ABORTION IN SHEEP AND GOATS – WHAT EVERY PRACTITIONER SHOULD KNOW

Paula I. Menzies DVM MPVM Dip. ECSRHM
Department of Population Medicine
Ontario Veterinary College
University of Guelph, Guelph, Ontario, Canada N1G 2W1

INTRODUCTION

Abortion as either an outbreak or enzootic disease issue is an important production-limiting disease of sheep and goats. The following describes how to approach diagnosis and management of the more common causes of abortion in USA and Canada, i.e. *Campylobacter fetus* subspecies *fetus*, *Campylobacter jejuni*, *Chlamydophila abortus*, *Coxiella burnetii* (Q fever), *Toxoplasma gondii* and iodine deficiency goiter. It also gives an overview of the less common causes of abortion in sheep and goats. Zoonotic implications of the various disease agents will also be addressed as well as how to reduce risk to humans.

Unfortunately significant losses associated with abortion in sheep and goats are quite common but fortunately laboratory diagnosis is often more rewarding than with other livestock species. In this presentation, I will cover how to approach diagnosis and management of small ruminant abortion, the more common causes and zoonotic implications.

DEFINITION OF ABORTION

The definition of abortion is any pre-term loss of a foetus or embryo. Pregnancy loss prior to day 12 after conception will result in an early return to oestrus when it occurs during the breeding season. Later pregnancy loss will present variably as one or more of the following observations: delayed return to oestrus; a female believed to be pregnant but that fails to give birth and no abortion is observed; a blood-tinged vaginal discharge observed but no foetus or placenta is found; abortion occurs and a foetus and/or placenta is found; lambs / kids born at term (> 142 days of gestation) either stillborn and/or weak. Sometimes all of these may present in the same outbreak. The foetus may be delivered macerated, mummified, decomposed or freshly dead, and alive.

INVESTIGATING THE ABORTION PROBLEM

Presenting Complaint:

The classical presenting complaint is usually numerous abortions clustered in time, generally starting one to two weeks before the ewes / does are due to start giving birth. Normally, no more than 2% of females should abort each gestation. A rate of 5 to 10% is abnormal and is seen in flocks with endemic disease. Abortion “storms” can affect a significant portion of the pregnant flock within a short period of time. But the veterinarian should remember that any of the following could indicate an abortion disease issue: decreased lambing/kidding rate; non-viable foetuses seen on ultrasound; increased rate of return to oestrus; and / or increased still-birth rate and poor viability of newborns.

History:

The history will help create an appropriate list of diagnostic hypotheses. The following information will help:

- Number, proportion, type, age and source of animals aborting;
- Plotting out on a calendar when abortions occurred (clustering) and gestational age;
- Recent introductions to the flock/herd (even virgin replacement females) or sharing of animals in the last year including rams or bucks;
- Previously diagnosed abortions or illness on the farm;
- Vaccination history, including abortion vaccines and timing and frequency of administration;

- Nutritional and grazing history, e.g. salt / mineral supplementation, silage feeding or toxic weed exposure;
- Potential exposure to toxins including drugs, e.g. an anthelmintic with a known teratogenic effect;
- Environmental factors, e.g. extreme heat during early pregnancy, stress, predation, presence of cats, rats on the property;
- Clinical illness in the individual ewe / doe before, during or after abortion.

General Inspection:
Assess the environment in which the animals are housed. Are the animals in good bodily condition, is there evidence of diarrhea, do they have access to water, palatable free-choice salt and mineral. Is there evidence of rodent, cat feces in vicinity?

Clinical Examination:
The Adult: Examine both the pregnant at-risk animals as well as aborted. If the females are ill, this might support some hypotheses such as listerial abortion or salmonellosis. Often the females look healthy while aborting but may have had a history of being transiently off-feed such as with *Campylobacter* spp.
The Fetus: Determine how premature the fetus is (size, crown-rump length, evidence of wool/hair coat). Was the fetus alive when aborted? Is the abortion fresh, macerated or mummified? Look for skin lesions that may indicate a mycotic or bacterial infection. Are there congenital defects such as arthrogryposis, spinal bifida, cleft palate, microopthalmia, etc? Is the fetus meconium stained?
The Placenta: Examine the cotyledons for evidence of inflammation (swelling, hyperemic, purulent debris), necrosis or calcification. Check the intercotyledonary space for evidence of inflammation, thickening, necrosis, hyperemia, etc. Normal intercotyledonary placenta should be transparent and thin, e.g. can read the paper through it.
Submission for Diagnostic Testing:
In almost all cases laboratory support is necessary to make a definitive diagnosis. Have the producer gather up all the abortions and placentas available. They should be placed in a clean, water-proof sac (e.g. garbage bag) and kept cool and away from scavenging animals (e.g. rodents, cats, and dogs) prior to submission. Instruct the client regarding the zoonotic risk and to wear gloves and protective clothing. Submission of placenta is critical.
When submitting, it is very helpful to indicate a list of diagnostic hypotheses and to direct the pathologist on what you consider the most likely cause(s) of the abortion. In many diagnostic laboratories, the pathologist cannot run tests unless you request them. For this reason, your input is critical for the success of the diagnostic investigation.
Submission of Entire Fetuses and Placentas: Gently remove debris but do not wash off; submit all specimens available; do not freeze before submitting but do keep chilled; submit in leak-proof clean containers.
Necropsy and Submission of Specimens from Fetuses and Placentas: It is not always possible to submit entire fetuses and placentas. In this case, perform a gross necropsy, make note of any abnormal findings, and submit as outlined below or follow your state diagnostic labs recommendations on appropriate samples to submit. Record the weight of the fetuses, estimated gestational age, and history.
Submit a separate tissue sample for each lab section, i.e. bacteriology, virology and mycoplasmology in separate, sealed and labeled Whirl-Pak bags. Keep fresh tissues chilled using ice packs in insulated leak-proof containers. Submit tissues showing typical lesions.
- **Placenta formalin-fixed**. At least 2 cotyledons + intercotyledonary area. Include areas with obvious lesions.
- **Placenta - fresh.** Place into separate sealed bag. *Coxiella burnetii* may be present in large

numbers on the placenta and should be carefully handled. PCR for *C burnetii* and *Chlamydophia abortus*

- **Fetal Tissues - Formalin-fixed**. Eyelid, skeletal muscle, thyroids (submit with tracheal section), thymus, lung, myocardium, liver, kidney, adrenal, spleen, jejunum, spiral colon (with meconium), and brain. Immunohistochemistry can be requested for several of the disease agents, e.g. *Toxoplasma gondii.*
- **Fresh in one bag.** Lung, spleen, liver, thymus and thyroid for isolation of Border Disease virus (BDV).
- **Fresh in one bag** - lung and spleen for *Chlamydophila abortus* ELISA (do not include placenta).
- **Fresh in one bag** - lung and liver for bacterial and mycoplasma culture.
- **Stomach content** (in sterile leak-proof container) for culture.
- **Fresh in serum tube** - fetal thoracic fluid or heart blood (serum) for analysis for titres to BDV and *Toxoplasma gondii.*

Serum from Females: Serology is not as rewarding as a titre doesn't always indicate causality and it is usually costly. However, if aborted fetuses and placenta can't be obtained, serology may offer some clues as to why the abortions occurred. Sample all aborting females and a portion (minimum of 10%) of pregnant ewes/does. Submit paired sera - acute and convalescent (10 to 21 days after acute sample) to demonstrate a rising titre.

Managing the Aborting Flock:

While developing a therapeutic plan, there are actions that may influence the severity of the outbreak or may reduce the risk to humans. Remove the *pregnant* females from *aborted* females, which should remain in the contaminated pen or pasture. If you have a working diagnosis, it may be prudent to initiate specific control measures before the diagnosis is confirmed. If the aborted females are to be culled, they should be sent directly to slaughter once the vaginal discharge has cleared, to avoid the risk of being taken into another flock as a breeding animal.

Zoonotic Risks:

Be aware of the zoonotic risks from many of the infectious agents. Advise the producer and others working with the animals to wear gloves, boots and protective clothing that are changed before managing the rest of the flock. These clothing items should never go in the house but should remain in the barn and should only be used when managing the aborting animals. Fitted N95 respirators are also recommended as some of the disease agents can be aerosolized. These masks should be worn when assisting a birth or removing an abortion or cleaning the barn. Pregnant women or immune compromised people should not assist at birthing and should, if possible, not have contact with the pregnant, aborting or newly lambed /kidded females and offspring.

CAUSES OF ABORTION IN SHEEP AND GOATS

There are many regional differences in diseases that cause abortion in these species. Sometimes, knowing the history of an area can help refine the list of reasonable hypotheses. Others can be eliminated based on clinical findings. But having a knowledge base of what can occur is critical to being able to propose an appropriate therapeutic plan.

In Canada and the USA, most cases of infectious abortion in sheep and goats are linked to infection with *Campylobacter jejuni*, *Campylobacter fetus* subsp *fetus*, *Chlamydophila abortus*, *Coxiella burnetii* or *Toxoplasma gondii* and sometimes a combination of those diseases. Most cases of non-infectious abortion are linked to iodine deficiency - at least in parts of North American that are iodine deficient such as the Great Lakes region and (in Canada) the Prairie Provinces. There are many other uncommon causes which can also result in dramatic losses. Some are briefly covered here. A more complete list is presented in Table 1.

Campylobacter Species

The Agent: Both *Campylobacter jejuni* and *C. fetus* subsp *fetus* have been implicated in ovine abortion. Abortion in goats from these agents is unusual. The bacteria reside in the intestinal tract of sheep and many other species, including dogs and birds, including domestic poultry. Recent research (Dr. Paul Plummer and associates) suggests that *C jejuni* has a serovar which most commonly associated with ovine abortion. In central and eastern North American, it is the most prevalent cause of campylobacter abortion. *C fetus* subsp *fetus* appears to be more common in western North America in large range flocks and in New Zealand. There are several serotypes of *C fetus* subsp *fetus* (A2 and C reported in western USA versus B1 in New Zealand) although no recent work has been published on the types in North America it is likely that strains to vary over time, which may affect vaccine efficacy. Recently the Animal Health Laboratory at the University of Guelph has found an equal distribution between *C. jejuni* and *C. fetus* subsp *fetus* (Dr. D. Slavic, personal communication April 2012)

Transmission and Pathogenesis: The organisms are harboured in carrier sheep in the intestine and gall bladder. Sources of infection are from contamination of feed or water from faeces, foetuses, placenta and vaginal discharges from aborted ewes. Carrion birds such as crows may enable transmission between flocks. Incubation ranges from 8 to 60 days.

Clinical Picture: Most abortions occur during the third trimester about 3 days following foetal death with occasional stillbirth and weak lambs. Foetuses are expelled well preserved with the placenta. Ewes may have transient diarrhoea and fever. The placenta tends to be oedematous with congested swollen cotyledons. The foetus may have subcutaneous oedema and an enlarged abdomen with pleuritis, peritonitis and hepatitis with occasional target lesions of hepatic necrosis. Abortion levels of up to 70% have been reported but levels of 10 to 20% are common in enzootically infected flocks. Immunity from one species of campylobacter species is not cross-protective to another but natural immunity of up to 3 years can occur.

Diagnosis*:* Stain impression smears of cotyledons or foetal stomach contents will demonstrate the organism. The placenta and foetal abomasal contents should be cultured. Antibiotic sensitivity testing must be done on any isolates as resistance to tetracyclines is very common with *C. jejuni* in North-Central USA and in Ontario.

Control and Prevention: In the face of an outbreak, all pregnant ewes should be treated with an antibiotic that is likely to be efficacious. Long acting oxytetracycline at the label dose once (20 mg/kg) or in the feed (250-300 mg/head/day) until lambing is finished, can be used but a recent study found that most abortion isolates of *C. jejuni* are resistant to tetracyclines. The authors did find that the isolates were susceptible to tilmicosin, florfenicol, tulathromycin and enrofloxacin, and 97% were sensitive to tylosin. The feed recommendations are empirical and there is no published information on efficacy of this route of control. In Canada, there is one commercial vaccine available but no published efficacy information. Vaccination can be done in the face of an outbreak but takes two weeks to develop sufficient immunity to stop abortions. In flocks in which campylobacteriosis has been diagnosed it is preferred that vaccination be routinely done according to label directions. This involves vaccinating all breeding ewes with a bivalent killed bacterin twice, the first before breeding and the second injection 60 to 90 days later. Annual re-vaccination before breeding should be maintained, although some debate if this is necessary. A lack of efficacy of the vaccine may be due to several factors. The vaccine may not contain the correct serotype of *Campylobacter fetus* subsp *fetus* or the abortion is due to another species of campylobacter bacteria.

Chlamydophila Abortus (Enzootic Abortion)

The Agent: *Chlamydophila abortus*, previously *Chamydia psittaci* sheep abortion strain, is one of the most common causes of abortion in sheep and goats in North America.

Transmission and Pathogenesis: Transmission may occur from exposure to aborted materials, vaginal discharge or from environmental contamination and ingestion. Infected males may have *C abortus* isolated from the semen and seminal vesicles. When the infected

female becomes pregnant, the organism moves via a haematogenous route to the placental chorium. By 95 days of gestation, the infection has spread from the cotyledons to the intercotyledonary areas. The fetus also becomes infected but pathological changes are minor.
Clinical Picture: Abortion rarely occurs at less than 100 days gestation but can occur. Females infected in late gestation or when not pregnant, will abort at the subsequent pregnancy – making the purchase of replacement stock from infected flocks an important method of disease introduction. Immunity post-abortion lasts at least three years but organisms may be shed in vaginal secretions during oestrus. The placenta is necrotic, with lesions affecting both cotyledons and intercotyledonary spaces. Foetuses may be aborted necrotic, well preserved, rarely mummified, stillborn or alive and weak. Abortion levels can be very high (up to 30%) in the first year of the disease but decrease in subsequent years to 10 to 15% and then may only affect ewe-lambs / doelings and new introductions in subsequent years.
Diagnosis: The severe placentitis is typical but not diagnostic. Smears made from the chorionic villi and stained with appropriate stains will demonstrate clumps of intracellular elementary bodies. The organisms can be confused with *Coxiella burnetii*. Immunohistochemistry can differentiate these infections. Use of a specific quantitative PCR can be used on placental tissues. *C. abortus* organisms can be present when it is not the cause of abortion but numbers tend to be lower. Serology must use acute and convalescent samples to differentiate from vaccination titres.
Prevention and Control - Vaccination: Only an inactivated vaccine, a whole cell killed vaccine, is available in North America and is approved for sheep only. The vaccination procedure is to initially inject 60 days prior to breeding and administer a second dose 30 days later. Annual revaccination is required. Inactivated vaccines do not prevent shedding of chlamydiae in birth fluids so the environment remains contaminated serving to continue the infection cycle.
Prevention and Control - Antimicrobials: In an outbreak or commencing after 80 days of gestation in sheep flocks known to be infected, injections of long acting oxytetracycline at label dose (20 mg/kg bw) and repeated every 2 to 3 weeks, may prevent some abortions, although likely fewer than 50% and there is disagreement whether multiple treatments is justified. Often the placental damage is so severe that antimicrobial therapy is of limited efficacy in the face of an outbreak. Replacement females should be managed as a separate flock/herd until after lambing/kidding to reduce exposure. In chronically infected flocks, feeding levels of 250 to 500 mg/head/day of tetracycline, starting 60 days prior to the first expected lambing/kidding date, is thought to be helpful. Higher levels are generally fed during an outbreak of abortion and lower levels are fed prophylactically - although there is no published information on true efficacy and there is a risk of development of antimicrobial resistance. If dairy animals are treated with antimicrobials during gestation, the veterinarian must assure that at lambing or kidding no antimicrobial residues remain in the milk. Canadian gFARAD has established that the milk withdrawal for a single injection of long-acting oxytetracycline should be a minimum of 7 days but it is strongly recommended to test individual goats prior to shipping the milk.
Zoonotic Risk: Occasionally *C abortus* can cause significant human disease, particularly in pregnant women working with aborting or lambing/kidding females.

Coxiella burnetii (Q fever)
The Agent: *Coxiella burnetii* is a gram negative intracellular bacterium which can infect a wide range of hosts including ruminants (cattle, sheep and goats), swine, guinea pigs, cats, dogs, wildlife, rodents and humans as well as birds and ticks. It has two forms, the Large Cell Variant – which exists intracellular and grows logarithmically in an infected host, and the Small Cell Variant (SCV) – which is spore-like, is shed in birth fluids, semen, milk and feces, and exists outside the host. The SCV is very resistant to heat, freezing and desiccation and can survive many months in dust, soil, manure etc. It is this form that infects animals.
Clinical Picture – Animal: Goats appear to be most clinically affected, followed by sheep and

then cattle. Abortion rates can vary from 5% to 35%. Severe suppurative placentitis along with abortion, stillbirth and weak lambs / kids is commonly seen. Abortion in subsequent years is less common due to flock / herd immunity although stillborn and weak lambs / kids may persist. Cattle may also abort but it appears not to be as common as in sheep and goats. There is evidence that *C. burnetii* infection in cattle is associated with an increase in somatic cell counts (i.e. subclinical mastitis) but its role in infertility has not been determined. What is important to understand about this agent, is that infection is much more common than disease. However, because with infection, the organism is readily shed into the environment, it is important to consider when determining appropriate control measures that include protecting the people that work with infected animals.

Transmission and Pathogenesis: Inhalation of contaminated air or dust, or mucous membrane contact with aborted materials, vaginal fluids and membranes from normal birthing can serve as a source of infection. A cloud of organisms is present around aborting animals or even during normal parturition. Ticks may also shed the organism and contaminate the wool. The number of organisms shed is much higher when abnormal birth events occur but can still be significant when birthing is normal. The organism is shed for weeks after an abortion or normal parturition and for months in the feces and / or milk. Sheep intermittently shed in the milk but goats and cattle are persistent shedders – sometimes for several months, particularly cattle. Pasteurization will kill the bacteria in the milk. The organism can be present in the contaminated bedding and manure for months and be a source of infection when manure is spread on dry, windy days. The bacteria have been detected up to 3 miles (5 km) downwind of an infected farm.

Diagnosis in Animals: It is important to be able to differentiate *C. burnetii* is the cause of disease or if present only as infection. When determining if it is the cause of abortion, demonstration by immunohistochemistry and / or PCR is highly reliable method of diagnosis when combined with histopathology. There should be evidence of inflammation and the pathologist should confirm that if intracellular bacteria are seen on histopathology, that they are confirmed as *C. burnetii* – as they may be confused with intracellular *Chlamydophila abortus*. Culture of the bacteria is rarely done as it requires a Level 3 containment facility because of its zoonotic nature. Serology can be done to support a suspected clinical diagnosis as animals which have aborted due to *C. burnetii* are often – but not always – seropositive as determined by IFA, CF or ELISA. However, an animal may be seropositive and have a normal kidding / lambing. Additionally serological status is a poor predictor of shedding. Sheep and goats may shed vaginally, in the milk or in the feces and be seronegative. They may also be seropositive and not shed. For this reason, serology is only useful for establishing the infection status of the herd or flock and not for determining which animals in the flock are infected and shedding. It should never be used in a test and cull program.

Treatment of Animals: The current level of knowledge suggests that long-acting oxytetracycline, when injected twice at 20 mg/kg bw during mid to late gestation, is not effective in reducing the level of abortion due to *C. burnetii*. However, the studies have been small and more work should be done in this area. What is certain is that any administration of antimicrobials appears not to influence the level of shedding of the organism. For these reasons, there is no justification to recommend the administration of antimicrobials in the feed or water either in the short or long-term to control *C. burnetii* in an infected herd or flock.

Control of Infection in Animals: Because of the environmental contamination and the longevity of the organism in the environment, control should focus on lowering the level of challenge from the environment by lowering sources of contamination. As use of antimicrobials will not affect shedding in goats ore sheep, nor prevent abortion – when most of the shedding occurs, then it is important to investigate the use of vaccines in those species. No commercial vaccine is available in North America although a phase I inactivated vaccine (Coxevax, CEVA Santé Animale) licensed in Europe appears to prevent abortion and reduces shedding in goats and cattle. Less work has been published in sheep and – to-date, long-term studies of efficacy

have not been published in any species. However, there is sufficient evidence that at this time vaccination is recommended as a control measure for preventing abortion and reducing risk to humans. Cats, and rodents may also be a source for continuing re-contamination of the environment. The organism can remain viable in a dried state in the environment for months and be a source of reinfection. So at this time, continued vaccination is recommended until further research indicates if the organism can be eradicated from a premise.

Q Fever in Humans: The disease in humans makes this organism very important to control in livestock. Although most people who become infected do not become clinically ill (60%), approximately 40% develop flu-like illness with about half of those becoming ill enough to seek medical attention. Approximately 5% of people with Q fever are hospitalized. A similar percentage may go on to develop chronic Q fever. The time from exposure to clinical signs is 2 to 3 weeks but many patients do not suspect Q fever, believing instead that they have the 'flu' and may delay seeking medical attention for another week or two. Signs of acute illness include undulating fevers, headaches, malaise, nausea, rashes, shortness of breath indicating an atypical pneumonia, and sometimes hepatitis and more rarely meningitis. Acute Q fever is very responsive to the appropriate antimicrobial therapy (usually doxycycline) if provided in a timely manner. Pregnant women who develop Q fever, are at risk of severe fetal disease and should be treated as a medical emergency. Chronic Q fever is also very dangerous to the health of the human and more difficult to effectively treat and has a high case fatality rate. It is associated with chronic fatigue syndrome and endocarditis in people with pre-existing heart valve problems. Diagnosis is based on serological response of the patient (from a serum sample) and the level of Phase I and Phase II IgG and IgM antibodies are measured and interpreted. In a very early case, PCR performed on blood may be positive before antibodies are detected. It is important to note that many physicians are unfamiliar with the disease and the vagueness of the signs may delay appropriate therapy.

Control of Q Fever in Humans: Because of the degree of shedding, the highest risk for human infection is likely from working with parturient small ruminants – particularly if abortion is occurring. People may become infected from breathing in contaminated aerosols, handling infected placentas and lambs / kids, from being present in the barn during parturition, and from windborne organisms from infected premises or dried organisms in the dust of barns. Biosecurity precautions are very important to reduce risk to humans. Milk from dairy ruminants on infected farms is commonly infected with *C. burnetii* and so the consumption of raw milk is discouraged because of this. Pasteurization temperatures will kill the organism.

It is prudent for people who are considered high risk for infection, e.g. people with weakened immune systems (e.g. the elderly, those on immunosuppressive drugs), have pre-existing heart valve conditions, are pregnant or are very young – to lower their risk. This can be done by not attending lambings or kiddings and staying out of the area of the barn where ewes/does are giving birth.

However, Q Fever can occur in healthy young to middle aged adults so all people should consider that they are at risk. When in the barn, all people caring for livestock should wear protective clothing that is dedicated for animal use. This includes coveralls or overalls, boots, coats, mittens, hats or any other piece of clothing worn in the livestock areas of the barn. That clothing should remain in the barn and not be brought to the house except in a plastic bag for purposes of laundering. Laundering of the clothing should be done carefully with hot water and soap and no other clothing items be washed at the same time. All lambings / kiddings / calvings should be done weaning shoulder-length plastic sleeves and afterwards, hands and arms washed thoroughly with a disinfectant soap (e.g. chlorhexadine). When potentially exposing themselves to aerosolized organisms (at birthing events or when moving manure or contaminated bedding), it is advisable to wear a fitted N95 respirator which will filter out the bacteria. A renovator's mask is not sufficient.

Manure should be composted for at least 3 months and only moved or spread on still days to avoid creating contaminated dust. If moved off property, the manure should be covered while

transported – again to avoid exposing people to the wind-borne bacteria. Aborted fetuses, placentas or stillborn kids and lambs should be properly disposed – e.g. covered composting or burning if allowed. They should not be disposed in the manure pile.
Owners and employees on infected farms, as well as service providers that must handle the animals (e.g. veterinarians, shearers, livestock truckers, abattoir workers), should consider themselves at high risk of becoming infected with *C. burnetii*. In addition to taking proper precautions, they should visit a physician if they have signs of fever, malaise, headaches or lower respiratory disease. Because many physicians are unfamiliar with the disease, it is advisable to provide fact sheets on the signs of Q fever, its diagnosis and treatment to the physician at the office visit. These factsheet can be obtained from a number of on-line sources including the Centre for Disease Control (USA). A human vaccine is not readily available in Canada or the USA although a licensed vaccine is marketed and widely used in Australia.
Management of a Q Fever Outbreak: On occasion, both animal and human disease due to *C. burnetii* infection is significant with more than one human case occurring in a short period of time, or evidence of spread of infection beyond one farm. The Q Fever Working Group of the National Association of State Public Health Veterinarians (NASPHV) is in the process of developing a document which can be used by veterinarians to assist in managing disease in animals as well as risk to humans. The document contains details beyond the scope of this article. At the time of writing this document, the recommendations are still in draft form but should be available soon.

Toxoplasma gondii

Agent: *Toxoplasma gondii* is a protozoan parasite that in many countries, it is one of the most commonly diagnosed causes of ovine and caprine abortion. The sexual part of its life cycle is completed only in domestic and wild cats. The oocysts shed in their faeces are infective for up to 18 months when protected from desiccation and sunlight. The asexual component of its life cycle may occur in any warm blooded animal.
Transmission and Pathogenesis: Non-immune cats, particularly kittens first learning to hunt, may become infected by ingesting food or animals containing cysts , e.g. rodents, offal from slaughtered farm animals, birds and aborted foetuses and placentas. Cats will shed millions of oocysts from 4 to 12 days after ingestion and then become immune. Oocysts, which will remain infective for up to 6 months are ingested by small ruminants through contamination of feed, water or pasture. Naïve non-pregnant animals develop immunity without disease but naïve pregnant animals will also experience foetal infection as the organism has a predilection for the pregnant uterus. Infections in mice populations are persistent due to vertical transmission.
Clinical Picture: Infection of the foetus prior to 40 days results in resorption, between 40 to 120 days in maceration, mummification or abortion, and after 120 days, stillbirths or birth of weak or healthy immune offspring. Abortion levels within the flock may vary from 5% to 100%. The history usually includes contact with kittens, either directly or from faecal contaminated forages or grain. A few weeks to days before the expected onset of lambing / kidding, ewes/does may start to abort. Often all levels of infection happen within the same outbreak and sometimes within the same litter, e.g. a weak lamb may be born with a mummified fetus. Ewes / does do not appear ill. Sheep are immune but goats may abort in subsequent gestations.
Diagnosis: The foetuses may be mummified or aborted in a decomposed state. Subcutaneous oedema and blood tinged fluid with strands of fibrin in the body cavities may be seen. The placental cotyledons appear bright to dark red and speckled with white calcified foci of necrosis 2 mm in diameter. The intercotyledonary placenta appears normal. The toxoplasms are not numerous and are often seen only on the periphery of lesions. Immunohistochemical techniques help to demonstrate organisms more clearly, even in decomposed tissues. Approximately two weeks post-infection, antibody titres rise and remain high for several years. A positive titre in a ewe / doe is not diagnostic. However a positive titre from an aborted fetus indicates congenital infection. Negative titres in the dam will aid in ruling out toxoplasmosis.

Control and Prevention: Often little can be done during an abortion "storm", so control measures are important. Feeding monensin at a dose rate of 16.8 mg/head/day or decoquinate at 2.0 mg/kg/day in the final 14 weeks of pregnancy has been shown to be an effective prophylactic. Lasalocid does not appear to be effective. A modified live vaccine has been developed which confers excellent immunity for at least 18 months and probably for life but is not available in North America. Removing all cats from the farm or spaying all the queens to eliminate new crops of kittens will reduce contamination of the environment. Cats should also be prevented from defecating on hay and grain. Kitty litter boxes can be kept in the barn near where the cats tend to congregate in order to encourage their use over the feed. The top layer of hay bales should be fed only to non-pregnant animals and the grain stored in metal containers to prevent access by rodents. All of these measures may help to reduce incidence but will not prevent all possible routes of exposure. Feral cats, purchased feeds and vertical transmission within rodents may help to perpetuate infection within a farm.
Zoonotic Disease: Pregnant non-immune women are the most at risk of disease. *T. gondii* can cause congenital neurological disease and blindness in human foetuses. It is an important cause of encephalitis in humans suffering from Acquired Immunodeficiency Syndrome. Most humans probably become infected from consuming undercooked meat although handling of cat faeces should be considered as a source. Freezing meat to -12 ^{o}C for 1 day or cooking meat to 67 ^{o}C will kill tissue cysts. Microwave cooking is uneven and may leave some cysts viable.

Congenital Goiter due to Iodine Deficiency

The entire Great Lakes region has soils deficient in iodine. Most outbreaks are associated with failure to provided iodized salt to gestating females, or the salt is not readily available free choice (e.g. buried under hay or bedding, spoiled with feces).
Clinical Picture and Diagnosis: Abortion and stillbirth due to iodine deficiency goiter occur often at very high levels. The foetuses exhibit swellings on either side of the neck region that are shown to be enlarged goitrous thyroid glands. Normal thyroid glands are very small (< 2-3 mm) and enlarged glands can measure up to 2-3 cm in size. Absence of wool or hair is also commonly seen with late term abortions and in severe cases; myxedema is evident in the limbs and abdomen. Lambs and kids born alive are very weak and, unless supplemented with iodine, usually die. Boer goats appear to be more at risk of congential goiter.
Control and Prevention: Once diagnosed, the rest of the pregnant group should be supplemented with oral iodine. It has been suggested that Lugol's iodine be added to the water or that 1 to 2 ml of tincture of iodine be painted on skin once/week. To prevent problems, iodine should be fed at a rate of 0.5 to 0.8 mg/kg DM diet with the higher end recommended for lactating ewes and does (NRC, 2007). Sheep grazing goitergenic brassicas such as kale, cabbage or turnip tops may require higher levels of supplementation up to 2 mg/kg DM. These plants interfere with I uptake or interfere with T_4 - T_3 conversion. Toxicity will occur at levels ~ 90mg/kg DM but at lower levels iodine may be excreted in the milk of dairy animals which may pose a risk to human health. Sources of I are EDDI (~800 gm/kg) and KI (~600 gm/kg). Another source of I commonly used is kelp. Analyses vary with different products but average ~ 0.05% or 500 mg/kg DM. Adult dairy does during lactation need between 2 and 4 mg/day. Therefore kelp products should not be feed at a rate higher than 4 to 8 gm/day (~ 1/5 to 1/4 of an ounce). There is no zoonotic risk.

Listeria monocytogenes

Agent: *Listeria monocytogenes* causes abortion, encephalitis and septicaemia in sheep, goats and cattle and humans. It is found widespread in the environment and may contaminate feed and objects, as well as be present in rodents and insects. *L. ivanovii* has been implicated only in sheep abortions.
Transmission and Pathogenesis: *Listeria* spp grow well in poor quality, alkaline silage (pH > 6.0). Contamination of the silage occurs from soil and mouse, bird or other animal faeces which

are inadvertently ensiled. Outbreaks have also occurred on pasture, where it is likely that the pastures were contaminated with faeces containing the organism. *Listeria* causing encephalitis probably enters through the oral cavity and invades up the facial nerves to the brain. Abortion occurs less commonly, 7 to 30 days after infection.

Clinical Picture & Diagnosis: Abortion is accompanied by metritis with marked illness. The liver and brains of aborted foetuses reveals microabscessation of those organs. Culture will confirm the diagnosis.

Control and Prevention: In an outbreak, prophylactic treatment with long acting oxytetracycline (20 mg/kg) may avoid further cases. Prevention should focus on providing only good quality silage and taking measures to avoid soil contamination and ensure anaerobic conditions are maintained. Addition of acidifying agents at ensiling time may help reduce the pH thus inhibiting growth of *Listeria*. Holes in bags or a wide front to the bunker, will invite growth of Listeria. Silage that appears spoiled should not be fed to sheep or goats.

Zoonotic Disease: Listeriosis is an important foodborne disease, particularly of improperly pasteurized milk products (e.g. soft cheeses) and processed meats. Additionally, the organism can grow at refrigerator temperatures. It is not known if disease in a flock or herd increases the risk of food borne transmission.

Border Disease Virus

The Agent: Border Disease virus (BDV) is a pestivirus closely related to Bovine Viral Diarrhoea virus (BVDV). There are many strains of BDV isolated from clinical cases of BD and occasionally some of the outbreaks to be actually due to infection with BVDV from exposure to persistently infected cattle. Mostly the disease occurs in sheep but can also occur in goat herds.

Transmission and Pathogenesis: After the virus invades immunologically naive females, it attacks the placenta but does not cause illness in the females. Most fetuses infected in utero prior to day 60 to 85 of gestation are reabsorbed, aborted, macerated or mummified. Those that survive are congenitally damaged. Myelination of nerve cells is disturbed, particularly in the cerebellum. Hair follicles will produce hair rather than wool. Surviving lambs are born persistently infected (PI) with BDV, and shed virus in urine, feces and saliva. Fetuses infected after day 85 may abort or be born weak or unaffected, virus negative and with a pre-colostral titre to BDV. It is possible to have several levels of signs present in the same litter of lambs.

Clinical Picture: Abortion is seen in the flock followed by the birth of congenitally affected and/or weak PI lambs. These lambs have shortened facial and long bones with a wool coat that is frequently hairy and darkly pigmented, particularly over the shoulders and neck. They may show mild to severe body tremors and are commonly termed "hairy shakers". Case fatality rates in PI lambs are very high.

Diagnosis: BDV can be isolated from aborted fetuses and from the buffy coat of affected lambs. Antigen capture ELISA can be used on the sera of suspected persistently infected adults and lambs older than 2 months. Serum titres from the aborted ewes tend to show high antibody levels although this is not diagnostic. If an aborting ewe is a PI, then antibody levels will be low or absent but virus can be isolated or detected from the serum or buffy coat.

Control and Prevention: After an outbreak of BD abortion, all congenitally affected lambs should be kept separate from the breeding flock and sent to slaughter as young as possible. Replacement lambs older than 2 months, should be screened for virus. Cattle and sheep should be kept separated, including no shared feeders or water sources, and an effective vaccination program for BVDV should be maintained in the cattle herd. There is evidence that cross protection is poor between BVDV and BDV, therefore there is no advantage to vaccinating sheep with a killed or modified live BVDV vaccine unless BVDV has been isolated.

Zoonotic Disease: BDV is not zoonotic.

Uncommon Causes of Abortion in North America

Salmonellosis: Many Salmonella serovars have been implicated in ovine abortion. Three serovars *S* Abortusovis, *S* Montevideo and *S* Arizonae have a predilection for ovine pregnancies. Abortion rates in these outbreaks can be very low (< 1%) to as high as one-third of the pregnant flock. Many other species of salmonella have been implicated in ovine abortion with significant adult mortality. *S* Dublin*, S* Typhimurium*, S* Schwarzengrund, *S*. Brandenburg as well as others, have been reported as associated with abortion and ewe death due to metritis and septicaemia. These organisms may be introduced through contaminated feed, carrion birds and carrier animals such as dogs, cats, rats and livestock. Usually these sporadic, opportunistic outbreaks disappear from the flock and don't persist to the following season. All serovars of Salmonella can be considered zoonotic and care should be taken.

Brucellosis (*Brucella ovis* and *Brucella melitensis*): *B. ovis* is a common cause of epididymitis in rams in some countries and occasionally causes abortion in ewes. The organism is spread through contact between mucous membranes (vaginal, preputial, conjunctival) both ram to ewe and ram to ram, but not ewe to ewe. Abortion is rare but levels of 25 to 35% have been reported. Placentitis is marked with the organism easily cultured. Examination and serology of the breeding rams should also be performed. Rams should not be shared between flocks. *B melitensis,* exotic to USA and Canada should be considered as a rule out, particularly in states bordering Mexico. *B. ovis* is not zoonotic but *B. melitensis* is an important zoonotic disease in many parts of Asia, Africa and Mediterranean countries.

Leptospirosis: The most common agent in sheep is *Leptospira interrogans* serovar Harjo although L Pomona has been isolated from cases. It is an unusual cause of abortion in sheep but should be considered in areas where leptospirosis is a problem in cattle, pigs or wildlife. Ewes abort or have stillborn lambs and may also have high fevers, flaccid agalactia, haemoglobinuria and jaundice. An important rule out is copper toxicosis.

Bluetongue Virus: Bluetongue virus (BTV) is an arthropod transmitted orbiviral disease of domestic and wild ruminants. The virus requires an arthropod midge vector *Culicoides veriipennis* for transmission. In North America, it is found endemically in southern and western United States, as well as parts of the Caribbean, Mexico and Central America. At this time it is exotic to Canada. Clinical expression of the disease tends to be seasonal (fall) and linked to the midge's seasonal cycle which prefers a wet and warm environment. Sheep infected with BTV show signs of high fever, swollen ears, face and tongue, oral and nasal ulcers and lameness. Up to 20% of fetuses infected in early gestation have varying degrees of hydranencephaly and skeletal deformities and may be aborted or carried to term.

Cache Valley Virus: Cache Valley virus (CVV) is an arthropod-borne virus associated with ovine abortion. In North America, CVV has been implicated in outbreaks of congenital deformities in the south west of the USA particularly Texas, and occasionally in the north east and southern Ontario. CVV is transmitted mainly by mosquitoes that feed on wild e.g. deer, or domestic large ruminants. Abortion occurs in early January as the ewes must be in early gestation during the fall when the viral load in the mosquito population is at its peak. Signs are early embryonic death, mummification, arthrogryposis, and CNS deformities. A titre in the fetus is diagnostic and occasionally tissues are positive on PCR. Lack of a titre in the aborting ewe, rules out the disease. If CVV is a regional problem, avoid breeding sheep in mosquito infested pastures, or wait until after a killing frost before turning in the ram. CVV has only rarely been implicated in encephalitis in humans, but is not caught from sheep but rather from infected mosquitoes.

Neospora canis: *N. canis*, a protozoal parasite of livestock and companion animals, is one of the most commonly diagnosed causes of abortion in dairy and beef cattle. However, there are few reports in the literature of naturally occurring *Neospora* infection in sheep or goats. However, sheep and goats are susceptible to this organism, so it should be considered as a diagnosis if toxoplasmosis is ruled out in cases of protozoal abortion.

Sarcocystosis: Abortion due to Sarcocystis species is unusual but there are difficulties with

definitive diagnosis. Sheep may be infected by four species of Sarcocystis. Two are pathogenic - *S tenella* and *S arieticanis,* and may cause abortion during the acute phase infection. Detection of infection with pathogenic species can be done using a species-specific PCR on blood samples of infected sheep and will help to differentiate infection with other protozoal parasites of sheep.

Selenium Deficiency: This has been associated with early embryonic death and abortion due to congenital white muscle disease. Se supplementation in many regions of North America is nutritionally critical for sheep and goats as deficiency in the soils is widespread, particularly eastern and western regions. Se should be added to the ration at a rate of 0.1 to 0.3 mg/kg DM diet with 0.25 mg/kg the probable optimum level for supplementation when feeding deficient rations. The Se may also be fed as a supplement or salt-mineral mixture to be consumed at a rate of 0.23 - 0.46 mg/sheep/day from breeding period forward. Injection of the pregnant ewe of Se commercial preparations at a rate of 0.056 mg/kg body weight one month prior to lambing, has been shown to decrease the risk of congenital myopathy but needs to be repeated every 2 weeks.

Energy / Protein Deficiency: At the time of breeding, energy and to a lesser extent protein deficiency is associated with embryo loss. If the female is not fed adequately for the first and second trimesters, this will adversely affect placental growth and attachment to caruncles. Reduced birth weights and mummification are outcomes of this. Inadequate energy in the third trimester will inhibit foetal growth. Lambs /kids will be born small with poor body fat reserves and be more at risk of hypothermia.

Overnourishment of Adolescent Ewes: If peripubertal ewe lambs are fed a balanced ration intended to promote rapid growth, there is a marked detrimental effect on placental mass, the number of cotyledons per placenta and subsequent birth weight of the lambs. Laboratory data on these lambs reveals elevated serum urea and on post mortem, these lambs have abnormal kidneys and gut.

***Astragalus* spp and *Oxytropis* spp (locoweed):** Ingestion of locoweed can cause abortion, birth defects such as arthrogryposis, right heart hypertrophy resulting in hydrops amnios and hydrops allantois, reduced birth weight and weak lambs. The severity of the effect is dose dependent on the amount of plant ingested and the time of gestation. The toxin is the indolizidine alkaloid swainsonine. It appears to cause these pathologies through delaying embryo implantation, interruption of vascular development and inducing alternations in foetal fluid balance. It has been suggested, that for some geographic regions where these plants grow commonly, i.e. the western United States, locoweeds are a very important cause of abortion and reproductive failure.

Stress / Trauma: Abortion can be seen after an unusual stress, e.g. predator attack, shearing, inappropriate handling. While it is possible to handle late gestation ewes and does, care should be taken. Associated with inappropriate handling is the risk of foetal trauma (ruptured liver or kidney). This may happen in a chute or being run through a gate but fighting and shoving between females at a feeder is also a cause.

Habitual Abortion of Angora Goats: A suspected genetic cause of abortion in Angora goats is linked to finer fibre diameter and overproduction of maternal cortisol. This results in 3rd parity plus does aborting at 90 to 120 days of gestation. It may be a genetic linked to fibre quality, or it may be management. Regardless, when investigating abortion problems in Angora goats, it is best to first eliminate the possibility of other causes.

Sheep-Goat Hybrids: Does housed with rams can become pregnant but most often abort by 60 days but occasionally later. The cotyledons are large and unusual in appearance and the fetus may have an anasarca appearance. Occasionally, the abortion may be associated with dystocia and maternal death. For this reason, it is important not to house mature does with rams. It appears that ewes bred by bucks do not conceive.

There are many other miscellaneous and sporadic causes of abortion in sheep and goats.

Prompt and appropriate investigation of an abortion problem will often lead to a diagnosis and thus will yield valuable information to the practitioner in helping control the abortion problem and protect the health of the producer and family.

Additional Sources of Information

Blasco JM, Molina-Flores B: Control and eradication of *Brucella melitensis* infection in sheep and goats. Vet Clin North Am Food Anim Pract 27:95-104, 2011.

Menzies PI: Control of important causes of infectious abortion in sheep and goats. Vet Clin North Am Food Anim Pract 27:81-93, 2011.

Menzies PI: Vaccination programs for reproductive disorders of small ruminants. Anim Repro Sci 130:162-172.

Stuen S, Longbottom D: Treatment and control of chlamydial and rickettsial infections in sheep and goats. Vet Clin North Am Food Anim Pract 27:213-234, 2011.

Table 1. Causes of Abortion in Sheep and Goats in Canada and USA.

INFECTIOUS	NON - INFECTIOUS
Common	
Campylobacter jejuni – S, G (rare)	Congenital goiter due to iodine deficiency – S, G, R
Campylobacter fetus subspecies *fetus* - S	Trauma – S, G
Chlamydophila abortus – S, G	
Coxiella burnetii – S, G	
Toxoplasma gondii – S, G	
Border Disease virus – S, G (rare)	
Listeria monocytogenes – S, G	
Uncommon	
Bacillus spp – S, Sp	Selenium deficiency – S, G, R
Bluetongue virus – S, R	*Astragalus spp and Oxytropis spp (locoweed)*– S, R
Brucella ovis – S, R	Starvation – S, G
Cache Valley Virus – S, R	Overnutrition – S, G
Caprine Herpes Virus I – G	Heat shock – S, G, R, Sp
Leptospira interrogans serovars – S, G, R	Sheep / Goat hybrids – G, Sp
Mycoplasma mycoides subsp. *mycoides* - G	Congenital enzootic ataxia (Cu deficiency) – S, G, R
Mycotic – S, G, Sp	Habitual abortion of Angora goats – G
Neospora canis – S, G	Drug toxicities such as levamisole - G
Sarcocystis tenella and *S arieticanis* – S, Sp	Prostaglandin products - G
Salmonella –pathogenic serovars – S, G, Sp	
Streptococcus dysgalactia – S, Sp	
Yersinia spp – S, Sp	
Infectious Causes Exotic to Canada and U.S.A. at Present	
Akabane virus – S	
Anaplasma phagocytophilum (Tick-borne fever) – S, G	
Brucella melitensis – S, G	
Nairobi Sheep Disease virus – S, G	
Peste des Petits Ruminants virus – S, G	
Rift Valley Fever virus – S, G	
Salmonella enteritica – abortifacient serovars (Montevideo, Abortus Ovis) – S,	
Schmallenberg virus – S, G	
Wesselbron Disease virus – S, G	

S = sheep; G = goat; Sp = sporadic; R = regional incidence