necropsy.

in many cases, the inhibited larvae all maturate at the same time causing clinical disease. This is called Type II disease.

Considerable controversy exists among scientists on why and how inhibition occurs. Data from Louisiana indicate that inhibition in the south begins in May whereas northern inhibition occurs later in August much like the European model [11]. Necropsy shows that when some larvae are simply slow to develop where other larvae turn into a state of complete inhibition. (Necropsy shows that some larvae continue to slowly develop in the adult stage while other larvae remain in a state of complete inhibition.) These inhibited larvae usually stay in an inhibited state until winter time when transmission stops, the gut begins to improve and then these inhibited larvae begin to return to normal and emerge into the gut tract. The same phenomenon occurs in the southern United States but triggered by drought conditions rather than winter conditions (see Figure 1.48). As parasite burdens build in the gut, the physiology of the abomasum changes. As more and more gastric glands are invaded by newly ingested and acid production stops, the pH in the gut starts to rise. The ideal pH at the surface of the abomasum is between a pH 2 and pH 3. As the abomasum pH rises too high, an increasing number of gastric glands are shut down, digestion slows and eventually stops altogether, and the animal dies.

If a developing larva in the gland realizes that the condition of the gut is no longer conducive for development (as the pH raises in the lumen of the abomasa), the larva stops developing and undergoes arrested development and becomes inhibited. Once the conditions of the abomasa improve, development resumes. It is not in the best interest for the parasitized animals to die because they also die. Inhibition is therefore a protective mechanism to prevent overwhelming their host. Two key periods cause a redevelopment of these larvae: a prolonged drought and natural winter conditions. So, as cattle undergo a severe drought or winter condition, they are no longer exposed to infective larvae. As winter or drought conditions persist, the old worms gradually die off and conditions in the gut improve, triggering the inhibited larvae to begin maturation again.

The inhibited or arrested state of developing larvae occurs when the invading larvae stop development in the host animal and remain in a stage of arrested development only to begin development later in the year such as during the middle of winter when external conditions are better. The cattle parasite that is most well known for becoming inhibited is *Ostertagia* (also known as the Brown Stomach worm); however, *Haemonchus* (also known as the barber's pole worm) for sheep and goats and small strongyles in horses undergo an inhibited period in their development from

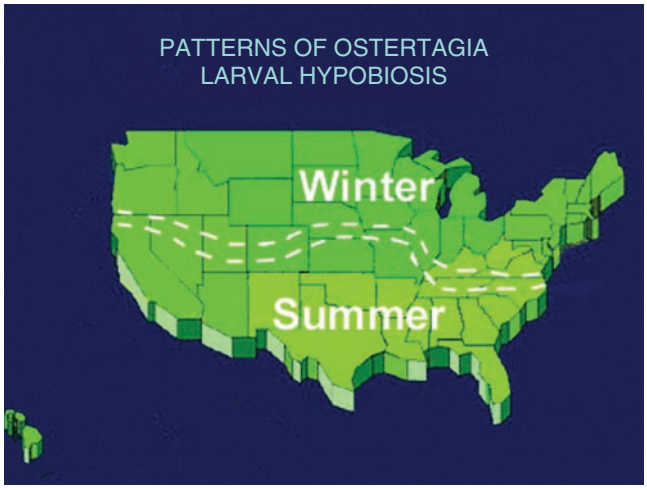

FIGURE 1.48 Patterns of inhibited larvae, northern versus southern climate.

infective larvae to an egg-laying adult parasite. This inhibition state occurs primarily in weaned calves and yearling cattle. Inhibition is almost always found later in the season starting mostly in mid-summer lasting until the end of the season.

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 6 months depending upon the level of worm burden present and conditions of the gastrointestinal tract at the time of larval ingestion. The triggering mechanism that tells the inhibited larva is the pH of the abomasum. If it is in the normal range of pH 2–pH 3, larval development is normal. During inhibition, infective exsheathed third-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 is not 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 gastrointestinal tract changes making conditions such as high pH affecting conditions such that conditions are no longer right for larval development, and development stops following which new incoming larvae begin to undergo arrested development. 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; as worms mature and older worms die off, the gastrointestinal conditions begin to improve. Previously inhibited larvae now begin to develop, emerge, and mature to adult egg-laying worms.

The buildup of parasitic infections on pasture probably accounts for most of the variations 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 buildup does not occur until late August or early September with inhibition occurring from September onward, which coincides with the northern inhibition period. A very important point to consider when talking about pasture contamination is to keep in mind, the condition of the pasture in terms of rainfall (or irrigation) and stocking rate has more influence on parasite population and reinfection rate for the animals grazing that pasture than geographical location of the pasture (see Figures 1.49 and 1.50).

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 is broken down into two separate types: Type I and Type II disease.

Type I disease is caused by a heavy infection during the grazing season for horses, cattle, sheep, and goats. This disease outbreak is defined as parasitic gastroenteritis, which is caused by a heavy infection of adult worms and is easily diagnosed since it occurs during the middle of grazing season. Type I disease is a due to a buildup of larval contamination on the pasture and occurs in late spring/early summer in southern regions of the country and in mid to late summer in the more temperate regions of the country. Type I disease is most common in non-treated, heavily infected, poorly managed animals on a low plane of nutrition. Often, only several animals out of a group will first show symptoms for Type I disease. With sheep and goats, Type I disease usually occurs where the stocking rate is high and parasite contamination becomes overwhelming for the animals. Fecal worm egg counts are a great indicator whether parasite worm burdens are reaching a clinical threshold (see Figure 1.51).

FIGURE 1.49 Low stocking rate (in West Texas) means less parasite exposure.

FIGURE 1.50 High grazing density in rotational grazing setup.

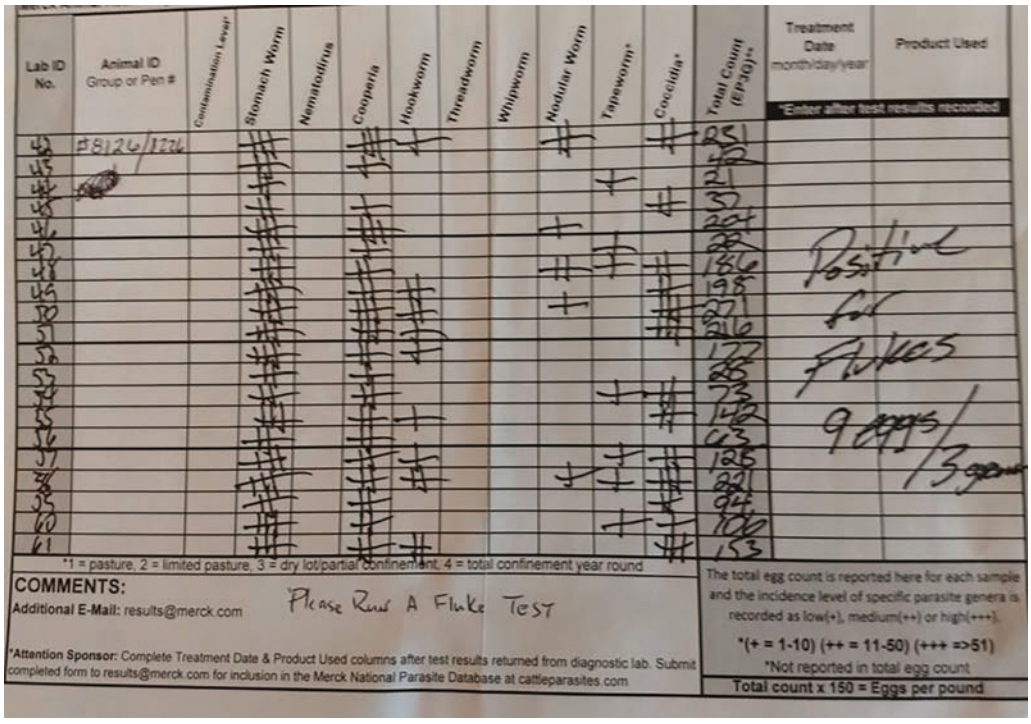

FIGURE 1.51 Texas cattle showing high worm egg counts.

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. Type II disease can also occur in the south during hot dry summers when contamination of the pastures are low parasite transmission due to poor grass or "brown out" conditions. The mechanism, which causes this sudden emergence of larvae, is not fully understood but is often associated with poor nutrition or other stress-related conditions. Type II disease, therefore, usually occurs either in the middle of the winter when animals are housed in crowded conditions being fed poor quality of hay and feed or during the summer during "brown out" conditions in Southern United States.

The inhibited phase is a survival mechanism that gastrointestinal 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 veterinary parasitologists have compared this inhibition mechanism to the diapause phenomenon in insects which works like a biological clock, which programs the larvae to cease development until a more favorable environment is present.

THE PATENT PERIOD

It is the survival and reproduction time of adult worms (both female and male worms are required) in its host animal. This the reproductive phase of parasitism. Each female worm can lay thousands of eggs during her adult life, which may be as short as a few weeks and as long as several years. Some parasites such as *Trichuris* have been reported to live in an animal for several 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 change this process when new larval ingestion slows down or stops; this is the time of the year when inhibited larvae begin development and emerge, keeping the parasite life cycle going.

Worm egg shedding can vary from animal to animal. Equine seem to mix their manure better internally, so repeated worm egg counts from one animal is often very similar, whereas with cattle you can take five divots from the same paddock and get five different answers. Multiple fecals are often required to get a reliable result; therefore, 5–10% of each animal grouping should be random sampled. Biostatisticians tell us 20 samples are the maximum number of samples needed regardless the size of the group sample [14].

The patent period is the period when the worm parasites lay eggs and the fecal count becomes positive and then the first question is how many eggs do I need to find to make treatment worthwhile? From a parasitologist standpoint, this can be frustrating or it can be the golden moment when people begin to understand the economics of parasitism.

How do all the different gastrointestinal parasites ensure survival? Parasites survive by contaminating the environment of the host animal to ensure propagation of their species. Mostly, all animal species are infected by more than one species of parasite at any given time. In grazing cattle, it is very common to find eight or nine different species present upon necropsy. Monospecific infections are rare and usually only seen with young animals infected with what is called "barnyard infections." Most problematic parasites such a *Haemonchus* in goats or small strongyles in horses survive because of the high degree of fecundity by these parasites and their ability to lay thousands of eggs keeping the environment of these animals heavily contaminated, especially in late summer.

It is not uncommon for equine to shed over 500,000 eggs per animal per day back in their environment. There are parasites, like the whipworm, which are noted for being low-egg shedding parasites, however, because their eggs can survive in the environment for many years and they too have very good survivability of their species. The whipworm larvae remain protected in the egg shell until it is eaten or the infective larvae is released from the shell. Each parasite seems to have found its niche in being able to survive. Another factor that helps survivability is the ability of these parasitic larvae to accumulate or buildup in the animal's environment over a summer grazing season.

During the parasite life cycle, pastures are contaminated 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 into 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 into 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 very short period of time.

Factors that affect the level of pasture contamination include:

1. The level of worm egg excretion and total number of eggs passed in the manure.
2. The stocking rate or density of grazing animals. The more animals present, the greater possible number of eggs being shed on the pasture.
3. The survival rate of different worm eggs and hatched larvae.

4. The survival rate of infective larvae. Larvae that remain in the manure in late fall have the greatest chance of surviving until the following spring.
5. Pasture management variations. Number of trees or shaded area of the pasture, whether or not harrowing of the pasture is practiced, and whether pastures are grazed down completely can all affect the infection rate.
6. Anthelmintic treatment, the brand of dewormer used, and time of treatment can all affect pasture contamination level or control thereof.

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*, has 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 (at best) 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 under very dry or very cold conditions.

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."

What is the meaning of positive fecal worm egg counts in beef cattle? The first step in conducting fecal worm egg counts is to determine the types and prevalence of various parasites in a particular area to help establish product and treatment times necessary. The first step in developing treatment strategies is to determine the presence of parasitism under natural field conditions.

A total of 17,973 non-treated cattle were examined using the "Modified Wisconsin Sugar Flotation Method" fecal exam from nearly 2000 cattle operations (see Figures 1.52–1.57). Results indicated the prevalence of a number of parasites such as tapeworm (*Moniezia*), *Nematodirus*, and whipworm (*Trichuris*) was considerably higher than expected. Results demonstrated that up to 71.4% of the operations with nursing/weaned calves were positive for *Nematodirus*. Feedlot

Prevalence of Parasites found in 5981 Non-Treated Beef Cows from 427 Beef Operations:

Parasites	Number of Farms	Number of Cattle	Percent of Farms Infected (%)
• All parasites	427	5981	N/A
• Stomach worms	377	3765	88.3
• *Nematodirus* spp.	47	101	11.0
• Threadworm	27	96	6.3
• *Cooperia* spp.	292	2178	68.4
• Hookworm	14	18	3.3
• Whipworm	10	22	2.3
• Nodular worm	104	361	24.4
• Tapeworm	135	274	31.6
• Coccidia	268	1674	68.4

FIGURE 1.52 Prevalence of gastrointestinal parasites in non-treated beef cows.

Prevalence of Gastrointestinal Parasites found in 1102 Non-Treated Nursing Calves from 101 Separate Midwest Ranch Operations:

Parasites	Number of Farms	Number of Cattle	Percent of Farms Infected (%)
All parasites	101	1102	N/A
Stomach worms	82	796	81.2
Nematodirus spp.	53	240	52.5
Threadworm	8	33	7.9
Cooperia spp.	72	625	71.3
Hookworm	0	0	0
Whipworm	12	45	11.9
Nodular worm	10	39	9.9
Tapeworm	34	129	33.7
Coccidia	72	535	76.3

FIGURE 1.53 Prevalence of gastrointestinal parasites in non-treated nursing calves.

Prevalence of Parasites found in 1,773 Non-Treated weaned "backgrounder" Calves on 105 Ranches:

Parasites	Number of Farms	Number of Cattle	Percent of Farms Infected (%)
All parasites	105	1773	N/A
Stomach worms	96	1292	91.4
Nematodirus spp.	75	479	71.4
Threadworm	13	71	11.9
Cooperia spp.	93	1245	88.6
Hookworm	6	8	5.7
Whipworm	31	77	29.5
Nodular worm	24	168	22.9
Tapeworm	54	215	54.4
Coccidia	87	986	82.9

FIGURE 1.54 Prevalence of gastrointestinal parasites in non-treated weaned calves.

cattle operations were 60.3% positive for *Nematodirus*, 22.2% positive for whipworms, and 47.6% were showing tapeworms. Many of the dewormers used today lack efficacy for parasites such as whipworms, *Nematodirus,* and tapeworms, so producers are actually selecting these parasites to become more prevalent and, of course, become more of an undetected economic problem.

The fecal worm egg count is really a predicable value because it determines what the future infection of the animals that are sampled is going to be. The following are the key points to the question on the true meaning of fecal worm egg counts in beef cattle:

1. The first step is to determine the shedding rate in terms of fecal worm egg output on a daily basis, for example, cows averaging 10 eggs/3 g sample × 150 1500 eggs/lb of manure × 60 lb per day equals = 90,000 eggs/day/cow.
2. The second step is to determine if the animal has been treated in the previous 14 days while on pasture to determine if dewormer last used was effective (positive worm egg counts should not be seen for at least 14 days following treatment in any aged animal). Also, if the animal is in confinement or is on a winter pasture since the last treatment, the previous treatment

Prevalence of Parasites found in 3447 Non-Treated Replacement Heifers and Stockers from 213 Operations:

Parasites	Number of Farms	Number of Cattle	Percent of Farms Infected (%)
All parasites	213	3447	N/A
Stomach worms	194	2633	91.1
Nematodirus spp.	90	459	42.3
Threadworm	18	39	8.5
Cooperia spp.	160	1902	75.1
Hookworm	11	40	5.2
Whipworm	31	88	14.6
Nodular worm	54	496	25.4
Tapeworm	72	269	33.8
Coccidia	160	1677	75.1

FIGURE 1.55 Prevalence of gastrointestinal parasites in non-treated yearling cattle and stockers.

Prevalence of Parasites found in 2835 Non-Treated Feeder Cattle from 126 Operations:

Parasites	Number of Farms	Number of Cattle	Percent of Farms Infected (%)
All parasites	126	2835	N/A
Stomach worms	122	2056	96.8
Nematodirus spp.	76	482	60.3
Threadworm	6	18	4.8
Cooperia spp.	119	2055	94.4
Hookworm	1	1	0.8
Whipworm	28	66	22.2
Nodular worm	27	167	21.4
Tapeworm	60	252	47.6
Coccidia	112	1589	88.9

FIGURE 1.56 Prevalence of gastrointestinal parasites in non-treated feedlot cattle.

still should be effective and fecal worm egg counts should remain negative until the treated animals are moved to an infective pasture.

3. The third step is evaluating the egg count based on the season of the year.
   - **A. Wintertime:** There is no need for cattle to harbor parasites through the winter. The first reason is that winter often has the highest maintenance cost for cattle and so why have parasites increased this cost and, second, wormy cattle during the winter will shed eggs on the spring pasture re-establishing the infection level of this pasture.
   - **B. Springtime:** Any shedding in the spring is bad because it re-established the infectivity of the pasture. The goal of strategic deworming is to keep the animals worm-free during the winter and the first six weeks of spring grazing. If the cattle are worm-free from Thanksgiving to 4 July, then they are basically free of parasite problems for the rest of the year.
   - **C. Summertime or early fall:** There are no worries here because parasite level will be low if winter/spring control strategy is successful.

Combined Summary of Gastrointestinal Parasites Recovered from 17,973 Non-treated Midwestern Cattle – all age groups:

Parasites	Number of Farms	Number of Cattle	Percent of Farms Infected (%)
All parasites	969	17,973	N/A
Stomach worms	903	16,733	93.1
Nematodirus spp.	450	3177	46.4
Threadworm	93	336	9.5
Cooperia spp.	810	13,593	83.5
Hookworm	52	127	5.3
Whipworm	154	426	15.8
Nodular worm	289	2487	29.8
Tapeworm	450	1957	46.4
Coccidia	764	9929	78.8

FIGURE 1.57 Prevalence of gastrointestinal parasites – all age cattle summary.

1. What category of animal is the count from: cows, calves, stockers, heifers, bull, or feeder cattle?
2. What time of the year is it in terms of each season of the year: winter, spring, summer, or fall?
3. Are the animals in confinement or on permanent pasture, do they go to mountain pasture, high desert pasture, an irrigated pasture, or are they rotationally grazed?
4. When were the animals last treated?
5. What product was used for the animals' last treatment?

See: Chapter 2 on strategic deworming strategies for beef cattle.

What is the meaning of positive fecal worm egg counts in dairy cattle? An important first question for dairy cattle is: are the milk cows, dry cows, replacement heifers, or calves on pasture or have exposure to pasture?

1. Milk cows and heifers not on pasture for over six months do not need treatment.
2. Positive Dry Cows should be treated prior to or at the time of freshening to reduce stress at calving and help keep cow in best shape for the start of producing milk.
3. All positive calves and yearling cattle on pasture should be dewormed strategically.
4. Baby calves of up to three to four months old should be checked for "barnyard infections."
5. Milk cows with exposure to pasture should be checked annually during the first trimester to make sure parasites are now interfering with milk product and reproductive efficiency.

Parasites reduce dry matter intake and reduce gastric gland function, so any milk cows with positive counts should be treated during the first trimester of milk production.

See Chapter 3 for more information on deworming dairy cattle.

What is the meaning of fecal worm counts in horses? There is nothing good about gastrointestinal parasites in horses. Horse owners with small numbers of horses can eradicate parasites in their horse altogether. First of all, worm-free animals on worm-free pasture stay worm-free. Horses made worm-free are then brought on to a farm where horses have never resided will stay, that way providing no new horses are brought on to the location. Any horse leaving the property has a chance to bring parasites back to property. Routine fecal exam will prevent this from

happening. Horses less than six months of age should be checked with a fecal exam and may need monthly treatment to remove any threadworm (*Strongyloides*), strongyles, or roundworms (*Parascaris*) if any of these worm eggs are found from a fecal examination.

See Chapter 4 for detailed information on deworming horses.

What is the meaning of fecal worm egg counts in swine? All sows should be worm-free at the time of or just prior to farrowing. Sows that are not worm-free at the time of farrowing will immediately infect their baby piglets. Purchased grower pigs should always receive a fecal worm egg count to make sure they are worm-free. A second fecal worm egg count should be conducted when the grower pigs are around 100 lb. Show pigs should be routinely checked because parasitized swine are harder to maintain good outward appearances for showing.

See Chapter 5 for more deworming information on parasite control in swine.

What is the meaning of fecal worm egg counts in small ruminants and hoofed wildlife? Small ruminants are relatively new to the deworming scene. Never move a parasitized animal. If you are buying or selling, always make sure a fecal worm egg count is conducted. Over the past 30 years, thousands of people bought "wormy" goats and brought them home to where goats have never lived and within 2 to 3 years, they have dead goats from heavy Haemonchus infections and now they are living with a nightmare of seasonal Haemonchosis but during the summer (Type I disease) and during winter early spring (Type II disease).

See Chapters 6 and 7 for more deworming information and deworming strategies for small ruminants and hoofed wildlife.

Summary on why gastrointestinal parasitism is so important for food animal production as well as for companion animals? A parasitized disease state can be a resultant condition occurring either from or during the establishment process of a parasitism. The condition may be prevalent whether the endpoint or the encounter results in rejection or death of the parasite or in the concession of accommodations to it by the host. The situation occurs where clinical detection is insufficient because it lacks the sensitivity to detect the existence of harmful parasitic infections unless this infection has progressed to the state where clinical signs are present. The parasite encounter, therefore, often remains undetected by humans unless a specific and sensitive laboratory test is available. In many cases the presence of parasitism means economic loss to a producer or owner, but in other cases where companion animals harbor parasites, the concern may strictly be due to the ability of the owner to know whether or not parasites are present and then being able to remove the parasites and improve the animal's health and well-being.

Parasitism can masquerade in many forms and cause extensive losses directly or indirectly. The extreme of parasitism may result in intestinal irritation with diarrhea, anemia, severe loss of general condition, and eventually death. Parasites have often been described as ubiquitous, unseen, and of great variety and abundance, and because of their effects are generally not apparent, they undermine the health of countless thousands of animals and a constant hazard to efficient profitable production for food animals. Parasites can cause damage to the animal and loss to their owner by:

1. Reduced yield and depreciation of animal products such as milk, eggs, hides, and wool.
2. Condemnation of animal parts such as liver, intestines used for casings, and meat carcass to federal meat inspections. Value of hides can also be reduced with cutouts due to parasite damage.
3. Waste of feeds, labor, and space to bring animals to mature productivity or market.
4. Interference with breeding, reduced reproduction efficiency, diminished fertility, reduced litter size, and lower egg-laying or poultry.
5. Reduced quality of animal – lower grades of market stock and reduced sale.

6. Lowered efficiency or work animals such as horses and mules. Reduced longevity in animals used for labor in third-world countries.
7. Depreciation of capital items – breeder animals, farm properties, and abandonment of properties. Cost of plowing down parasite-contaminated pastures and reseeding.
8. Inefficient utilization of pastures, barns, and pens by unproductive stock.
9. Lower resistance of infected stock to other diseases and parasites such as coccidiosis. Reduced efficiency of vaccines in heavily parasite-infected animals.
10. Death, suffering, and anxieties imposed on humans by parasites transmitted from domestic animals or by disease carried by parasite that are primarily animal rather than humans.
11. Reduced performance by "competition animals" such as race horses, hunters, jumpers, rodeo animals, and negative impact on physical appearance by show animals.
12. Expenditures for worthless or inefficient drugs, and related treatments and equipment. Cost of failure to control parasites because of using "dewormers" at the wrong time of the year.
13. Pain and suffering by the parasite-infected or parasite-infested animal themselves.

Animals suffer daily from parasites, especially by those parasites that infect the gastrointestinal tract that can only be controlled through treatment with an anthelmintic or dewormer. Oftentimes, the longer an animal goes untreated, the greater the infection becomes with increased pain and suffering.

The economic importance of parasitism is changing as animal production becomes more efficient due to continued improvements in genetics, nutrition, implant technologies, and disease control measures. A recent study from Iowa State University identified parasite control as the single-most import factor in producing beef efficiently. This study identified parasites as a primary detriment to efficient production and that gastrointestinal parasites are responsible for adding as much as $190.00 per animal to the cost of raising beef cattle from birth to slaughter. A second comprehensive study by Dr. Judith Capper was conducted on the environmental and economic impact of withdrawing parasite control (fenbendazole) from the US beef production. The summary study from published field studies reported 10% better pregnancy rates, 8.5% better weaning weights (+46.2 lb), 11.8% lower feed cost (−187 lb), 15.4% better land and water utilization, 17.1% less fossil fuel used, 13.3% less greenhouse gas emission, and an over 15.4% better land and water utilization [15, 16]. A third comprehensive study was conducted monitoring deworming treatments given to grazing cattle versus only treating cattle upon arrival in the feedyard. There was a clear statically significant advantage in deworming cattle on pasture versus only treating after arrival in the feedyard in terms of both better gain and feed efficiency but also in terms of significantly better health data (stronger immune function). The economics of parasitism calculated for this analysis came from the effects of parasitism upon reproductive efficiency, rate of gain, feed efficiency, carcass quality, milk production, and the immune system through reduced mortality and morbidity.

These measurable parameters most importantly do not consider the amount of pain and suffering that can take place within infected animals (which also goes unnoticed) during the early phase of infection. Parasite-infected animals are often seen standing around or lying down rather than up grazing, this is because they do not feel well even though the signs of an infection is not yet visible when looking at the physical condition of the animals in question. Leaving parasitized animals untreated or allowing them to become heavily infected before giving treatment can, therefore, sometimes become an issue of concern regarding animal welfare. The harmful effect on the animals themselves can be seen in the following photo (see Figure 1.58).

The parasite-infected cattle are on the greenest pastures showing less grazing due to appetite suppression caused by the parasites. The pastures grazed by the treated cattle are grazed down

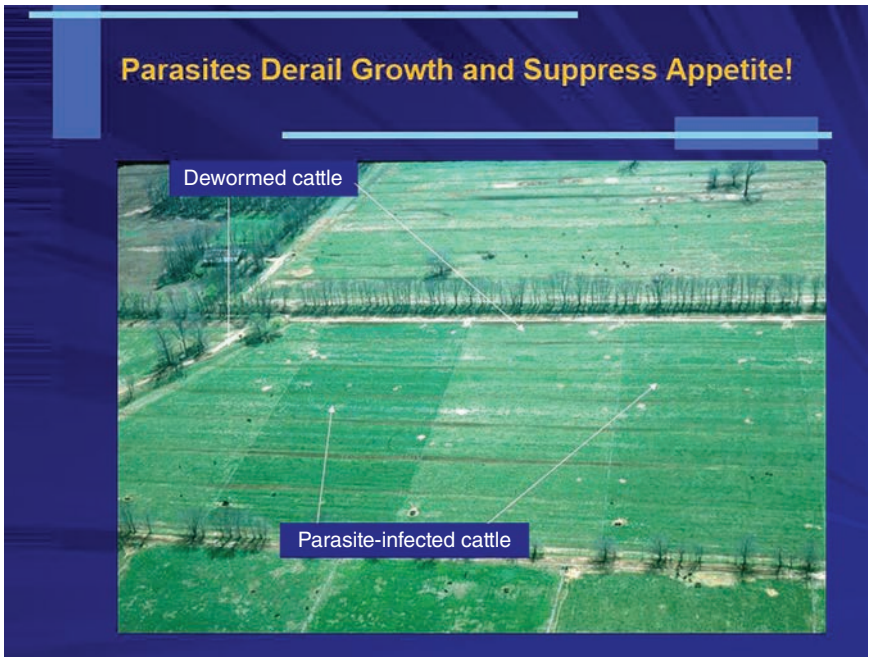

FIGURE 1.58 Pasture view showing the effect of parasites on appetite.
Source: Gasbarre [17] with permission from ELSEVIER.

because the cattle feel well and, therefore, are eating well. The cattle on the grazed pasture averaged +60 lb heavier than their herd mates grazing the parasitized pastures.

A popular theory prevailing in the United States is that treatment for parasites should only occur or be recommended for treatment after infections level reach a high point [18]. This is touted as a way to prevent parasite resistance from occurring. This theory is simply a theory not proven by research data of any kind, instead it is quickly seen as a very harmful theory for the animals themselves endure pain and suffering caused by the parasite before treatment is instituted. Parasitism is a process by which one organism invades and lives off another organism and thereby causes harm throughout this invasion process. The damage usually begins immediately upon contact with the parasite or parasitic stage that is involved with the invasion and continues throughout the life cycle of that organism. There is nothing good about parasites and the process of parasitisms, the animal under attack most often suffers damage long before the presence of the parasite is diagnosed.

Internal parasite can also adversely affect the immune system. Recent data indicate that gastrointestinal parasites have a strong effect on the animal's immune system [19–21]. One benefit to deworming that is often overlooked is its impact on the effectiveness of vaccinations. Cows that are infected by parasites have compromised immune systems caused by the negative nutritional impact gastrointestinal parasites have on the immune system. In addition to this indirect effect, some parasites have a direct effect on the immune system through mechanical damage they cause to the animal itself.

Immunosuppression occurs when parasites actively hinder one or more of the host's defense mechanism. Because the *Ostertagia* larvae damage the glands of the abomasum during the development, they disrupt metabolism and are thought to affect development of immunity simply by reducing the necessary substances such as protein and trace minerals. It has been shown that some parasites in cows create immune cells that shut down the production of antibodies and macrophages, key components in a functioning immune system. Such measures ensure that the

parasite will survive and be able to reproduce in the cow. These immune-suppressive tactics that protect the parasite leave the cow susceptible to other invaders such as bacteria or viruses. As noted previously, immunosuppression interferes with the host's ability to respond to a vaccination, our most effective tool for preventing infectious diseases [19, 20].

Cooperia spp. has now become of one the most prevalent parasite species in US cow/calf operations as observed by our data (see Figures 1.52–1.57). This is at least in part due to the widespread use of endecticides that have minimal activity against these parasites. The effects of *Cooperia* spp. on cattle productivity has not been studied until recently [22]. This study demonstrated the *Cooperia* has a deleterious effect on both appetite and nutrient uptake or utilization. Mesenteric lymph nodes were increased in size and the small intestinal mucosa was thickened with an increased amount of mucus. The most prominent histological changes in the small intestine involved mild to moderate numbers of intraepithelial lymphocytes and globule leukocytes as well as aggregates of eosinophils within the lower lamina propria (see Figures 1.59 and 1.60).

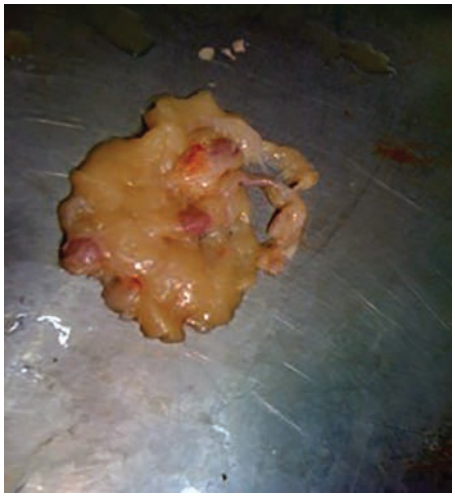

FIGURE 1.59 Normal small intestines from "parasite-free" calf.

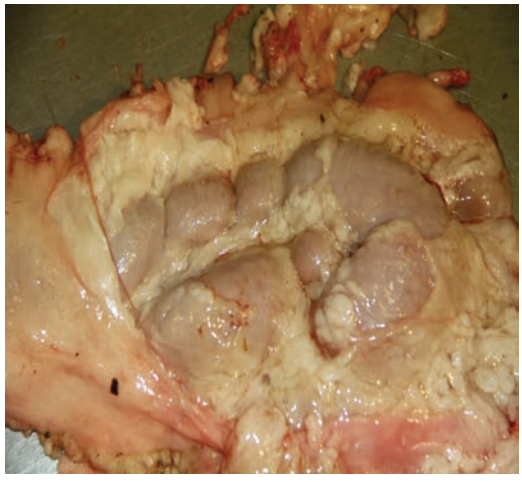

FIGURE 1.60 Enlarged lymph nodes in small intestine from artificially induced *Cooperia* infection in young calf.

What are the goals for parasite control? The monetary cost of parasite control depends upon the economic benefit achieved minus the cost of treatment. With pets and companion animals, swine, or cattle in total confinement and, in many cases, horses that are isolated from other horses, eradication of the parasites is possible. This eradication effort can be coupled with key strategies. Another area where eradication of parasites would be excellent is with the expansion of the goat market, producers that had never had goats on their operation before, have the opportunity to thoroughly deworm every animal before arrival, preventing these animals from contaminating the new place.

The eradication of parasites of grazing animals is not a practical proposition. Strategic deworming is designed to reduce parasite contamination throughout the grazing season. Fecal worm egg count monitoring is excellent here to help devise the correct treatment timing. Fecal checks during the winter months are an excellent time to make sure all animals are parasite-free. Fecal check during this spring is conducted to make sure animals are not shedding eggs during the early part of the grazing season. Fecal checks during the summer and fall are designed to provide information on whether the parasites are being controlled. A high worm egg count during this period indicates treatment failure and that a re-evaluation of what deworming product was used and when the treatment was given are both necessary.

Veterinarians routinely perform fecal examinations for cats and dogs but not for other species. Why not? Providing companion animal and food animal clients with up-to-date practical advice that will help them keep their animals healthy and improve the efficiency of their operations is the key to a successful practice. Conducting fecal examinations is a professional value-added service that provides scientific basis for diagnosis or treatment recommendations and can determine whether a particular treatment was successful. The service fecal exams provide separates the veterinarian advice from layman's suggestions. The veterinarian can help protect clients' profits and minimize their losses from parasite infections by building a long-term parasite-control strategy. An accurate and simple fecal examination(s) will help accomplish this goal.

Internal parasites are most small organisms that live, for at least part of their life, within a larger host animal, maintaining their survival at the expense of their host. Internal parasites have two main functions of life; one is to survive by finding and living off the host, while the second is to reproduce by excreting ova (eggs), cysts, oocysts, trophozoites, or larvae, which pass out of the host contaminating the host's environment which is necessary to sustain future generations of these parasites. Since detecting live parasites from within a host animal would require killing the host animal to recover and identify the parasites, the best way to detect parasitic infections is accomplished by finding eggs, oocysts, or larvae in the feces, urine, blood, or mucus of the infected animals. An adage that states "where there's smoke there's fire" is the same for internal parasites where it can be said that "when parasite eggs are found, adult parasites must be present."

What is the importance of parasite diagnosis? There are many reasons why providing an accurate parasite detection service has become very important to the veterinary field. One of the first reasons is that many of the pharmaceutical companies bringing new products or supporting existing products are spending millions of dollars on advertisements, thus creating an increased awareness to the producers or animal owners about the importance of controlling parasitic diseases. This awareness raises concerns by producers and pet owners about potential parasite problems in their animals and they are turning to the veterinary profession for help and advice. Secondly, the economic importance of treating internal parasites in domestic animals has gained increased emphasize in recent years as products to remove these infections have become highly efficient and the negative economic effects of parasitism has become more clearly established.

Improved production parameters through new technology for breeding, nutrition, and animal health presents a third reason for increased importance of accurate parasite detection. This increase in efficiency in animal production means that the need for parasite control also increases since parasitism is one of the greatest deterrents to efficient production. It often takes

fewer parasites to cause a problem in a highly efficient animal than a poor performing animal. A good example of this is that a high-producing lactating cow exposed to just a few hundred infective *Ostertagia* larvae (which invade the gastric glands causing an increase in abomasal pH and decreasing digestion efficiency, producing a subsequent drop in dry matter intake) can experience plummeting milk production. Low-producing cows, on the other hand, can often carry relatively high levels of parasites before negative effects on production can be detected. One of the reasons for this is that poor producing cows often have management, nutritional, or health problems that mask the negative effects of parasitism.

More accurate diagnosis leads to healthier animals: A more recent reason that parasite diagnosis is gaining important is that over the past 25 years, internal parasites have been shown to affect a multitude of economic parameters in domestic livestock, but now new data regarding the effect of parasites on the immune system show an even stronger effect than previously thought. These data demonstrate that parasites can cause a suppression of the immune system negatively affecting an animal's ability to fight off other diseases. Not only do the animals suffer directly from the presence of parasitic infections but also these infections allow other disease problems to become more significant. Having the ability to accurately diagnose the presence of parasitic infections is, therefore, very important to the overall health of an animal.

Parasite diagnosis and control are also a very important part of animal health because nearly all animals encounter parasites sometime in their life. Although parasite exposure is highly variable, depending upon environmental contamination, many of these animals are exposed to high levels of parasitism at various periods throughout their lives. Often, the types of parasites and the level of parasitic infections that develop vary with the age of the animal and are influenced by the animal's immune system, the environmental and management conditions they are raised under, and the level of parasite contamination present in the animal's environment. Parasite exposure can also be influenced by many other factors, including housing or pasture conditions, contamination history, stocking rate or degree of animal concentration, individual animal behavior activities, and weather. The failure to detect the presence of parasitism, whenever it occurs in an animal's life, can have serious economic ramifications on production parameters or serious emotional importance for those animals raised as pets or with other special attachment.

Parasite resistance found in cattle, equine, and small ruminants throughout the world increases the need for better diagnosis: Overall, probably the most important reason for providing sensitive and accurate testing for parasites by veterinary clinics all across the country is that parasite resistance has now become a widespread problem in nearly all species of animals. Knowing whether or not treatment is successful can only be done through post-treatment monitoring. Parasite resistance to dewormers has been known to occur in equine and small ruminants (sheep and goats) for several decades, but during the past few years it has now become a widespread problem in cattle. Monitoring treatment to ensure success can be worth millions of dollars to producers and animal owners throughout the United States. Identifying treatment failures allows follow-up treatments before the resistant population can propagate and cause serious problems [19–21]. Fecal worm egg reduction tests conducted over the past 20 years show widespread resistance (efficacy less than 90%) for macrocyclic lactone pour-ons and injectible products while the efficacy of multi-formulations of fenbendazole have to maintain a high level of efficacy during the same period of time (see Figures 1.61 and 1.62). The combination or concomitant use of macrocyclic lactone pour-ons and injectables with fenbendazole, on the other hand, have demonstrated a high efficacy value for this combination use (see Figure 1.63).

Diagnoses for liver flukes: There are two different types of liver flukes of veterinary importance that can infect cattle in the United States. These flukes are *Fasciola hepatica*, the common liver (bile duct) fluke of cattle and *Fascioloides magna*, the giant deer liver fluke. Both flukes are completely different from each other in terms of their distribution, their infection process, diagnosis, and economic importance. *Fasciola hepatica* is endemic, mostly only in coastal areas of the country, but

Trial Summary for Fecal Egg Count Reduction Tests Reported on the Merck National Data Base Conducted with FDA Approved Macrocyclic Lactone Products (Updated May, 2021).

			Egg Counts/3 g*		
Products	**No. of Trials**	**No. of Samples**	**Pre-Rx**	**Post-Rx**	**Percent Efficacy (%)**
Injections					
Ivomec® Inj.	25	1352	70.1	37.1	47.0
Ivomec® Plus	17	823	102.6	55.7	45.7
Dectomax® Inj.	44	1791	64.1	15.4	76.0
Cydectin Inj.	12	614	36.9	5.3	85.7
Ivermectin Inj.	13	630	90.0	45.6	48.3
Ivermectin Plus	5	193	97.5	48.6	50.1
Inj. summary:	116	5403	76.8	34.6	54.9
Pour-ons					
Ivomec® PO	21	823	61.8	27.0	56.3
Ivermectin PO	81	3378	62.6	29.2	53.4
Dectomax® PO	23	941	67.9	23.7	65.1
Cydectin® PO	25	1044	60.9	14.5	76.1
Eprinex®	5	224	38.1	25.8	32.2
PO summary	155	6410	58.3	24.0	58.8
Overall summary:	271	11,813	68.4	29.8	56.4

* All samples taken at Rx and again 2 weeks post-RX.

FIGURE 1.61 Trial summary for fecal egg count reduction tests for macrocyclic lactone pour-on and injectable products.

Trial Summary for Fecal Egg Count Reduction Test Reported on the Merck National Data Base Conducted with Various Formulations of fenbendazole (Safe-Guard® and Panacur® – Merck Animal Health).

			Egg Counts/3 g*		
Product	**No. of Trials**	**No. of Samples**	**Pre-Rx**	**Post-Rx**	**Percent Efficacy (%)**
Panacur® Drench	32	1296	59.3	0.7	98.8
Safe-Guard® Drench	88	3694	62.1	0.8	98.7
Summary Drench	120	5110	60.7	0.8	98.7
Safe-Guard® feed	29	1459	51.6	0.1	99.1
Safe-Guard® 1.96%	19	803	38.6	0.7	98.1
Safe-Guard® mineral	16	620	30.7	1.1	96.2
Safe-Guard® paste					
Blocks, liquid feed	20	835	38.1	1.6	95.8
Overall summary:	175	7516	53.9	0.7	98.7

* All samples taken at Rx and again 2 wks post-Rx.

FIGURE 1.62 Fecal worm egg reduction test showing efficacy of multi-formulations of fenbendazole (Safe-Guard® or Panacur®).

Trial Summary for Fecal Egg Count Reduction Test Reported on the Merck National Data Base Conducted with Safe-Guard®/Panacur® in Combination with Various Endecticide Formulations (Updated May, 2021).

Combination Product	No. of Trials*	No. of Samples*	Egg Counts/3 g		Percent Efficacy (%)
			Pre-Rx	Post-Rx	
Safe-Guard/Panacur Drench plus:					
Ivomec® PO/Inj./Plus	21	805	79.4	0.4	99.4
Ivermectin PO/Inj.	34	1424	81.3	1.1	98.6
Dectomax® PO/Inj.	7	263	97.9	0.1	99.8
Cydectin® Inj.		1 41	134.2	0.7	99.4
Cydectin® Pour-on	11	447	64.0	0.2	99.7
Combination summary	74	2980	91.4	0.5	99.4

*Updated May, 2021.

FIGURE 1.63 Fecal worm egg reduction test showing efficacy for combination treatments*.

can be found in some limited river valleys away from the coast or on irrigated pastures. *Fascioloides magna* has a more widespread prevalence found throughout some 25 states (mostly in the midsection and upper Midwest region of the country), wherever the natural host, the white-tailed deer, are prolific. The deer fluke can be found in the upper Great Lakes region, lower Mississippi and Southern Atlantic seaboard, the Gulf Coast, the Rocky Mountain trench, and Northern Quebec and Labrador. Both flukes depend upon the distribution of an intermediate host, the lymnaeid snail, thereby limiting where enzootic areas are located. Even though fluke-infected animals are often moved throughout the country, these infections will not propagate unless the intermediate host snails are present.

The amount of economic loss caused by liver flukes in cattle is not well defined because infections are seldom uniform throughout a herd and the level of infection (number of flukes) in a particular animal cannot be quantified. Conducting fecal fluke egg counts is time consuming, and even if eggs are found, there is no way to know how many animals in a particular group are infected unless all animals are tested and the level of infection is impossible to know without necropsy. Even though damage by liver flukes cannot be accurately quantified, the fact that these flukes invade and live in a vital organ, their overall importance is seldom questioned by the veterinary practitioner. The questions for most producers are "How do I know whether flukes present in my herd, and, if so, which type of flukes do my cattle have, what are the economic consequences, how do I control these flukes and is treatment economically justified?"

Recent pharmaceutical advertisements indicate that the common fluke, *F. hepatica*, is spreading across the United States and has become a problem everywhere; however, there are no published documentation indicating that this is occurring [23]. Several USDA reports on liver condemnations indicate an increase in livers being condemned since 1973, a time when no approved products were available to flukes, but do not indicate whether this increase is normal due to weather fluctuations, better inspection techniques, or due to ineffective treatment. Technical experts (from a corporate sponsor) on a recent TV show told a producer from Minnesota and one from Virginia that treating cattle for flukes in their area was very important, however, they forgot to tell the callers that the liver flukes found in Minnesota and Virginia are almost exclusively deer flukes (*F. magna*) and that their product was not approved for use to control this parasite.

I. *Fasciola hepatica*, the common liver fluke of cattle is found mainly in Florida, Louisiana, the gulf coast of Texas, parts of California, Hawaii, the coastal Pacific Northwest, and some river valleys and irrigated pasture in the Northwest as far east as Montana (see Figure 1.64). Although this fluke can infect some wildlife species such as deer and small ruminants such

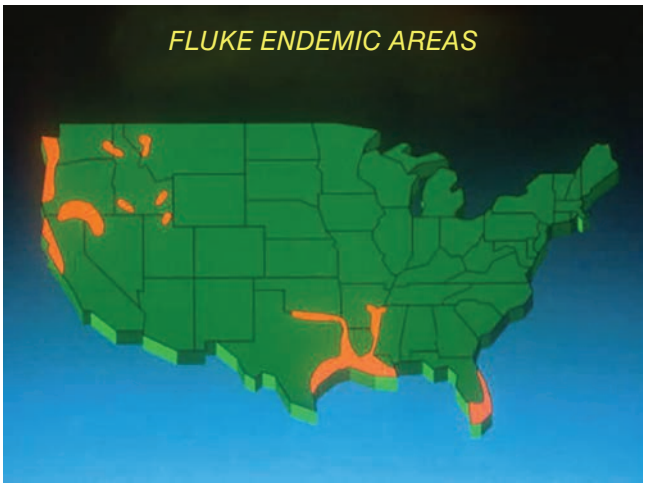

FIGURE 1.64 Map showing prevalence of the common liver fluke (*Fasciola hepatica*).

as sheep and goats, cattle are the main host and continued environmental contamination by cattle is required to perpetuate the infection.

A. Life cycle: Adult *F. hepatica* lives in the bile ducts of the liver and eggs are passed through the bile into the feces and back into the environment. When these eggs are deposited in warm moist environment, they hatch and develop into free-swimming organisms (miracidium) which invade a particular type of snail (*Lymnaea*) for further development. Once infected snails shed a tadpole-like organism (cercaria) which then migrates onto green plants where they form a protective cyst (metacercaria) and are consumed by grazing animals. Pastures showing ideal conditions for liver flukes survival on pastures requires moisture for a large part of the season (see Figure 1.65). The cysts will

FIGURE 1.65 Pasture showing ideal conditions for liver flukes if *Lymnaea* snails are present.

die off after the water recedes and hot dry summer conditions arrive since these cysts are very susceptible to dry conditions. Snails survive cold winters or hot dry periods by burying themselves in the mud and waiting until favorable conditions return. Infected snails often release the cercaria in the spring when they emerge from the mud as temperatures begin to warm up and spring rains bring moisture which helps the infection process. Moving fluke-infected cattle to feedlots or areas of the country where lymnaeid snails are not present to complete the life cycle does not spread the infection. The yearly transmission cycle for *F. hepatic* on pastures is shown in Figure 1.66.

B. **Ecological requirements:** High rainfall areas, irrigation, or wet lands are required for fluke transmission and for the survivability of the intermediate snail host. Light loam or sandy soil is not conducive for snail survivability: therefore, the presence of liver flukes in Gulf States like Mississippi and Alabama is limited or nonexistent. Also, snails cannot survive in acid soils such as peat soil but prefer neutral, well-buffered heavy clay soils such as those found in Louisiana. Snails survive in shallow depressions in fields, springs, seeps, and slough that hold water over 180 days per year. Sustained heat and summer droughts end infection season.

C. **Prevalence:** Prevalence is relative low nationwide according to surveys conducted with feedlot cattle demonstrating a 5% condemnation rate throughout the United States [24, 25]. Surveys conducted with beef cattle operations demonstrated 19.2% (range 5.9–52.7%) prevalence in fluke endemic states as listed above but a much lower rate when prevalence is calculated with states where *F. hepatica* numbers are low or nonexistent [4]. The number of flukes per infected liver was not determined in this study. Recent advertisements indicate liver fluke prevalence is on the rise, spread by moving hay from infested to un-infested areas, but this claim is not substantiated and highly unlikely since metacercaria are killed during the drying process. Furthermore, if this statement is true, it would indicate that current approved products despite extensive use are not sufficiently effective to impact the prevalence of this parasite.

D. **Economics:** The economic threshold of liver fluke infections in terms of fluke burden and stage of infection from the common fluke has not been established. One study indicated economic loss occurred in cattle when a mean fluke count of 60.3 flukes per liver

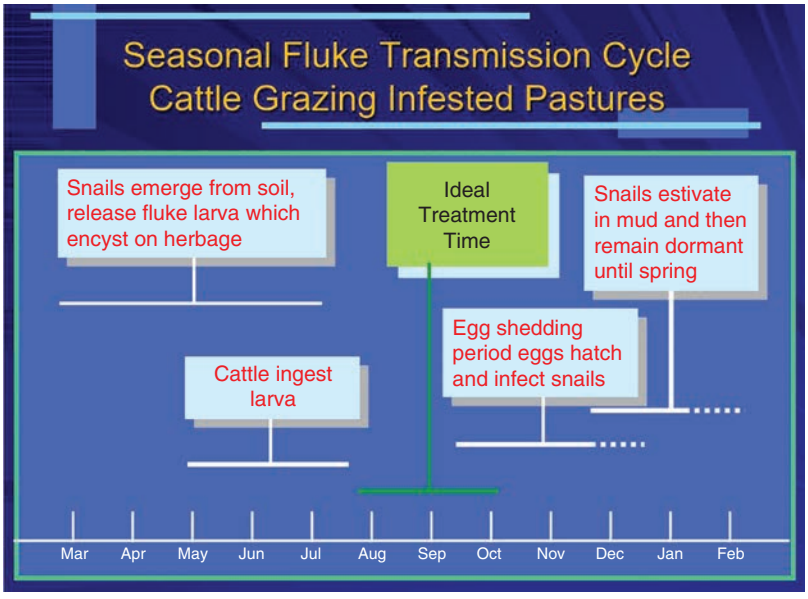

FIGURE 1.66 Fluke (*Fasciola hepatica*) yearly transmission cycle on pasture.

was found while clinical conditions developed when a mean fluke count of 171.2 flukes per liver was found [23]. The problem is that it is impossible to determine when fluke numbers are high enough to be a problem. In feedlots, the success from treatment for flukes in arrival cattle is confounded both by the age of the flukes upon arrival and recovery time of the liver following treatment even if treatment is successful. If the flukes are immature when treatment is given, these flukes will be missed by treatment and continue to develop and livers will be condemned at slaughter. Even if all flukes are killed, but the animals are sent to slaughter before livers have regenerated from previous fluke damage, the livers will be condemned despite treatment. In both cases, the economic loss due to liver condemnation is equal.

A study conducted at Louisiana State University with fluke-infected feedlot calves comparing albendazole (Valbazen®-Pfizer) and thiabendazole demonstrated better gains for albendazole [26]. The study was somewhat compromised because albendazole is a more efficient dewormer than thiabendazole as well as demonstrating some fluke efficacy. Two further studies conducted in 2001 and 2002 showed no benefit in treating flukes with ivermectin plus clorsulon @ 2 mg/kg (Ivomec® Plus – Merial) while using a full dose of clorsulon @ 7 mg/kg (Curatrem® – Merial), an economic benefit was realized [27, 28]. In the first study, young calves exposed to liver fluke-infested pastures gained significantly better when treated with doramectin injectable (Dectomax® – Pfizer) compared to a combination of ivermectin and clorsulon @ 2 mg/kg (Ivomec Plus-Merial). In the second (four-year) study, body condition score, weight gain, and pregnancy rates for heifers grazing fluke-infested pastures treated for gastrointestinal nematodes alone (injectable endecticide), flukes alone (clorsulon @ 7 mg/kg), both nematodes (injectable endecticide) and flukes (clorsulon @ 7 mg/kg) versus non-treated controls demonstrated significant improvement for those heifers treated with full-dose clorsulon (7 mg/kg) plus endecticide versus endecticide alone. Heifers treated for flukes alone did not have significantly higher pregnancy rate than untreated control heifers or heifers treated only for gastrointestinal nematodes.

E. Diagnosis: Finding *F. hepatica* eggs in fecal samples. Checking manure samples for *F. hepatica* eggs is just an extension of the Modified Wisconsin Sugar Flotation method. The residue in the bottom of centrifuge tube contains fluke eggs if any are present. To find any fluke eggs that are present, simply mix the residue with tap water and pour through the Fluke Finder and any fluke egg present will be on the final screen (see Figure 1.67).

FIGURE 1.67 Commercial Fluke Finder with filtration device.
Source: FLUKEFINDER.

These eggs are identified (see Figures 1.68 and 1.69), counted, and reported as the number of fluke eggs present in a 3 g sample (see Chapter 2).

F. Treatment: For successful treatment of *F. hepatica* in endemic areas of the country, the flukes need to be sufficiently developed for the clorsulon (at 7 mg/kg) to work (>56 days old); therefore, strategic treatment for flukes should be given as soon as a majority of the invading flukes are sufficiently mature to be killed by the flukicide of choice, but before the flukes are mature enough to begin laying eggs back onto the pasture. With albendazole or ivermectin plus clorsulon (at 2 mg/kg), the flukes need to be mature (>90 days old) for treatment to be effective. Freedom of Information (FOI) studies indicated that in 100% of the studies reported, fluke eggs were found following treatment with albendazole. Since mature flukes are laying eggs back into the environment, treatment with these two products need to be given later in the year and most likely will not prevent pasture recontamination. Most transmission takes place in the spring when moisture

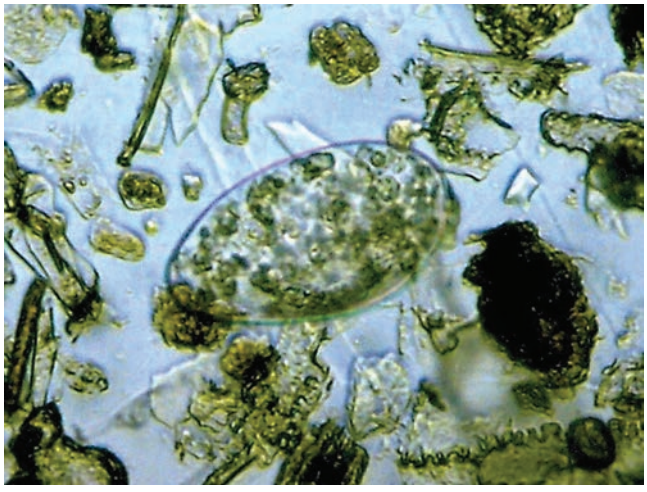

FIGURE 1.68 Fluke (*Fasciola hepatica*) egg found in fecal sample (40×).

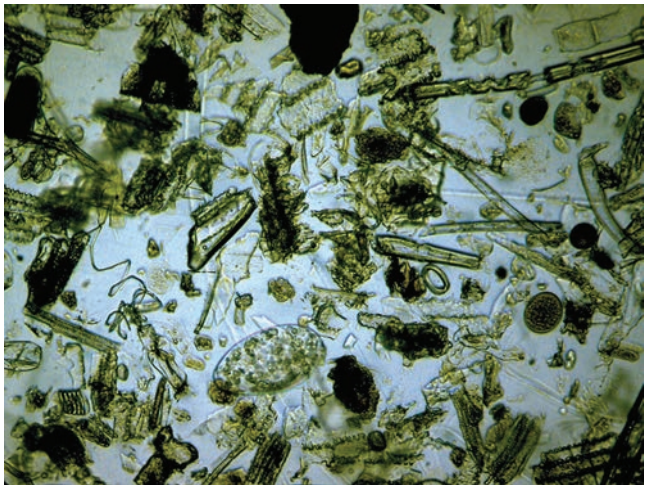

FIGURE 1.69 Fluke (*Fasciola hepatica*) egg found in fecal sample (10×).

levels are at the highest and before temperatures turn hot; so, in southern coastal states where flukes are present, depending upon spring moisture, flukes can mature by early to late July whereas in the Pacific Northwest, maturity usually takes place later. Most producers, however, apply fluke treatment in late fall or early winter after pasture recontamination has already taken place.

Fluke treatment for the common fluke, *F. hepatica*, is best applied where the infection occurs on pasture. Clorsulon (at 7 mg/kg) is currently the only effective treatment based on production trials. Ivermectin plus clorsulon (at 2 mg/kg) was shown to be no more effective than doramectin alone with fluke-infected animals [28]. Ideal treatment should be given to herds with greater than 25% prevalence levels and with greater than 40 flukes per animal. Treatment timing should occur just prior to maturation of the migrating fluke to adults.

Treatment for flukes in the feedlot is difficult because it is nearly impossible to know which cattle need treatment because flukes may not be mature. Also, cattle coming from fluke endemic parts of the country may not have sufficient level of infection to warrant treatment. Some of these cattle may have already received fluke treatment. Treating cattle upon arrival in the feedlot is usually too late to prevent damage to the livers and production loss in the animals since the flukes were consumed on the pasture and most likely the flukes have already completed the migration phase by the time the cattle arrive in the feedlot. Also, treating cattle for flukes on arrival in the feedlot does not prevent liver damage unless treatment is effective against all stages of the parasite [28, 29].

II. *Fascioloides magna*, the liver fluke of deer which can infect cattle and other domestic species such as sheep and goats. Deer are the natural host for this parasite. The flukes are flat, elongated worms found while slicing the liver usually surrounded by a fibrous capsule.

A. Prevalence: *Fascioloides magna* occurs throughout the United States, but is mostly found in the Great Lakes area where deer populations are high and the necessary snail intermediate host is present to keep the infections going. In cattle, the encapsulation prevents eggs from escaping in the liver and therefore fecal checks for deer fluke eggs is not possible.

B. Economics: There are no economic data demonstrating losses in cattle due to the deer fluke other than the cost of liver condemnation at slaughter. A field study reported from the Michigan State University Veterinary Extension under the Michigan Beef Improvement program demonstrates excellent slaughter results in heifer calves born and raised in Northern Michigan with nearly 100% of the livers infested with *F. magna* liver flukes [30]. It is assumed that liver damage in cattle has to be extensive (>90%) before clinical disease is observed. This parasite, however, is lethal in sheep and goats since the parasite is not encapsulated and continues migration through the liver until the organ is destroyed and the animal succumbs.

C. Treatment: There is no approved treatment for *F. magna* in deer or cattle. High doses of albendazole and clorsulon showed some activity but albendazole levels (20–46 mg/kg) necessary for even marginal efficacy were dangerously close to lethal levels (>4.5 times recommended dose).

Treatment for the deer fluke, *F. magna*, in cattle is currently not available and based on recent production studies probably unwarranted [29]. Limiting cattle's access to wet areas of a pasture by fencing off the creeks and areas where standing water may be present for a major portion of the summer may be the only way help reduce exposure to the deer fluke.

Dung beetles: Many of the anthelmintics (dewormers) on the market today may have a negative impact on dung beetles. The residue of a number of anthelmintic products passed in fecal material following treatment have been shown to have an impact on the natural

development of parasite fauna in the fecal pats excreted by cattle. This impact may range from destroying fly larvae and the development of these flies to the inhibition of eggs and larval stages of the dung beetle. Most experts agree that the destruction of fly larvae is a good thing; however, the destruction of the eggs or development stages of the dung beetle may not be as universally acceptable for a number of reasons outlined below [31, 32].

The anthelmintic products that have been determined to have a detrimental effect on the dung beetle fauna are ivermectin, doramectin, and eprinomectin [33]. No differences were observed between the injection and pour-on formulations [31]. These avermectins showed larval mortality, mortality of immature adults, and reduced egg production for periods up to one month following treatment. In experiments performed under temperate and tropical conditions, the aging of the dung pat did not lead to significant lowering of the concentrations of ivermectin [32]. There are approximately six months of the year when treatment of cattle with avermectins would affect mortality of newly emerged dung beetles and three months of the year when avermectin would affect dung beetle oviposition or larval survival [34–36].

Fenbendazole, albendazole, and moxidectin have shown no effect on the dung beetle or its offspring [33, 37]. Even when fenbendazole was administered in a sustained release bolus, no detrimental effect on dung beetles was observed. At 42-days post-treatment, the solid matter of the control and fenbendazole-containing cow pats were reduced to crumbling, granular texture, while the cow pats from the ivermectin-treated animals were solid and compacted.

The dung beetle (see Figure 1.70) has been identified as environmental aid for the degradation of the fecal pat, which provides the re-fertilization of the pastures and aids in the natural destruction of infective parasitic larvae. Recent research has demonstrated that the dung beetle is

FIGURE 1.70 Heavy infestation of dung beetles on a single manure pat.

responsible for the natural destruction of infective larvae present in the fecal pat (see Figures 1.71 and 1.72), which develop from eggs passed from animals infected with gastrointestinal parasites. It is easy to spot pastures where ivermectin products have be administered to animals and intact fecal pats are visible. This is especially true of horse pastures found throughout the United States

FIGURE 1.71 Intact manure pats seen in Oregon.

FIGURE 1.72 Intact manure pats on horse pasture in Wisconsin.

(see Figure 1.73). A number of studies have indicated that dung beetles naturally destroy from 60% to 80% of these larvae in any given fecal pat (see Figure 1.74). This may turn out to be an extremely important event that researchers have only just discovered since destruction of the dung beetle could lead to higher levels of parasite contamination on pastures grazed by avermectin-treated cattle [34, 37, 38].

Veterinary clinics are often involved in many situations where the early detection of potential parasite problems allows the clinics to alter or adjust their recommendations to prevent or otherwise curtail the development of a problem in the animals being treated. The degree of science that accurate parasite diagnosis by a veterinary clinic provides is hard to quantify but by simply having the knowledge and ability to detect certain parasites, these clinics can often prevent a high level of economic loss or unnecessary suffering to their clients' animals.

Books on clinical parasitology written for veterinarians describing the biology and treatment of parasites are numerous; however, a book specifically written for veterinarian technicians containing up-to-date information describing the newest and best techniques leading to the diagnosis of both clinical and subclinical parasitic infections is lacking. Such a book for veterinary

FIGURE 1.73 Fresh fecal pat in West Texas with active dung beetles.

FIGURE 1.74 A second species of dung beetles found in fresh manure pat in West Texas.

technicians is especially important to help livestock producers strive for maximum efficiency for their animals and informed pet owners strive to protect their animals from as many harmful parasites as possible. Furthermore, many of these harmful parasitic infections require highly sensitive laboratory diagnosis in order to detect their existence. The goal of this book on parasitology, written for veterinary technicians, is to ensure veterinary clinics all across the country have the best, most sensitive, and specific laboratory tests to detect both clinical and subclinical economically important parasitism when and wherever they occur in both domestic animals and wildlife. We want to provide the best science available for veterinary clinics and veterinary diagnostic laboratories to help diagnose and treat parasitisms.

In summary, gastrointestinal parasites include a wide range of economically important organisms that infect nearly all domestic animals and wildlife sometime in their life. These parasites develop natural infections in animals depending upon environmental contamination causing anywhere from minor to major health problems which can even lead to death by the host animal if the infection becomes overwhelming. One of the keys to successful treatment is early detection; however, when the parasites exist in a subclinical state, detection can be a significant problem and economic losses or physical suffering can occur without the knowledge of its owner or the producer raising the infected animals. Veterinarians play a key role in diagnosing and prescribing treatment to control or prevent damage caused by these organisms. The first step in this process, however, is for veterinary technicians to know and have available the best and most sensitive technique in order to find and identify these organisms so proper treatment can be prescribed by the attending veterinarian.

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