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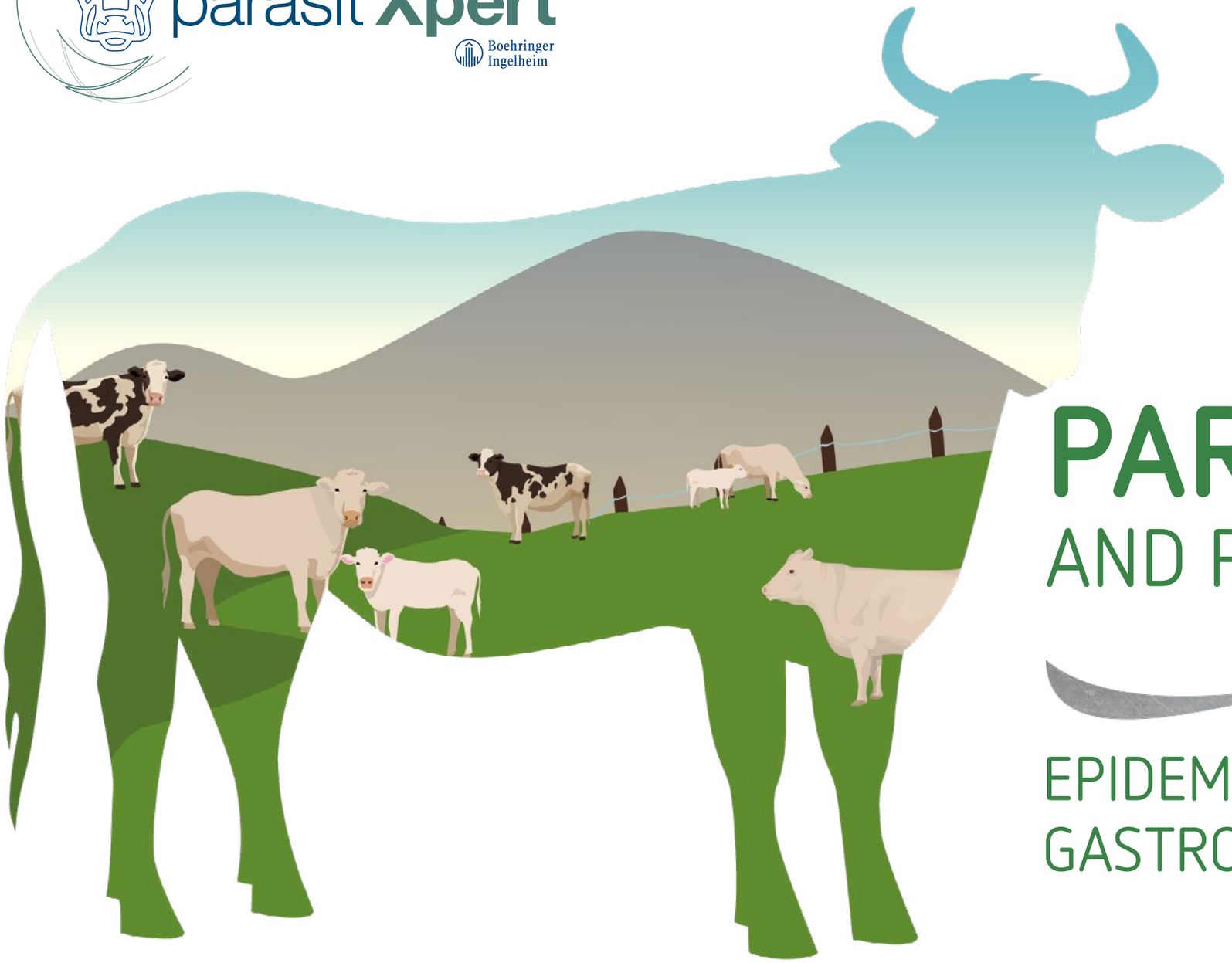

PARASITES AND PARASITE BURDEN

EPIDEMIOLOGY OF CATTLE GASTRO-INTESTINAL PARASITES

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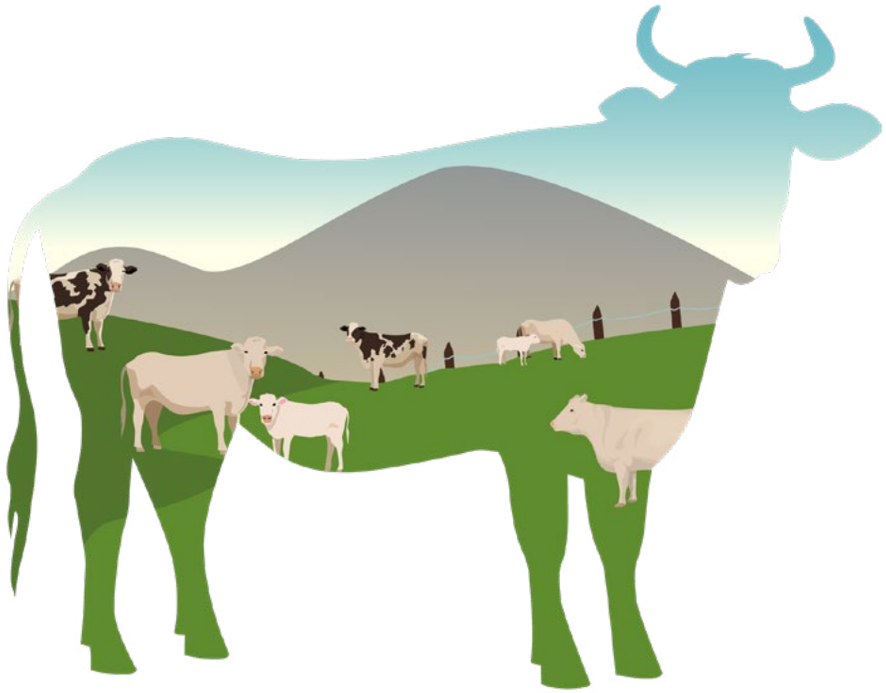

PARASITES
AND PARASITE BURDEN

EPIDEMIOLOGY OF CATTLE
GASTRO-INTESTINAL PARASITES

CATTLE PARASITES HAVE A HUGE IMPACT ON PRODUCTION

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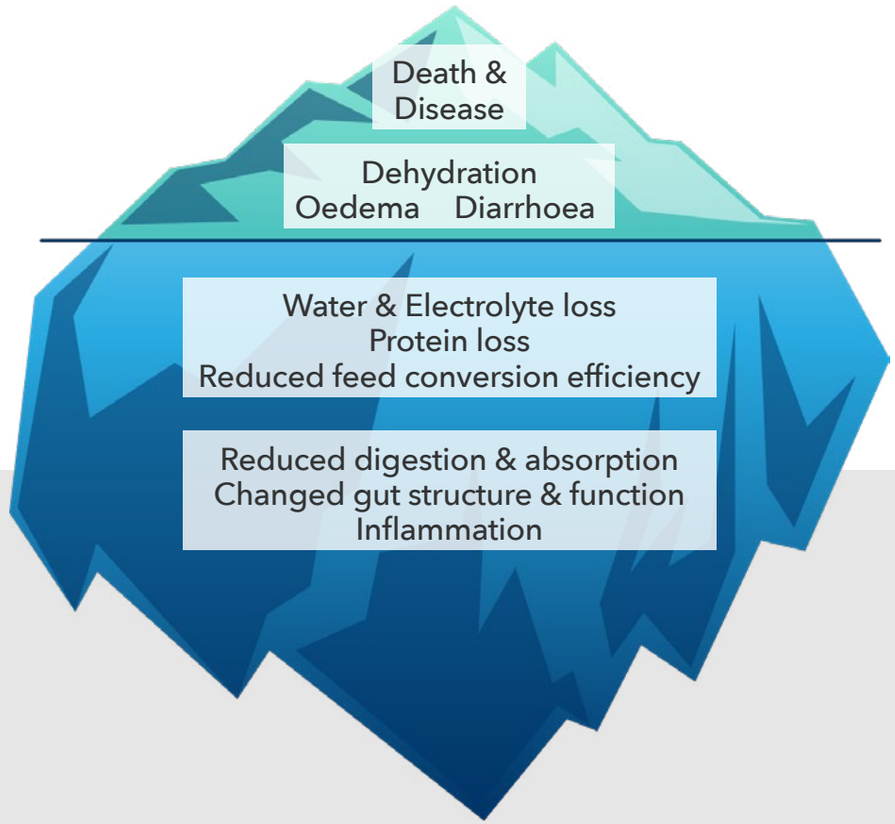
Gastro-intestinal parasites can be associated with clinical signs -dehydration, oedema and diarrhea, and even in some cases death. But they are also responsible for numerous subclinical signs, which can lead to severe economic losses: water and electrolyte loss, protein loss, reduced feed conversion efficiency, reduced digestion and absorption, changed gut structure and function, and inflammation.

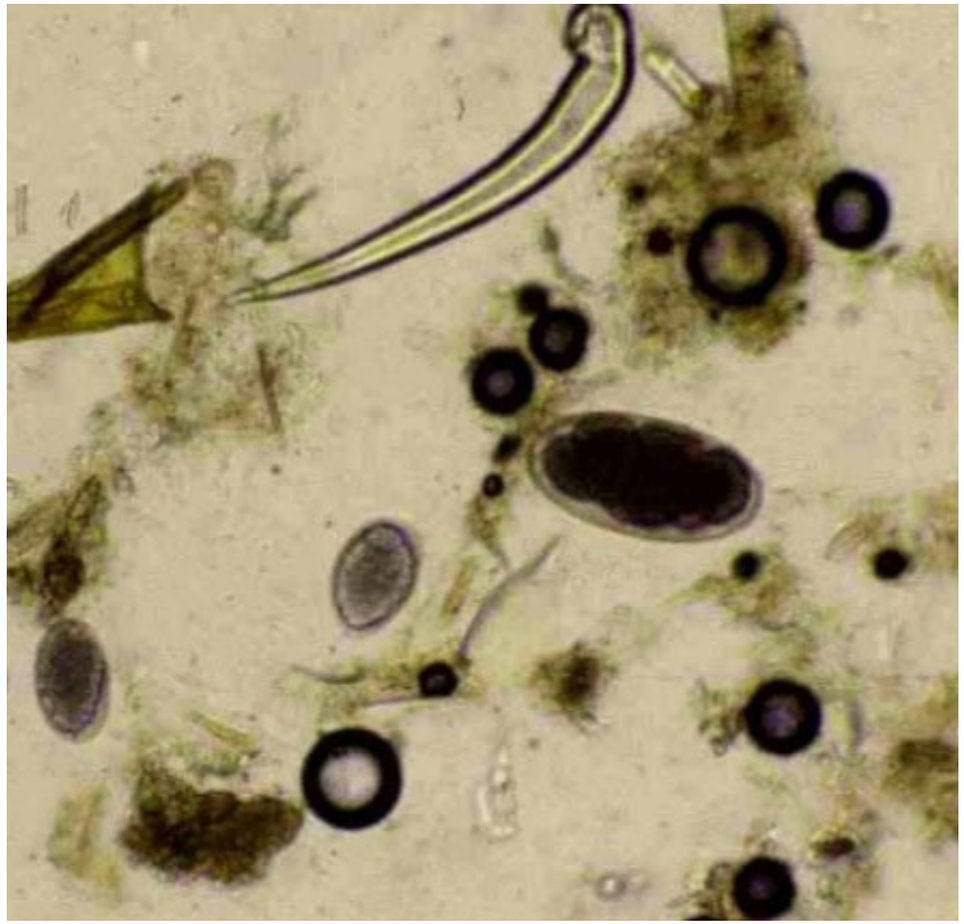
See the **impact of parasite burden**.



Factors affecting parasite burden

Diagnosis is essential to determine which parasite is involved and to assess the associated impact on the animal.
Several types of diagnostic tools can be used. Let's focus on 2 major ones:

FAECAL EGG COUNTING (FEC)



OPTICAL DENSITY RATIO (ODR)

which is carried out in milk samples to detect *Ostertagia*.

POSSIBLE RESULTS AND CONCLUSIONS

COMMON PARASITES IN CATTLE

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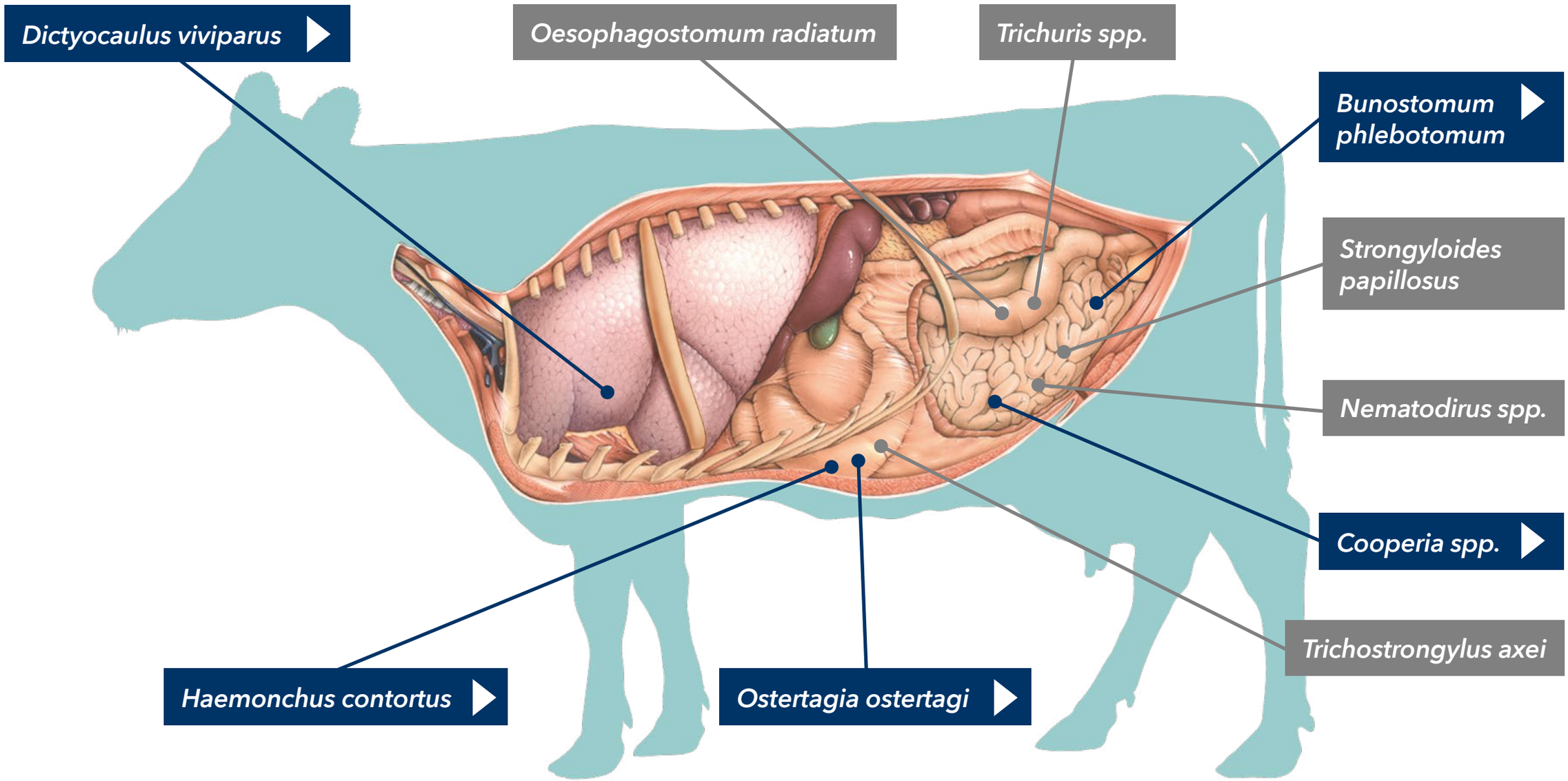
Cattle may be infected by a variety of gastrointestinal parasites, affecting different organs. Here are some examples:



Ostertagia ostertagi has the strongest impact on production.

Pathogenicity

- Lesions of gastric glands.
- Hyperplasia of abomasum mucosa.
- Dilatation of affected glands.
- Reduction of HCl production.
- Increase of pH in abomasum lumen (reduction in the digestion of protein).
- Increase of pepsinogen production.
- Plasma protein leakage to gastric lumen.

Lifecycle

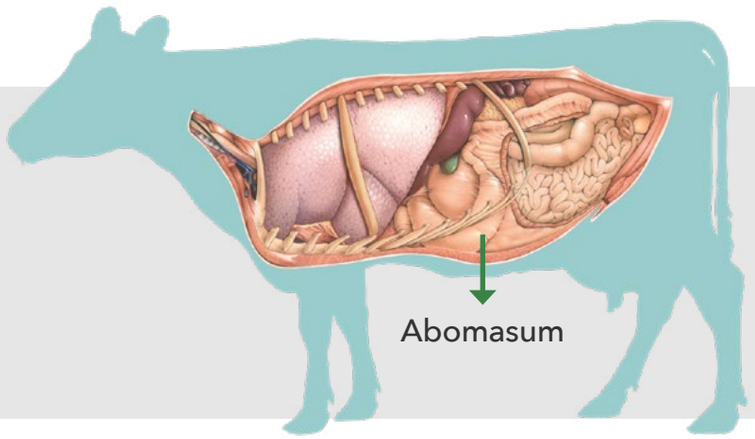
The endogenous cycle, that-is-to-say the time period from the ingestion of L3 larvae until the production of eggs by an adult worm, is also referred to as the prepatent period, and lasts 2 to 3 weeks for *Ostertagia*.

The length of the cycle depends on the climate.

SEE THE IMPACT OF CLIMATE

The economic impact of *Ostertagia* infestation

- Decreased productivity.
- Decreased milk production.
- Decreased fertility.



Establishment of immunity

When animals are put on pasture, some are more susceptible to parasites than others.
This can be explained by the fact that the development of immunity is progressive.
The appropriate parasite control programme will thus vary depending on the animal's exposure and level of immunity.

FIRST GRAZING SEASON CALF ▶ **SECOND GRAZING SEASON CALF** ▶ **ADULTS** ▶ (Time of pasturing > 8 months)

IMMUNITY

Why care about immunity?

The installation of immunity is progressive.

When established, it is linked to: a reduction in the size of the adult female parasites, a reduction in egg laying, the inhibition of training cysts of fourth stage larvae, a reduction in the establishment of third generation larvae in the lining of the abomasum, and the expulsion of adult worms.

These factors contribute to the reduction of the parasitic load in animals.

It is time to build up immunity

PREVENTIVE MEASURES ▶

Cooperia are small reddish nematodes that are up to 10 mm long. They are distributed worldwide, and typically the most prevalent parasite found in cattle, although their impact is often unclear. *Cooperia* are also the dose limiting species for most anthelmintics.

Pathogenicity

Cooperia oncophora is a mild pathogen in calves, but has been associated with inappetence and poor weight gains.

On the other hand, *Cooperia punctata* and *Cooperia surnabada* penetrate the epithelial surface of the small intestine, typically the duodenum.

They are responsible for:

- Catarrhal enteritis and the thickening of gut.
- Villous atrophy and malabsorption.
- Heavy infestations sometimes resulting in diarrhea.

Clinical signs

Lifecycle

Cooperia has a direct migratory lifecycle.

Dictyocaulus viviparus are slender, thread-like worms measuring from 4.5 to 8 cm. They are ovoviviparous, and lay larvated eggs.

They are highly pathogenic nematodes that cause parasitic bronchitis.

It is estimated that around 1/3 of exposed naïve youngstock develop clinical disease, but the rate can be even higher during outbreaks.

The clinical signs are well known – the weight loss and/or dip in productivity can be dramatic, as animals are choosing between breathing and eating.

Severity is related to the number of larvae the animals is exposed to (pathogenic dose: 300-500 larvae).

Clinical signs

The clinical signs are well known – the weight loss and/or dip in productivity can be dramatic, as animals are choosing between breathing and eating.

The migratory lifecycle

Impact of climate

Pilobolus fungi

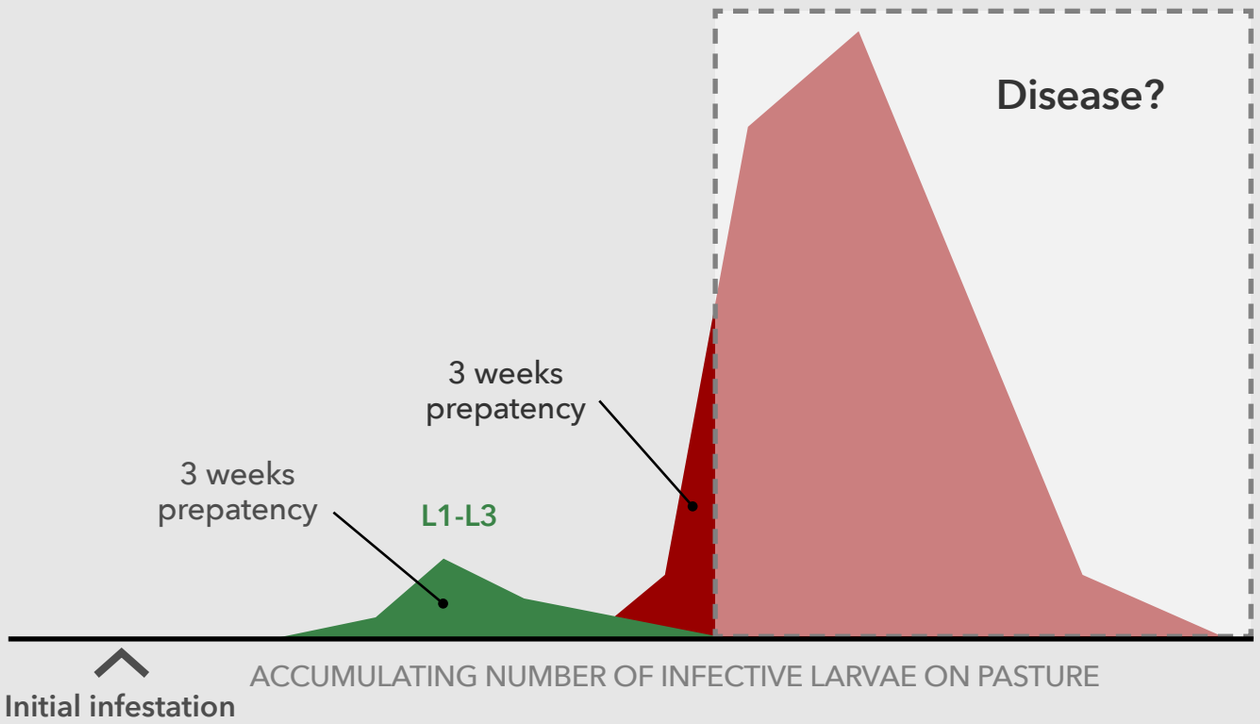
1 **After turnout**, which corresponds to the initial infestation, the first wave of infestation leads to L1 passed out onto pasture after the prepatent period of 3 to 4 weeks. Whilst these numbers are generally low, they represent new infestation for the grazing animals.

2 **After another 3-week prepatent period**, following on from the initial L3 produced after the first infestation, the number of L3 rises to much higher levels, potentially high enough to result in disease.

3 The box outlines the period when **outbreaks of disease** may occur, but this is dependent on the immunity of the challenged livestock.

Non-symptomatic carrier animals represent up to 70% of herds.

There is no guarantee that the disease will occur or not, or whether an animal will develop an immunity preventing the disease from developing.



Diagnosis

Diagnosis is largely based on clinical signs, such as coughing, significant drops in milk production, or even death in some cases, although it is important to remember that there are other causes for these clinical signs. Some tests can be used **to confirm** the suspected diagnosis:

Animals exposed to lungworms may develop 2 levels of immunity.

1. **Immunity to the initial infestation**, which develops rapidly between 8 to 11 days after a high primary infestation, but wanes over 6 to 12 months. As it wanes, the initial infestation may persist, and large numbers of larvae may hit the lungs. This scenario is certainly a contributor to disease in older animals.
2. **Immunity to maturation** of juvenile larvae develops between 10 to 30 days after infestation, and does not wane. Note however that it requires adult infestation, and may not develop if animals are treated early in the course of disease before adult worms have developed.

Type 1 immunity protects against infestation, while type 2 immunity protects against the development of adults. It is thus important for animals to develop both types of immunity.

Parasite control

Lungworm control relies on 2 key aspects:

Balance between challenge and immunity

Treatment

Practitioners should also be aware of the **re-infection syndrome** when defining a treatment strategy.

General presentation

- *Haemonchus contortus* is a species infecting both cattle and sheep, one of the most pathogenic parasites.
- It actually affects **sheep more than cattle**.
- It is called 'Barber's pole worm' due to its appearance.
- It is the **largest gastrointestinal nematodes of the abomasum**: 14 mm - 27 mm.

Clinical signs

Lifecycle

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