

Cattle Gutworms - the facts

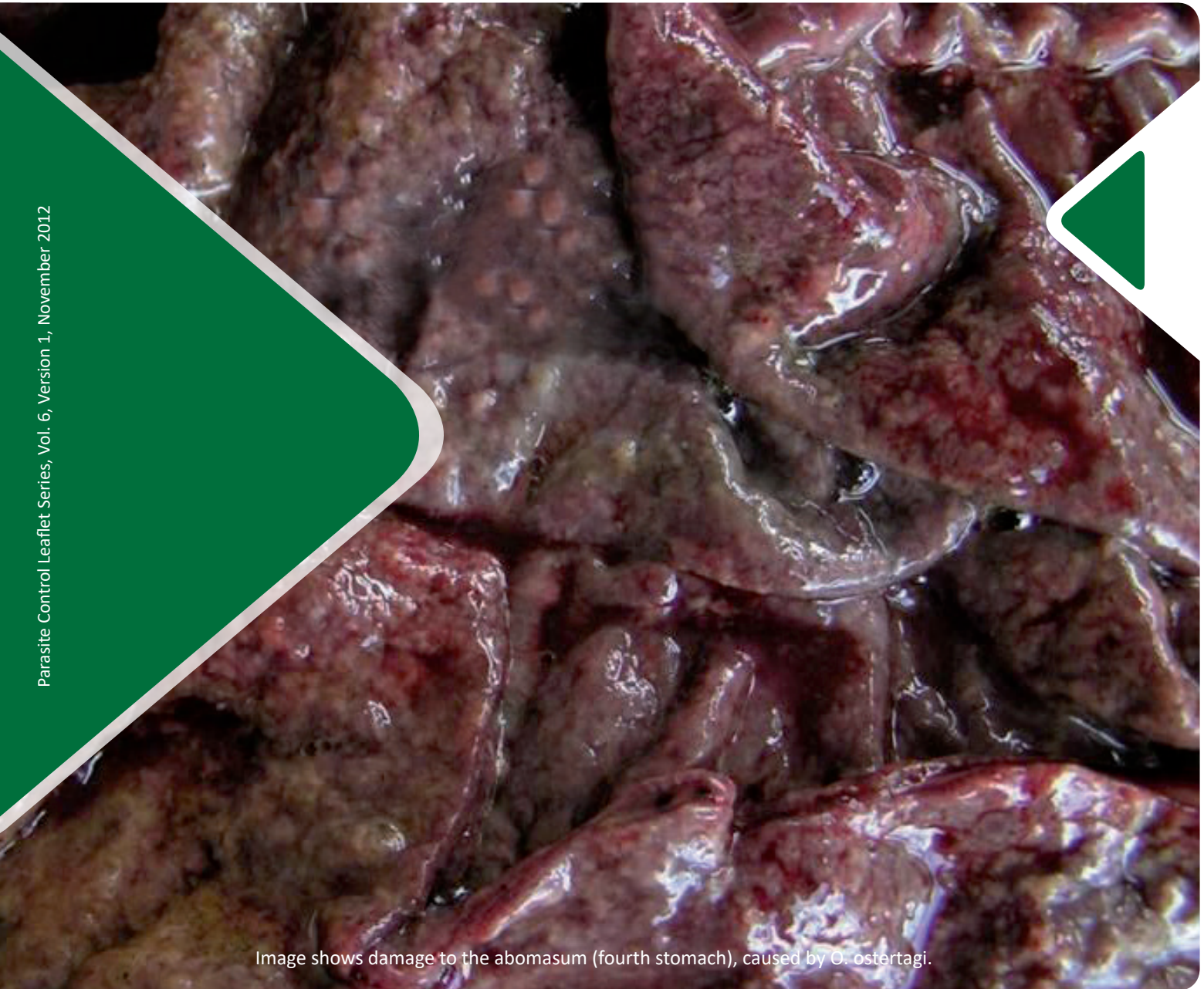


Image shows damage to the abomasum (fourth stomach), caused by *O. ostertagi*.

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PARASITE CONTROL PROGRAMME



Parasite Control
Animal Health Ireland.ie

Animal Health Ireland, 4-5 The Archways, Carrick-on-Shannon, Co. Leitrim, N41 WN27

AHI gratefully acknowledges the financial and other contributions of our stakeholders.



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Introduction

There are approximately 20 species of worms, known collectively as gutworms, that live in the intestines and stomach of cattle. These worms cause the condition known as parasitic gastroenteritis (PGE), which is common in cattle (and sheep) of all ages and can result in poor thrive and liveweight gain, ill health, poor production and financial losses. The two most economically important species of worms in Ireland are *Cooperia oncophora* and *Ostertagia ostertagi*.

C. oncophora

- It lives in the small intestine.
- It is the main contributor to faecal egg counts, certainly up to mid-summer of the first grazing season (FGS).
- Animals appear to develop an immune response to this parasite; consequently both intestinal worm burdens and faecal egg counts tend to decline towards the end of the FGS and remain low subsequently.
- This parasite can worsen the effect of *O. ostertagi*, particularly in young calves.

O. ostertagi

- It lives in the fourth stomach (abomasum).
- Like *Cooperia*, infections can be acquired from turnout in the Spring by ingesting over-wintered larvae on grass.
- Immunity to *Ostertagia* takes longer to develop than in the case of *Cooperia* and animals are not normally considered to be immune until they have completed two full grazing seasons.
- Adult animals are commonly infected with *O. ostertagi*, though the worm burdens are typically lower than in calves.

The importance of parasitic gastroenteritis (PGE) in cattle

Young animals (particularly calves) may develop severe clinical infections that result in a reduction in feed intake, diarrhoea and rapid loss of condition. However, more commonly, PGE is present subclinically, which means that it may not be easily detected and that farmers and veterinary practitioners may therefore be unaware of the associated production losses.

Gutworm-related production losses are often due to a loss of appetite from a sub-clinical PGE, **which means that animals simply do not eat enough to support higher levels of production**. Table 1 highlights the negative effects that subclinical PGE can have on different animal classes. Monitoring animals performance throughout the season (weight gain and production) and using suitable diagnostic tests (Table 2) is essential to establish a strategy for control of gutworms.

Table 1. Production Losses Associated with Sub-clinical Parasitic Gastroenteritis

Animal type	Effects on production
First grazing season (FGS) dairy calves	Reduced growth rate
Housed cattle	Lower Feed Conversion Efficiency
Second grazing season cattle	Reduced growth rate
	Delay in puberty & reduced conception rates in replacement heifers
	Reduced in-calf rate in heifers
	Loss of carcass yield and quality
Dairy cows and first calved dairy heifers	Decreased milk yield
	Longer calving to conception interval
	Decreased in-calf rates
Beef cows and first calved beef heifers	Decreased milk yield
	Longer calving to conception interval
	Lower calf weaning weight
	Decreased in-calf rates

Life cycle

The lifecycle of these worms can be considered in two phases - inside the host animal and outside. Figure 1 shows the whole life cycle. Figure 2 highlights the stages of the parasite development within the host animal, and Figure 3 focuses on the development stages outside of the animal.

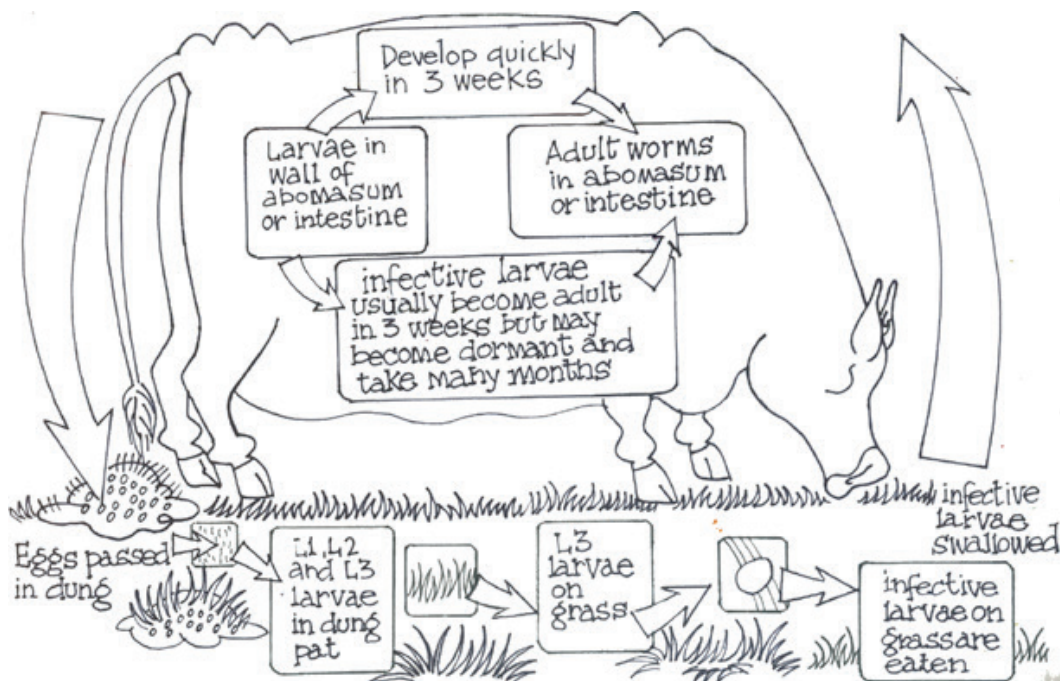


Figure 1: Gutworm life cycle

The parasitic stages inside the animal

After the infective L3 larvae have been ingested with grass, they lose their outer covering (exsheath) in the rumen, then move to their chosen sites, which for *Ostertagia* are the glands of the abomasum, and for *Cooperia*, the tissue of the small intestine. There, the worms develop and emerge as adults in a period of 15-18 days in the case of *C. oncophora* and 18-21 days in the case of *O.ostertagi* after which they start laying eggs (Figure 2). Generally for planning worm control programmes and monitoring the effect of these through dung sampling, a three week interval between initial infection and the appearance of eggs in the dung can be expected.

A change in the standard life cycle in *Ostertagia* occurs in the autumn and over winter. Larvae acquired during the latter part of the grazing season, instead of proceeding through the normal development to adult worms, enter a period of hibernation in the gutwall. Development resumes after several months, towards the end of the winter, and the emergence of the larvae from the gutwall at this time can result in severe clinical disease in some animals - a condition known as type II Ostertagiosis. The risk of this disease can be virtually eliminated through the administration of an effective anthelmintic (wormer) at housing (see Table 3).

Figure 2. *Ostertagia ostertagi* Life Cycle; Transmission and Parasitic stages



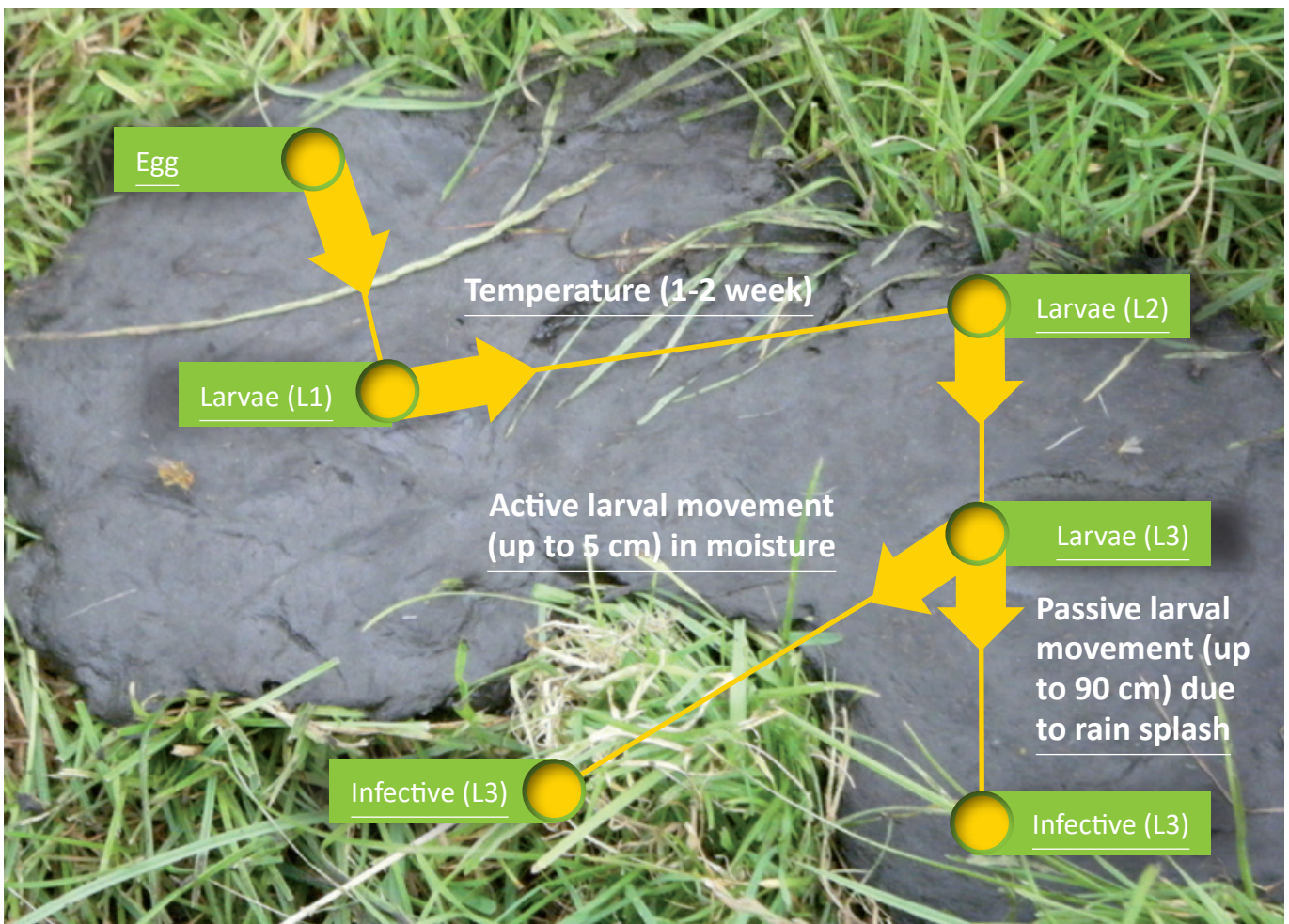
The parasitic stages outside the animal

Worm eggs are excreted in the dung, hatch and then undergo further development within the dung pat (Figure 3). The free-living stages of both *O. ostertagi* and *C. oncophora* are very similar. Hatching and larval development are mainly dependent on temperature, although moisture is also required as these stages are susceptible to drying out. It can take up to 3 weeks for the eggs to develop to infective larvae under field conditions over the period of April to October, while development during the winter and early spring takes longer. The larvae are hardy and able to withstand environmental and temperature fluctuations which allows them to survive in the soil in the long term. *O. ostertagi* and *C. oncophora* L3 larvae can be recovered from pasture up to two years after deposition in the dung, although their numbers are greatly reduced after one year.

Studies have shown that most larvae move no more than five centimetres on grass, with the majority of larvae being found in the lower five centimetres of the sward. However, because grass heights can commonly range between five and eight centimetres, there is plenty of opportunity for stock to ingest infective larvae, particularly if they graze close when grazing pressure is high.

Rainfall is important in facilitating movement of larvae away from dung and onto pasture. An initial wetting and softening of the dry crust, which typically forms on dung pats, is followed by the infective larvae being splashed out in droplets. This can account for 90% of the movement of larvae from the pat to the pasture and larvae can be found up to 90 cm from the pat. Spread of larvae beyond this range takes place passively through water flow and transport hosts, including earthworms, insects, birds and cattle themselves (viable infective L3 larvae can be found in samples of encrusted faeces on the feet and limbs of grazing cattle).

Figure 3. The worm larvae that develop in the dung are a source of infective larvae for grazing animals

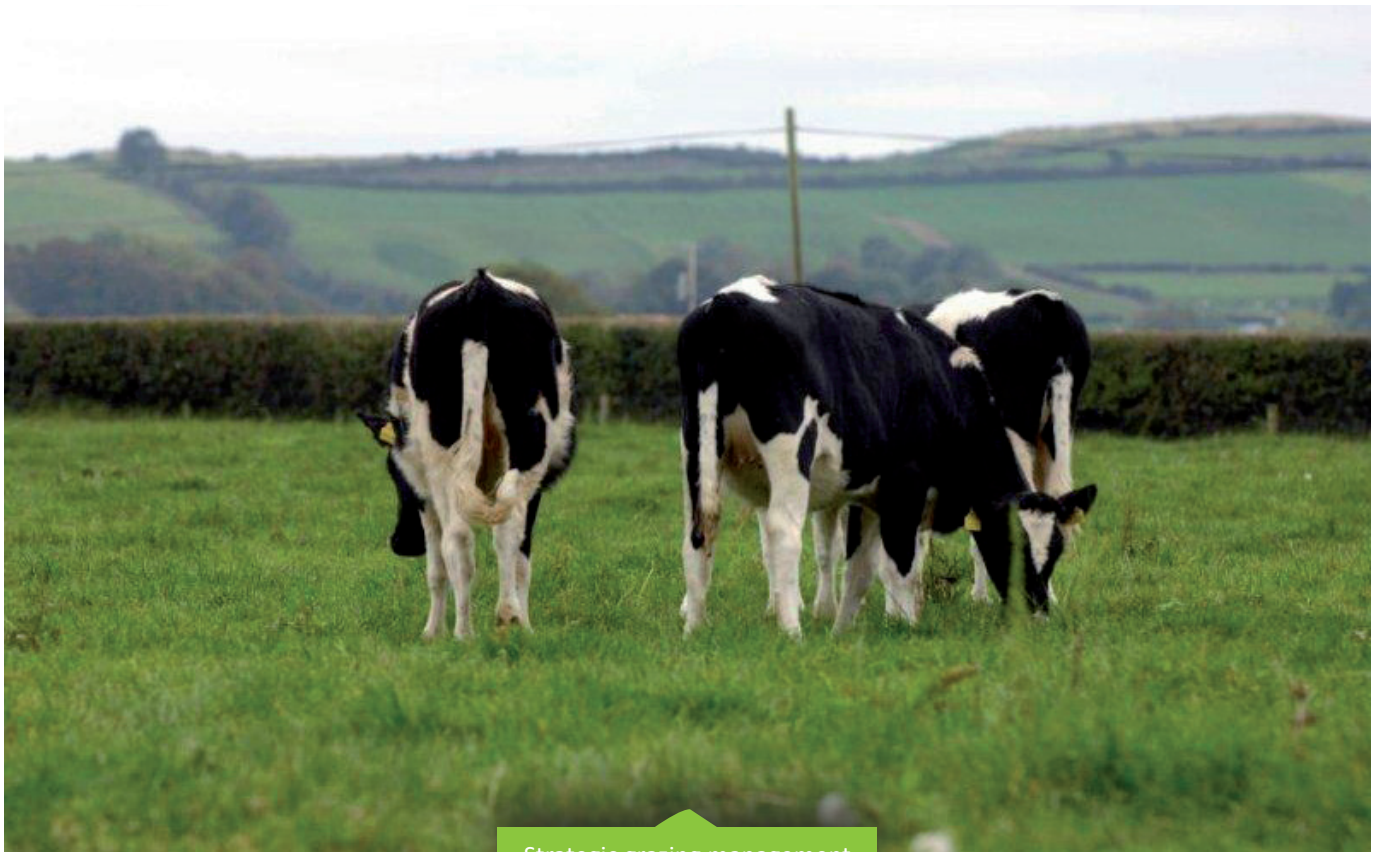


Management Systems

The Irish spring-calving pattern and extended grazing seasons determine the patterns of worm infection and the control options that can be used. For control programmes, different approaches are taken with first grazing season and second grazing season animals. Detailed information on the treatment and control of stomach worms is available within the AHI Parasite Control Leaflet Series 'A Guide to Parasite Control at Housing', 'A Guide to Parasite Control at Turn-Out' and 'A Guide to Parasite Control at Grazing'. These leaflets are available on the AHI website www.animalhealthireland.ie.

Dairy Farms

Larvae that have survived over winter in grass may cause disease in cattle following turn-out in springtime. At this time, the numbers of larvae will have declined from the levels present at housing the previous autumn, but because the fields may have been ungrazed by cattle for a relatively short time (2 to 3 months), over-wintered larval survival may be relatively high (compared to systems when cattle are housed for up to 6 months over winter). Hence, calves and older animals may be exposed to infection early in the year. If uncontrolled, ingested worms will go on to complete their life cycle, produce more eggs and further contaminate the pasture in a cyclical fashion, so that after a couple of months, pastures can be highly contaminated and the grazing animals consequently at risk of high levels of infection. Newly re-seeded pastures or aftergrass that have had no cattle for at least 6 months should pose



Strategic grazing management
will help in gutworm control on
dairy farms

Beef Farms

In spring-calving beef breeding herds the cows are typically immune and excrete low concentrations (<100 eggs per gram), of worm eggs in their faeces. Nevertheless, because of the large amount of dung (25-30 kg freshweight) produced by adult cattle, they can contribute significantly to pasture contamination, by adding to already present larval populations (which have survived over winter) already present in the grass.

The young beef suckler calf is fully susceptible to these pasture dwelling gutworm larval stages from birth. However, as its diet is mostly milk for the first few months of life, parasite infection from pasture may not be a large issue. It's not until the calf is nearly six months old and close to weaning that pasture dry matter (DM) intake exceeds milk DM intake. This large intake of grass is what is most likely to expose the calves to large numbers of parasites. These suckler calves are also susceptible to lungworm and other parasitic infections - refer to the leaflets on the Animal Health Ireland website www.animalhealthireland.ie for further details.

Overall, second grazing season beef calves may have low levels of immunity and may experience poor performance and show signs of disease from parasites because of their relatively limited exposure to worms in their first year of grazing.

Recommendations

The effects of PGE in cattle underline the importance of control of these for the farmer and veterinary practitioner. However, because PGE, especially in older animals, is commonly expressed in its sub-clinical form, it may be given less attention than more 'dramatic' clinical diseases. Detailed information is available from the AHI Parasite Control Leaflet Series on the treatment and control of stomach worms (and other parasites) at housing, turn-out and during grazing. These leaflets are available on the AHI website www.animalhealthireland.ie.

Treatment and control is based on monitoring (through observing animal performance and diagnostic testing), appropriate grazing management strategies and treatments.

The approach to gutworm control will tend to vary from farm to farm. An understanding of some of the important details of their life-cycles, combined with observation of animal performance (weighing etc), regular sampling (faecal and bulk milk tests) and reviewing the results in consultation with your vet, should enable you to limit their impact in an economic and sustainable way, through measures such as strategic pasture management and the targeted use of anthelmintics.

Lungworm and liver fluke may also be present on a farm at the same time as gutworms and control plans for all of these parasites may need to be integrated with each other. Further information on these parasites is available



Beef animal performance should be monitored closely to avoid economic losses from gutworms

Table 2. Technical information for diagnostic sampling of Gutworms

Stock Class	Sample Type	No. of Animals Sampled	Tests
First grazing season calves	Faeces	10-15*	Faecal examination for worm and fluke eggs and lungworm larvae
	Blood	10-15	Plasma pepsinogen Stomach worm
In-calf heifers (Second GS)	Faeces	10-15*	Faecal egg count Fluke
	Blood	10-15	ELISA Stomach worm
Adult cows	Bulk milk	1	ELISA Stomach worm Fluke Lungworm?

*Faecal samples from individual animals can be pooled in the laboratory, thereby reducing costs (though losing some valuable information on individual values and variability).

Table 3. Treatment options for Gutworms

Parasite	Animal Age	Significance	Treatments
Stomach & other gut worms	All ages, immunity is typically less in first grazing season animals compared to second grazing season animals. In turn, immunity is less in second grazing season animals than in adults.	Suboptimal performance	Benzimidazoles* Endectocides* Levamisole* only effective against adult worms
Inhibited larvae of stomach & other gut worms (e.g. Ostertagia ostertagi)	All ages	Ostertagiosis Type II disease	Some Benzimidazoles* (check label) Endectocides* N.B. levamisole not effective

*Available in combination products

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Front cover abomasum image is courtesy of Regional Veterinary Laboratories, Dept of Agriculture, Food & Marine.

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