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YOU CAN VIEW OLDER ISSUES OF *ag animal health*:
<http://vetextension.wsu.edu/newsletter/index.htm>

From the Editor – Here I go again. Only this time it's not residues. I am still harping on drug use, but this time it is on the development of resistance by bacteria when we use antibiotics. So, how about the FDA's consideration (*not rule, please note*) that we should only give antibiotics to animals based on prescription? In short, that means NO Over-the-counter (OTC) drugs. We already need approval by or a prescription from a veterinarian for many of the antibiotics we use on farm. So consider the possibility of no OTC drugs and think about what it might mean to you as a producer or veterinarian. What are all the consequences of maintaining the way we currently use antibiotics? What are the consequences of going to prescription-only? Does it make a difference? This would be a great lunch-time conversation to have with your colleagues.

Featured Faculty – Dr. Ahmed Tibary, College of Veterinary Medicine

Congratulations, Dr. Tibary! --- For being named the "Theriogenologist of the Year" by your colleagues at the 2011 Annual Society for Theriogenology and American College of Theriogenology conference. For those of you who do not know (or have not yet "Googled" it), theriogenology is the study of animal reproduction and covers a wide range of activities - from basic research to herd- or flock-level issues in reproduction. And - Dr. Tibary has been engaged in quite the gamut of theriogenology activities. From research, to resident and graduate education, to teaching and clinical service and paying attention to all species, and, in particular, his work with camelids. Thanks for all you do!



Dr. Ahmed Tibary



WSU Highlights at the AABP

The 2011 Annual Conference of the American Association of Bovine Practitioners in St. Louis, MO, was a watershed event for many WSU College of Veterinary Medicine folks. First, we had great participation from our students, many of whom submitted abstracts. One of our 4th-year students, Chelsey Johnson ('12) **won** the AABP student case presentation competition with her presentation on *Effects of Varying Bunk Space on Feedlot Performance and Behavior of Yearling Steers*. Jennifer Wilson ('12) presented her work: *Comparison of Three Antimicrobials Used for Treatment of Undifferentiated Disease in Dairy Replacement Heifers 15 to 60 Days of Age on a Commercial Calf Ranch*. Craig Louder ('12) presented a talk in the Research Summaries section on: *Evaluation of Response and Safety to Parenteral Trace Mineral Supplementation in Idaho Neonatal Dairy Hutch Calves*. Alea Hoffman ('14) presented a poster for Research Summaries titled: *Preliminary Evaluation of Two Methods for Estimating Lameness Prevalence on Western U.S. Dairies*. Katrina Hartman ('13) and Jen Wilson ('12) served as student delegates. Tim Gibbs ('13) and Ben Werkhoven ('12) were scholarship recipients. Supporting our group was Kat Chew ('12), Tracy Quirk ('14) and Caitlin Himmel ('13). Drs. Bill Sischo (VCS) and Tom Besser (VMP) were presenters in the General Sessions and Dr. John Wenz was a moderator, Dr. Schneider was the student advisor and Dr. Moore served as AABP General Sessions Program Chair. Dr. Giebel and Dr. Wenz manned the WSU Vet Med Extension booth along with many of the students.

Chelsey Johnson



Does early palpation result in pregnancy loss in dairy cows?

Romano et al. 2011. *JAVMA* 239(5):688. Looking at almost 1000 dairy cows in one study, researchers tried to determine the effect of palpation per rectum by use of 1 or 2 fetal membrane slips (FMSs) for pregnancy diagnosis during early gestation on pregnancy loss in dairy cattle. All cattle were determined to be pregnant by use of ultrasound at about day 31 after heat and randomly allocated into 2 groups (control group [n = 476 cows] and palpation group [452]). The control group was not subjected to pregnancy diagnosis via rectal palpation. The palpation group was subdivided into 2 groups, one palpated with 1 membrane slip and one palpated with 2 performed by 1 veterinarian between days 34 and 43 after heat. All cattle were reevaluated by ultrasound on days 45 and 60 to determine viability of the embryo and fetus. Overall pregnancy loss between 31 and 60 days was 14.1%. Pregnancy loss for the control, 1 slip, and 2 slip groups from days 31 to 60 was 14.5%, 12.6%, and 14.9%, respectively. Embryonic loss for the control, 1, and 2 slip groups was 12.4%, 9.1%, and 9.5%, respectively. Fetal pregnancy loss for the same groups was 2.4%, 3.8%, and 5.9%, respectively. **Conclusions – Pregnancy diagnosis by 1 or 2 fetal membrane slips performed during palpation in early gestation did not increase pregnancy loss in dairy cattle.**



Issues of Thin Sheep

By: Dr. Steven M. Parish

Key Points

- *Thin sheep can be caused by many things*
- *Bacterial causes, parasitism, nutritional deficiencies could all be responsible*
- *Producers, working with their veterinarian, can assess all the possible risk factors and develop prevention plans*



A common complaint from sheep producers concerns ewes that are thin and failing to thrive. This often necessitates early culling from the flock. Typically, such ewes are often in the prime of their life and come from any sized flock. The condition can occur in a single ewe or this may be a flock problem with numerous ewes affected on an annual basis. Weight loss can be so debilitating in some ewes that they may either die or need to be destroyed. Sometimes the thin ewe comes to light at shearing time as a surprise to some owners or may present as a ewe in late pregnancy that is found down with no other metabolic disease. The problem occurs across all breeds and not only affects ewes but also rams. Historically, the problem has been called the “Thin Ewe Syndrome” or the “Ill Thrift Syndrome”. The most important goal for producers is to define and eliminate the reason for the failure of these animals to prosper. A single animal noted to have weight loss and ill thrift may represent the potential cases likely to develop making it even more important to define the underlying problems.

Historically the term “Thin Ewe Syndrome” can be at least in part attributed to Dr. Norm Gates and others from the U.S. Sheep Experiment Station at Dubois, Idaho. In 1977 that group published a scientific paper in Journal of the American Veterinary Medical Association where they defined the common reasons for thin ewes in a major western range flock of sheep. The paper also detailed the underlying issues on reproductive efficiency. They described three common causes of weight loss in mature ewes of white face breeds. Based on necropsy findings they noted that Ovine Progressive Pneumonia (OPP), Caseous Lymphadenitis (CL) and other internal abscesses caused by *Arcanobacterium pyogenes* were the three most common underlying reasons for ill thrift in the ewes. Regardless of management programs, these three causes are still recognized as major causes of weight loss in populations of sheep under all modes of management. Many industry and university groups have addressed the importance of these diseases however they still represent major causes of economic losses in sheep production. In an attempt to control these diseases, producers routinely test for OPP and CL. They then base management decisions on the results of the testing.

It is important to realize though that there are far more individual and flock causes of ewes and rams who fail to prosper. For instance, individual animals may be affected by chronic infections such as mastitis or uterine infection negatively affecting the animal. The overall nutritional program of the flock is another one of the most important parameters affecting the productivity of all sheep. If protein and energy in stored feeds or even pastures are not adequate, then animals will fail to prosper. Without normal dentition, sheep will not be able to eat and utilize nutrients that are available. Harsh grazing circumstances are a common cause of premature teeth wear in foraging sheep.

Micronutrient deficiencies are also part of a potential nutritional basis of ill thrift and may include selenium, iodine, cobalt and even copper deficiency. Some producers attempt to avoid excessive copper and possible toxicity but actually create a deficiency instead. Internal parasites affecting the abomasum and intestine and even the liver should be considered. Other chronic disease processes can lead to individual and sometimes flock issues such as Johne's disease which is characterized by intestinal changes that lead to malabsorption of ingested nutrients. Certain breeds of sheep have unique and possibly heritable issues that lead to debility during the prime of life such as the abomasal emptying defect shown in Suffolk sheep. Intoxicants from the sheep's environment, particularly poisonous plants, could account for liver or kidney issues at an individual or flock level.

The "Thin Ewe Syndrome" is both a specific and non-specific spectrum of disease processes that can lead to either individual or flock level debilitation." The producer, working with a veterinarian, can use the breadth of physical and diagnostic tools to assess the underlying cause or causes of less than desirable performance and design a program to lessen the affects of this myriad of disease processes.

More on Beef Heifer Reproductive Technologies From WSU

Artificial insemination at 56 hours after intravaginal progesterone device removal improved AI pregnancy rate in beef heifers synchronized with five-day CO-Synch+CIDR protocol

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The objective of this study was to determine whether timed artificial insemination (TAI) at 56 h after CIDR removal, in 5-day progesterone-based CO-Synch protocol with two doses of prostaglandin F2a (PGF) at progesterone withdrawal, would yield improved AI pregnancy rate in beef heifers compared to TAI at 72 h after CIDR removal. Angus cross beef heifers (N = 1098) at 9 locations [WA (5 locations; N=634), ID (2 locations; N=211), VA (1 location; N=193) and WY (1 location; N=60)] were included in this study. All heifers were given a body condition score (BCS), and received 100 µg of gonadorelin hydrochloride (GnRH; Factrel®, Pfizer Animal Health, New York, NY) and a controlled internal drug release (CIDR; Eazi-Breed™ CIDR® Cattle Insert; Pfizer Animal Health, New York, NY) insert on Day 0. The CIDR insert was removed and two 25 mg doses of PGF (Dinoprost; 5mL Lutalyse® sterile solution; Pfizer Animal Health, New York, NY) were given, first dose at CIDR removal and second dose at 6 h later, on Day 5. Within farm, heifers were randomly allocated to 2 groups and were inseminated at 56 h (N=554) on Day 7 starting at 4 PM or at 72 h (N=544) on Day 8 starting at 8:00 AM. Heifers were given 100 µg of GnRH concurrently at the time of AI. The data were analyzed using PROC MIXED procedure of SAS (SAS Version 9.1, Cary, NC). The variables included in the model were treatments (56 h vs. 72 h), locations (1 to 9), BCS (≤ 6 vs. > 6), and treatment × location and treatment × BCS interactions. AI sires and AI technicians were included as random variables in the model. Insemination at 56 h after CIDR removal significantly improved AI pregnancy rate compared to insemination at 72 h (66.2% vs. 55.9%; P<0.001; 1-β=0.94). The locations, BCS and location × treatment and BCS × treatment interactions did not influence AI pregnancy rate (P>0.1). The AI pregnancy rate for heifers with BCS ≤ 6 vs. > 6 was 61.8% vs. 60.1%, respectively (P>0.1). The AI pregnancy rate among locations varied from 56.0% to 69.2% (Figure 1; P>0.1). In conclusion, **heifers inseminated at 56 h after CIDR removal in a 5-day CIDR-CO-Synch protocol yielded an average of 10.3% higher AI pregnancy rate compared to heifers inseminated at 72 h after CIDR removal.**

Clinical disease in dairy cows during transition

Chapina, et al. 2011. The association of serum metabolites with clinical disease during the transition period. *Journal of Dairy Science*. 94(10):4897-4903.

The objective of this observational field study was to validate the relationship of serum concentrations of nonesterified fatty acids (NEFA), β -hydroxybutyrate (BHBA), and calcium with disease in early lactation across different management systems. Fifty-five Holstein freestall dairy herds located across the United States and Canada were selected and visited weekly for blood sample collection from 2,365 cows. Only diseases that were consistently recorded across herds and blood samples collected before the disease occurred were considered. Metabolite concentrations in serum in wk -1 relative to calving were considered as predictors of retained placenta (RP) and metritis, and metabolite concentrations in serum in wk -1 and wk +1 relative to calving were considered as predictors of displaced abomasum (DA). For each disease, each metabolite, and week of sampling in the case of DA, a critical threshold was calculated based on the highest combined sensitivity and specificity and used to categorize the serum concentrations into high and low risk categories. Multivariable logistic regression models were built for each disease of interest and week of sampling, considering cow as the experimental unit and herd as a random effect. Cows with precalving serum NEFA concentrations ≥ 0.3 mEq/L were more likely to develop RP [odds ratio (OR) = 1.8; 95% confidence interval (CI) = 1.3 to 2.6] and metritis (OR = 1.8; 95% CI = 1.5 to 2.9) after calving than cows with lower NEFA concentrations. Precalving NEFA ≥ 0.5 mEq/L (OR = 2.4; 95% CI = 1.5 to 3.7), postcalving NEFA ≥ 1.0 mEq/L (OR = 2.7; 95% CI = 1.7 to 4.4), and postcalving calcium ≤ 2.2 mmol/L (OR = 3.1; 95% CI = 1.9 to 5.0) were associated with subsequent risk of DA. In conclusion, elevated serum NEFA concentrations within 1 week before calving were associated with increased risk of RP, metritis, and DA after calving. Serum NEFA and calcium concentrations in the 2 wk around calving in combination were associated with the risk of DA.



What does this mean for dairy producers?

- High NEFA levels indicate fat tissue breakdown - cows are losing weight too fast.
- Look at the body condition score of your dry cows and intakes of your close-up cows.
- Fat cows have a tendency to lose weight faster in transition.
- Rapid fat tissue breakdown may lead to a number of fresh cow problems.

WSDA Corner by Dr. Leonard Eldridge, State Veterinarian

WSDA has a new Foreign Animal Disease Diagnostician

Dr. Dana Dobbs (WSDA Field Veterinarian/ Ellensburg) completed the Foreign Animal Disease Diagnostician (FADD) training in June. This training is a two-week long intensive training on Plum Island, New York (the site of the Foreign Animal Disease Diagnostic lab for USDA and Homeland

Security). FADD's are trained to recognize symptoms of foreign animal diseases and are able to perform the necessary testing to rule out these economically devastating conditions. WSDA now has seven FADD's strategically located around the state. This addition will benefit the State as Dr. Dobbs is responsible for region three; Okanogan, Chelan, Douglas, Kittitas, Grant, Yakima and Klickitat counties.

Animal Disease Traceability

On April 29, 2011, the Governor signed SHB 1538, an Act relating to animal health inspections, which amended RCW 16.36 and 16.57. The Act directs the department to, adopt by rule, designations of when animal health documents, livestock inspection certificates, and certificates of permit or other transportation documents require a physical address for the animal's destination. The Animal Health and Livestock Inspection programs are proposing rulemaking to align their rules with the adopted legislation. Proposed amendments designate when documents must identify a physical address of a destination, when animals must be delivered or transported to the physical address stated, and establishes penalties for physical address and diversion violations.

SHB 1538 also authorizes the department to adopt by rule a per head fee on cattle to administer animal disease traceability activities for cattle. In response, the Animal Identification program is proposing rulemaking to adopt a new chapter; Chapter 16-29 WAC Animal Disease Traceability. The purpose of this chapter is to allow the department to assess a forty cent per head fee on all cattle sold or slaughtered in the state or transported out of state except for;

- a) Cattle moving interstate for grazing purposes and return per WAC 16-86-017; and
- b) Female dairy cattle moving interstate for feeding purposes and return.

The question becomes whether Washington State needs animal disease traceability and what is the cost of having it and not having it? To answer this question, I would pose the issues around just one of the multitude of animal diseases I worry about. If one animal is infected with Foot and Mouth Disease (FMD) on a ranch, the virus is so contagious that all cloven hoofed animals on that ranch must be depopulated because they all will become infected with the disease. The philosophy that, I got my herd and leave me alone because it won't affect me and I am safe, is not reality in today's world. On this year's anniversary of 9-11, our U.S. officials were concerned about terrorists putting bombs in cars and planning to blow them up in a crowded area. To bomb, an individual must have the ability to obtain the explosives and have the savvy to cause them to explode effectively. To spread FMD, one does not need personal protective equipment or special weaponization of the agent, or the savvy to make something work. All that is needed is to have a vial of FMD in ones shaving kit, transport it into the United States in a less than 3 ounce container, dump it in a feed bunk, and because of the virulent nature of the FMD virus, it will spread naturally. This tells us that it is not **if** FMD will happen in the United States, it is **when** it will happen. The cost of not having traceability that facilitates early identification and quick containment will be billions to our cattle industry and affect us all. We are all in this together.

WSDA has written proposed rules and now needs your participation to develop or reject a preventive response system for Washington. The most important part of that system is being able to quickly determine where our animals came from, where they went, and which ones are exposed; we call it animal disease traceability.

Now let's talk about the cost of an animal disease traceability system in Washington. As long as a cow remains in the state the cost will be 40 cents, one time, during the time you own that cow. When cattle leave the state on a grazing permit or a dairy heifer feeding certificate of veterinary inspection, and then returns to the state, the 40 cents will be refunded, so one still only pays 40 cents one time while owning the cow. We spend many times that much over the life time of the cow on animal health products and preventative medicine keeping cattle healthy so they are profitable. If we are unable to perform the traceability necessary to do early identification and

quick containment our state and country will become an economic disaster just as we have watched FMD in other countries become an economic disaster.

The animal disease traceability system will aid in all disease outbreaks. Just look at what BSE did to the cattle industry and all because Washington could not find cattle that entered the state from Canada that ate the same feed in Canada. Tuberculosis and Brucellosis was costly in Washington in years past and is still costly today to states that are dealing with these diseases. There will never be a 100% guarantee; however, for less than the price of one cup of coffee, one time in the ownership of a cow (if the cow does not leave the state) we can purchase insurance that will provide additional protection by facilitating animal disease traceability in Washington.

Brucellosis

I received information that the USDA is reducing the brucellosis slaughter surveillance in Washington to just one slaughter plant. This eliminates all financial support for our regulatory laboratory at the Washington Animal Disease Diagnostic Laboratory and they are recommending we stop vaccination for brucellosis. I believe this increases the risk to Washington when we know we receive a significant number of breeding cattle from the Greater Yellowstone Area states (GYA) where brucellosis exists in wildlife. Another herd of cattle was identified as infected this last week in Montana. We must continue brucellosis vaccination of our female cattle and continue slaughter surveillance at all plants if we are to continue receiving cattle from the GYA states. I will be listening to the cattle industry at fall meetings on suggested options on how we can minimize our risk of another resurgence of Brucellosis such as happened in the nineteen seventies and eighties.

What's New at WADDL? -- Q Fever Testing at WADDL

A recent outbreak of Q Fever in goats has piqued interest in Q-Fever testing of flocks. This article describes the disease and the kinds of testing done for it.

What is Q- Fever? Query or Queensland Fever (Q Fever) is a bacterial infection affecting a variety of animal species as well as human beings. Q Fever is caused by *Coxiella burnetii*, an obligate, intracellular, rickettsial organism that can survive in a dried condition for extended periods.

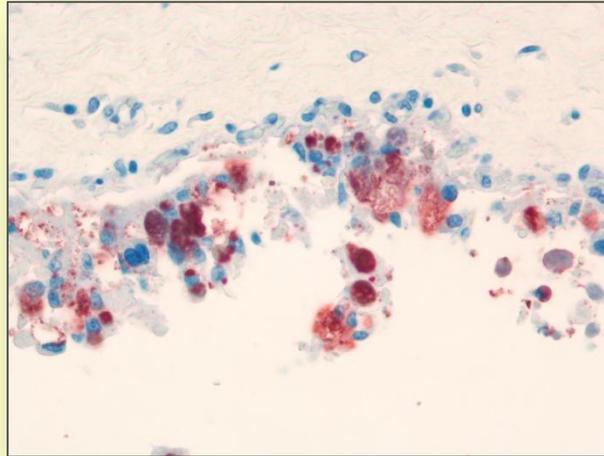
Is Q Fever widespread? *Coxiella burnetii* infection of sheep and goats is nearly worldwide in geographical distribution and is thought to be endemic in most continents. *C. burnetii* cycles in a wide variety of wildlife species and their ectoparasites. The true incidence of Q Fever infection is unknown in the Pacific Northwest region of the USA. WSU-WADDL is currently conducting a research study to determine the geographic distribution and herd prevalence of *C. burnetii* infection in goats in Washington state.

What are the clinical signs of Q Fever? In livestock the disease is usually subclinical. However, occasional abortion outbreaks caused by *C. burnetii* have been reported in goats and, less commonly, in sheep. Susceptible pregnant females develop necrotizing placentitis (inflammation and necrosis of the placenta), which results in abortion, whereas non-pregnant females do not develop clinical signs. Some ewes and does abort without apparent clinical signs, whereas others show loss of appetite and depression 1 to 2 days before aborting. After the initial abortions or infections, animals become immune to abortion but can remain subclinically infected. After the infection is established, the female can carry the organism indefinitely, sporadically shedding it in milk and at lambing or kidding.

How is Q Fever abortion diagnosed in the laboratory? Diagnosis of Q Fever abortion requires laboratory testing of aborted fetuses and placenta from aborting does or ewes. Diagnosis is based

on identification of lesions in the placenta (gross and microscopic pathology) together with identification of the organism by non-culture methods. Culturing *C. burnetii* in the laboratory is not feasible because of the particularly contagious potential of the organism in laboratory cultures to laboratory technicians. Therefore, diagnosis of Q Fever abortion at WSU-WADDL is based on special non-culture methods such as immunohistochemistry, which uses color-linked *C. burnetii*-specific antibody probes to visually identify (under the microscope) *C. burnetii* within the infected placenta (Figure 1) or molecular diagnostic PCR methods that uses amplification of *C. burnetii* DNA directly from infected placenta.

Figure 1. *Coxiella burnetii* (Q-Fever) organisms (red) within infected placenta cells (blue).



How is Q Fever transmitted? Cattle, sheep, goats, and wildlife may carry the organism, which may be shed in placentas, uterine fluids, colostrum, and milk, especially from aborting animals. Inhaling contaminated dust can infect animals and human beings. Contact with aborted material, vaginal discharge, and mucous membranes of infected animals are other modes of contamination. Grazing contaminated pastures and tick bites present another source of infection.

What is the potential for Q Fever transmission to humans? Q Fever can be transmitted to human beings by ingesting milk from infected animals and having contact with placentas or feces. Disease in human beings is characterized by influenza-like symptoms. The majority of human cases have a history of contact with infected sheep or goats. The organism is killed by pasteurization but can be readily transmitted in non-pasteurized milk. All persons should wear masks and gloves when removing manure from the barn, assisting with lambing and kidding, and handling aborted fetuses.

Is there a diagnostic test for Q Fever infection in an animal with subclinical infection (no clinical abortion)? There are a number of serology tests for Q Fever that identify a host immune response to *C. burnetii* infection indicative of a previous infection with *C. burnetii*. However, serology tests do not indicate whether or not an infected ewe or doe may be shedding organisms. WSU-WADDL uses both a complement fixation and ELISA for Q Fever serology. According to the World Organization for Animal Health complement fixation antibody titers of more than 1:20 indicate exposure to *C. burnetii*, and various commercially available ELISA tests have established test cutoffs for positive, negative, and suspect animals.

Highly sensitive PCR tests (that amplify organism DNA) are currently in use at WSU-WADDL and can be used to diagnose *C. burnetii* shedding in body fluids on subclinically infected animals. However, since shedding is sporadic (does not occur at all times) a negative PCR tests cannot rule out *C. burnetii* infection.

For information on “Best Practices to Control Q Fever visit the Washington State Department of Agriculture website:

<http://agr.wa.gov/FoodAnimal/AnimalHealth/Diseases/QFeverManagementPractices.pdf>

For information on human illness, go to: <http://www.cdc.gov/qFever/index.html>

By: Tim Baszler, DVM, PhD, Professor and Director, Washington Animal Disease Diagnostic Laboratory

Continuing Education

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