RED DEER IN A FARM SYSTEM

Health

Maintaining good deer health is crucial to production.

Good animal health is crucial to reaching production goals. Deer can be affected by both gastrointestinal and thoracic parasites, as well as external parasites like Ticks. Also from consuming contaminated pastures, these include Facial eczema and Ryegrass staggers.

Other diseases include Tuberculosis, Johne's disease , Yersiniosis, Leptospirosis and Abscess/fusoformus. Trace element deficiencies can also limit production.

Parasites

Internal parasites (sometimes known as worms) can limit production and can be complex to manage. Having an animal health plan, developed with a veterinarian, can help minimise the effects of parasites.

- Parasites of the deer stomachs and intestinal tract are known as Gastrointestinal tract parasites.
- Parasites of the airways and lungs (such as Lungworm) are known as Thoracic parasites.

Parasites in deer stomachs are known as gastrointestinal (GI) nematode parasites. Some nematode parasites are specific just to deer but some affect deer, sheep and cattle. They are sometimes known as 'worms' or ‘gutworms’ and include roundworms, stomach worms and nematodes.

How does the problem spread?

Parasites move from paddock to paddock when their hosts (such as deer) are shifted and eggs are excreted with faeces onto the new pasture. GI parasites can also over winter on pastures. However warmth and high humidity are required for larvae to develop. The life cycle of GI parasites start when eggs are shed in deer faeces. The eggs hatch and develop on the pasture through to a larval stage. They are then ingested by the deer and continue to develop in the gut (although winter can arrest their development for a period).
Moderate to severe burdens of immature parasites in the lining of the abomasums (fourth stomach) can lead to reduced acid production. This causes raised pH and poor protein digestion, resulting in chronic weight loss. Parasitised deer will fail to thrive, have reduced growth rate and shows signs of a rough coat, soft faeces, soiled tail and diarrhoea. Severe GI parasitism will be fatal.

Low levels of protein in the blood can sometimes result in oedema (fluid collecting) under the jaw. This is referred to as bottle jaw.

Effect on deer production

This effect can be very serious, especially in young deer. Adult hinds can be affected when put under stress and experience weight loss. Stags can be affected after the stress of the rut. There appears to be breed differences in susceptibility to GI parasites - Fallow deer are relatively immune/resistant to GI parasites while Wapiti deer for example are more susceptible . Red deer sit in between.

Diagnosis

Diagnosis of GI parasitism in a live animal is difficult. This is because the relationship between faecal egg counts and total worm numbers is not well understood. The most risky stage for the deer tends to be when parasites are immature and lodged in the stomach lining. However, as they are not shedding eggs at this point, then their presence is not identified in a faecal egg count. For this reason faecal egg counts are not reliable. Faecal egg counts may be useful in fawns but only in autumn prior to them developing immunity to the parasites. In weaners visual signs of infection such as loss of condition and weight gain that is lower than expected can be a useful indicators. The lining of the abomasum is thickened and pitted and has a “Moroccan leather” appearance on post mortem.

Control and Treatment

Treatment with anthelmintics is complemented by pasture management and grazing management.
To help deer naturally resist the effects of parasitism, good nutrition is required (both good quality and high availability). Pastures high in legumes or chicory can provide good nutrition.

Which stock to treat

Once a problem is either suspected or predicted (either through a faecal egg count and/or visual signs, past experience of a fields

history or time of the year) then control should follow. Young deer are susceptible in autumn, winter and into early-spring.

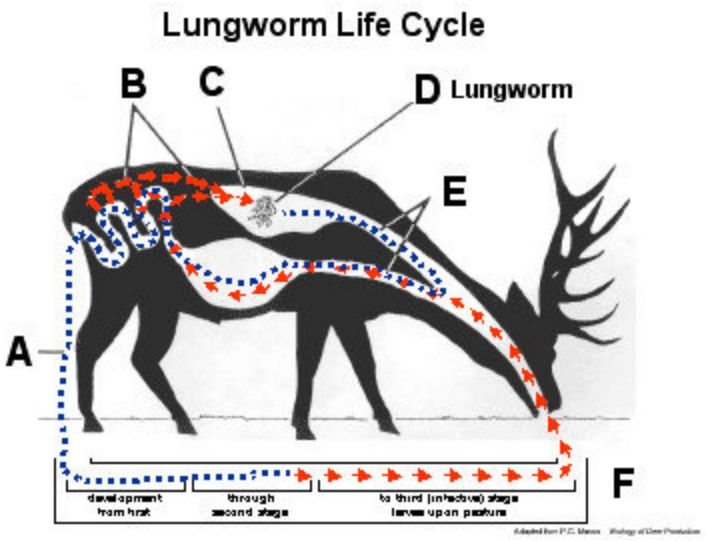
Thoracic (Lungworm)

Lungworm (Dictyocaulus eckerti) is the most important and serious parasitic disease of young farmed deer. The cattle lungworm (D. viviparus) has also been shown to affect deer, although it is less well adapted to red deer.

How does the problem spread?

Adult worms live in the air passages in the lungs. They look like 40-50mm threads. Outlined below is the life cycle.

Diagram A: Life Cycle of lungworm in Red Deer



1. First-stage larvae passed in faeces.
2. Infective third-stage larvae on pasture are eaten. They pass down through the stomach, penetrate intestinal mucosa and migrate via lymphatic and blood circulation to the lungs.
3. Development to fifth-stage larvae and maturation to adulthood in air passages of the lungs.
4. Adult worms inhabit airways and lay eggs.
5. Eggs coughed up and swallowed, hatch to first stage larvae and are passed out in faeces.
6. Infective larvae consumed with herbage.

   Cold weather or hot dry weather slows larval development on pasture. However lungworm are hardy and can over-winter in cold climates, surviving at 4°C for a year.

What are the symptoms?

Parasitism can be confused with other deer health issues, particularly gastro-intestinal parasites. Coats look rough and growth rates drop. A more particular sign to lungworm infection is a soft bronchial cough (usually indicating a heavy parasite burden).

Effect on deer production

Young deer aged three to five months in their first autumn are the most susceptible to infection. This is because they have not yet developed full immunity and lungworms are often in high numbers in autumn. All age groups may carry infestations at any time but generally young stock or heavily stressed animals that are untreated will develop severe infestations. Moderate infestations may cause production losses and heavy infestations may be fatal (because of blocked airways). Pneumonia can result from damaged lungs.

Diagnosis

The faecal larvae count (FLC) is currently the only laboratory test for lungworm. It requires 5 to 10 grams of faeces collected from

the rectum and submitted to a diagnostic laboratory in a small pottle. Usually samples are collected from 5 to 10 deer to get a good estimate of parasite burdens in the group.
FLCs can be used to monitor lungworm burdens in young (2 to 6 month) old deer in late summer/autumn. Regular counts are needed to detect when lungworm burdens first occur and to monitor changes over time. Start drenching as soon as there is a sudden rise in FLC. FLC are really only useful in young deer in their first autumn and can be misleading in the following spring.

On farm poor growth rates and coughing (especially when moving a mob) indicates lungworm.

Control and treatment

Control and treatment should go hand-in-hand. Control should focus on:

- reducing the reinfection challenge to young deer; and
- eliminating the existing lungworm burden with anthelmintics.
- Control options to reduce reinfection challenge include-
- grazing at-risk blocks with older deer, adult sheep or cattle;
- replacing older pastures with new pasture species less likely to harbour parasites, such as brassicas;
- treating deer with anthelmintics before there is an outbreak to avoid building up large parasite populations on pasture; and
- providing optimum nutrition to increase young deer's ability to resist the effects of parasitism.
- Treatment of parasitic infestations by anthelmintics is important for the maintenance of deer production.

Which stock to treat

The class of stock requiring treatment are typically young deer (three to six months old) in their first autumn. Immunity builds after this time. The whole mob should be treated, not just those showing signs of parasitism.
Adult red deer are relatively resistant to lungworm, as long as they are well-fed and not suffering environmental stress. This means they will have minimal lungworm burdens, which will not have any effect on production. However, some red deer that have been stressed, such as red stags after the rut and hinds lactating during drought conditions, may be susceptible and require treatment.

When to treat

Late summer/autumn (September to December) is the key risk period for young deer so treat at intervals during this time, according to the anthelmintic used. Seek veterinarian advice on this. The aim is to break the lungworm life cycle.
The timing for treatment depends on both the faecal sample results and the season. Infection rate usually increases during late summer and early winter. However, if the summer is particularly warm and wet then worming may be required earlier. The interval between treatments depends on the persistence of the anthelmintic in the animal (see below) and the level of challenge by infective larva .

Note it is very unwise to leave young deer undrenched in autumn because they can develop large burdens very quickly if untreated. They have not had time to develop immunity.

Key times to consider treating adults is when some stress affects their condition eg hinds before calving and/or weaning, and stags after the rut. Stocking rate also dictates the timing and number of treatments, with more intensive grazing systems needing more monitoring and perhaps more treatments than extensive systems. Adult deer with poor trace element status and tight feed conditions might develop clinically significant lungworm burdens. Similar deer with good nutrition may not. For this reason an integrated approach to managing animal health is best.

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Johne's disease

Johne's disease is a common cause of death and reduced growth rates among deer. It is also known as paratuberculosis and there are two main strains. Deer are mostly affected by the cattle strain.
The disease is caused by a bacterial infection (Mycobacterium avium subspecies paratuberculosis).The infection starts in the small intestine and spreads to the lymph nodes. If the disease advances the intestinal walls can thicken, interfering with digestion and absorption of nutrients.

How does the disease spread?

Johne's disease is most often introduced onto an uninfected farm through newly purchased deer.
On farm, infection occurs through deer eating pasture or drinking water contaminated with contaminated faecal material. Hinds can also pass the bacteria on to their fawns through the placenta, colostrum or milk.

What are the symptoms?

Clinical signs are muscle wasting, ill thrift and diarrhoea. Stress can bring on clinical effects (for example during lactation, the rut, or during drought). Sometimes enlarged lymph nodes or cheese-like abscesses can be seen. Typically infected deer show only subclinical symptoms (slow growth rates) rather than obvious symptoms. This means it can be hard to recognize the problem.

Effect on deer production

Johne's disease outbreaks typically affects young deer aged 8-15 months, with losses of up to 20% recorded. Sporadic cases in mixed age deer occur.

Diagnosis

There are several diagnostic tests. These involve taking a blood sample. These are best used for confirming Johne's in a deer with clinical signs of the disease.
Faecal samples can be cultured but it takes up to 60 days. As shedding of the bacteria in the faeces is intermittent, not all infected animals (especially those with minor infections) will show up as positive. Culture is the best to identify different strains.

If the animal is going to slaughter, Johne's can be confirmed by culturing intestine and lymph nodes. Often the first indication of Johne's in a herd is from the processing plant. Enlarged lymph nodes at slaughter are noted by meat inspectors and the number of animals with lesions reported back to farmers. Farmers can use this information to help put a control programme into place.

Preventing introduction of Johne's disease

Steps to prevent the introduction of Johne's disease include-

- running a 'closed' deer herd (not buying in deer)
- only introducing deer that have tested negative .
- only introducing new genetics through artificial insemination (rather than risk bringing in the disease via a stag)
- Controlling the spread of Johne's disease
- Once introduced, Johne's disease can't be easily eradicated. Control requires a combination of measures including
- culling affected animals
- vaccination
- testing to detect deer with subclinical symptoms (for culling)
- Minimising cross-infection between age groups.
- using older animals - which are more resistant - to clean up contaminated pastures.
- avoiding the grazing of weaned animals on areas contaminated by clinically affected deer, cattle or sheep.