Johnes

Johnes Disease is a chronic wasting disease caused by a bacteria called *Mycoplasma Avium subspecies Paratuberculosis (MAP).* This infection can affect any ruminant animal, including cattle, sheep, deer, rabbits and other wild life. It is thought up to 30% of herds are infected with Johnes and it can have major financial and animal welfare implications.

It is contagious gastrointestinal disease. The infection is picked up as a calf but they may appear to stay healthy for 2-5 years before any clinical signs develop. The 3 main routes of infection are;

- Faecal ingestion (from sucking contaminated dams teats in the calving pen).
- Colostrum
- Across the placenta

Once ingested, the bacteria are able to localise to part of the small intestine where it multiplies and causes a thickening of the gut. This thickening prevents nutrient re-absorption and also leaking of important proteins from the gut. The clinical signs of this disease include 'pipe-stem' diarrhoea, chronic wasting and emaciation, and milk drop even with a continuing appetite. However these signs may only occur later on in the animal's life but it will have been spreading the bacteria up to this point without any sign of disease.

MAP infection is difficult to detect, particularly when the animal shows no clinical signs due to fluctuating antibody levels and no way of detecting the bacterium itself. The tests available are based on a blood sample, a faecal sample or a biopsy of the small intestine plus using clinical signs and history of the herd to make a diagnosis. We often take a blood sample and a faecal sample to increase the likelihood of getting a true diagnosis.

Controlling Johnes in your herd:

1. **Test and Cull**
   Gaining Johnes Accredited Status can be a long process. All breeding animals over 24 months old are blood tested on a yearly basis and any positive animals and their offspring should be culled or fattened (but isolated, so as not to spread disease). However, not all infected animals will show a positive result on their first test and it can take up to 3 annual clear tests before an accredited status can be given

2. **Controlling Spread of Infection**

Controlling the spread of Johnes infection is based on controlling the 3 routes of infection, hygiene management (faeces), colostrum management and culling your positives offspring.

- Calving pens should be as clean as possible; this is the MAIN area where calves will pick up infection.
- Dairy Farms – Snatch calving is a good way of reducing the risk of calves picking up infection from sucking dirty teats of cows or their infected colostrum.
- DO NOT use colostrum from known infected cows, or pooled colostrum you do not know the status of. ONLY use colostrum from cows that are not infected, particularly for the heifer calves likely to be kept for breeding. Pasteurisation can be a helpful tool to help reduce bacterial load.
- Do not feed waste/dump milk to calves.
- Do not graze stock on fields where slurry has been spread, the bacterium can survive in slurry for up to 8 months. It is very resilient to disinfectants, heat and cold.
- Cull any positive animals and do not retain their offspring for breeding.

3. **Vaccination**

Vaccination is not available in the UK and the vaccine would have to be imported from abroad. However, vaccination is an option to reduce the number of clinically infected animals in your herd.

BVD

Bovine Viral Diarrhoea (BVD) infection has a major economic impact for cattle farmers as well as cattle welfare. Many countries have introduced eradication schemes with varying success. It can cause numerous issues on farm, including poor reproductive performance, poorer immunity and increased susceptibility to other diseases as well as mucosal disease. Mucosal disease (MD) is typically the end stage of the infection of a persistently infected (PI) animal around the age of 6-12 months. Animals will have diarrhoea, wasting, depression and fever, with ulcerations throughout their mouth which will be widespread throughout their digestive tract. Euthanasia by this stage is essential if not done before this stage has been reached.

Infection can be acquired from coming into contact with a PI animal; it can also be sexually transmitted. Adult animals exposed to the virus tend to undergo a transient infection with mild clinical signs (mild fever and milk drop). However, they usually recover and build up immunity. The problem arises when an exposed animal is pregnant; the virus is very adapted to crossing the placenta and infecting the calf. If a cow is less than 120 days in gestation, the foetus won't have developed its own immune system to be able to recognise a virus as foreign. This allows the virus to become part of the calf's normal DNA and when the PI calf is born, it will excrete massive amounts of virus for the rest of its life. A PI animal, although live born, will be smaller, less thrifty and less healthy than the rest of its herd mates and will never fatten well. Getting rid of these animals is the main aim of most eradication schemes as they are the major source of infection. A PI cow will always give birth to a PI calf so it is critical in controlling the disease to not keep any PI animals.

Early infection can also present as repeat breading from early embryonic loss and reabsorb ion. Older foetuses infected by BVD may be aborted, may be born with eye and nervous system defects or born prematurely/weak.

Important Points to help minimise bringing infection on to your farm:

- Do not keep any PI animals. They are the source of infection!
- Any animals with an unknown BVD status isolate and test for BVD virus and only introduce into the herd when a clear result is received.
- Only buy in from BVD free or CHeCS Accredited herds
- Vaccinate: Vaccination is a great aid in protecting your herd, although if a PI animal is present, even vaccination will not give complete protection. There are many vaccinations are on the market and appropriate programs can be discussed with your vet.

The Scottish BVD Eradication Scheme is set up into 4 stages and we are currently in Stage 4.

Stage One: Subsidised screening.

Ran from September 2010 to April 2011. The Scottish Government provided £36 towards testing for BVD for each herd, and a further £72 towards further testing or veterinary advice if the result was positive. Around 4,000 herds took advantage, at a cost of £180,000.

Stage Two: Mandatory Annual Screening.

All keepers of breeding cattle herds were required to screen their herds for BVD by 1 February 2013, and **annually** thereafter. A range of testing methods is available. Also, where there are calves born in non-breeding herds, they must be tested within 40 days.

Stage Three: Control Measures (Reducing the spread of infection)

Early in 2012 the Scottish Government consulted on proposals to introduce control measures, including movement restrictions. These control measures came in to force in **January 2014**, and include:

- A ban on knowingly selling/moving cattle infected with BVDV;
- Requiring the herd's BVD status to be declared before sale; and,
- Restrictions on untested herds/animals

Stage Four: Enhanced testing and further movement restrictions

- Controls introduced on 1st June 2015 include:
- Movement restrictions on 'not negative' herds
- A reduction in the number of testing options available
- The requirement to test replacement animals from untested herds
- Assumed negative status for dams of calves which have tested negative

Bovine Respiratory Disease

Calf Pneumonia

Bovine Respiratory Disease (BRD) is estimated to cost the UK cattle industry £80 million annually (between £30 for mild cases to £500 when the animal dies). Financial costs can be from antibiotic treatment and mortality, but the largest cost of to the farmer comes from the poor weight gain and performance of the animal.

Pneumonia tends to be most commonly seen during the first month of housing with the autumn calves being more affected than the spring-born calves. BRD is a multi-factorial disease, with the infectious causes (viral and bacterial) being exacerbated by management factors (housing design, ventilation, concurrent disease etc).

Viral causes of BRD include:

- Parainfluenza-3 (PI3)

- Respiratory Syncitial Virus (RSV)
- Infectious Bovine Rhinotraceitis (IBR)
- BVD may also play a part, causing immune-suppression

Bacterial infections tend to come in and cause secondary infection once viral agents have caused damage to the respiratory tract. The main bacterial causes of BRD are:

- *Mannheimia haemolytica*
- *Pasteurella multocida.*
- *Haemophilus somni*
- *Mycoplasma Bovis*

Clinical signs of pneumonia are typically a high temperature, nasal discharge, coughing, not coming forward to eat and depression. Antibiotic treatment and an anti-inflammatory prescribed by your vet is based on previous history

Testing for BRD can be done using a blood sample or paired blood samples that allow the lab to detect if there are rising levels of antibody indicating disease. Post mortem can also allow us to diagnose if pneumonia was the cause of a sudden death, however would not tell you what the cause of the pneumonia is.

Vaccination for BRD is an excellent too to help reduce the chance of calves developing pneumonia and building resistance. However, vaccination alone will not prevent pneumonia developing , it needs to be carried out correctly using the manufacturers guidelines as well as having good husbandry; including ventilation, stocking densities and stressful events such as dehorning or speaning that need to be taken into account as these will put pressure on the calves at housing time.

Come in and ask one of our vets about the different vaccination programs and what would be best for your production system.

Husk

Husk is another name for lungworm and can be a cause of pneumonia in adult cattle. These animals tend to have a white nasal discharge, a fast resting respiratory rate and there will be a milk drop (as much as 50%) associated with the condition in dairy animals. A cough will also be noticeable after short bouts of exercise or when walking to and from the parlour. In severe cases animals will become reluctant to move, extend their neck and have a low head carriage.

Diagnosis of lungworm is based on a faecal sample and demonstration of the lungworm larvae.

Prevent of lungworm is best achieved by building immunity through vaccination and exposure. Exposure to lungworm and picking up infection during grazing season allows immunity to develop and then with controlled use of wormers, clearing any lungworm infection before clinical disease occurs.

Calf Scour

The incidence of calf scour is dependant upon the level of colostral antibody protection the calf has and the level of hygiene within the calving pen and calf shed. It is recommended that calves receive 4 litres of

colostrum within the first 4 hours after birth. If no colostrum has been given to a calf within 6- 12 hours of birth, the ability of the calf's gut to absorb the antibodies decreases and the more likely the calf is to develop disease. Many factors can contribute to a calf not receiving enough colostrum soon after calving, for example; recumbency due to a difficult calving or nerve damage, a dopey calf who is slower to get up and suck or long droopy teats that calf struggles to suck.

The major causes of calf scour are:-

- Rotavirus
- Corona virus
- E. Coli and Salmonella
- Coccidiosis
- Cryptosporidium

The viral causes of calf scour are some of the first to cause disease, often a severe, acute watery yellow to green scour is seen within the first week or two of life. These calves will be slow to stand, unwilling to drink and become recumbent and dehydrated quite quickly.

E.coli will cause scour in the very young calves only 1-3 days old because they are exposed to it from the environment. Vaccination for Rota, corona and E.coli combined is available and should be used annually, particularly for dairy calves where these causes are more common.

Cryptosporidium tends to cause issues in older calves 1-3 weeks old, once they have been grouped together and pen contamination builds up much more quickly. Calves will have a watery scour with mucus, blood and undigested milk, be weak, lethargic and dehydrated.

Coccidiosis generally affects weaned calves, although can be as young as 3-4 weeks old. Clinical signs don't tend to show until 2 weeks after a calf has been infected, so damage has already been done by the time you know you have a problem. Calves will also be stunted, dehydrated and strain which can lead to a rectal prolapsed in some cases.

Treatment of calf scour is very similar for all causes:

- **Isolate:** all sick calves should be isolated in a warm, dry well bedded pen away from all the other calves to reduce the chance of spread to others. A heat lamp raised above helps to keep the calves warm.
- **Fluids**: Scour results in the loss of vital salts, fluids and energy so 1-2 litres of oral electrolyte fluids should be given every 3-4 hours. Electrolytes should be given at body temperature Hydration of the calf can be assessed whether the calf has sunken eyes, a skin tent and if a suck reflex is still present.
- **Energy:** Although fluids are essential to restore the calf's salts and hydration status, the calf still requires energy, which is provided by the milk feeds. Milk and oral fluids should not be fed any less than 2 hours apart, this is to allow the milk to clot correctly and not curdle in the calf's stomach which would also lead to a dietary upset.

If calves are extremely flat and dehydrated, you should contact your vet. Intravenous fluids will be administered to the calf to ensure rapid re-hydration as quickly as possible.

Prevention

Prevention is better than cure.

Ensuring calves receive adequate colostrum is vital. The 3 Q's of colostrum are;

- **Quality –** Ensuring good quality colostrum, from dams that have been vaccinated provides the correct antibodies for calves. Quality can measured using a refractometer on farm which assesses the density of the colostrum.
- **Quantity –** 3L in 3 hours - If colostrum is of a lower quality then feeding a larger volume of it can help to combat any deficiencies.
- **Quickly –** Calves should receive colostrum within 2-4 hours of life. After 6-12 hours, the calf's ability to absorb antibodies from the colostrum through the stomach wall reduces. If a calf does not receive any colostrum within 12 hours, that calf will be very vulnerable to many infections and will struggle to survive.

In addition to colostrum management, hygiene management is also of paramount importance. Make sure calves drink from clean buckets and calf feeders. Pens should have fresh bedding that is dry and pens should ideally be steam cleaned and refreshed after each batch of calves.

Cattle Parasites

Parasitic gastroenteritis (PGE) is most commonly seen in young grazing stock but it can also be seen in adult cattle too. Cattle are able to build some resistance to worms after 2-3 grazing seasons but these animals can still experience weight loss. The main causative of PGE is a roundworm called *Ostertagia. Ostertagi,* larvae of which burrow into the abomasum and cause pathology in the cow's stomach. There are two types of ostertagiosis, Type 1 and Type 2.

- **Type 1**:- Affects your first season grazers and spring calves. These calves will show signs in the 2nd half of the grazing season, once they have ingested worm eggs and put more onto the pasture during the first season in a cyclical fashion. As well as pasture contamination from calves (as is the main culprit for dairy units), on a beef production system, the main contamination is obviously from the adult cattle. These calves will have profuse, green scour, weight loss, loss of appetite and a lot of the young stock will be affected.

- **Type 2:-** Clinical disease is seen in the autumn born calves in late winter and often housed when disease occurs. Disease is caused by the re-emergence of all the inhibited larvae that have burrowed into the stomach lining. These inhibited larvae all re-emerge at the same time causing a lot of damage to the stomach lining and is associated with a higher rate of mortality that type 1. The larvae go into an inhibited state due to having been on the pasture and taken up by the cow later on in the grazing season when it's too cold for the parasite to survive on grass. These animals tend to have intermittent diarrhoea and weight loss with loss of appetite and it can be fatal.

Immunity to worms is developed after 1-2 grazing seasons. However, although adult cattle are said to be immune, sub clinical disease can still be present and production losses may still occur.

Diagnosis of PGE in cattle is based on clinical signs, farm and animal history alongside faecal egg counts (FEC) and pathology, (from a post mortem at the lab or from abattoir feedback).

Control is based on good grazing management and the correct use of wormers. Ideally you want to limit the exposure to parasites, take into account which animals are most susceptible and which fields are most likely to be contaminated. The highest risk would be in animals in their 1st grazing season, grazing permanent pastures. New leys, aftermaths and co-grazing help to reduce infection and pasture contamination.

Treatment:-

- Ivermectins are good for young-stock out at grass. With a 3 week persistency, you would treat animals 3 weeks post turnout and then at 8 and 13 weeks post turnout.

- Worming animals frequently can lead to resistance so targeting your treatment to animals that may be most affected and leaving the biggest animals can be a regime worth thinking about.

For the best plan and more details on treatment and control on your farm, come in and speak to one of our vets, who will be more than happy to help and give you the latest information.

<u>Fluke</u>

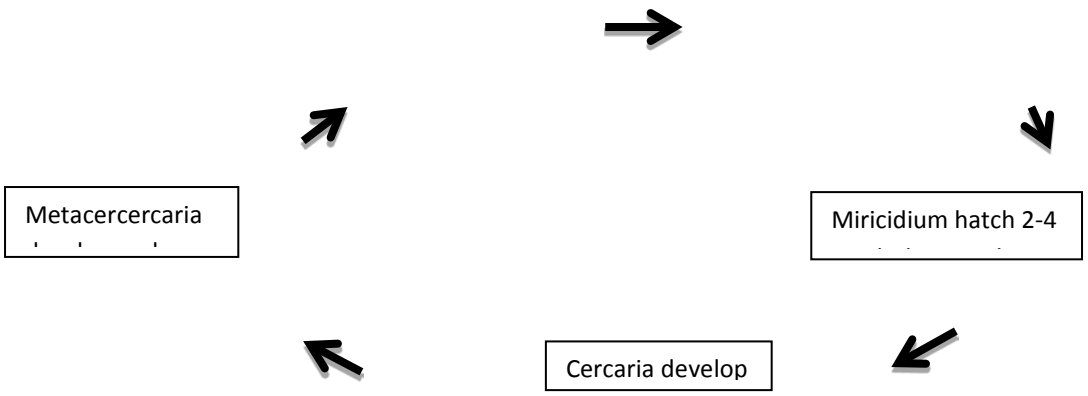
Fluke is and always has been an ever increasing problem, particularly in the south west of Scotland. Abattoir feedback of damaged livers and live fluke is greatest between January and March and also September-October.

There are many obstacles when it comes to fluke control:

- Cattle don't develop a natural immunity to fluke infections
- Environmental control is not always practical and can be costly e.g. draining, fencing
- Flukicide products have their limitations, few medicines act against the jeuvenile fluke, withdrawal times can be lengthy making dairy cows harder to treat.
- Triclabendazole resistance in increasingly being diagnosed.

The **life cycle** of the fluke in a 12 week turn over and a mud snail is used as an intermediate host, which adds to the difficulty in control of fluke, you need to control the snail too.

The immature	Eggs are



In the first 8 weeks of infection, the jeuvenile fluke have migrated to and penetrated the liver; however, it is very rare that during this stage, any clinical signs will show. Cattle will still appear to be performing well. Between weeks 8 and 12 of infection, the fluke are maturing in the bile ducts. But, calves will start to show a reduced performance and weight check 12 – 13 weeks after being infected

Snails will carry developing fluke through the winter, as will untreated stock, and both will contribute to spring pasture contamination. The breeding season for the mud snail is May-October and these will become infected with miricidium during the summer months. However, with the developing fluke requiring moisture and warmth as well as the sail, the season for infection is extending.

The **effects** of fluke on your cattle include:-

- Reduced weight gain
- Anaemia and liver damage
- Reduced food conversion efficiency.
- Milk yields will be affected
- Reduced pregnancy rate and increased time to conception

Diagnosing fluke can be done a number of ways, although some are less useful than others. Either by looking for evidence of the parasite on faecal examination, looking for an immune response to the infection or the pathology the fluke leaves in your animals.

Treatment depends on the stage of fluke that you're targeting and the time of year.

- Triclabendazole is the only drug effective against the very early stages of fluke in the animal. Killing fluke as young as 2 weeks old.
- Closantel and nitroxynil kill immature fluke 6-7 weeks old and above.
- Oxyclozanide, clorsulon and albendazole kill fluke 10 weeks old and above.
    - Oxyclozanide also kills rumen fluke.

The limitations of these medicines are that a lot of them are unsuitable for dairy cows, with long withdrawals or no license at all in milking cows.

Oxyclozanide ('Zanil') is an option for dairy farmers, it only has a 72 hour milk withhold so can be used in milkers if required. Albendazole is another suitable option and only has a 60 hour withhold.

Controlling fluke is done in a number of ways;

- Grazing – if high risk pastures are on your farm, remove animals from them in august or graze them with cattle for 8 weeks before moving and treating.
- **Winter housing** – Is the cornerstone for treatment, with options such as:-
    - Triclabendazole 2 weeks post housing
    - Closantel/nitroxynil at housing and/or 8 weeks post housing.**
    - Albendazole/oxyclozanide at housing and/or 12 weeks post housing**
- Out wintered stock must receive a dose in January if not before.

**- There is a risk of loss of production is animals are heavily infected.

Rumen Fluke

There is an increased frequency of rumen fluke, which also uses a mud snail as its intermediate host. Clinical disease is seen in a loss of production due to ill thrift and is thought to be caused by the immature stages in the small intestine.

Control of rumen fluke is combined with liver fluke treatment. Treatment is best achieved using only oxyclozanide (Zanil or Levafas).

Lameness

Lameness is one of the 3 main reasons for culling in the dairy industry. Lesions can cause intense pain and are a major animal welfare issue, not to mention that it can have an immense economic impact on the dairy farmer. Losses come from a reduced production of milk, increased calving to conception interval and cost of treatment. A lame cow can produce 1.7-3L less a day for up to a month before and after treatment.

There are many factors which influence the incidence of lameness; you can influence these factors to help reduce lameness in your herd.

- Walking surfaces
- Standing times
- Hygiene in the shed and parlour
- Foot bathing regime
- Cow comfort
- Nutrition
- Genetics

Typical causes of lameness in cattle:

- Digital dermatitis
- Sole or toe ulcers

- White line disease
- Foul in the foot
- Toe Necrosis
- Laminitis

Locomotion scoring of your herd is a good way of seeing what level of lameness is present; this would be carried out by one of our vets. Foot trimming can be a great way to keep on top of any lameness issues and provides you with an idea of the common types of lesions that occur and why on your farm.

Mastitis

Mastitis is inflammation of the mammary tissue within the udder. There are a number of causes of mastitis in cattle including chemical, thermal or mechanical damage or a bacterial infection. Some examples of the sources of infection can be; contaminated hands while milking, the clusters/milking parlour as well as the environment the cattle are in. 99.9% of cases of mastitis are causes by bacteria ascending the teat canal and therefore it is of upmost importance to strive to reduce the bacterial contamination and transmission.

The cow has many defence mechanisms to help prevent getting mastitis, including the skin of the teat and the sphincter within the teat.

1. Healthy skin will have an environment that is hostile for bacteria to survive on its own, so if it becomes cracked or chapped, it's a starting point for bacterial growth.
2. The teat has a ring of smooth muscle within which opens and closes, allowing milk let down and also acts as a defence to bacteria ascending into the udder. On average it takes 30 minutes for this canal to close post-milking, but can take up to 2 hours. Hence, fresh feed is promoted after milking.
3. Keratin is a type of protein that forms a plug within the teat, again to act as a barrier to bacteria. Between 12-40% of this plug can be lost after each milking and has to build up again.

On top of this, the cow also has her own immune system which fights the infection once bacteria start to take over in the mammary gland. White blood cells are produced and put into the circulation and travel to the udder to fight the bacteria and clear the infection. This is also known as the somatic cell count (SCC) and is a good indicator of the level of infection a cow has.

- Subclinically infected cows will show no clinical signs, but an increase in the SCC may be the only indicator. A California milk test (CMT) is a cow-side test the farmer can carry out to determine if a cow is sub-clinically infected.
- Clinically infected cows will have high SCC of over 200,000, with a hard, red hot swollen udder and visible clots in the milk. She will typically have a reduced milk yield and depending on the severity, be quite sick.

Treatment will often be a course of intramammary tubes and/or followed by systemic antibiotics if the tubes don't clear the infection. Non-steroidal anti-inflammatories (NSAIDs) are great for pain relief and reducing the inflammation and oxytocin and stripping out the infected quarter is necessary to remove the dirty milk and bacteria within.

- The exception to this treatment protocol is if you have a case of E.coli mastitis, also known as toxic mastitis. This is an emergency and you must call your vet immediately.

Controlling mastitis is done numerous ways;

1. Dry cow therapy. The aim, at the end of a lactation, is to clear any remaining infection with the use of an antibiotic, and then to seal the teat using a teat sealant. Dry cows are often put somewhere out of the way from the rest of the farm but these cows are actually very important. The start and end of the dry period are the 2 biggest risk times for infection to occur. A staggering 50% of intramammary infections occur in the first 100 days, these infections being picked up in the dry period.
2. Hygiene –The parlour hygiene is paramount, as is the milker's hands and the cubicles. Reducing the bacterial contamination and transmission is key.

For more information about mastitis or if you think you have an issue and would like it investigated, why not drop in and speak to one of our vets.