Johne's disease in sheep and goats

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Abstract

Johne's disease in sheep and goats appears to be common in Ontario dairy sheep and dairy goat herds. The bacterial infection is difficult to diagnose prior to clinical signs appearing. Vaccination will reduce losses due to the disease and will also reduce fecal shedding. Test and cull is expensive, and will take years to reduce the prevalence of disease. It is important to combine any control program with sanitary measures that reduce the transmission of the bacteria to replacement animals and other uninfected animals in the flock.

Résumé

La maladie de Johne (ou paratuberculose) chez les ovins et les caprins semble un problème courant dans les troupeaux de brebis et de chèvres laitières de l'Ontario. Toutefois, l'infection bactérienne est difficile à diagnostiquer avant l'apparition des signes cliniques. On sait que la vaccination réduit les pertes dues à la maladie et l'excrétion du microbe dans les fèces. Le dépistage et la réforme des animaux coûtent cher et prendront du temps avant de diminuer la prévalence de cette maladie. C'est pourquoi il est important de combiner tous les programmes de lutte dont les mesures sanitaires visent à réduire la transmission de la bactérie chez les animaux de renouvellement et les animaux non infectés du troupeau.

Introduction

Johne's disease in sheep and goats has been less well studied than the disease in dairy cattle, although it may be that the clinical effect is worse and the prevalence of disease is more widespread in those species. Recent research in Ontario dairy sheep and dairy goat flocks examined the prevalence of Johne's disease through the use of several different diagnostic tests. At least one fecal sample was positive by PCR in 24 of 29 goat herds and by culture in 23 of 29 goat herds. At least one fecal sample was positive by PCR in 15 of 21 sheep flocks (culture data not yet available). This suggests that in Ontario, Johne's disease is a common and important infectious disease of dairy small ruminants. Control is hampered by a lack of good diagnostic tools, suitable vaccines, and an understanding of how to limit infection. This talk will attempt to shed a little light on this topic, and hopefully will aid veterinarians working with small ruminant clients.

The Agent

Johne's disease is caused by *Mycobacterium avium* subspecies *paratuberculosis* (MAP), although there are different strains that infect sheep, goats, and cattle. Ovine strains tend to infect sheep and less often cattle, and cattle strains infect both sheep and goats, although how infectious they are to sheep remains to be further elucidated. There is current evidence in Ontario that cattle strains do infect sheep. Goats can also become infected with sheep strains, but disease from cattle strains appears to be worse.[12] Type I (sheep strain – also called "S" strain) is very slow growing and Type II (cattle strain – also called "C" strain) is much faster growing and less fastidious.[15] There is also described an intermediate strain – a sub-group of Type I, called Type I/III that is even more slow-growing than Type I.[15]

Transmission and Pathogenesis

Young animals are more susceptible than adults to infection, although adult sheep may be infected in the face of high environmental challenge [16]. The dose of bacteria is also linked to the likelihood of infection, as is the genetic susceptibility of the animal [16]. The presence of anti-MAP antibodies is not protective and may actually enhance uptake of the bacteria in the gut [16]. Route of infection is usually oral, either from feces or MAP infected/ fecal contaminated colostrum/milk in the neonate. The bacteria may invade either through the tonsils or Peyer's patches. The bacteria may also infect transplacentally. Macrophages contain the bacteria and either eliminate or slow its replication. If the bacteria are not eliminated by the animal's immune system, then the disease progresses. The granulomatous lesions are due to the chronic immune response to the bacteria's multiplication. MAP at this time may spread to other organs including lungs, liver, uterus, and mammary glands, and bacteria are shed in the faeces.

Clinical Signs

In both sheep and goats, the signs are vague and not pathognomonic for Johne's disease. Clinical signs may appear as young as 12 months of age – much

younger than cattle.[12] Animals lose weight in the face of a moderate appetite, and feces are pelleted or soft in consistency. Terminally a proportion may develop diarrhea, bottle jaw and/or moderate anaemia, and generally death is secondary to severe wasting. There are many other diseases that may present very similarly, e.g. dental disease, lentivirus infection, caseous lymphadenitis, internal parasites, and scrapie. To further confuse things, adults with early clinical Johne's may also be more likely to suffer from internal parasites because of a weakened immune system. Because of the vagueness of signs, it is very important that adults with chronic wasting be subjected to a necropsy and that samples are taken to detect MAP infection. Not all animals on a given farm are equally susceptible to infection, and work looking at candidate genes for resistance in goats and sheep is being carried out.

Diagnosis

Necropsy

Necropsy provides the best diagnostic test. On necropsy, the changes to the intestine may be very subtle depending on the form present in the animal. They are often classified as Type I, II, IIIa, IIIb, IIIc (see Table 1 for full explanations of the forms).[10,11] Lesions are often restricted to the distal ileum and ileo-caecal and mesenteric lymph nodes. MAP infections may be focal and delimited, multi-focal and diffuse. The diffuse form can be either lymphocytic or non-lymphocytic.[2] The lymphocytic form tends to have a better cell-mediated immune response versus the non-lymphocytic, in which humoral responses are stronger. Macrophages are the predominant inflammatory cells in the non-lymphocytic form, it is multi-bacillary, and granulomas may be seen in the Peyer's patches. The intestinal wall appears thickened grossly, and regional lymph nodes (ileo-cecal and mesenteric) may be enlarged. The lymphocytic form is paucibacillary and lymphocytes are the predominant inflammatory cell.[3] Diffuse mixed can also occur. Goats appear to be more prone to diffuse forms than sheep. Culture of tissues and histopathology can be used to confirm the diagnosis. Use of immunohistochemistry will more reliably identify intracellular MAP bacteria compared to acid-fast staining, which can lack specificity.[4]

Fecal culture

MAP takes weeks to months to grow, with Type I taking three to four months and Type I/III up to six months, compared to eight to 12 weeks for Type II.[15] The media requirements and whether to use solid or liquid media also varies by strain type. Sheep strains tend to grow better in liquid media (radiometric). It appears that in goats, fecal shedding is detected earlier than immune responses and goats can be cultured positive as

early as nine months of age. At least 25% of goats may shed bacteria but be antibody negative, depending on the stage of infection.[9] Better results are obtained when culturing multibacillary versus paucibacillary forms.

Pooled fecal culture is used in sheep to reduce costs of screening large flocks.[5] Sensitivity is reported to be high at 92%[14]. Generally seven pools per flock are necessary if detecting a prevalence of 10% or higher. Each sheep is represented by one fecal pellet. The number of samples per pool should be substantial, as this will represent a greater proportion of the flock. Fifty sheep are generally recommended. However, for smaller flocks, smaller pool sizes will accomplish the same goal, as long as the number of pools remains seven or greater. If the prevalence is suspected of being lower than 10%, more pools per flock are required and pool size should be lower.[5] Sensitivity of pooling is also affected by the type of lesions (multibacillary having a much higher sensitivity than paucibacillary).

Polymerase Chain Reaction (PCR)

The advantage to using PCR is that it is rapid, and most diagnostic laboratories are able to perform the test. It is more expensive than serology but for goats at least, appears to have a higher sensitivity. Specificity depends on the primers used, with the most commonly reported as *IS900* sequence. Although it will detect low levels of bacteria, it may cross-react with other mycobacteria. More specific genes are used, but may be single-copy and so may lose sensitivity. However, a quantitative PCR has the advantage of determining high shedders and targeting the correct animals for culling. The *hspX* gene is currently being used in diagnostic PCR tests.[a] It appears to be very specific and sensitive.[1] PCR can also be used on bulk-tank or individual milk samples in goats.[6] More work on the performance of these tests in Ontario dairy sheep and dairy goat herds will be presented at the meeting.

Total protein

One of the clinical features of small ruminant paratuberculosis is bottle jaw, an indication of advanced disease. With the disease in sheep, it has been found that with both the multi- and paucibacillary forms of Johne's, affected animals have low total protein (51.4 versus 70.4 g/liter) due to a profound hypoalbuminemia (14.1 versus 32.7 g/liter), with a normal gammaglobulinemia (37 g/liter) when compared to healthy adult ewes.[13] This quick "sheep-side" test can be used to as a diagnostic tool when investigating chronic wasting in adult sheep.

Antibody tests

Antibody tests, usually performed on sera but sometimes performed on milk, appear to have a moder-

Table 1. Classification of pathological lesions of Johne's disease in sheep.

Type	Site	Lesion Description	Cell Type	Bacterial Numbers	AGID +ve	ELISA +ve
1	Ileocaecal aggregated lymphoid follicles (Peyer's patches) and occasionally lymph nodes	No gross changes. Histopathology: Small granulomas	Macrophages	Rare	0%	12%
2	Peyer's patches and associated intestinal mucosa	No gross changes. Histopathology: Small granulomas	Macrophages	Present in variable amounts & only in the granulomatous lesions in the lamina propria	33.3%	33.3%
3a	Peyer's patches, and associated intestinal mucosa as well as intestinal mucosa distinct from Peyer's patches	Grossly only slight enlargement of serosal lymph vessels in ileum. Histopathology: Small multifocal granulomas not associated with lymphoid tissue, mostly in the ileum and occasionally jejunum. Also in caudal mesenteric lymph nodes and ileo-caecal lymph nodes.	Macrophages		0%	80%
3b		"Multibacillary". Grossly the intestinal wall was thickened and corrugated in ileum and occasionally jejunum. Dilated lymphatics on serosal surface. Mesenteric and ileocaecal lymph nodes enlarged. Animals are emaciated and some with bottle jaw Histopathology: Mosaic appearance to the mucosa. Widespread in the lamina propria. Villi are wide and flat with occasional erosions. Lymphangitis and lymphangiectasis present on serosa.	Large numbers of macrophages with few lymphocytes and giant cells	Abundant bacteria present in lesions	95%	95%
3c		"Paucibacillary". A diffuse granulomatous enteritis but cell types are different than 3b. Lesions of the serosa and mucosa are similar to 3b but edema of the submucosa is also frequently present so that the intestinal wall appears more swollen.	Large numbers of lymphocytes with scattered macrophages and giant cells	Absent or present in very low numbers	20%	20%

Adapted from Perez V, Garcia Marin JF, Badiola JJ. Description and classification of different types of lesions associated with natural paratuberculosis infection in sheep. *J Comp Path* 1996;114:107-122, and Perez V, Tellechea J, Badiola JJ, Gutierrez M, Garcia Marin, JF. Relationship between serologic response and pathologic findings in sheep with naturally acquired paratuberculosis. *Am J Vet Res* 1997;58:799-803.

ate to poor sensitivity, regardless of which test is used. The traditional test is the agar gel immunodiffusion (AGID) test, and sensitivity in clinical animals is usually quoted to be around 40%, with better performance in goats.[8] Some ELISA tests report slightly better or similar sensitivities.[8] If one wishes to use an antibody test to determine if Johne's is present in the herd, a large proportion of the group (usually those animals > two years of age), need to be sampled. For example, if the test sensitivity is 40% and the specificity is 100% (as is believed with the AGID), to detect one infected animal in a herd with a prevalence of 10%, you must sample

50 animals in a herd size of 50, 65 animals in a herd size of 100, or 72 animals in a herd size of 500.[b] More work on the performance of these tests compared to fecal culture and PCR in Ontario dairy sheep and dairy goat herds will be presented at the meeting, which should aid in a decision on which diagnostic test to select and how to best use it.

Control of Johne's Disease

The usual goal is to control Johne's disease in a flock or herd rather than eradicate. Persistence of the organism in the environment or in local wildlife, poor accuracy in many of the diagnostic tests as well as the high cost of those tests, makes eradication of MAP infection very difficult. However, the potential that MAP may play a role in Crohn's disease in humans, and the presence of MAP bacteria in milk and meat products from infected farms, strengthens the argument that eradication is most prudent or at least the goal to reduce shedding and contamination of food products should be pursued. Many control programs have focused on prevention of infection of replacement animals. This includes identifying infected, shedding adults and culling, reducing environmental contamination with manure, reducing environmental and colostrum/milk challenge with MAP to replacement stock, and selecting low-risk replacements. Individual testing of adults and culling, when done, should use the most accurate (fecal culture, fecal PCR which can be used in vaccinated animals), and/or the most economical (ELISA on serum or milk) diagnostic tests. However, for small ruminants, the cost of individual diagnostic testing is very expensive when compared to animal value. Vaccination allows improved prevention of transmission to replacements and is more cost-effective than using a test-and-remove strategy, given the low sensitivity of currently available diagnostic tests.[7] Producers and veterinarians should use a test-and-cull protocol as much as they can afford, but should also use sanitary measures to reduce challenge to young-stock.

Vaccination

In the US and Canada, there is no vaccine approved for use in sheep and goats. However, vaccination as a method of control is appealing, as no diagnostic testing is required beyond confirmation of the disease in the flock, and the clinical effects of the disease are mitigated. The vaccine will not allow for eradication of the disease and may not always suppress shedding, so that vaccination must continue in replacement stock for control to be effective.[8] Vaccination is a component of Johne's control in many countries including Australia, New Zealand, and Europe, with the only drawback that vaccination may

interfere with tuberculosis control programs. Commercial vaccines are killed, and contain irritating adjuvants to enable continuous liberation of antigens.[2] Lambs and kids are generally weaned and two to four months of age when vaccinated, and not older than eight months. Vaccination in either sheep or goats lowers mortality from MAP infection, reduces the isolation rate of MAP from the feces or tissues at necropsy, and reduces the severity and prevalence of pathogenic lesions,[2,8] indicating that commercial vaccines provide a real benefit in the control of Johne's disease in those species.

Preventing Introduction of Infection to a Herd

This is very difficult, as diagnostic tests have low sensitivity, are expensive, and the incubation period is long. Producers who purchase even one replacement per year (e.g. a ram or buck), can easily introduce Johne's disease. It may be years after that initial introduction before the producer becomes "annoyed" at the number of cases of chronic wasting that are occurring. It may be that the original animal was not even diagnosed if it left the herd before developing signs. Recommend that replacements be single sourced from a herd or flock that is closed, and has a known low-risk Johne's disease status through monitoring of culls and deaths of adults through necropsy (ideally) as well as culture, PCR or antibody testing.[12] If this can't be accomplished, using semen collected at an approved facility will be a low-risk way of introducing new genetics.

Slowing the Spread of Infection in a Herd

Below are some recommendations written for dairy sheep and goat producers that can be instituted in dairy or meat operations. They may need to be tailored to fit the management constraints of the producer, but can serve as a starting point.

How is MAP transmitted?
- From the environment. MAP is very resistant to destruction in the environment. It is shed in the manure of infected animals, six months to a year before the goat shows signs of disease. These bacteria – which have a very tough outer wall – can survive in a barn and on pasture for more than one year. This means that any manure-contaminated object or substance (feed, soil, bedding, water, teats, hair, housing equipment, feed handling equipment – the list goes on) can serve as a source of infection to the animal. So any control program has to focus on the fecal-oral route of transmission (i.e. risks of the animal ingesting anything contaminated with manure).

- In the uterus. When the disease is advanced in the adult, the bacteria will move throughout the body and can infect the fetus while still in the uterus so that it is born infected.
- Through the colostrum and milk. The MAP bacteria can be shed in the colostrum and milk. This is a very important source of infection to the newborn. It may also pick up the bacteria from nursing on dirty teats, suckling on dirty hair/wool or being born into a dirty pen.

MAP is not transmitted through the air, and likely not effectively transmitted through milking or multi-use needles. However, it may be transmitted through saliva; more work is needed to determine how important this risk is. Although infected males can shed it in the semen, the bacteria shed in the male's manure are likely more important as a method of transmission.

How to control Johne's in your flock / herd

What follows are guiding principles. Appropriate modifications can be made to optimize the program in a specific herd. It will not be possible in the short term to clean up the current adult herd without performing testing on all adult animals. This program focuses on preventing transmission to the replacement animals and establishing a "new" lower-risk herd that will eventually replace the current herd.

This means it is critical to have sufficient labor to attend all births at kidding/lambing as one of the keystones to the success of this program. The best idea is to have females checked every hour or two with the intent of immediately removing and processing newborns ("snatch and rear"). It is best to focus these measures only on female lambs or kids and male lambs/kids that have ram/buck potential, leaving market animals with their dams longer to drink colostrum. If in doubt regarding which animals will be replacements, it is best to snatch and rear all of them.

Animal identification

Easily readable, permanent unique identification is critical to the success of this program. Radio frequency identification tags will reduce labor and mistakes, fit nicely into a milk recording program, and will reduce the need to grab ears and attempt to read tags. A better ID system will pay back immediately not only in terms of properly tracking animals, but also in terms of labor savings.

Breaks in the program

Because breaks in this program will occur, if the kids/lambs did suckle or a kidding/lambing shift went awry, it is ideal to tag those offspring differently (e.g. different colored tags) to differentiate between those that are suitable replacements (i.e. made it through the "snatch and rear" program) versus those that should be sent to market (i.e. either unsuitable as replacement stock or likely nursed from their dam).

Reduce exposure to environmental bacteria

Make sure the young-stock are not exposed to manure from potentially infected adults.
- Remove the kids/lambs immediately at birth; this will prevent them from ingesting contaminated colostrum or milk, and will reduce exposure to adult manure.
- Avoid build-up of manure on dam's udder as this will help avoid fecal-oral transmission if the kid/lamb nurses.
- For dams with long hair or wool, clip the udder and escutcheon area prior to birthing. This prevents fecal material from building up in this area and having the kid/lamb come into contact with it if they do nurse.
- Keep feed equipment, feed and water troughs free of manure; routinely dump sediment that gathers at the bottom of water troughs. Keep the area around feeders and waterers clean and dry.
- Pen replacement kids/lambs in penning that is cleaned and disinfected thoroughly between groups. As this group moves through the barn, use an all-in, all-out system to facilitate this. Do not pasture or dry-lot in areas which have previously had any goats, sheep or cattle (including young-stock) from the infected herd.

Process newborns quickly and appropriately:
- Prevent kids/lambs from nursing dams.
  - Removing as soon as born is ideal, but sometimes difficult to do 100% of the time. Teat tape applied to dam's teat ends prior to birthing will temporarily frustrate the kid/lamb from successfully nursing, but is not a substitute for timely "snatch and rear".
  - Removing immediately is critical – as soon as it slips out of the dam. Don't even let her lick it off.
- Move the kids/lambs to a warm location and dry off with clean, freshly laundered towels. Dip navels in 2½% tincture of iodine. Administer a vitamin E/selenium injection if selenium is not already present in the dam's late-gestation diet. Insert suitable ID tags, and immediately feed colostrum.
- Some producers will use washable "Rubbermaid®"-type tubs that can be purchased from home hardware stores. Put some straw in the bottom for absorption and traction for the kids/lambs. These can be easily hosed out and disinfected when they become soiled – and can be stacked

for storage when kidding/lambing is done. Cardboard boxes can be used as well. When they get dirty, they can be burned or composted.
- Once dried, processed, and fed their first feeding of colostrum, kids/lambs can be moved to the rearing area. Until one week of age, try to keep them in small groups (< 10 per group) so that you can more easily detect sick animals.
- Ideally, the person caring for the newborns should not be the same person that treats sick animals, as they can easily transmit bacteria, parasites, and viruses that cause diarrhea. If labor is short, have this person change coveralls between these groups, wash hands, and wear disposable gloves when handling.

Prevent young-stock from ingesting contaminated colostrum or milk
- If possible, feed colostrum from test-negative animals if certain of their status.
- There are a few different options for feeding "safe" colostrum:
- *Heat-treat colostrum from infected dams or dams of unknown status.*
  - It is best to collect colostrum from healthy-appearing older dams.
  - Collect "first-milking" colostrum only, regardless of what subsequent milking colostrum looks like.
  - When collecting colostrum from these animals clean the udder thoroughly before collection, including disinfecting the teats. This is to reduce the chance of MAP-containing manure contaminating what might be healthy colostrum. Wear disposable gloves when milking to prevent contamination from hands.
  - Heat-treat colostrum by warming to 133 - 140°F (56 - 60°C) for 60 minutes. This is a cooler temperature than pasteurization of milk, and will kill some but probably not all bacteria. Do not allow the temperature to become hotter than 140°F (60°C) or cooler than 56°C. If cooler, more MAP will survive. If hotter, the immunoglobulins (also called gamma globulins, antibodies, or IgG) in the colostrum will "cook", resulting in inactivation, and ruin the colostrum for feeding.
  - Time the 60 minutes from when the temperature of the colostrum first reaches 133°F (56°C).
  - While these temperatures are known to effectively kill CAE virus, some MAP bacteria may survive this process.
  - Other bacteria are also killed by this process, with the benefit that immunoglobulin absorption by the neonate is actually improved by the heat-treating process, thus better protection from disease.
  - Water bath-type pasteurizers (e.g. Weck canner) are able to keep the temperature constant. There are also home milk pasteurizers that claim they are also suitable for heat-treating colostrum (not all pasteurizers are suitable). These may cost $300 to $700, but are worth it in terms of labor savings and improved health of the offspring.
  - Heat evenly in small batches, then freeze and label with the ID of the donor ewe/doe. This is important in case that animal becomes clinical with JD in the next few months. That colostrum would have been higher-risk than colostrum from a healthy female, and kids or lambs that were given that colostrum should be identified and possibly put on the market list.
- *Colostrum from cows.* There are advantages and disadvantages to this:
  - Advantages are that large volumes of good-quality colostrum can usually be easily sourced.
  - Disadvantages are the immunoglobulins are not to the "farm bugs"; the cows may also be infected with MAP, therefore the colostrum should also be heat-treated; cows should be vaccinated against clostridial diseases to assure protection of the kids; and unusually, we sometimes see anemia develop in kids a few weeks after feeding cow colostrum.
  - Heat-treat cow colostrum as with goat or sheep colostrum. Obtain from healthy older cows – first milking only – and ideally from cows that are only moderate milk producers. This is to reduce the dilution of the immunoglobulins with milk.
  - Label each batch with the ID of the donor cow in case you later identify problems. If so, the remaining colostrum can be discarded.
- *Use colostrum replacement products.* Again, there are advantages and disadvantages:
  - The biggest advantage is that there are tremendous labor savings as safe colostrum is always available.
  - The disadvantages are the high cost, and that the source of the colostrum is bovine, so it may not have immunoglobulins to the "farm bugs".
  - It is important to use appropriate volumes as under-feeding will result in kids that are more susceptible to diseases such as pneumonia, diarrhea, coccidiosis, and soremouth.

- It is important to only use products licensed and labeled as colostrum replacement (not supplement). Colostrum substitutes should all have bovine immunoglobins present as at least 10% of the volume by weight of the product.
- Freezing and thawing colostrum.
  - Freeze in volumes that will be easy to thaw (e.g. ice cube size).
  - Once frozen, move to plastic freezer bags that are labeled using a permanent marker (Sharpie). Double bagging will reduce "freezer burn".
  - Store in a chest freezer at -4°F (-20°C).
  - Use oldest colostrum first (should not be older than six months).
  - Thaw at room temperature or in the refrigerator (not the microwave, as it will unevenly heat and will destroy the immunoglobulins). Thawed colostrum can be stored in the refrigerator for up to one week. Best is to purchase a chest freezer and refrigerator just for the kid/lamb-rearing operation. This will assure that you have lots of space to properly store colostrum.
- Proper amount of "safe" colostrum to feed
  - At the first feeding, which should be within an hour of birth (the sooner the better), the volume of colostrum should be 22.7 ml/lb (50 ml/kg) body weight. If an average-sized kid or lamb is ~ 8 ½ lb (4 kg), then it needs to drink 200 mL or almost seven ounces. Bigger kids/lambs need more.
  - It can be fed by nipple bottle (must be cleaned and disinfected between uses and stored away from flies) or esophageal feeder tube (ditto for cleaning). Bacterial contamination of feeding equipment will prevent proper absorption of the immunoglobulins and will make the kid/lamb sick.
  - Colostrum may be sticky and thick – if too thick for the tube or nipple, it can be diluted slightly with clean, warm water, but the original volume of colostrum must be maintained.
  - Over the first 24 hours of life, the kid/lamb needs to consume 90 mL/lb (200 mL/kg) body weight (~ 3 ounces/lb) of colostrum, or 20% of its total body weight. The best way is to repeat the second feeding three hours after the first, and then the next two feedings six and 12 hours later.
  - Always use first-milking colostrum for the first 24 hours of life. If second to sixth milking colostrum is used to feed neonates older than 24 hours, it must also be heat-treated prior to feeding.

- For commercial products, follow directions on the label as a minimum recommendation.
- Do not feed unpasteurized milk to kids/lambs; use either pasteurized milk or milk replacer. For batch pasteurization of milk, heat to 145°F (63°C) for 30 minutes. For flash pasteurization, heat to 162°F (72°C) for 15 seconds.

Prevent spread of MAP bacteria on the farm
- Reduce risk of potentially infected replacements from transmitting the disease.
  - Sell young-stock born to dams diagnosed with JD directly to slaughter; do not keep as replacements or sell as breeding stock. These animals are more likely to have picked up JD while still in the uterus.
- Provide clean, dry environments for kidding.
  - Use deeply bedded, clean pens that are free of manure for birthing. Clean the pen after the group has finished giving birth, or if the pen becomes contaminated with manure.
- Disinfect pens and equipment between groups of animals.
  - MAP is susceptible to five minutes of exposure to a phenolic germicidal detergent. This will help to reduce the number of MAP in the environment. All visible manure must be removed from the surface that this product is to be used on before applying the product.
- Quarantine any unthrifty animals in the herd and contact the herd/flock veterinarian.
  - Test any animals you suspect may be showing signs of JD or CAE; severely wasted animals should be sent for a postmortem exam by a veterinary pathologist to confirm the cause of death. This is very important to do for the new "low-risk" herd/flock.
- Keep the low-risk herd separately housed from the infected herd.
  - The low-risk herd is comprised of replacements fed safe colostrum and milk, and not reared on the dam. Ideally, they should be animals that did not have an opportunity to nurse does/ewes or that spent more than an hour or two exposed to manure that may contain MAP bacteria.
  - Kids/lambs destined for market (i.e. allowed to nurse or fed regular colostrum or milk) should ideally be housed separately from low-risk animals. Although opportunities for infection are low while this young, there is always a chance the kids are already infected, and you have spent too much labor and time to risk this. Market animals should leave the herd before four months of age.

- Milk the low-risk herd first to reduce risk of exposure to the infected herd.
  - Between milkings, animal traffic areas of the parlor should be cleaned and hosed down to prevent manure buildup.
  - Feeders in the parlor should either be removed entirely or disinfected between milkings.
  - Waterers should be in the pens only (not holding areas) so that does of differing health status don't share a waterer.
  - When moving the low-risk herd past the infected herd (or vice versa), make sure no opportunity exists to share feeders or waterers. Do not hold animals where any contact between groups can occur.
- Manage exposure to manure properly
  - Manure clean-out should be from the low-risk herd first. Clean all feeding and manure management equipment after using it in the infected herd.
  - Do not share pastures or dry lots between low-risk and infected herds.
  - Compost all manure a minimum of five months; turn for thorough heating. Make sure runoff from manure does not enter dry lots, pastures or water sources.
  - Do not spread manure on pastures or hay-fields, but rather fields intended for growing crops. Manure should be plowed in before seeding.
  - All water sources should be from drilled wells (rather than dug wells or surface water) so they are at low risk of being contaminated with manure or manure runoff.

Cull the infected herd as quickly as possible

After making a diagnosis in a herd or flock, it is prudent to assume that all animals are either infected or sufficiently exposed to be a risk. This means that the goal is to quickly buildup a low-risk herd, eliminate the infected herd – also quickly - while staying in business. Below is an example of how this could be done in a dairy goat herd:
- Normal cull and adult mortality in a goat herd is 25% per year (20% cull and 5% mortality).
- The goal is to raise 0.7 healthy low-risk doe kids for every doe in the herd, e.g.
  - 1.7 kids born per doe kidding
  - 10% kid mortality = 1.5 kids reach weaning per doe kidding
  - 50% are doe kids = 0.75 doe kids reach weaning per doe kidding
  - One in 20 (5%) are not successfully "snatched and reared" (e.g. nurse) or are born to clini-

cally JD does, so they are moved to the market kid group.
- This leaves 0.7 potential replacements for each doe kidding. Some of these kids will not be suitable replacements (parrot-mouthed, poor doers etc).
- Pregnancy rates are lower in doe-kids and so you may expect 90% of retained, suitable kids to give birth by 12 to 15 months of age.
- With this number, it is possible to replace at least 40% and as high as 50% of the herd each year. This allows the producer to put quite a bit of selection pressure on the infected herd. All does should be sent to slaughter only.
- Within three to four years, depending on reproductive and kid-rearing success, the producer can totally eliminate the infected herd. The faster, the better.

Monitoring the low-risk herd/flock

The best test is to necropsy any thin adults. Pooled fecal culture is the next best tool, and could be done annually. If any sample is positive, then the contributors to the positive pool(s) should be tested individually and removed from the flock/herd. Serology could be done in this case, but not if vaccination is also used.

Conclusions

Johne's disease in sheep and goats appears to be common in Ontario flocks and herds, particularly dairy. Vaccination is likely the best method of control, but if not available, control is done by test-and-cull, combined with reducing transmission of infection to replacement animals.

Endnotes

[a]Tetracore VetAlert™ Johne's Real-Time PCR, http://www.tetracore.com/index.html
[b]FreeCalc Version 2, http://www.ausvet.com.au/content.php?page=software

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