Emerging Issues in Small Ruminant Health: A Look At A Few Zoonoses Old And New

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Overview

Most bacterial diseases associated with sheep and goat abortion have some potential for zoonotic transmission. The main diseases of concern as identified by Edmondson et al (2012) are: Brucella melitensis, C. burnetii (Q fever), Chlamydia abortus, Salmonella, Toxoplasma gondii and Campylobacter jejuni. Knowledge of abortifacient zoonotic pathogens, their clinical signs, gross lesions and their potential risk to humans is critical to prevent zoonotic disease to veterinarians and small ruminant owners. The focus of these proceedings will be Coxiella burnetii as there has been growing public health concern over the risk to the human population beyond simple occupational exposure, along with a mention of some of the major foodborne zoonotic pathogens and a quick review of Contagious ecthyma (orf). A recent Coxiella/Q fever outbreak in the Netherlands resulted in 4000 human cases and the euthanasia of 50,000 goats (Menzies et al 2013).

Coxiellosis, “Q fever”

Coxiella burnetii, the causative agent of coxiellosis in animals or “Q fever” (Query fever) in humans, is an obligate intracellular gram-negative bacterium and zoonotic pathogen. Ruminants act as a reservoir and are replicating hosts of the organism. Cats, dogs, and wildlife may also serve as reservoirs (Whitney et al 2009). C. burnetii has been identified in fish, wild birds, rodents, marsupials, horses, swine, camels, marine mammals, ducks, turkeys, geese, cats, dogs, and rabbits but was first isolated from a tick and has been identified in ~40 species of tick subsequently (Davis and Cox 1938). The disease is subclinical in most animals, with adverse pregnancy events reportedly the most common clinical sign. These abortion rates can vary from a few cases up to 90% of the herd (Palmer et al 1983, Arricau-Bouvery et al 2005) Recently, C burnetii has been identified as a biological-threat agent due its low infectious dose and high rate of transmission, and is listed as a group B bioterrorism agent by the CDC in the US (Oyston and Davies 2011). This organism is present in ~90% of dairy cattle operations in the United States and a significant number of sheep and goat operations (Plummer 2012). Humans are infected via inhalation of aerosols or contaminated dust from infected ruminants or through exposure to infected animal products (Whitney et al 2009). Exposure and seroconversion may result from consumption of unpasteurized dairy products (Whitney et al 2009). Horizontal human-human transmission is rare but has been reported through sexual contact and aerosol transmission (Whitney et al 2009).
Bacteriology and epidemiology

*C. burnetii* occurs in two predominate variants, a large cell variant which replicates in cells, and a small cell variant that survives in the environment. *C. burnetii* undergoes phase variation, which is associated with alterations in its cell wall components, and have been called phases 1 and 2. The changes in cell surface antigens with subsequent phase variants has clinical implications for serologic testing due to the production of phase 1 and phase 2 antibodies to *C. burnetii* at different stages of natural infection. Phase 1 is highly virulent and can replicate in immune-competent hosts (Roest et al 2013) and phase 1 bacteria are internalized and survive intracellular killing, whereas phase 2 bacteria are efficiently phagocytized and then killed (Roest et al 2013).

The primary route of transmission to both animals and humans is via aerosolization, and the organism is highly infectious; an infectious dose of 1 organism is reported for humans (Jones et al 2006). It has been suggested that the organism is capable of aerosolizing up to 5 km in appropriate environmental conditions. The bacterium is shed from ruminants in fetal membranes, fluids of parturition, vaginal secretions, aborted fetuses, milk, urine and feces (Berri et al. 2001; Arricau-Bouvery et al. 2003, Guatteo et al. 2006), and can be spread from infected carcasses at the time of slaughter (Porter et al., 2011). Bacteria colonize the uterus of the pregnant female, and the trophoblasts in the allanto-chorion of the placenta are the reported target cells of *C. burnetii* (Roest 2012). Long-term shedding in milk, up to 32 months post-infection, has been reported (Rodolakis et al.2007, Berri et al. 2007; Angelakis and Raoult, 2010). Persistent shedding in urine and feces is reportedly up to 20 days post-partum (Angelakis and Raoult, 2010). Abortion storms are most common in the first year of introduction into herds/flocks when the animals are immunologically naïve with increased herd shedding and fewer abortions observed in later years (Plummer 2012). This is especially true for goats where chronic subclinical shedders often remain in the herd. In sheep, shedding in vaginal secretions, feces and urine has been documented for up to 60 d post-partum (Rousset 2007), with 7-14 d of intermittent milk shedding (Berri 2001, Rodolakis 2007, Astobiza 2010b) but more information regarding excretion of *C. burnetii* leading to shedding in feces, vaginal secretions, urine and milk is needed.

Clinical signs

Acute infection is typically asymptomatic in small ruminants (Angelakis and Raoult 2010), and most shedding occurs in the complete absence of clinical signs in infected herds/flocks. Clinical disease is generally associated with adverse pregnancy events (abortions, still-born, weak kids). Rearing of these kids may be complicated by respiratory or GI problems (Wouda and Derksen 2007). Animals that abort generally have no signs of systemic illness and the aborted fetuses/placenta are often grossly unremarkable, although metritis may be present (Wouda and Dercksen 2007). Asymptomatic live born kids may shed high loads of *C. burnetii* into the environment. Up to 98% does in naïve herds have been reported to abort (Berri et al 2007) from
Coxielllosis. According to Plummer (2012), *C. burnetii* is one of the top three causes of goat abortions and is also commonly associated with abortions in sheep; however, *Campylobacter, Chlamyphila* and toxoplasmosis are more often seen.

**Diagnosis**

Abortion is not specific for Coxielllosis. For direct diagnosis of the etiologic agent samples from aborted placental tissue, vaginal discharge or an aborted fetus should be collected and tested, but *C. burnetii* is capable of being shed in animals without histopathologic evidence of infection (Rousset et al 2007, Plummer 2012). Immunohistochemistry on infected placenta can detect Coxiella to confirm abortion due to coxiellosis. (Roest et al 2013) Coxiella shedding confirms its presence on the farm, and the farm should be considered positive, and a zoonotic Q fever risk. PCR tests are available and validated for use on vaginal fluids, milk, feces and placental tissues (Roest et al 2013). These tests are highly sensitive and specific.

There are also a number of indirect diagnostic tests. The World Health Organization for Animal Health (OIE) recommends the complement fixation (CF) test as the preferred serologic assay (OIE 2010) but ELISA and IFA tests are reportedly more sensitive tests (Rousset et al 2007), Plummer. Negative serology tests cannot be used to rule out infection and shedding of an individual animal due to the fact that ~10-15% of infected animals do not develop a measurable immune response. Berri et al. (2001, 2005) found that 57% of parturient ewes shed the organism on vaginal swab PCR despite being ELISA sero-negative.

A combination of direct and indirect diagnostic testing methods are recommended, such as PCR of aborted tissue and/or vaginal mucus (2-6 animals) within 8 days of abortion in addition to serology (6-10 animals) from animals that had given birth at least 15 days prior (Roest et al 2013). Increasing the number of samples submitted and repeated sampling around kidding/lambing season will increase the sensitivity for detecting an infected herd, according to Plummer (2012). Bulk tank testing in lambing/kidding season along with herd serology may also be of value in dairy herds. Serology is more cost-effective but PCR gives direct evidence of shedding. At the herd level, serology is helpful to determine if the population is infected; however, individual animal results must be interpreted with caution and negative results do not rule out infection or shedding as shedding is intermittent. According to Roest et al, (2013) PCR is the most sensitive technique to detect *C. burnetii*, whereas ELISA is the most sensitive technique to detect *C. burnetii* specific antibodies.

**Prevention and Control**

Antibiotic treatment and vaccination are currently the 2 methods available for control of Coxielllosis. The addition of in-feed tetracycline or injectable oxytetracycline pre-partum has not been shown to prevent *C. burnetii* shedding in milk, feces, and vaginal secretions (Berri et al 2007, Astobiza et al 2010b). Due to the lack of evidence supporting antibiotic efficacy and the
fact that prudent use of antibiotics is needed to avoid resistance, they should not be used for treatment of Q fever in animals at this time (Roest et al 2013).

Vaccination with a phase 1 C. burnetii, inactivated vaccine is reportedly effective for abortion prevention, and the reduction of bacterial shedding in goats and cattle (Arricau-Bouvery et al 2005, Guatteo at al 2011), and is most effective in primiparous animals. The vaccine is provisionally licensed for cattle and goats in the EU (EFSA 2010a) but there is currently no vaccine in the US. Vaccination may not be effective in animals that are already infected (Guatteo et al 2008, Rousset et al 2009). A phase 1 vaccine is currently available for human use in Australia, and has been recommended for high-risk, occupationally exposed, sero-negative individuals (Arricau-Bouvery and Radolakis 2005).

C. burnetii can survive for several weeks outside a host in warm, moist environments (Tissot Dupont et al 1992, Marrie and Raoul, 1997). As ruminants are the main reservoir, controlling endemic Coxiellosis in livestock may play an important role in reducing disease in populations living in close contact with infected animals (Schelling et al 2003). Angelakis and Raoul (2010) suggest that controlling environmental contamination through the control of infected ticks and biosecurity measures may reduce introduction of C. burnetii to naïve farms. Composting is the preferred method for handling manure and bedding and a 90 day minimum decreases the Coxiella burden and risk of spread (Menzies et al 2013). Manure should be covered during transport, stored in a location where water run-off and water contamination is minimal. Transport and spread of manure should be done when wind is minimal to reduce the risk of spread through aerosolization (Tissot-Dupont et al 1999, 2004). Composting carcasses and aborted tissues and contaminated bedding on site is preferred (Menzies et al 2013). Quarantine and mass euthanasia is not recommended for the following reasons: C burnetii is endemic in the US, and is a ubiquitous and persistent organism in the environment. It is thereby almost impossible to completely eliminate infection from a herd and to completely decontaminate, and there is no known treatment to stop shedding in infected animals. (Menzies et al 2013).

Zoonotic transmission: the human-animal interface

People usually acquire Q fever via inhalation of environmental C. burnetii. Domestic ruminants seem to be the primary source of infection for human Q fever, although companion animals must also be considered a source as several human outbreaks were related to pregnant dogs and cats (Roest et al 2013). The role of horses, ticks and wildlife remains unclear at this time. Outbreaks have been reported frequently, are world-wide and result in approximately 400-500 lab-confirmed human cases/outbreak. Sheep are usually the main source in most outbreaks, and goats are the second most commonly identified source. Contrary to this was the Dutch Q fever outbreak that lasted from 2007-2010, that had mainly dairy goats as the source of infection (Roest et al 2011). 18 reported outbreaks in 12 different countries involving 2 to 289 people were reported between 1999-2004 (Angelakis and Raoul, 2010) and were attributed to exposure to sheep, goats, goat and sheep manure, wild animals, and dogs and cats. However, in humans Q
fever results from the inhalation of contaminated aerosols. These aerosols can travel up to 5 km and infect humans who have no direct contact with animals, and several outbreaks have demonstrated this (Roest et al. 2013).

*C. burnetii* poses a significant zoonotic risk to those exposed to ruminants (veterinarians, farmers, stock-breeders, livestock truck drivers, wool shearers, slaughterhouse workers), medical and paramedical practitioners, laboratory technicians with *C. burnetii* exposure, rural or semi-rural residents, and those handling contaminated manure fertilizer (Tissot-Dupont et al. 1999, Berri et al. 2003), and possibly any individuals within a few kilometers’ distance from infected farms (Plummer 2012). High-risk individuals must be aware of the risks and take necessary precautions such as barrier protection, face masks, and hand-hygiene. Approximately 25% of veterinarians at the 2006 AVMA conference were seropositive for *C. burnetii* exposure (Whitney et al. 2009). The results of this study found that large animal veterinarians or those individuals with contact with standing bodies of water were also at increased risk, but the relationship to standing water was unclear.

Consumption of raw milk or the selling of unpasteurized milk to consumers is not recommended as *C. burnetii* is shed in the milk of infected animals. Pasteurizing milk at 145F for 30 min, or 161F for 15 sec is enough to destroy *C. burnetii* and many other raw milk pathogens.

**Clinical presentation of Q fever in humans**

There are 3 primary clinical presentations: acute Q fever, chronic Q fever, and a post-Q fever fatigue syndrome (QFS). The majority of Q fever cases are asymptomatic post-exposure (60%), while 40% will be symptomatic and present with what has been described as a non-specific, self-limiting illness. Fever, headache, chills, pneumonia and hepatitis are observed in more severe acute cases. In the Dutch outbreak, the mortality rate was reportedly low (1.2%) and any fatalities were all patients suffering from severe underlying disease. Chronic Q fever develops in 1-5% patients, and may not be seen for years after primary infection. Symptoms include fatigue, fever, weight-loss, night sweats, hepato-splenomegaly and endocarditis. *C. burnetii* is not detected in people with QFS and antibody levels are low. Symptoms include: fatigue, arthralgia, myalgia, blurred vision and peripheral lymphadenopathy. Why some people develop long-term presentations of Q fever is unclear but acute Q fever requiring hospitalization was identified as a risk factor (Roest et al. 2013). Diagnosis includes combinations of PCR and serologic testing. The treatment of choice is doxycycline.


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Foodborne zoonotic pathogens

The most common zoonotic diseases in the US are gastro-intestinal illnesses. A number of foodborne pathogens have been identified in raw milk that are of clinical significance: *Campylobacter jejuni*, Shiga toxin-producing *E. coli*, *Listeria monocytogenes* and salmonellae (Oliver 2009), with *Listeria* and *Salmonella* spp being the most commonly reported. Several studies have assessed the incidence of these organisms in sheep and goat milk and have found the same organisms along with enterotoxigenic *Staphylococcus aureus*. Raw milk sales are currently allowed in 30 states (Indiana is NOT one of them). Roughly 35-60% of farm families and their employees consume raw milk (Oliver 2009) but consumption in urban communities is unclear.


**Campylobacter**

*Campylobacter jejuni* is a leading cause of bacterial food-borne gastroenteritis worldwide and is considered to be a major public health problem. Clinical signs of *Campylobacter* infections in humans include self-limiting watery/bloody diarrhea, abdominal cramps, nausea, and fever; however, severe neurological disease and sepsis may be seen. *Campylobacter* is widespread in food animals, and animal products are often contaminated. *Campylobacter* outbreaks are also commonly associated with consumption of raw milk. Ruminants contribute significantly to sporadic cases and human disease outbreaks. A tetracycline-resistant, highly virulent *C. jejuni* clone has emerged as the predominant cause of *Campylobacter*-abortion in sheep which has also been associated with multiple human gastroenteritis outbreaks linked to raw milk consumption. Currently there is no evidence implicating the clone as a cause of human abortion, but it is possible given the virulence of the clone in pregnant animals and the fact that *C. jejuni* has been implicated in human abortion, stillbirth, and neonatal mortality. This study also supports prior studies that implicate *Campylobacter* as the most common cause of milk-borne disease outbreaks in the US, with unpasteurized milk and associated dairy products as major sources of human infection.


**Shiga toxin-producing Escherichia coli (STEC)**

Shiga toxin-producing *Escherichia coli* (STEC) has emerged as an important zoonotic pathogen, particularly *E. coli* O157:H7 which is responsible for several human diseases, including hemorrhagic colitis (HC), and in children the hemolytic-uremic syndrome (HUS) (Oporto et al 2008). Most outbreaks and sporadic cases of HC and HUS have been attributed to strains of *E. coli* O157:H7 STEC. Hard cheeses are considered to present a lower risk for growth and survival of pathogens than soft cheeses; however, STEC can survive in hard-ripened raw milk cheeses up
Cheese may be manufactured in the United States using raw milk, provided the cheese is aged for at least 60 days at temperatures not less than 35 °F (1.7 °C) (Brooks et al 2012) but there is currently increased concern among regulators regarding the safety of raw milk cheese due to the potential ability of foodborne pathogens to survive the manufacturing and aging processes. A recent study sampled feces from 28 goats and 20 sheep on 7 city hobby farms and found STEC strains in 45/48 (94%) of samples (25/28 goat, 20/20 sheep). Other prior studies cited had found lower prevalence of STEC in sheep (30-67%), and (17-75%) in goats (Schilling et al 2012). While no animals or humans had reportedly shown any clinical signs, silent carriers of this organism can serve as a disease reservoir and are a risk for zoonotic disease.


**Listeria monocytogenes (LM)**

LM is an intracellular, non-spore forming Gram-positive coco-bacillis that belongs to the genus Listeria. There are 6 species including LM, but only two are considered potentially pathogenic: LM and *L. ivanovii*. LM is the major pathogen of listeriosis and the only species that poses a serious public health risk. It causes invasive and often fatal CNS infection in ruminants, horses, dogs, pigs, deer, South American camelds, cats, and men. *L. ivanovii* is considered only mildly pathogenic and seems to affect almost exclusively ruminants, causing abortion, still-births, and neonatal septicemia, but not CNS infections. LM is ubiquitously distributed and grows in soil, water, plant matter, food items, and intestinal tract of mammalian hosts. LM produces a biofilm making it more resistant to hostile environmental conditions such as low pH, high salt concentrations and low temperatures, and food-processing and food-preserving procedures. LM has emerged as an important foodborne pathogen and is a major cause for large food recalls due to bacterial contamination. Although LM is able to infect a wide range of animal species, it occurs primarily in ruminants and humans. The link between ruminant and human listeriosis is not completely understood. Listeriosis is considered to be a zoonosis, but direct transmission between ruminants and humans rarely occurs. Most cases of direct transmission are associated with cutaneous infections via contact with infected cattle or aborted tissue. However, the literature suggests that ruminants may act as a natural reservoir for strains causing human infections given that the same epidemic clone responsible for a large number of human epidemics has been repeatedly isolated from ruminants with listeriosis.


**Staphylococcus aureus** food poisoning (SFP)

*S. aureus* is a ubiquitous pathogen found in air, dust, sewage, water, environmental surfaces, and in humans and animals and is associated with both human and animal diseases including mastitis, and staphylococcal food-poisoning (SFP). In rare cases, it has been associated with more life threatening illness such as toxic-shock syndrome (TSS), endocarditis, and necrotizing pneumonia. Clinically and subclinically mastitic animals can be a source of transmission of staphylococcal enterotoxins into milk. Clinical signs of SFP in humans include sudden onset of nausea, vomiting, abdominal cramps and diarrhea, and is caused by ingestion of food containing heat-stable staphylococcal enterotoxins. Other contamination rates in meat products have been reported from 17.1-48.1% in one study and 96.2% in goat bulk milk and 37.8% in raw milk products in a Norwegian study. Small ruminant owners consuming raw milk, cheese or meat from their animals may be at risk for this disease.


**Toxoplasma gondii**

Toxoplasma gondii infections are highly prevalent in humans and food animals. Felids are the key species in the life cycle of this parasite as they excrete the environmentally resistant oocyst in their feces. Humans become infected via ingestion of tissue cysts from undercooked meat, consuming food contaminated with oocysts, or by accidentally ingesting oocysts from the environment. Clinical toxoplasmosis in humans has been linked to ingestion of *T. gondii* in food, and foodborne transmission is one of the major sources of *T. gondii* infection. Clinical disease resulting from *T. gondii* infection occurs as acquired infection in immune-competent persons (usually mild), disease in immunosuppressed persons usually due to recrudescence, congenital disease, and ocular disease (congenital or acquired). In a recent assessment of foodborne illnesses in the US, toxoplasmosis was identified as the second leading cause of foodborne illness–related deaths and fourth leading cause of foodborne illness–related hospitalizations. Results of a recent study and previous surveys indicate the prevalence of *T. gondii* in lambs can be high. Although pasteurization will kill *T. gondii* in goat’s milk, unpasteurized raw milk is sold by small goat farmers and goat cheeses made from raw milk could be a source of *T. gondii* infection.Little is known of the excretion of *T. gondii* in goat’s milk. Goat meat is also very popular with many ethnic groups in the US. In a recent study, the seroprevalence of *T. gondii* antibodies in goat meat destined for human consumption in the US was found to be 53.4%.


**Salmonella**

Sheep and goats can be carriers of different Salmonella serovars, including *Salmonella enterica* serovar Enteritidis and *Salmonella enterica* serovar Typhimurium, the most important serovars for human infections. However, salmonella prevalence in sheep and goat herds is reportedly very low (Schilling et al 2012). In 2008, 2.25% of the sheep flocks and 1.97% of the individually tested sheep in Germany carried Salmonella spp. The prevalences in goat herds were even lower, at 0.51% of the herds and 0.78% of the individually tested goats. In another study, 1.3% of the sheep carried Salmonella spp., with slightly higher ratios in sheep that were kept on limited pastures and had contact with wild birds. Small ruminants appear to play a minor role in the
transmission of *Salmonella* to human beings, and general hygiene management should be adequate for limiting the risk of acquiring *Salmonella* infections from sheep and goats. (Schilling et al 2012)


**Contagious ecthyma (CE), orf, soremouth, scabby-mouth, contagious pustular dermatitis**

CE is a contagious, zoonotic disease of goats, sheep (and camelids) with a world-wide distribution. It is an epitheliotropic parapox virus that enters the goat via skin abrasions, which then replicates in regenerating epidermal keratonocytes and spreads to lymph nodes, bone marrow and liver during primary viremia. The virus may become generalized and through a secondary viremic phase, spread to the head, extremities, udder, genitals, and lungs.

**Epidemiology**

Morbidity in kids can be close to 100%, with a mortality rate from secondary infection and starvation reportedly up to 20% but rarely exceeds 1%. Death is usually due to secondary complications such as pneumonia or starvation and not from CE itself. Persistently infected carrier goats and sheep have been demonstrated as an important source of disease and infection can recrudesce during times of stress.

**Clinical signs**

The incubation is 3-8 days post-infection. Papules progress to vesicles, pustules and scabs. Proliferative, crusty lesions typically form on the lips, but can be seen on the face, ears, coronary band, scrotum, teats, or vulva. Other rarer distributions on the neck, chest, and flank and caudal hind legs have also been reported. Scabs may have secondary bacteria and infections can develop. Lesions regress in 3-4 weeks according to Smith and Sherman (2009) or 4-8 weeks (Musser 2012). In more severe cases, lesions have reportedly extended down the respiratory or GI tracts and led to pneumonia or gastro-enteritis respectively but this is rare.

A persistent form referred to as malignant contagious ecthyma has been reported in a small number of sheep flocks. Lesions are found in atypical areas (distal limbs, feet) and fail to regress and may continue to enlarge, making secondary complications more common.

Adult goats with lesions on the lips generally continue to eat and milk well, but animals with teat lesions may affect udder health and predispose to mastitis. Painful teats may also lead to rejection of the kid when trying to suckle, or painful lip lesions may prevent adequate suckling. Kids/lambs can also developed more generalized disease and secondary bacterial infections and
lesions can also contain maggots if complicated by fly strike. Boer goats and Boer goat crosses seem to develop a more generalized and persistent form of CE, but it is unclear if it is due to a different strain or immunologic response.

**Diagnosis**

Diagnosis is usually based on CS but skin biopsy and histopathologic exam can confirm the diagnosis. Electron microscopy, IHC or serology can also aid in diagnosis or distinguish CE from the other differentials.

Differential diagnoses include bluetongue, ulcerative dermatoses, capripox (sheep and goat pox) or foot and mouth disease (FMD).

**Treatment**

As the disease is typically self-limiting and lesions resolve within 3 weeks, treatment is not often performed. The benefits of treatment must be also weighed against the zoonotic risk of infection. Antibiotics have been used in cases of secondary bacterial infections. If lambs or kids are not suckling well, anesthesia and local debridement of lesions with either electrocautery or cryotherapy have been successfully reported in lambs. Topical astringents or ointments may actually delay resolution of lesions.

**Zoonotic transmission: the human-animal interface**

Human lesions are caused by direct inoculation of infected material, through scab contact from either natural disease or from live vaccine and are reportedly very painful. Sheep and goat owners, farmers, and veterinarians are at risk and CE is an occupational disease among sheep shearsers, butchers and slaughterhouse workers. CE lesions occur mainly on fingers and are usually seen as large, painful vesiculo-pustular nodules (1–2 cm). A low fever and swelling of the draining lymph nodes may also occur (Grey 1949). Lesions (up to 5 cm) have been observed. Spontaneous recovery occurs in 3–6 weeks but repeat attacks are reportedly common. Diagnosis is confirmed by examination of collected material (from a vesiculo-pustule or a scab) by negative electron microscopy, which shows characteristic virions (LaChapelle 2012).

**Prevention and control**

Avoid buying animals from CE-infected farms if possible. Minimize transport stress. Quarantine any new arrivals on the farm until CE infection can be ruled-out. Scabs maintain large amounts of virus and protect it from environmental inactivation from months-years (Musser et al 2008), and provide a source of pasture and shed contamination.

Vaccination is generally not recommended in non-infected herds as vaccination with live un-attenuated virus will introduce disease. In herds where purchase and introduction of new animals or showing occurs frequently, vaccination may prevent an outbreak during the show
season or in milking animals. If vaccination is utilized, it should be done at least 6 weeks prior to the show season so any scabs will not be present, or at least 8 weeks prior to lambing/kidding so the dams are immune when suckling lambs/kids. Vaccinated ewes/does should ideally have all of the scabs fall off before moving them to the lambing/kidding area so neonates are not born into a high viral load. If a CE outbreak occurs on a previously uninfected premise, affected animals should be isolated and vaccinating unaffected animals may limit the duration of the outbreak. Subsequent vaccination of all kids in conjunction with annual revaccination of late-gestation does is suggested. It is thought that no colostral antibody transfer occurs with CE for passive immunity in lambs/kids, therefore, timing of vaccination does not appear to be a factor.

Commercially available preparations are of un-attenuated, live virus or tissue culture strains marketed as vaccines. Autogenous vaccines are made by crushing scabs in saline, filtration of the suspension through a cheese cloth, along with several drops of antibiotic added to prevent secondary bacterial overgrowth. Skin in a hairless region such as axillae, inner ear pinnae, under the tail is scarified and the vaccine is rubbed in. Scabs should appear in 1-3 days if the inoculation was successful. Lack of response suggests a pre-existing immunity.

Most vaccines are not labeled for use in goats and research has shown that the use of sheep vaccine was not protective in goats (de la Concha et al 2003). A goat vaccine was created (Musser et al 2008) and continues to be protective if the vaccine-derived CE strain but is not cross-protective for antigenically distinct strains (Musser et al 2012). Reasons for vaccine failures have historically been attributed to physiologic variability among individual animals and breeds, genetic factors, nutritional differences, environmental conditions, the method of inoculation, viral titers at the time of inoculation and challenge exposure, incorrect vaccine administration, and antigenically distinct strains of the orf virus (Musser et al 2012) but this study suggests it is due to the latter.
