WASHINGTON STATE UNIVERSITY
VETERINARY MEDICINE EXTENSION

ag animal health

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WASHINGTON STATE UNIVERSITY EXTENSION & WSU COLLEGE OF VETERINARY MEDICINE

In This Issue

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From the Editor – "I'm from the government, and I'm here to help you."

Although most of us laugh about this and don't feel comfortable with too many regulations and government interventions, I have been amazed at the help producers in the state of Washington actually do get from their state veterinarian's office. Most of the help comes in the way of education – what's happening with disease outbreaks, some informational bits to let you all know what's coming in the way of regulation, but also what the easy steps are to comply with animal and public health regulations to keep us out of hot water and to help us make the right decisions. This month, the WSDA is sponsoring three meetings for dairy producers and veterinarians on FDA's proposed new milk testing (Page 5). They *are* from the government and they *do* help!

Featured Faculty – Dr. Margaret Davis, Epidemiologist

Dr. Margaret Davis

Dr. Davis graduated from the WSU College of Veterinary Medicine in 1985 and practiced small animal medicine for 3 years. She obtained a Master's of Public Health (MPH) in Epidemiology (1992) at UW and worked as an epidemiologist for the Seattle-King County Department of Public Health from 1990-1996. She returned to WSU for a PhD in molecular epidemiology (2002). Her research interests include molecular epidemiology and transmission of the major zoonotic enteropathogens *Campylobacter, E. coli* O157:H7 and *Salmonella*. Public health impacts of these pathogens are often complicated by antibiotic resistance and her research area includes the relationships between resistance genes and resistance phenotypes in *E. coli* and *Salmonella*. She is currently PI of a USDA-funded project titled "Minimizing antibiotic resistance transmission: the dairy farm as a model system" which involves development of a communication model for antibiotic use messages on dairy farms, exploring the pre-weaned calf environment for ways to minimize transmission, and comparing genetic determinants of resistant *Salmonella* from cattle to those from people. Dr. Davis is currently serving as the Washington State University's Veterinary Teaching Hospital Infection Control Epidemiologist and will be teaching epidemiology to veterinary students beginning 2012.

Mycoplasma Mastitis and Beyond: Dissemination of the pathogen within the cow and the herd

By: Larry Fox, PhD, WSU

***Mycoplasma bovis* can cause more than just mastitis** – it can also result in pneumonia, otitis media, arthritis, and vaginitis (Pfutzner and Sachse, 1996; Maunsell and Donovan, 2009). The complex of diseases caused by *M. bovis* is collectively termed *M. bovis*-associated disease (**MbAD**) (Maunsell and Donovan, 2009). The two most prevalent and costly forms in dairy cattle are pneumonia and mastitis. Nicholas and Ayling (2003) discussed losses due to **MbAD** in a recent review and estimated that in Europe, more than 150 million Euros are lost each year to *M. bovis* respiratory disease. In the US it may be only $30 million from respiratory disease but over $100 million from mastitis.

Almost 10% of dairy herds in the western USA have cases of mycoplasma mastitis and appears to be a function of herd size (Anonymous, 2003a and b).

Key Points

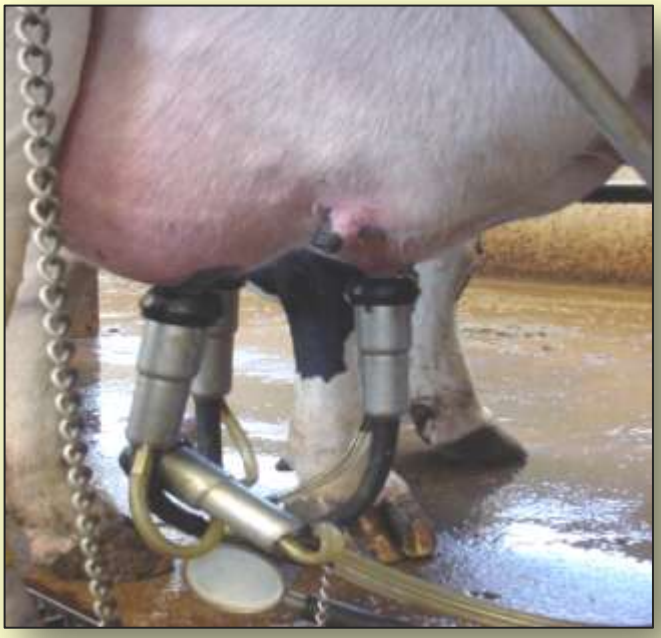
- ***Mycoplasma causes a range of disease problems including mastitis, pneumonia and ear infections***
- ***Weekly bulk tank cultures are recommended so as not to "miss" cows that are shedding Mycoplasma***
- ***Strains of Mycoplasma "unique" to the herd can cause an outbreak***
- ***Some cattle can carry the organism and not be sick***
- ***The risk of developing Mycoplasma mastitis in the hospital pen was 100 times greater than the home pen***

The regional estimate in the Pacific Northwestern states is about 8% of herds (Fox et al., 2003). Although there are a number of *Mycoplasma* species that cause mastitis, *M. bovis* is the most prevalent, and most mycoplasma mastitis caused by this species (Gonzalez and Wilson, 2003).

Maunsell and Donovan (2009) recently reviewed the literature on *M. bovis* infections in young dairy calves. **They indicated that *M. bovis* bovine respiratory disease (BRD) is a significant problem in US dairy calves**, although there are no accurate estimates of the extent of the problem in the US. However, they suggested that the problem of BRD by *M. bovis* in dairy calves in the US is more prevalent than in Europe where it has been estimated that 25-35% of calves are affected, perhaps as many as 30 million cattle (Nicholas and Ayling, 2003). A US survey indicates that 12.4% of unweaned dairy calves are affected by BRD and 46.5% of weaned calf deaths are due to BRD (Anonymous, 2003b). We have been studying the **MbAD**, links between diseases in the herd between all age groups with specific reference to mastitis.

Our research at Washington State University has shed some new light on *Mycoplasma bovis* transmission, especially within the animal itself. We purchased 10 cows with mycoplasma mastitis and milked them for 30 days before slaughter, testing their milk

daily (Biddle et al., 2003). We found that 5 cows shed the pathogen consistently in high numbers, such that even in a large herd of more than 1000 cows, the *Mycoplasma bovis* would be shed in sufficient numbers such that the bulk tank milk would still be positive to the agent. The other 5 cows did not shed the pathogen in high numbers, and thus cows with mycoplasma mastitis could be missed by monitoring the bulk tank infrequently. From this, **we recommend weekly bulk tank culture to monitor herd mycoplasma mastitis.**



At slaughter, many body sites (lungs, urogenital tract, lymph nodes, ears, and heart) had the same clone, or strain, of mycoplasma that was causing mastitis. In other words, the type of *Mycoplasma bovis* that was causing mastitis was also the one colonizing many body sites. The UC Davis research group (Jain et al., 1969) reported years before that mycoplasma appeared in the blood of cows with mastitis, and was likely to spread to different body sites. Our findings confirmed this spread and we found other strains not involved with mastitis. Although a small minority, the non-mastitis-causing strains of *Mycoplasma bovis* could be found throughout the animal.

The **dissemination of *Mycoplasma bovis* throughout the body** was confirmed in some of our other studies (Fox et al., 2008, Punyapornwithaya et al., 2010) and yet we always noted a predominant strain in various organs. An outbreak of mycoplasma mastitis in one herd was preceded by pneumonia and arthritis in calves. The strain causing the calf diseases was the same that caused the mastitis. When we went to sample the herd, we found that one-third of calves and half the cows were shedding the mastitis strain of *Mycoplasma bovis*, mostly from their noses. We continued to sample animals for a year and shedding virtually disappeared to less than 3% of animals. Not only did shedding sharply diminish over time, but when we found *Mycoplasma bovis* shedding in the herd, most often it was a different strain or species than what had caused mastitis. Cases of mastitis disappeared along with shedding, and the disappearance of mastitis coincided with a sharp reduction in appearance of the outbreak strain. This phenomena can be referred to as a "bloom". The unique strain of *Mycoplasma bovis* will be spread to both cows and calves, colonize animals but cause disease in only a few. How does it spread? Given that it spread quickly and caused disease in calves suggests that it was spread by aerosols from the nose. Transmission from cow to cow by udder in the milk parlor is possible and cannot be ruled out. The fact that half the cows and a third of the calves were shedding the strain which was found most often in nasal swabs, **suggests that nose to nose contact was the main route of spread**.

What was the source of the mycoplasma mastitis outbreak in our study? Calves were raised off-site at a local dairy herd. Additionally, some fresh heifers were obtained from a local herd and milked in the study herd. **The animals imported into the study herd could have carried the mycoplasma strain and spread it to other cattle.** None of the imported fresh heifers ever developed mastitis, and none of the calves reared off-site became diseased. Only the home herd animals became diseased. A larger

percentage of animals directly exposed to the off-site animals became carriers of the mycoplasma strain. We think the source of the strain was the local herd that had carrier animals that spread it to the study herd, and then it became widespread. But why did the spread and apparent carriage of *Mycoplasma bovis* stop? Why was the disease limited? We hypothesize that local herd animals had developed immunity and were able to keep the mycoplasma strain in check, and simply carried it without symptoms. The study herd was naïve to the strain, and when first exposed to these asymptomatic carriers, lacking immunity, they became infected and some became sick. Eventually the study herd animals developed immunity and the disease and colonization apparently went away. Did the strain entirely disappear? This is unlikely, because it was still found in a few animals a year after the outbreak.

With this **"bloom" theory of development of mycoplasma carriage and spread**, leading to disease, one might wonder: What is the best method to control mycoplasma mastitis on a dairy? At the study herd, the first cows, half the cows that developed disease, were immediately culled once they were determined to have mycoplasma mastitis. The other half were kept in the herd and milked separately. They eventually cured themselves and were free of the disease in their next lactation. Does this mean that the test and slaughter method of control should not be followed? Every cow is not the same, some are good milk producers, some not. Not all *Mycoplasma bovis* are the same; some are more virulent than others. Given that *Mycoplasma sp*. in general, is difficult to culture, and transmission may occur rapidly, many producers are tempted to cull quickly and ask questions later.

We examined control measures likely to be associated with rapid time to clearance of mycoplasma mastitis in a herd. In 18 herds we studied control practices after a very recent and new bulk tank mycoplasma positive result. Nine of the herds aggressively tried to find the infected cow(s) and cull them. The other herds decided to monitor the bulk tank and wait to determine if things improved. **In the monitoring herds, none had recurrent cases, and their mycoplasma situation resolved without intervention. Thus the test and slaughter approach did not seem to be critical to controlling the mycoplasma mastitis outbreaks and no single milking time hygiene practice seemed to separate herds with shorter vs. longer recovery periods.** Only 1 of the 18 herds continued to have an outbreak lasting more than 2 months. This herd not only had cases of mycoplasma mastitis, but concurrent cases of arthritis and pneumonia in lactating cows where the mycoplasma was the same strain causing mastitis (Punyapornwithaya et al., 2011). The herd's mycoplasma mastitis outbreak lasted 5 months. The manager of this herd aggressively tried to determine which cows had mycoplasma mastitis. The suspect mycoplasma cows were kept in a newly-formed hospital pen. The trouble was that all mastitis cows went into the hospital pen, and stayed there until the mycoplasma culture results were known. On average, cows stayed in the hospital pen about a week. Cows with mastitis other than mycoplasma developed mycoplasma mastitis in the hospital pen. More cows developed this disease than had this disease in the home pens. In fact, **the risk of developing mycoplasma mastitis in the hospital pen was 100 times greater than in the home pen**. Thus the hospital pen appeared to be a fertile area of spread of this disease. The herd manager was very much aware of the need to maintain excellent milking time hygiene for the hospital pen cows. Was the spread between hospital pen cows due to nose to nose contact, or via udder to udder spread at milking time? We will never know for certain. The point is amongst the 18 herds, one herd appeared to have a more virulent strain, and the very act of trying to contain the problem may have actually contributed to the spread. It could be argued that although the spread seemed to be heightened in the hospital pen, it rarely spread beyond the pen and thus the formation of the hospital pen might have contained the problem. Additionally, this herd had outbreaks of pneumonia and arthritis in their

cows, and all the isolates from mastitic quarters, nares, and joints were the same identical strain of *M. bovis*.

Research from the Washington State University mastitis group indicates that cattle can carry *M. bovis* asymptomatically, and such carriage can be associated with an outbreak of **MbAD**. It appears that a test and slaughter approach to control mycoplasma mastitis might not be the only approach to control this disease. Some herd managers relied on close monitoring and the disease seemed to "run its course". It could even be argued that the formation of a hospital or isolation pen for segregation of *M. bovis* animals might not aid in the control of this disease. **We need to rethink our "one-size-fits-all" strategy for control of this disease in dairy cattle.**

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Testing for Residues in Dairy Cattle

"Feds: Wash. dairy cows had unlawful drug residues

By Dairy Herd news source | Sunday, January 16, 2011 *SEATTLE (AP) — Federal authorities have sued a northwest Washington dairy that they claim has a long history of selling cows for slaughter even though their tissues contained drug residues deemed unsafe to eat. …"*

The above article cites poor record-keeping and improper drug administration as the major reasons for residues found in market cattle from the farm. FDA will soon begin a milk sampling program aimed at approximately 900 dairies across the US that have had a meat residue violation in the past three

years. These dairies will be randomly selected from a national pool of 1600-1800 dairies. The FDA is concerned that operations that have marketed an animal with a confirmed meat residue may be at higher risk of selling milk with violative residues, particularly with non beta-lactam (beta-lactams = penicillin, ceftiofur) drugs. Of particular interest are the anti-inflammatory drug flunixin meglumine (Banamine®) and the sulfa family of drugs.

Since the announcement of FDA's intent, dairy organizations and co-ops have provided information to producers, and many veterinarians and dairy producers have been looking at their treatment protocols and withholding times more closely. However, there are still some questions producers have about the process and the consequences of the testing program. And, probably more important is: What are the reasons for the residues seen and what should we look for to tighten up our drug use procedures?

The *Washington State Department of Agriculture* and *Washington State University Extension* are sponsoring 3 meetings throughout the state for **dairy producers** and **veterinarians** to help address this issue. The meetings will start at *11:00am* and lunch, sponsored by Pfizer Animal Health and Northwest Dairy Association, will be provided on the following dates:

- ***Wednesday -- March 30, 2011*** *- Bon Vino's Bistro, 122 N. 16th St, Sunnyside, WA*
- ***Thursday -- March 31, 2011*** *- Kit Carson Restaurant, 107 Interstate Ave, Chehalis, WA*
- ***Friday -- April 1, 2011*** *- Fairway Café, 1726 Front St, Lynden,*

Meetings will begin promptly at 11:00 AM at each location. Please come! At each of these three meetings, we invite dairy producers and veterinarians to discuss the following:

1. ***What are the facts about the FDA's concern and what is the proposed process for milk testing?*** Claudia Coles, Food Safety Program Manager, and Dr. Paul Kohrs, Assistant State Veterinarian, WSDA – 30 minutes
2. ***What are the primary reasons for a residue from specific drugs and how does that inform prevention strategies?*** Dr. Dale Moore, Director, Veterinary Medicine Extension, WSU – 30 minutes
3. ***How can I reduce disease and treatments, determine if treatments are working, and better track drug withdrawal times? Your cows have the answers; learn how to understand what they are saying.*** Dr. John Wenz, Assistant Professor, Field Disease Investigation Unit, WSU – 30 minutes
4. ***What are the current requirements for handling non-ambulatory cows, what role do they play in food safety, and what are some of the reasons for down cows?*** Dr. Paul Kohrs, Assistant State Veterinarian, WSDA – 30 minutes

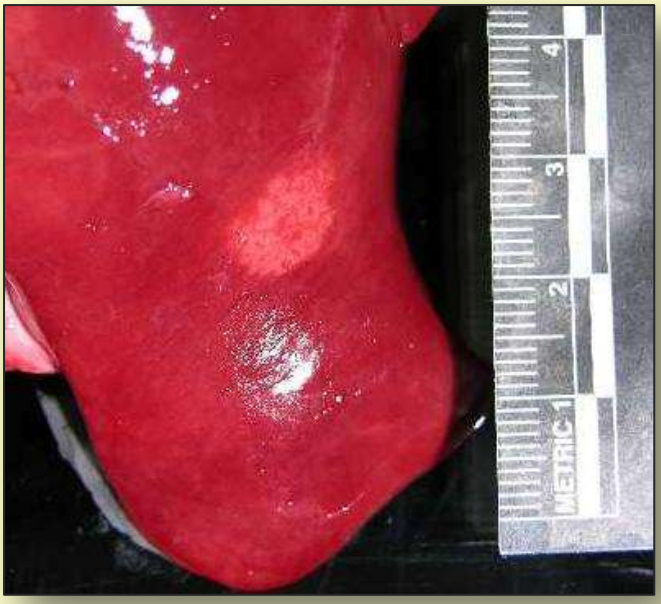
What's New at WADDL? Sheep Abortion Outbreaks

by Drs. Besser and Potter

An emergent strain of Campylobacter causing abortion in sheep is resistant to tetracycline, the drug usually used to treat animals in the face of an outbreak. WADDL recently diagnosed *Campylobacter* abortion in two sheep flocks, one in Idaho and one in Washington. The Idaho flock was a well established Suffolk flock with no history of previous abortions. The flock experienced premature birth of weak or dead lambs starting in early February. The Washington flock was composed of crossbred ewes, including recent introductions two weeks prior to the onset of abortions. This flock experienced 2-3 abortions per day. Lesions identified by histopathology included

severe placentitis (inflammation of the placenta) in both cases and fetal meningitis (inflammation of the covering of the brain), epicarditis (inflammation of the heart sac) and bronchopneumonia. In both cases, *Campylobacter* type organisms were identified on Victoria blue stains of placental smears and *C. jejuni* was isolated in high numbers from fetal tissues and abomasal fluid. Isolates were resistant to tetracycline.

Three species of *Campylobacter* can cause abortion in sheep and cattle. *Campylobacter fetus* subsp *venerealis* causes genital infections resulting in infertility and abortion, primarily in cattle. It is a true venereal infection. *C fetus* subsp *fetus* and *C jejuni* cause intestinal infections that result in transient bacteremia (bacteria in the bloodstream) and are particularly problematic in sheep and goats. Transmission is by the fecal-oral route. If non-immune ewes are pregnant during the bacteremic phase they may abort. Characteristic gross lesions are placentitis centered on cotyledons (seen in both cases here) and grossly evident multifocal hepatic necrosis in fetal livers (not always present). Abortion rates in newly infected flocks range from 5 to 50% but average 25%.



Sheep fetus liver with typical necrotic lesion from C fetus
(not seen in the current Washington or Idaho cases).

Typically, infection with *Campylobacter* bacteria during an outbreak results in immune responses that provide considerable immunity at least during the first year or two after an outbreak. Vaccines for both *C. jejuni* and *C. fetus* are available, although their effectiveness is not well established.

C. fetus was the predominant cause of sheep campylobacter abortion through the late 1980s, but subsequently *C. jejuni* has emerged to become the more predominant agent. Almost 90% of *Campylobacter* associated sheep abortion outbreaks were due to *C. jejuni* in a 2002-2007 Iowa study, and the same study reported 95% and 100% of recent *Campylobacter*-associated sheep abortion outbreaks in Idaho and California, respectively, were due to *C. jejuni.* Furthermore, diverse molecular typing methods show that across the US, a single strain type of *C. jejuni* accounts for the vast majority of these outbreaks.

Treatment with appropriate antibiotics during a *Campylobacter* abortion outbreak can dramatically reduce losses. Unfortunately, the emergent *C. jejuni* stain type is characteristically resistant to the

tetracycline class of antibiotics, the only drug labeled for use in infectious ovine abortion. Antibiotics with better activity against this *C. jejuni* strain include tilmicosin (approved for use in sheep), tylosin, florfenicol, or tulathromycin, which if used in extra-label fashion must be administered with consideration for the requirements of the Animal Medicinal Drug Use Clarification Act (AMDUCA) regulations (http://www.avma.org/reference/amduca/amduca1.asp).

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The Nature of Vaccines Used in Veterinary Medicine by Dr. George Barrington

Vaccination is the process of inoculating an antigen into an animal. The term does not imply that an immune response will occur, but it is frequently assumed. *Immunization* is the process of inoculating an antigen into an animal and the animal responds with a detectable immune response. While the term indicates that an immune response has occurred, it does not guarantee protection, though that is often assumed.

Veterinary vaccines must meet specific requirements for purity, potency, safety and efficacy. For most, data has been submitted to and approved by the Center for Veterinary Biologics (USDA) which demonstrates the vaccine provides sufficient protection to support the label claims. These claims can span from "prevents infection…", to "for use as an aide in the reduction of disease…". Differences in label claims illustrate the variation in protection that may be expected from various vaccines. Clearly, consumers must be aware of the limitations in the ability of vaccines to protect animals from infection and disease.

An ideal vaccine would have the following characteristics: a) produce a strong immune response similar to natural infection; b) protect against clinical disease; c) protect for a defined period, preferably lifetime; d) minimal side reactions; e) simple administration; f) favorable cost to benefit ratio. The majority of vaccines in use today contain either killed or modified live whole bacteria or viruses (MLVs). Most killed vaccines use either aluminum hydroxide or some form of oil as an adjuvant. The adjuvant is used to help stimulate a stronger immune response to the antigen. Advances in molecular biology, immunology, microbiology, and chemistry have led to newer approaches for developing more safe and effective vaccines. A brief description of these newer vaccine technologies follows.

Subunit vaccines contain specific antigens that are either purified or genetically engineered in expression vectors. Advantages include a more pure product with more of the required antigen and less irrelevant antigens or other substances that might cause inflammation or immune suppression. They remove/reduce immunosuppressive or inflammatory components, and remove/reduce antigens associated with hypersensitivity.(redundant with prior sentence - OMIT) Disadvantages of subunit vaccines include their inability to replicate, limited antigenicity (ability to produce an immune response), and the frequent requirement for use with potent adjuvants.

Gene-deleted vaccines utilize pathogens that have had specific genes either deleted or inactivated. They can be either killed or modified live products. These vaccines share many of the advantages of conventional modified live and killed vaccines, but have specific virulence factors removed. Gene-deleted vaccines have been used effectively as marker vaccines in eradication programs because the immune response to the vaccine can be differentiated from the parent disease. Similar to MLV's, gene-deleted vaccines have the potential to recombine in the animal and regain virulence. Gene-deleted vaccines have been developed for pseudorabies, bovine herpes virus 1, and hog cholera.

Live vector vaccines are produced by incorporating a protective antigen(s) from one pathogen into another organism (vector) that can safely reproduce itself. Advantages include improved safety, use as marker vaccines, and the use against pathogens that poorly replicate during an infection. Viral vector vaccines have been developed for rabies, Newcastle disease, and canine distemper virus.

DNA vaccines involve the incorporation of genes that code for specific antigens that are engineered into bacterial plasmids. This plasmid DNA is then purified and directly given to the animal. The plasmid DNA utilizes the host animal's cellular machinery to produce a protein. The protein then acts as the immunizing antigen. Advantages of DNA vaccines include low cost, stability (not needing refrigeration), and safety as they cannot revert back to virulent form. Concerns with DNA vaccines include variation between animals in their response and if integrated genes could potentially induce cancer. DNA vaccines are under investigation for bovine herpes virus, bovine viral diarrhea virus, pseudorabies, porcine and equine influenza viruses, avian influenza virus, rabies, Newcastle disease, *Anaplasma marginale*, and *Cowdria ruminatium*.

Transgenic plants can be engineered to produce various antigens from pathogens as well as monoclonal antibodies for use in animals. High concentrations of antigen or antibodies could be delivered directly to the animal in its food to help reduce signs of disease.

There are many types of vaccines available for animal health. The key to using them effectively is to know the research behind them, results of field and challenge trials, and label requirements for storage and use.

WSDA Corner by Dr. Leonard Eldridge, State Veterinarian

Malignant Catarrhal Fever -- On December 9th, a call was received from a local veterinarian that had examined a dairy cow that was exhibiting signs of Malignant Catarrhal Fever (MCF). That dairy cow had been at the Puyallup Fair when there was sheep present, in spite of recommendations from WSDA and other experts on MCF. Dr. Pospisil completed an investigation and determined that it was most likely MCF, however, we completed testing and this proved to be the case. The disease was

confirmed at both Plum Island and WADDL laboratories. The Fair management decided, after numerous requests, to have the dairy display cows remain in the same barn as the sheep during the final days of the fair. They agreed to take all of the risk if the owners would consent to leave these animals; 10 owners agreed. The fair management, in a letter sent to owners, has decided that the risk is not worth it any longer. These two species will again be separated in 2011 as they were in 2009 when there were no confirmed cases of MCF.

Trichomoniasis -- I recently traveled to Tonasket to talk to the area cattlemen about Bovine trichomoniasis (Trich). Some of the cattlemen had questions and concerns regarding methods of diagnosing and eliminating the disease. I conducted rulemaking in 2008 to eliminate infected bulls from entering the state by requiring a Trich test. I conducted additional rulemaking in 2010 to address the disease when it was diagnosed in herds within the state. At the meeting I received comments that additional rules and regulations should be put into place requiring all bulls in an area, where there is identified infection, be tested yearly. I do not believe in writing rules that cannot be enforced and WSDA does not have the resources to enforce testing of all bulls in an infected area. I know there are animal health and livestock inspection rules that are violated daily; so is the speed limit. There are consequences when caught speeding or breaking animal health and livestock inspection laws. I would echo County Commissioner Jim Detro's comments when he said; First there is no money to enforce such requirements that all bulls in an area be tested either at the state or county level and Secondly it is better for the local cattlemen to get together and govern themselves. I will listen to the industry and conduct additional rule making if there is popular support, however; the local authorities and cattle industry will need to monitor and assist in enforcement.

Tritrichomonas foetus is a sexually transmitted pathogen (protozoan) associated with reduced fertility and abortions in cows. Trich is one of the most economically devastating diseases that a cow-calf herd can get. It is caused by a protozoan and is a long-standing endemic disease in the western states and the rate of diagnosis has increased in recent years. It is an in-apparent infection in bulls and once infected the bull becomes a life-time "carrier". Trich causes "temporary infertility" and abortion in cows and heifers; however, unlike the bull it is a "self-limiting" disease and will clear infection in 2-6 months in most cows. It is estimated that 0.05-0.1% (1 out of 1,000 – 2,000) females may continue to be a "carrier" and the infection is more commonly a problem among beef cattle herds.

We expect a trend of increased diagnosis to continue due to recognition of T. foetus as a major problem in beef cattle in many states, thus leading to a screening requirement before moving bulls into neighboring state pastures on pasture permits or for import or export in Washington. It is important to increase awareness of the PCR test among clients and veterinarians. I want to remind them that during cold weather, it is important to collect the sample, inoculate the medium, such as In-Pouch TF (Trich-Pouch®), and send the sample as soon as possible after collection to the laboratory, paying special attention to prevent freezing or over heating of the sample during shipment. Freezing or excessive heat will destroy the agent, increasing the difficulty for detection by the test, especially when present in low numbers. Note: Washington requirements are the sample needs to reach the laboratory within 48 hours.

Signs of infection in a herd are:

- Repeat breeding (due to temporary infertility)
- Extended calving season (late calves)

- Early abortion (too early to find fetus) (>50%)
- Occasional late-term abortions (<5%)
- High % of open cows at pregnancy check (30-50%)
- Pyometra (pus-filled uterus) in 1-5% of cows
- Reduction in number of calves born by 30-50%
- Less uniform calf crops strung out over 3-8 months

The economic impact of increased veterinary expenses and the costs of culling and obtaining subsequent replacements, the lowered calving rate, and the cost of feeding an open cow for the winter, only to find out she is not going to have a calf, has caused many cattlemen to go out of business. Nationally, it is estimated that the loss approaches $650 million annually and a reduction of income in a herd with Trich by 22-37%.

Trich can be controlled by:

- Testing ***all*** breeding age bulls before the breeding season
- Using young virgin bulls – consider culling bulls greater than 4 years of age (due to preputial crypt development).
- Pregnancy check cows/heifers and open animals.
- Using separate breeding groups – heifers vs. cows. Early calving cows vs. late calving cows if late calving cows are kept.
- Maintaining a "closed herd" – All new bulls should be tested before entering a herd. Do not purchase culled adult cows or bulls. Do not rent or loan any breeding animals.
- Using Artificial Insemination, if practical – easier for dairies than beef cattle operations.
- Use vaccine in high risk situations where there is commingling of cattle herds.
- Comply with Trich Regulatory Programs.
- Good fences make good neighbors!!

Test your Bulls **--** I am routinely asked why test the rest of the bulls a second time if you find a positive bull on the first test and the remainder tested negative the first test. The answer is the percentages of identifying an infected bull increase with addition testing; first test -80%, second test - 96% and third test - 99%.

Washington State Requirements **–** Test all bulls over 1 year of age when entering the state.

- There are no requirements on in-state movement except a haul slip for ownership, and a livestock inspection at change of ownership.
- Bulls in the slaughter pen at a sales yard must test before selling back into production.
- If there is a positive herd test for Trich on an instate herd:
    - The herd will be quarantined pending an epidemiological investigation.
    - Positive bulls are S-branded and sold for slaughter.
    - Quarantine is removed – after a second negative qPCR test of all the remaining bulls after the positive bulls are removed.
    - Open cows from an infected herd must be identified and sold to slaughter only.
    - Information that cattle have tested positive for the disease may be supplied to county extension agents, accredited veterinarians, and industry representatives. The department may also publish information on the counties that have infected herds.
    - Samples must arrive at the laboratory within 48 hours.

Trich has been diagnosed in three Okanogan County ranches in the last three months. Accredited veterinarians are required to be certified to conduct trichomoniasis testing. Certification is accomplished by completing training and passing a proficiency examination provided by the department. Take the course at: http://vetextension.wsu.edu/programs/bovine/trich/index.htm

Continuing Education

Veterinarians

Academy of Dairy Veterinary Consultants Spring Meeting
Dairy FARM Welfare Certification Course. April 15-16, 2011, Half Moon Bay, CA. Go to:
http://www.vetmed.wsu.edu/orgADVC/upcoming.asp

Veterinarian Online CE for Official Trich Testing
To take the course and receive certification - go to:
http://vetextension.wsu.edu/programs/bovine/trich/index.htm

Veterinarian Online CE for TB Testing Certification
To take the course and receive certification - go to:
http://vetextension.wsu.edu/programs/bovine/tb/index.htm

Assessing Calf Housing and Environments: Part I and II for veterinarians at
http://vetextension.wsu.edu/courses/index.htm

Producers

DairyBeef: Maximizing Quality and Profits at http://dairybeef.wsu.edu

Assessing Dairy Calf Housing and Environments for producers and dairy employees at:
http://www.vetextension.wsu.edu/EnviLoad.htm

4-H Leaders

NEW "Providing clean and comfortable environments to optimize livestock health and wellbeing. *Volunteer Leaders* Online Program http://vetextension.wsu.edu/programs/4-H/index.htm

Send newsletter comments to the Editor:
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