WASHINGTON STATE UNIVERSITY EXTENSION

ag animal health

VETERINARY EXTENSION NEWSLETTER • VOLUME 1, NO.4

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From the Editor:

At the *American Association of Extension Veterinarians' Strategic Planning Meeting* (Kansas City, MO, May 2008), we discussed the future of veterinary extension and the issues with which we must deal in our extension programs. The major issues noted were: Food Safety, Food Security, Animal Welfare, Public Health, and Environmental Stewardship. *What happened to ag animal health?* If we pause to think about all the measures we put into place or could incorporate into our practices that help improve animal health and performance, whether we are a producer, county extension faculty, or veterinarian, we can see that they are harmonious with the major issues mentioned. Reducing antibiotic use by applying prudent use guidelines and preventive medicine improves the safety of food derived from our animals and lowers costs. Practicing disease prevention and securing our operations will help promote food security. Reducing stress such as from summer heat and preventing conditions such as lameness improves both animal welfare and herd performance. Reducing diseases that affect animals and people will improve the public's health. Applying sound nutrition guidelines for ration-balancing can reduce over-supply of some nutrients in the environment as well as improve animal performance. It seems that improving ag animal health is right on the mark with the major issues facing animal agriculture and veterinary extension.

By: Dale A. Moore, Director, Veterinary Medicine Extension

Featured Faculty: Dr. Larry Fox

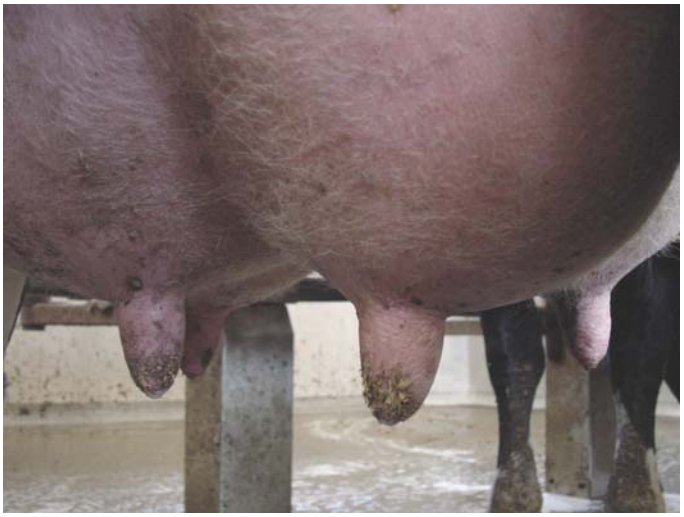
Dr. Larry Fox holds a split appointment between the College of Veterinary Medicine, Dept. of Veterinary Clinical Sciences and the Dept. of Animal Sciences. He is responsible for teaching and research but conducts significant work in outreach for both departments. His research is focused on understanding the dynamics and pathogenesis of contagious mastitis organisms such as *Staphylococcus aureus* and *Mycoplasma* spp. that will aid in development of prevention strategies.

Dr. Fox currently serves as the President of the National Mastitis Council whose mission is to provide a forum for education and global exchange of information on milk quality, mastitis and relevant research and communicate that information to the dairy industry to control mastitis and improve milk quality. http://www.nmconline.org/

Choosing the Optimum Dry Period Length

What is the optimum dry period length? This question has been debated for centuries. An 1805 text on dairy management in Britain discussed opinions where the range of recommended dry period lengths varied from 10 days to 2 months. In the early 1900's a survey of 10 widely used dairy management texts indicated that the recommended dry period length ranged from 4 to 10 weeks. Three studies conducted just before WWII where production records were analyzed retrospectively indicated that the minimum dry period to achieve maximum yield in the subsequent lactation was 30-39 days and that a 2 month period seemed most favorable. Several studies in the 1960s and 1970s substantiated the findings that the optimum dry period length was approximately 60 days, and that value became the standard length for decades.

The genetic merit of dairy cows has increased dramatically over the years so that some cows will produce more milk per day at the end of their lactation then cows decades ago would produce at their peak. So, why dry-off high producing cows early?



Several recent studies have examined the effect of shortening or eliminating the dry period. They considered lifetime yield, which would be influenced not only by milk production records in the lactation subsequent to a shorter dry period, but the production gained in the previous lactation by having a longer lactation. The results of these studies are mixed, with some suggesting that lifetime yield would not be affected by shortening the dry period to 30 days and others indicating that 60-day dry periods are optimum. One study examined the effect of eliminating the dry period altogether. In general, these studies revealed that shortened dry periods may best begin after the second lactation is completed.

The dry period is a time when cows receive intramammary antibiotic therapy as part of a recommended mastitis control program. Intramammary antibiotic dry cow products are generally formulated for dry periods longer than 30 days. Clearly many cows enrolled in 30-day dry periods will calve earlier than expected which would increase the risk of antibiotic residues contaminating the milk supply. We conducted a study to determine if reducing the number of days dry would lead to an increased incidence of intramammary infections and decreased cure-rate during the dry period for cows given either long or short dry periods and receiving intramammary antibiotic therapy at the time of dry-off.

Four herds participated in the study and cows were assigned to 30, 45, or 60-day dry periods. Cows with the 30 day (short) dry period received intramammary treatment with 200 mg Cephapirin Benzathine, a lactating product, and cows with the long dry period (45 or 60 day) received 300 mg of a cephapirin benzathine dry cow product immediately after the last milking.

Both the cure rate and the new intramammary infection rate over the dry period was similar for cows given either long or short dry periods, and ranged between 72%-81%. Coagulase-negative staphylococci and *Corynebacterium bovis* were the causes of most dry period infections. The milk somatic cell count for the first 6-7 months after parturition in the subsequent lactation was not affected by dry period length. Cows given the 30-day dry period produced less milk than those given a 60-day dry period in the next lactation. However, cows with a 45-day dry period did not produce significantly more milk than those with the 30-day dry period.

Although shortening the dry period to 30 days had no negative effects on mammary gland health as measured by intramammary infections or somatic cell counts, production may be adversely affected by shortening dry periods to 30 days. However, the additional milk production in the lactation previous to the shortened dry period may off-set the loss seen in the subsequent lactation.

By: Dr. Larry Fox, Dept. Veterinary Clinical Sciences
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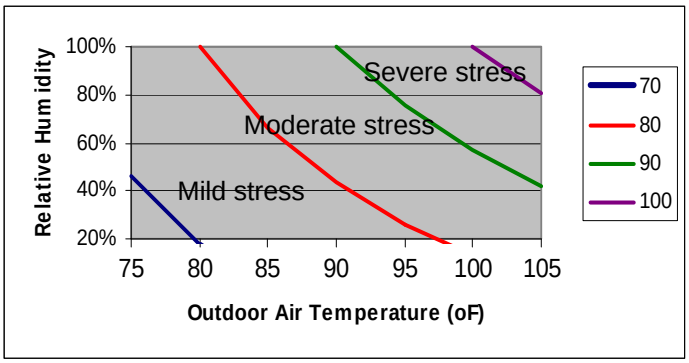
Preparing for Summer on the Dairy

Heat Stress Prevention

In the summer of 2006, over 30,000 head of cattle died due to severe heat stress effects in California. Deaths due to heat stress are unusual but what are more common are the effects that heat stress can have on cattle health, milk production and reproduction.



What is heat stress?

Heat stress is the combination of heat and humidity that negatively impact a cow's health and performance. It is often defined as a Temperature humidity index (THI) greater than 72. The ideal ambient temperature for a dairy cow is between 41 and 77° F. Moderate signs of heat stress may occur when the temperature is between 80° and 90°F with the humidity ranging from 50 to 90 percent. As ambient temperature approaches body temperature, sensible routes of heat loss (through respiration) are compromised which leaves only evaporative heat loss as the major route of heat dissipation. Because cows sweat very little we need to assist their heat dissipation. Heat stressed cows will often have the following behaviors:

- Seek out shade, and may not leave to drink/eat
- Increase water intake
- Reduce feed intake
- Stand rather than lie down
- Increase respiration rate
- Increase body temperature
- Increase saliva production

What are the effects of heat stress?

- Heat stress INCREASES: Respiration Rate, Rectal Temperature, Water Intake, Sweating
- Heat stress DECREASES: Rate of Feed Passage, Dry Matter Intake, Blood Flow to Internal Organs, Milk Production, Reproductive Performance.

Recent research on heat stress and reproduction showed that insemination at no to mild heat stress resulted in a Conception Rate (CR) of 38.8% but insemination under heat stress resulted in a CR of 17.6%. THI on day of insemination had greatest effect on non-return rate at 45 days.

Heat stress leads to **acidosis** through:

- Panting and loss of CO^2
- Decreased rumination
- Drooling and loss of salivary buffer (bicarbonate)
- Slug feeding (eating) in the cool part of the day leading to a drop in rumen pH

In one study, summer heat had the largest impact where:

- There was little or no heat abatement
- Bunk space and cow space were compromised
- Pen densities were high (overcrowded)
- No segregation of 1st lactation from older cows
- Transition management was less than optimal:
  a. Improper ration formulation and feed delivery
  b. Less than 3 feet bunk space for close-up cows.
  c. Fresh cows with less than 2.5 feet bunk space.
  d. Adequate cooling not in place during transition.

These factors all point to the importance of planning ahead and preparing for potential heat stress issues.

Managing heat stress effects on WA dairy cows:

The priority for heat stress management starts with the simple factor of increasing water availability to the more intensive cooling of cows in the facilities.

1. Water availability -- Cows need to increase water intake during times of heat stress to dissipate heat through respiration and by sweating. Water consumption will increase by as much as 50%. A 1400 lb cow producing 65 lb of milk at 3% butterfat needs at least 32 gallons of water per day at 80 degrees F. The same cow needs at least 36 gallons of water at 90 degrees F. If water supplies are not adequate or heat stress becomes severe, cows divert water normally used in milk synthesis to the processes of heat dissipation. Water intake will rise by 5-6 gallons on summer days due to temperature effects alone. To improve water availability:
   - Put waterers in the shade
   - Provide access to water right after milking. Cows drink 50-60 percent of their total daily water intake immediately after milking. When grazing, water should be located at the milking parlor exit and in each paddock so that animals are always within 600 feet of clean, fresh water sources.
   - Ensure enough waterer space by:
     - Having at least 1 station per 20 cows
     - Having a water supply with 3-5 gal/minute (cows can consume 6 gal./hr)
     - Maintaining 3 inches water depth
     - Providing 0.65 sq. ft. surface area per cow at single- or double-position waterers
     - At least one watering space or 2 feet of tank perimeter for every 15-20 cows and two feet of linear trough space per cow in return alleys.
   - Keep water tanks clean.
   - Monitor water temperature (cows prefer 70-86° F)
   - Water trough size and height: In an experiment, cows were given access to two water troughs that differed in height (30cm versus 60cm) and size (126cm × 68cm versus 139cm × 95cm), but were otherwise similar. Cows spent more time drinking, consumed more water and took more sips from the higher and larger of the two troughs.
2. Provide for maximum shade in the housing areas and holding pen (Lactating & Dry)
   - Are your shade structures sited North-South?
   - Are your freestall barns sited East-West?
   - Are you providing at least 25 to 50 square feet of shade space per cow?
3. Reduce walking distance to the parlor
4. Reduce time in holding pen
5. Improve holding pen and freestall ventilation
6. Add holding pen cooling and exit lane cooling
7. Cool close-up cows (3 weeks prior to calving)
8. Cool fresh cows and early lactation cows
9. Cool mid & late lactation cows

   Cooling cows might include:
   - Providing a cooler environment
     - Eliminate direct solar radiation
     - Decrease cow density
     - Cool the air (misting systems)
     - Create air movement (draw out hot air)
   - Cooling the cow

     Soak the cow with **sprinklers**:

     ✳ 0.33 gallons per cow/cycle
     ✳ operate when temperature greater than 70° F
     ✳ Duration depends on nozzle size (1-2 minuntes)
     ✳ Frequency: at 70-80°F (every 15 min), 81-90°F (every 10 min) greater than 90° F (every 5 min)
     ✳ Mounting height 6-12 inches above headlocks or 5 to 6 feet above floor
     ✳ Pressure in distribution line 15 to 20 psi
     ✳ Nozzle spacing every 6 to 8 feet
     ✳ Maximize number of wet-dry cycles/hour

Add **cooling fans:**

✳ Post spacing 24-30 feet (one 36-inch fan/post)

- Post spacing less than 20 feet – One 48-inch fan every other post
- Place fans over both free stalls and feed line

✳ Mount fans so that air flow is with prevailing winds and close to cow height

✳ Operate when temperature greater than 70°F

Adding fans provides little benefit unless a good soaking system is installed first.

10. Changes in feeding: Cow maintenance requirements *increase* with heat stress while dry matter intake goes down. Consider increasing feeding frequency (an extra feeding or two), feeding at a cooler time of day, or even changing the ration. Summer rations will require a buffer and better forage quality.

Preventing heat stress will help maintain higher milk production, better reproduction, improve animal health through reducing chances of acidosis and lameness, and improve animal well-being.

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What's New at WADDL?

The diagnostic laboratory posted their **annual report** on their website in April. To see the activities of the lab, go to: http://www.vetmed.wsu.edu/depts_waddl/

Pond scum – is it a problem or not?

Spring has returned to the Pacific Northwest, and with its return, come some problems that are of importance for domestic animals. Nitrate and lead poisoning in cattle are common to see in late winter and early spring, when cattle are turned out into pasture. Also, bovine pulmonary emphysema and edema [aka fog fever] is starting to rear its head. However, the one problem we generally see starting to arise in spring, and can last through summer, fall and into the early winter months is poisoning due to ingestion of cyanobacteria, commonly referred to as blue-green algae.

There are two main categories of cyanobacteria that are important to livestock: ones that cause neurotoxicity (nervous system effects) and ones that cause hepatotoxicity (liver effects). Both types of diseases, unfortunately, carry rather unfavorable prognoses. Cyanobacteria, formerly known as blue-green algae, are prokaryotic organisms which possess chlorophyll and carry out photosynthesis in the presence of light and air, producing oxygen. Cyanobacteria are found everywhere in the environment, and can be found in most water bodies but at very low densities. With appropriate environmental and water conditions [e.g. stagnant, eutrophic, high nutrient load, and calm weather conditions], the cyanobacterial organisms will rapidly proliferate to form a *bloom* which will rise to the surface of the water, giving the appearance of 'pond scum'. This bloom will look and have the consistency of pea soup or paint residing in the upper layers of the water. Livestock will be exposed when wind or water currents move the bloom to the shoreline, and animals go to drink.

Cyanobacterial blooms can come in a variety of colors – blue, green, red, purple, black – depending on the pigments that the organisms are synthesizing. Most blooms are blue-green in color, hence their name 'blue-

green algae'. These organisms can persist in the water for a variable length of time – hours, days, weeks or even months – depending on the weather and water conditions. There are many types of cyanobacteria, but only a few are commonly implicated in toxicities. *Microcystis* [*Anacystis*], *Nodularia*, and *Oscillatoria* species can produce hepatotoxins; *Anabaena*, *Aphanizomenon* and *Oscillatoria* species can produce neurotoxins. However, one cannot determine by visual inspection whether the cyanobacteria present in a bloom are producing the toxins – only testing can confirm the toxin's presence. There is no way to determine how much water livestock have to consume to receive a toxic or lethal dose, but based on clinical case reports, it appears to be easy for a cow to receive a lethal dose in a single exposure.

Bloom in eastern WA that was responsible for the death of over a dozen cattle on open range.

The hepatotoxins microcystin and nodularin are very specific toxins to the liver and cause massive liver failure within 4 to 6 hours of exposure. Often livestock are found dead near the contaminated water. The neurotoxins anatoxin-a and anatoxin-a(s) mimic the action of acetylcholine and inhibit acetylcholinesterase, and clinical signs of illness are often seen within a few hours of exposure. These signs can include excessive salivation, labored breathing, muscle fasciculations, weakness, recumbency, seizure, and eventually death.

Diagnosis of a cyanobacterial intoxication relies heavily on compatible signs, clinical pathological changes – gross and histological changes [only with the liver toxin blooms], history of access to a potentially contaminated water source [e.g., irrigation ditch, pond, lake, stream], confirming the presence of the organisms in the water or rumen / gastric contents of the affected animal, and confirming the presence of the toxin in the water or rumen/gastric contents and liver of affected animals.

There are some important misconceptions regarding blooms – one being that cyanobacterial blooms occur only during the hot dry months of August and September. It is true that this is the peak time to see cyanobacterial blooms in the Northwest; however, we have confirmed toxic cyanobacterial blooms in Washington and Idaho from the early spring to late January and into early February. Another myth is that if the bloom is toxic, you should see other dead animals around, and even in, the contaminated water [e.g., fish, coyotes, dogs, cats, deer, and elk]. Many times you do not see any other animals affected. Any fish that might be in the water will swim to other areas of the lake, pond or stream, and wildlife will apt to die away from the watering hole. Livestock, who typically loiter around watering holes, will be more apt to be found close by.

The key to preventing this poisoning in livestock is to know and regularly inspect all accessible watering sites. Though rare, cyanobacteria can potentially be found in shallow wells used for human consumption as well.
By: Dr. Patricia Talcott, Toxicologist, WADDL

WA Department of Agriculture Corner

Management of Animal Health Events using ID

For the Washington State Department of Agriculture, the focus has long been on disease prevention. But when animal health experts diagnose an animal disease, three questions asked are:

- Where has the animal been?
- What other animals have been exposed?
- What additional animals are at risk of exposure?

The answers to these three questions are critical to determine the size of the outbreak, identify the source of the disease, and reduce the spread and economic impact of the event. When fighting outbreaks of animal diseases, time is the enemy. The economic impact of any animal disease event is directly proportional to time of its containment.

Consider the cattle industry's worst nightmare – foot and mouth disease. The 2001 FMD outbreak in the United Kingdom cost $16 billion. No other single event has had such a negative economic impact since World War II. A similar FMD outbreak in the U.S. is estimated to cost $60 billion. The predicted impact of an FMD outbreak in the US is an additional $10 million for every hour we do not get containment. One scenario, which was modeled on actual livestock movements, predicts exposed livestock would be in 23 states by day 5 of the outbreak. The incubation time

for FMD, one of the most contagious diseases known to man, is just 5 days.

But the story doesn't stop there. Tuberculosis is spreading across the country. Cattle producers suffer hardship with increased testing requirements and decreased value of cattle originating in states that lose their TB free status. Yet today, the average time it takes to trace a TB case is still 125 days. It's no wonder that the disease is spreading.

In 2003, an avian influenza outbreak just across Washington's northern border in British Columbia cost producers $350 million, not to mention up to three times that in total economic impact to the B.C. economy.

In today's marketplace, we are moving livestock farther and faster. By tracing livestock using brand records, WSDA found that in two sales at Toppenish and Davenport in two days, the cattle move all over Washington and most of the Western states. That is in just two days.

The Challenges of Traceability

For many years, animal identification has been an integral part of animal disease monitoring, surveillance, and eradication programs. Today, participation in active disease programs has decreased as diseases have been eradicated. This decrease is most apparent in the cattle industry. In the past, when livestock diseases like brucellosis were widespread, cattle were commonly identified, tested and vaccinated. The process included officially identifying those animals and recording their information in the state and federal animal health systems, creating a high level of traceability for the cattle industry. Today, however, while Washington still has requirements for brucellosis vaccination, many states do not. Less than 12 percent of U.S. cattle population is vaccinated for brucellosis.

And even when animal ID information is available, it can't be used effectively during a response to an animal health event. States, industry, and USDA maintain separate animal identification information systems. Current animal identification and data collection approaches typically address individual objectives, such as specific disease eradication programs, interstate commerce, breed registries, and age and source verification. An animal can be identified multiple times, yet still not be fully traceable in a disease event because separate programs use distinct herd and flock identification protocols. These many sources of information cannot be accessed quickly.

Increasing Traceability

Traceability is the key to protecting animal health. In order to respond quickly and effectively to an animal disease event (whether it is a single incident or a full-scale outbreak), animal health officials need to know which animals are involved, where they are located, and what other animals might have been exposed. The sooner reliable data is available, impacted animals can be located, appropriate response measures can be established, and disease spread can be halted. Retrieving animal location and movement data within a 48-hour window is optimal for efficient, effective disease containment. The ability to find infected and exposed livestock in a rapid manner will:

- Increase states' disease response capabilities
- Limit spread of animal diseases
- Minimize animal losses and economic impact
- Protect producers' livelihoods
- Maintain market access

WA State Animal Identification System (WSAIS)

WSAIS was created because Washington producers wanted WSDA to house contact information, animal identification, and animal movement information in a state database. WSAIS is a Washington State based data system that stores livestock identification information such as Brands, Certificates of Veterinary Inspection, Bangs tags and test information (i.e. Brucellosis, Tuberculosis, etc). It also houses voluntary contact information in case of an animal health event.

If you choose to participate in WSAIS, you will be notified quickly when a disease event could affect your livestock. The state veterinarian's office will give you the information you need, when you need it most to protect your livestock and your investment. By registering, you will help speed up our efforts to identify and contain disease threats. Ultimately, this tool will help protect market access, gives you a competitive trade advantage and preserve the confidence of our trading partners.

Be assured that participating in either USDA's National Animal Identification System (NAIS) or the Washington Animal Identification System (WSAIS) is strictly voluntary. There are no penalties or other enforcement mechanisms associated with these programs. And under WSAIS, no information will be shared with USDA unless requested by a livestock producer or land owner. If you are interested in registering your contact information or premises, please contact my office at:

Washington State Department of Agriculture
Animal ID Program
PO Box 42560
Olympia, WA 98504-2560
(360) 725-5493
www.agr.wa.gov/FoodAnimal
wsais@agr.wa.gov
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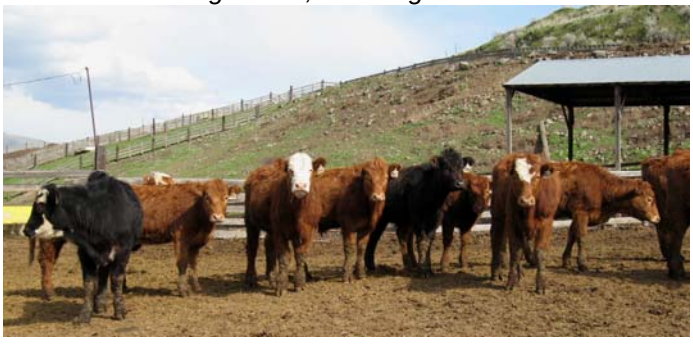

(USDA ARS Photo)

Porcine Reproductive and Respiratory Syndrome (PRRS) Virus

Porcine Reproductive and Respiratory Syndrome (PRRS) is a viral disease first recognized in the 1980's. The degree of reproductive loss or disease varies widely from herd to herd. Sows may abort or farrow early or have stillbirths. They may not have any milk and may develop mastitis. Pigs can develop respiratory signs, go off feed and may show diarrhea. There are many different clinical signs attributed to herd infection with this agent and some herds have no clinical signs at all. If the virus is circulating in the herd, pigs can become infected when maternal antibodies wane and these infected pigs can shed virus for 4 weeks.

A recent article in the *Journal of the American Veterinary Medicine Association* reports on the evaluation of contact exposure to acclimatize pigs to PRRS. Acclimatization is the process of deliberate exposure of young pigs to the farm-strain of PRRS to induce immunity before infection at the costliest time: late gestation. Inoculation has been successful but is relatively costly and time consuming. The study's objective was to determine if previously un-exposed 6.5 week-old gilts could become acclimatized to a farm-strain of PRRS is co-mingled with the same age gilts that were incoculated with the farm strain and if 10.5 week-old gilts could become acclimatized if commingled with inoculated or contact-exposed animals. The study outcomes were development of immunity to PRRS and effect on reproductive performance. The study resulted in all the gilts having evidence of infection by 19.5 weeks and developing antibodies to the farm strain by about 40 weeks of age. Litter size (about 10) did not vary between the three groups of gilts: inoculated at 6.5 weeks, contact-exposed at 6.5 weeks or contact-exposed at 10.5 weeks of age.

Once pigs become infected and develop immunity at a young age, the likelihood of outbreaks of the disease in the herd is greatly reduced. A ratio of 1 inoculated to 4 contact-exposed pigs appears to be an effective acclimatization strategy. However, biosecurity practices to prevent the introduction of *new* strains onto the farm are still essential because the degree of cross-protection appears to be unknown.

Reference

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(For general information on PRRS, visit the Pigsite at: http://www.thepigsite.com/diseaseinfo/97/porcine-reproductive-respiratory-syndrome-prrs)

By: Dale A. Moore, Veterinary Medicine Extension

Updates on Abortion Diseases in Sheep

According to the Merck Veterinary Manual, "abortion in ewes, as in cows, is not always easily diagnosed." A recent paper published in Spring 2008 lists all the infectious causes of abortion in various different species from cattle to dogs - we'll focus on sheep (ovine) for this article. It is important to note that many of the causes of sheep abortion are zoonotic, that is to say, they can infect humans, so any personnel working with possibly infected samples should employ measures to prevent infecting themselves.

Sheep abortion contributes to large economic losses to the industry. A recent study looked at patterns of late embryonic and fetal mortality and the association with several other factors. The number of ewes lambing and lambs born were compared with pregnancy diagnosis and counts of embryos by ultrasonography near d 25, 45, 65, 85 of gestation.[1]

- 20% of ewes had embryonic or fetal loss
- 21% of embryos/ fetuses lost d 25 to term
- 3.7% of embryos lost d 25 to 45
- 4.3% of fetuses lost d 45 to 65
- 3.3% from d 65 to 85
- 12% from d 85 to birth

Thus very early in gestation and very late in gestation appear to be the most likely times for pregnancy loss. The major infectious agents causing abortion in sheep are:

Campylobacter spp.	Border disease
Chlamydophila	Cache Valley
Toxoplasma	Bluetongue
Listeria	
Brucella	
Salmonella	
Coxiella burnetti	

Campylobacter spp. is the most common finding in abortions of sheep in North America. The bacteria *C. jejuni* (sporadic abortions) and *C. fetus* subsp. *fetus* (recurring abortion) often cause abortion late in pregnancy or stillbirths. Ewes do not show clinical signs but abort in the third trimester, have stillbirths, or give birth to weak lambs. Some ewes are persistently infected and continue to shed the organism in their feces.

Chlamydophila abortus (formerly Chlamydia psittaci) - Causes Enzootic Abortion of Ewes (EAE) and is characterized by late term abortions, stillbirths, and weak lambs. Abortions occur in the last 2-3 weeks of gestation regardless of when infection begins. Once the abortion occurs, the immune system will prevent subsequent abortions, but the organism can continue to be shed in vaginal secretions. Control consists of isolation and treating in-contact ewes with long-acting oxytetracycline or oral tetracycline. **NOTE:** *C. abortus* is zoonotic and poses risk to pregnant women, although rare.

Toxoplasma gondii (Toxoplasmosis) most comes from contaminated feed. Infection occurring <40 days of gestation results in embryo resorption. Infection at 40-120 days of gestation leads to abortions or mummification. Infection after 120 days can result in stillbirth or birth of weak or healthy lambs. A recent study showed that if a lamb aborted there was a high risk (55%) of abortion in the next lamb produced. These data suggest that life-long immunity to *T. gondii* infections may not always be acquired following an initial infection[3] as once thought. Keeping cat feces out of feed will help prevent exposure. **NOTE:** *T. gondii* can affect nonimmune people.

Listeria monocytogenes infection usually occurs through ingestion of poor quality alkaline silage or areas contaminated with feces containing this bacterium. Vaccine results have been equivocal and spoiled silage should be avoided. Ewes can abort 7-30 d after infection. Prior to aborting, ewes might show signs of fever and decreased appetite. Following abortion, ewes often develop metritis (inflammation of the uterus). *L. ivanovii* has also been shown to cause abortion in sheep. The bacteria are susceptible to penicillin (drug of choice), ceftiofur, erythromycin, and trimethprim/sulfonamide. **NOTE:** Zoonosis potential is uncertain because of findings where *Listeria* organisms were isolated from a significant number of healthy people as well as other animals. However, all suspected material should be handled with care, especially, aborted fetuses and necropsy of septicemic animals. Fatal cases have occurred where people have handled aborted material but is extremely rare (12 cases per million population per year).

Brucellosis such as caused by *B. ovis* rarely causes late-term abortions, stillbirths, and birth of weak lambs. *B. melitensis* is rare in the U.S. but causes abortion in places it has been found. *B. abortus* can also cause abortion in sheep. Ewes are generally without symptoms and abortions usually occur late in gestation, have stillbirth or give birth to a weak lamb. Most fetuses aborted due to *B. ovis* are mummified or autolyzed (absorbed). Ewes clear the bacteria within a few weeks of an abortion. A vaccine for *B. melitensis* is available in some countries. **NOTE:** *B. melitensis and B. abortus* are zoonotic, whereas *B. ovis* is not.

Salmonellosis abortion can follow an infection with *S. abortus-ovis, S. Montevideo,* or *S. arizonae.* Overcrowded conditions and shipping can predispose flocks to abortion storms with these organisms. Affected ewes can be without symptoms prior to aborting, or have clinical signs of fever, depression and diarrhea. Metritis and retained placenta can occur after aborting. **NOTE:** These pathogens are zoonotic.

Coxiella burnetti are usually transmitted by inhalation, contact with mucous membranes, ticks, and possibly seminal fluid. Infected ewes might develop placentitis and abort late in gestation, although abortion in sheep is not as common as it is in does. Abortion is not likely to occur the next lambing season due to development of immunity within the flock. Humans can become infected when handling infected placentas or lambs, and from inhalation of contaminated dust. The clinical signs of infection in people may include prolonged illness, fever that comes and goes, atypical pneumonia, hepatitis, myalgia, or endocarditis.

Yersinia spp. such as *Y. pseudotuberculosis and Y. enterocolitica* (serotype O) have been isolated from ovine abortion cases. Infection with *Y. pseudotuberculosis* leads to abortion, stillbirth, or birth of weak or healthy lambs. Infection with *Y. enterocolitica* has resulted in placentitis and abortion, with subsequent normal pregnancies. In humans, acute mesenteric lymphadenitis with fever, anorexia, vomiting, and diarrhea are common in people infected with these bacteria.

Border Disease Virus occurs worldwide and is an important cause of embryonic and fetal deaths, weak lambs and congenital abnormalities. The cause is a pestvirus closely related to Bovine Viral Diarrhea virus and Hog Cholera virus. Ewes infected before day 60-85 of gestation have fetal resorption, abortion, maceration, or mummification. If the fetus survives, it will be persistently infected with the virus, have potential cerebral brain damage, shortened facial and long bones, and produce hair rather than wool. If infection occurs after day 85, abortion, birth of weak or normal lambs, or birth of antibody positive lambs can occur. No clinical signs are shown in the dam. There are no vaccines available and inactivated BVD vaccines that are sometimes used on sheep are unproven in their effectiveness.

Cache Valley Virus is transmitted by mosquitoes and causes stillbirths, abortions, infertility, and multiple congenital abnormalities in sheep. The ewes display symptoms of fever, depression, and a reluctance to move. The virus is endemic in parts of the U.S., Canada, and Mexico. Stillbirths are the most noticeable effects and at the time of abortion, the virus is usually no longer viable. Diagnosis is by demonstration of antibodies in precolostral serum or body fluids. There are no vaccines.

Bluetongue Virus is an orbivirus and is transmitted by the biting midge (sandfly). Infected ewes can abort, have mummified fetuses, or deliver lambs with congenital defects (hydranencephaly – cerebral hemispheres are replaced with fluid-filled cavities; porencephaly – cavities in brain substance; cerebellar dysgenesis – defective formation of the cerebellum; and skeletal deformities). The ewe can show clinical signs of fever, lameness, oral and nasal ulcers, and swollen tongue, ears, or face.

From the Diagnostic Lab: Of 6 cases diagnosed between 2000-2004 two cases diagnosed as Vibrionic placentitis and abortion caused by *Champylobacter jejuni*; two were Bacterial placentitis and abortions caused by *Chlamydophila abortus*; and one caused by an ascending reproductive tract infection.

Summary

Abortions in sheep are caused by a number of different organisms, some of which can be prevented or treated and some that are difficult to control. If abortions occur with a group of sheep, an important step to prevention is to obtain the correct diagnosis. Sending samples such as the fetus and placenta to the diagnostic lab will help you make that diagnosis.

References:

1 – Dixon AB, Knights M, Winkler JL, et al; Patterns of late embryonic and fetal mortality and association with several factors; J. Animal Science 2007; 85(5): 1274-84.

2 – Daniel Givens M, Marley MSD, Infectious causes of embryonic and fetal mortality, Theriogenology. 2008; 10622: 5-7.

3 – Morley EK, Williams RH, Hughes JM, et al; Evidence that primary infection of Charollais sheep with *Toxoplasma gondii* may not prevent fetal infection and abortion in subsequent lambings; Parasitology. 2008; 135(2):169-173.

4 – Washington Animal Disease and Diagnostic Laboratory, Pullman, WA

by Brandon Freitas, MS, Extension Coordinator

Ag Animal Health Continuing Education

Veterinary Extension Website

Veterinary Medicine Extension has a website with unit activities. You can find it at: http://vetextension.wsu.edu/

Animal Health Risk Communication Training

The National Center for Foreign Animal and Zoonotic Disease Defense will host two training sessions on Risk Communication for Animal Health Events.

Dates: September 17, 2008 on the East side
September 18, 2008 on the West side

Contact: damoore@vetmed.wsu.edu for more information

Academy of Dairy Veterinary Consultants

The Academy of Dairy Veterinary Consultants (ADVC) is a group of dairy veterinarians who meet twice a year to discuss current issues in dairy herd health and dairy performance. Founded in California in the early '80s, the group has expanded to practitioners primarily in the West but members come from states as far away as Massachusetts. **Fall Meeting: SAVE THE DATE!** The Fall ADVC meeting will be held **October 24-25, 2008 in Santa Barbara, CA**. Topic: **"DEVELOPING THE STANDARD OF FOR CALF-REARING PRACTICE AND CONSULTATION".** For ADVC Membership and registration, contact Bill Sischo: mailto:wmsischo@vetmed.wsu.edu or (509) 335-7495 or Dale Moore: mailto:damoore@vetmed.wsu.edu (509) 335-7494.

WSVMA 97th Annual Veterinary Conference

October 3-5, 2008, Spokane Convention Center, Spokane, WA, Registration open August 4, 2008. http://www.wsvma.org/

Producer Education Meetings:

Northwest Junior Sheep Exposition, July 17-19, 2008, Grant County Fairgrounds, Moses Lake, WA

2008 WA State Sheep Producers (WSSP) Convention is going to be hosted by the Whitman County sheep producers in Pullman, October 31 – November 2, 2008. http://www.wssp.org/index.htm

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