WORM CONTROL IN DAIRY CATTLE

by

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Florida
Dairy Check-Off

Worm infection, or more properly parasitic gastroenteritis (PGE), is a condition characterized by weight loss/poor growth, loss of appetite, diarrhea, bottlejaw, and anemia in heifers during their first season at grass. PGE is caused primarily by the Brown Stomach Worm (*Ostertagia ostertagi*), the Barber's Pole Worm (*Haemonchus placei*), the bankruptworm (*Trichostrongylus axei*), and several species of Cooper's Worms (*Cooperia oncophora, Cooperia punctata* and others).

Seasonal patterns of parasitism

Northern U.S.: As a general rule, worms are a summer/fall problem in the northern and midwestern states. Worm eggs shed on pasture in spring develop to infective larvae by July and high levels of pasture infectivity persist through the fall. Overwintering is a problem for these parasites. *Haemonchus*, *Ostertagia*, and *Cooperia* are short-lived as adult worms (30-50 days), and they survive the winter as dormant ("arrested", "hypobiotic", or "inhibited") larvae in the host that will mature and commence egg laying in the spring. The onset of cold weather in the fall "conditions" the infective larvae on pasture to undergo arrest when ingested rather than develop straightaway to egg-laying adult worms (normally an 18-21 day process). Knowledge of the timing of this process is important since arrested larvae are resistant to most anthelmintics.

Southeastern U.S.: In the deep south things are a bit more complicated. Extremely hot summers will destroy infective larvae of most worm species (*Haemonchus* is a notable exception), so parasitism is primarily a winter/spring problem. This is further exacerbated in beef cattle by the nutritional stress that must be endured each winter (causes loss of acquired immunity to worms). In general eggs shed in the fall and winter develop into infective larvae in late winter/early spring. Infection and clinical signs appear shortly thereafter. Arrested development of larvae occurs during late spring and summer. In the fall arrested larvae resume their winter/spring gastroenteritis in southeastern cattle, outbreaks have also occurred in replacement heifers grazing highly improved summer pastures at a high stocking rate.

Western U.S.: In the west, worm transmission is influences as much by moisture as by temperature. Very little worm infection occurs in dry western rangeland where cattle do not graze irrigated pastures or moist river bottoms. Worm transmission is further confounded by the effects of altitude. At the risk of over simplification, one may assume that where pastures in the West are reasonably moist, whether by rainfall or irrigation, seasonal patterns of worm transmission follow that of the southeastern U.S. in areas having warm winters and that of the northern U.S. in areas where winters are cold.

Occasionally disease is seen at atypical times of the year. A condition known as **type II ostertagiosis** occurs when massive numbers of arrested larvae of *Ostertagia* simultaneously resume their development. (In contrast, **type I ostertagiosis** occurs when newly ingested larvae develop straight to adult worms in large numbers - the usual situation in late winter/early spring in the deep south).

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Treatment of PGE

Animals showing typical signs of type I disease should be dewormed and, if at all possible, moved to a worm-free pasture. Failure to move the animals may result in prompt reinfection and a return of clinical signs. Under these conditions retreatment at 3 or 5 (ivermectin) week intervals may be needed, especially with haemonchosis in small ruminants. When they again become available for cattle, a sustained release bolus would be helpful in this situation. For type II disease the treatment of choice is ivermectin. A move to safe pasture is usually not needed.

Prevention of PGE

Although routine deworming of lactating dairy cattle at calving has been widely promoted by pharmaceutical companies, not all properly controlled studies have shown a significant response to treatment. In contrast, dairy replacement heifers in their first season at grass are highly susceptible to parasitism, and prophylactic treatment yields big returns. In northern states heifers are best treated at 3 and 6 weeks after turn-out to spring pasture (3 and 5 weeks if ivermectin is used), or a sustained release bolus is given. This prevents the shedding of eggs onto spring and early summer pasture that develop into the big July rise in L_3 on pasture. An alternative commonly used in Europe is to treat cattle in July and move them to a safe pasture (usually one used for spring haying). No comparable programs have been worked out for the deep south. In general, replacement heifers in the southeast should be aggressively dewormed (repeated treatments or sustained release boluses) during the early part of their first season at grass.

Lungworms

Lungworms occasionally cause trouble in cattle, but disease is much less common now since the introduction of highly effective anthelmintics during the 1970s. Like *O. ostertagi* lungworm appears to be a cool season parasite in the deep south and a warm season parasite in the northern states. Since there is a rapidly acquired immunity to this parasite generally only young animals are affected. Calves given early, low-level exposure to lungworms normally acquire a strong immunity and never develop lungworm disease. However, if calves are exposed to large numbers of infective larvae on pasture or if the level of pasture contamination increases from a previous low level so rapidly that acquired immunity cannot keep pace, lungworm disease will occur. A typical history is that of young cattle grazing a pasture grazed earlier by young animals as commonly occurs with dairy replacement rearing. The first group contaminated the pastures with larvae, but acquired immunity before disease appeared. The second group received a massive exposure before immunity could be acquired. Alternatively, the mixing recently weaned calves from several sources may expose naive animals to larvae shed by "carriers". This is seen in some beef stocker or backgrounding operations.

Lungworm is effectively treated with ivermectin, levamisole, or any broad-spectrum benzimidazole, but not by thiabendazole or morantel. A repeat treatment may not be necessary since immunity is rapidly acquired, but animals should be moved to a new pasture if treating an outbreak of disease. Lungworm disease can be prevented by treatment of all mixed-source heifers before turnout to pasture to eliminate "carriers". Sustained release boluses or repeated treatments with conventional anthelmintics may be useful as well.