Parasitologists estimate that internal parasites cost American cattle producers more than $200 million each year. If subclinical losses could be measured the total loss would be much greater.

If cattle producers are not controlling internal parasites they are probably losing $10 to $40 per cow each year. Researchers estimate that 85 percent of all cattle are infected with brown stomach worms, and 67 percent are infected with large stomach worms (Table 1). Not surprising then, that research shows using dewormers can be highly profitable.

Cows that were dewormed systematically were 25 pounds heavier coming off summer pasture and bred back 6 to 9 days earlier. Infected calves that were dewormed while nursing weaned from 12 to 38 pounds heavier than controls. Stocker cattle receiving dewormers showed similar weight advantages, and improvements in feed conversion and average daily gain were shown in fed cattle. Achieving these results takes planning.

Most cattle producers recognize that internal parasite infestation can damage their livestock. Treatment with one or several dewormers is commonly applied. Too often deworming occurs when it is convenient or when the cattle are being handled. Producers must realize that such treatment is being applied after the cattle have been infected and the damage caused by internal parasites has occurred.

**Internal Parasite Epidemiology**

Internal parasite worm eggs are shed on the pasture or range by infected cows and calves. The eggs survive cold weather and drought, subsequently hatching when warm, moist conditions exist. Microscopic larvae are then dispersed, with some trickling deep into the soil and others carried onto surrounding grass. The free-living larvae feed on fungi and other soil and grass microorganisms and represent pasture contamination.

When these larvae have developed to an infective stage, they may be found in dewdrops on blades of grass early in the morning and seasonally in late spring and late summer/early fall. Cattle become infected while grazing. Upon passage of grass to the stomach, infective larvae penetrate into gastric glands. Larvae emerge from the glands as adults and the life cycle is complete.

Infected larvae that are picked up in March and April in southern climates often will remain in arrested development or as inhibited larvae for 4 to 5 months, effectively over summer until late August to October. These larvae then rapidly increase in size, emerge in large numbers, and cause massive destruction to the gastric glands, bringing stomach function to a virtual halt. The host cow or calf becomes clinically ill with anemia, accumulation of body fluid (edema) often seen as “bottle jaw,” and loss of appetite.

Some cattle are overwhelmed and die; others are set back for several months before adequate stomach repair can occur. In northern climates overwintering of inhibited larvae occurs with fall pickup of “arrest-prone” infective larvae followed by late winter (February-March) clinical ostertagiosis.

Thus, grazing cattle are infected, or re-infected, with internal parasites by ingestion of larvae that develops from hatching worm eggs. The larvae develop into adult worms in 3 to 6 weeks, and the female worms begin shedding additional eggs onto the pasture. Thus, cattle grazing pastures or rangelands contaminated with parasite eggs are infected and continue to shed eggs during the grazing season.

Pasture contamination levels increase during the grazing season, which exposes cattle to increasing levels of infection. The result is sub-clinical parasitism and, in some cases, clinical parasitism.

Parasitic infections are often more obvious in young animals than in older animals. However, older animals