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**From the Editor** - I texted a picture to a friend in Scottsdale, AZ, the other day of it snowing yet again on the Palouse with the caption “When will it ever end?” Well, we should be seeing Spring because the first official day of Spring is March 20<sup>th</sup>. With spring comes melting snow, rain and mud. Dr. McConnel provided us with some information on mud and livestock, and Dr. Allen gives us a peek at footrot. And, with Spring comes thoughts of Summer and heat stress. Dr. Adams-Progar and I have given you some thoughts on considering shade for cattle so you can be ready for Summer. Starting us out, is a summary of the weak calf syndrome cases the Field Disease Investigation Unit has been looking into and a request for additional cases from the field to help with the investigation.

*The ag animal health newsletter is devoted to the transfer of current, relevant information to food animal owners and veterinarians in the Pacific Northwest.*

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## Weak Calf Syndrome Investigation

By Drs. John Wenz and Craig McConnel, Field Disease Investigation

Weak Calf Syndrome (WCS) was first described in Montana in 1964 and since then observed across the US and in other countries. The summary provided in *Veterinary Medicine*, a text by Constable et al, nicely captures what has been commonly observed in the Northwest.

**Etiology:** Uncertain; probably multiple etiologies and multifactorial.

**Epidemiology:** Most commonly several cases on a farm; several farms affected in a geographic region in a single season; problem may not occur for several years and then occur as “epidemic” in a region.

**Clinical findings:** Calves may be born weak and unable to stand. More commonly, they are born apparently normal and stand but subsequently collapse with hypothermia and die within hours of birth.

In what was described as the “most common” situation, calves are born weak and die within 10-20 minutes after birth but “sometimes” live for up to a few days. The “earlier

descriptions” recounted in the textbook indicated about 6-15% of calves were affected by 10 days of age. About 20% of calves were affected at birth and calves that survived for a few days exhibited depression, weakness and a “hunched up back” if they stood. Mortality was 60-80% despite therapy.

This winter we worked with a beef herd in Idaho that reported 30% of calves affected. Most were first noticed “lacking vigor” at 3-5 days of age and presented as “dumpy, humped up and shivering” at 7-18 days of age. About 20% died, 20% remained with a “very rough appearance, poor condition and generally looking unhappy”. This outbreak was concurrent with a tough winter with more snow than usual and temperatures 10 degrees colder than normal. Some cases of WCS have been associated with “cold, hard winter weather”. However, a veterinarian in Oregon with several herds affected over the years reported no association with weather during the calving season. The difference could have something to do with management response to adverse weather. In the Idaho case, because of the snow and cold temperatures, the cows were moved to a smaller pasture at calving time and on some nights animals were locked in a straw-bedded corral for easy observation. Along with WCS-like cases they also experienced what was described as about 5 times more calving problems than usual (head back, foot back, placenta first). There have been anecdotal descriptions of more dystocias during bad weather attributed to reduced exercise of the cows just prior to parturition perhaps leading to more malposition/malpresentations.

Delayed calving associated with dystocia is one of the most common causes of stillbirth in cattle and the story described above could fit that possible cause with the resulting trauma associated with excessive force during assistance and fetal hypoxia (low oxygen levels). Of the proposed causes (see sidebar) delayed parturition with dystocia is likely a primary factor associated with WCS along with the others such as weather and management practices that could affect the clinical presentation ranging from stillbirths to “dumpy, humped up and shivering” calves at 2 weeks of age.

A text-book description of “dummy calves” exists that appears different from WCS but could present similarly. Similar to “dummy foals”, affected calves appear alert at birth, but if observed closely don’t rise for 1-2 hours and seem to lack the drive to suckle. Depending on how closely calving is observed it is possible these dummy calves fail to suckle and present like a WCS calf due to hypothermia and hypoglycemia. These calves would likely have failure of passive transfer of immunity from colostrum and if nursed through the initial problems would be at higher risk for infectious diseases. Dr. John

## Possible Causes of Weak Calf Syndrome

- Perinatal fetal infections (leptospirosis, BVD, adenovirus)
- Maternal nutritional deficiencies
  - Iodine deficiency resulting in hypothyroidism exacerbated by low selenium
  - Gross nutritional inadequacy (crude protein 7% during last trimester led to lower birth weights and thermogenic ability)
- Placental insufficiency resulting in intrauterine growth retardation (can lead to anemia and thymic hypoplasia and may be associated with low Estrone sulfate concentrations during late gestation).
- Trauma associated with excessive force during assisted calvings
- Fetal hypoxia associated with delayed parturition/dystocia

Madigan at UC Davis studies this problem in foals and has been investigating the potential role of neurosteroids that have sedative effects. His theory is that foals delivered too quickly don't experience adequate time in the birth canal to signal the normal downregulation of production of these sedative neurosteroids. Essentially, the foal is born sedated ("..good in the womb so they don't gallop.." as he is quoted saying) but could lead to the apparent lack of interest in suckling.

This peaked our interest in the possible role of neurosteroids in development of WCS. It turns out allopregnanalone is such a steroid, derived from progesterone and has been shown to be protective against hypoxic/ischemic brain injury during parturition in sheep and guinea pigs. Reduced levels of this neurosteroid have been associated with decreased myelination, white matter injury and lasting neurologic deficits. Interestingly, acute, short-term stressors (such as a single corticosteroid injection) *increased* allopregnanalone levels while chronic stressors (repeated corticosteroid injections) *decreased* concentrations. Is it possible that relatively longer stressors (weather, poor nutrition) reduce production of these neuroprotective steroids and also predispose cattle to delayed parturition? The result would be a calf less resistant to and more likely to experience hypoxemia during and shortly after birth.

These are all very interesting ideas, but in the end, how do we reduce losses associated with WCS? To date there have been many *general* suggestions targeting some of the potential risk factors: adequate nutrition (protein, energy and trace minerals), close observation and appropriate assistance at calving, vaccination, and provision of wind breaks and warming boxes during cold, harsh weather. Despite knowledge of these, WCS persists as a problem. Appropriate management of cattle is important but may not be the root cause. Maybe it is a seemingly appropriate response like bringing the cows up close into a smaller calving area so they can be better observed that inadvertently increases the risk due to lack of exercise thus more dystocia. In "bad" weather perhaps less WCS is seen in herds that leave the cows out. Again, lots of conjecture at this point, so how do we figure this out? Basic "gumshoe epidemiology" as Dr. Moore would say.

We need your help documenting as much as we can about your cow herds. Based on what we think are possible etiologies we have an idea of what information we need to gather. Because this is a sporadic problem we need to gather these data every year for multiple years so we can compare the good years to the bad within a herd and across herds. Although WCS is most commonly thought of as a problem of beef herds, it has been described in Irish dairy herds and very likely occurs on our dairies too. So we need dairy and beef herds. We also need to do thorough necropsies on all suspected WCS calves and get a complete set of diagnostic samples submitted. We have some funding that we may be able to use to subsidize or pay for diagnostic samples and may be able to get out in the field and do some data collection but will need to rely on your help to solve this problem.

### **What you can do**

If you are willing to help us with data and sample collection please contact John Wenz ([jrwenz@vetmed.wsu.edu](mailto:jrwenz@vetmed.wsu.edu), (509)335-0773) so we can add you to the WCS Team Roster. Our Field Disease Investigation group would first like to better characterize this syndrome and identify possible risks for this calving season. *If you have had any of these cases in your herd or your clients' herds, would you please complete the short, anonymous, online survey:* [https://wsu.co1.qualtrics.com/SE/?SID=SV\\_cUC2ymSXPUp2Z](https://wsu.co1.qualtrics.com/SE/?SID=SV_cUC2ymSXPUp2Z)

We will be using this information only to provide summary feedback to ranchers and veterinarians and to set the stage for more in-depth investigations. Let us know if you have herds (beef or dairy) that have experienced WCS. Share your WCS experiences and your ideas about potential causes and what you think we should do during the study to better understand the problem. Work with us to gather the needed data and samples to better understand WCS.

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## Managing Mud

by Dr. Craig S. McConnel, Extension Veterinarian

As Spring nears after a long and harsh Winter, it's time to consider what comes next. Mud. Everyone knows that mud is a nuisance and does what they can to limit its interference in day-to-day operations. But sometimes we forget just how detrimental mud can be to livestock and what it takes to limit its effects. There are numerous resources for gathering detailed information on mud control and management. This article will focus on some of the more pressing issues that rise to the top of the pile.

**Impacts on health.** Mud leads to added stress for livestock. Calves that are born in a muddy environment are more likely to struggle to get up and nurse, less likely to ingest appropriate levels of colostrum, and vulnerable to navel infections. Baby calves may also suffer an increase in gastrointestinal infections due to scour-causing and toxin-forming pathogens that are more easily transmitted and ingested in wet, muddy conditions. Add those to an already chilled calf with a muddy, matted hair coat and you have the recipe for an acute, often fatal intestinal infection.

Mature cows are less likely to spend time laying down in muddy areas, more likely to slug feed with consequent acidosis and laminitis, and are prone to increases in displaced abomasum, lameness, and mastitis. Bacteria lurking in the mud may cause coliform mastitis, digital dermatitis or foot rot. Wet conditions can also lead to softer hooves and increased levels of sole separation, bruising, and abscesses. Muddy conditions in the maternity pen can lead to contamination of the reproductive tract as well, with subsequent increases in the number of cows with metritis and follow-on reproductive issues.



**Impacts on animal productivity.** Mud increases energy demands on livestock by increasing exertion when moving from point A to B, and by decreasing insulating capabilities when it is caked on the hair coat. Housing cattle in mud can seriously diminish gains in feedlot cattle, particularly when the temperature remains below 40°F. A study out of the University of Nebraska's Institute of Agriculture demonstrated that under such conditions the potential loss of gain ranges from 7% in dewclaw deep mud, to 35% if the mud is belly deep. Even hock deep mud can reduce gains as much as 28%, and that can be all too easy to achieve in younger cattle. Similarly, dairy studies estimate that every inch of mud reduces dry matter intake by 2.5%, and lowers feed efficiency with predicted decreases in milk production. Add that to the impact of mud-related disease on production, reproduction, and overall well-being and it clearly makes economic sense to prevent and correct mud problems wherever possible.



**Management considerations.** Good dry-lot corral management should focus on water diversion, routine grooming, adequate slopes and drainage, and loafing mounds. The top layer of manure and soil can act as a sponge and must be routinely scraped without gouging and disrupting the slope. This is a good time for dairy managers to reassess freestall management as well. Particularly for those dairies that use compost bedding there is evidence that humid and rainy weather can lead to increased bedding moisture and decreased hygiene.

Correcting mud problems can be challenging at best after the soil has become saturated. For that reason, do what you can prior to Spring to prepare yourself for the rain and mud. Aside from the important corral management techniques mentioned above, this may be a good time to stabilize soils with wood chips, gravel, road base, etc. even if you only focus on high-traffic lanes, feed allies, and watering troughs. The more you do now, the less impact and interference mud will have on your operation over the coming months.

#### **Additional Resources:**

1. Great Plains Mud Control and Management Page (University of Nebraska)  
<http://gpvec.unl.edu/mud/>
2. CDQAP Ruminant: Managing Mud on Dairies (California Dairy Research Foundation)  
<http://cdrf.org/2013/11/30/cdqap-ruminations-managing-mud-on-dairies/>
3. How Feeding-Site Mud and Temperature Affect Animal Performance (Kansas State University)  
<http://gpvec.unl.edu/mud/MudImpactOnFeedlotPerformanceKSU%20mf2673.pdf>



## Dairy: Want More Milk Fat?

by Dale A. Moore, Extension Veterinarian, and Alison King (CVM Class of 2018)

As part of her senior paper project, Alison King (CVM Class of 2018) has been looking at all the factors that contribute to a great dairy feeding system to keep cows healthy and productive and what critical control points dairy veterinarians and advisors should monitor on a regular basis. One problem area on some dairies is maintaining milk fat levels, either for individual cows or for the herd. Not only can this affect the farm's bottom line because of the components for which dairy farmers are paid, it can also be an indicator of poor rumen health.

It's a matter of simple chemistry - the rumen's chemistry. Rumen pH needs to be maintained at a certain level or there are consequences. Normal rumen pH is about 6.5 to 7.0, pretty neutral. The pH fluctuates some during the day after a cow eats and becomes lower as the rumen produces volatile fatty acids (VFAs). If the VFAs are not absorbed fast enough, the pH can stay low (more acid) and carbohydrate fermentation can shift to lactic acid (lactate) production. If the pH repeatedly stays below 5.6 for long durations, the cow is said to have SARA - subacute ruminal acidosis. The consequences of SARA are reported to be laminitis, liver abscesses, reduced dry matter intake (DMI), and milk fat depression.

What are some of the factors that can lead to SARA? Although we can't cover all the considerations in this article, two very recent papers in the *Journal of Dairy Science* highlighted some management practices that could help dairy producers manage out of SARA and maybe milk fat depression in their herds.

**Eating too much too fast** - Feed deprivation can cause cows to overeat when feed is finally provided. The cycle of lack of feed and then provision of feed can increase the risk of SARA. But, the risk for SARA is not evenly distributed among cows in a herd. According to Macmillan et al. (2017), some cows are at higher risk for SARA than others. In their investigation, they were able to discern which cows were more tolerant or susceptible based on an "acidosis index". Knowing that increasing feeding frequency can modulate rumen pH, they fed high and low risk cows either once a day or 3 times a day. They found

that milk yield overall was not affected but that feeding more often reduced the severity of SARA for the high risk cows and that these cows had a more even distribution of pH. More importantly, increasing the feeding frequency increased the milk fat yield for all cows by almost a third of a pound per day. Dry matter intake did not really change overall, just the provision of smaller amounts more frequently.

**Sorting their feed** - Quite a bit of research has evaluated the sizes of particles in the Total Mixed Ration (TMR) that cows are provided and their ability to sort out the goodies (think small grain size) from the healthier forage with longer fiber particles. The long forage particles help to stimulate longer eating times, times spent chewing, and saliva production. Saliva helps to buffer (raise the pH) of the feed entering the rumen. Eating mostly grain (concentrates) can potentially set a cow up for a low rumen pH.

Making sure the TMR has adequate particle size is one critical control point. A Penn State shaker box can be used for that purpose. But, even more important is making sure that the first cows that eat from the bunk don't take all the small particles, leaving the long stuff to other cows. Miller-Cushon and DeVries (2017) looked at feed push-up frequency and its potential effect on sorting. Although pushing up feed from 3 to 5 times a day did not affect sorting the feed in their tie-stall barn study, if the cows did sort, it affected their milk fat percentage. For every 10% increase in sorting out the long particles, milk fat percent decreased by 0.1 %. If a cow was producing 3.3 % fat in her milk, decreasing her ability to sort 20% of the long particles out of her ration would increase her fat percent to 3.5%. Other research has found that pushing up feed more frequently will increase DMI.

**What does all this mean?** First, feeding frequency appears to have effects on maintaining a healthier rumen and milk fat that maybe just frequent feed push-ups do not. These two strategies don't seem to be doing the same thing. However, reducing feed sorting by cows by whatever means can have an effect on milk fat. One place to monitor this is at the feed bunk by examining the particle sizes of the TMR as well as the particle sizes of the feed refusals to see if sorting is taking place. If there is feed sorting, investigating the causes such as the chop lengths of the forages and corn silage, the mixing time, the mixer wagon function and stocking density could help pinpoint the cause.

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# Cattle: Made in the Shade

by Dale Moore, Extension Veterinarian, WSU and  
Amber Adams-Progar, Extension Dairy Management Specialist, WSU

It's only March but we are going to discuss heat stress and shade. We'll discuss it now so that dairy farmers and cattlemen have a chance to consider and maybe implement a strategy in time if necessary. What got me going on this topic were two recent (2016) papers I read - one in the *Journal of Animal Sciences* and the other in the *Journal of Dairy Science*.

**What's the problem?** Climate change? Global warming? How about *The Old Farmer's Almanac* for 2017? "April and May will be slightly warmer and drier than normal. Summer will be warmer and rainier than normal, with the hottest temperatures in late June and early to mid-July, from late July into early August, and in mid- to late August...." (<http://www.almanac.com/weather/longrange/region/us/15>). Whether any predictions come to pass, summer can be hot in parts of the Pacific Northwest and this hot weather can affect the health and welfare of cattle.

For **feedlot cattle**, heat stress affects Dry Matter Intake, and in some studies, Average Daily Gain. In a study by Hagenmaier et al. (2016), heat stress affected post-harvest dressing percentage. This study reported that heat stress effects can be more severe if the feedlot is feeding a beta-adrenergic agonist during the end of the feeding period. Welfare implications are the observable signs of heat stress, such as open-mouthed breathing but the subtle stressors are also there.

In the **cow-calf herd** on pasture, heat can lower conception rates by affecting bull fertility or result in embryonic losses during the breeding season for spring calving herds or affect calf viability for the fall calving herd. Cows affected by heat stress in the last months of gestation can calve prematurely (Wright et al., 2014).

A lot more research has been done looking at the effects of heat stress on **lactating dairy cows**. Effects on conception rates are commonly seen as well as Dry Matter Intake drops and lower milk production (West, 2003; De Rensis and Scaramuzzi, 2003). Heat stress can also affect the **dry cow and springing heifer**. It can influence mammary tissue development and lead to low milk production in the next lactation as well as increase the cows' risk for post-partum disease. But, in addition to affecting the cow, heat stress can disturb the **calf in utero**, its immune system (Tao and Dahl, 2013), birthweight and body weight up to one year of age. Long term effects from heifer calf heat stress such as subsequent fertility and milk production in that heifer's first lactation have been measured (Monteiro et al., 2016). For the **pre-weaned calf**, heat stress will affect feed intake, lower Average Daily Gain, and increase risks for disease and mortality (Jones and Heinrichs).

**How well does shade help with the problem?** The rules of heat stress remediation start with access to water, access to shade, a change in diet, and then some system of ventilation and cooling. These strategies are applicable to any class of cattle. Recent research looking at shade during hot temperatures shows how this strategy might pay for itself.

**Feedlot steers** - In the Hagenmaier (2016) study, pens of cattle received shades or not. Cattle in those pens with shade had better Dry Matter Intake, reduced open-mouth breathing and better post-harvest dressing percentage. In another study of Angus steers, shade improved Average Daily Gain, Gain to Feed Ratio, Hot Carcass Weight and average panting score (Gaughan et al., 2010). At the Meat Animal Research Center investigators looked at the steers' environment and found that as ambient temperature increased, the ground temperature under the shades could be as much as 30°C lower than the ground temperature not in the shade, allowing for a more comfortable resting spot for the cattle. In addition, body surface temperatures were lower with shaded cattle by almost 4°C, on average, but depended on the cattle coat color (Brown-Brandl et al., 2013).

**Cows in pasture** - Schutz and others have looked at cow preference for shade and the responses of grazing cows to shade. Dairy cows preferred shade to sprinklers even if the sprinklers reduced their heat load more efficiently (Schutz et al., 2011). The bottom line was that they use it, they pant less, they lie down less and they want more shade (Schutz et al., 2014).

**Lactating dairy cows** - It was 1947 when Smeath and Miller said, "In each case shade alone, as provided by the barn, was effective in reducing materially the body temperatures of the ... cows." Dairy cows in free-stall barns already have shade and can stay cooler unless the barn is poorly-ventilated or the stalls are uncomfortable resulting in some cows choosing to stay out in the exercise lot, if afforded to them. For lactating cows in dry lots, shades are provided and benefits of additional cooling systems such as sprinklers at the feedbunk have also been employed.

**Dry cows and springing heifers** - Access to shade for dry lot-housed dry cows and springing heifers should be considered. In a study we did a decade ago, close-up cows already had access to corral shades and sprinklers over the feedbunk but providing additional shade over the bunk and a set of fans led to greater 60-day milk production after they calved and had an economic benefit (Urdaz et al., 2006). For these animals on dry-lots, adding shade is the first step to improve comfort as well as subsequent performance of them and their calves.

**Pre-weaned calves** - Calves in hutches already have shade but research has shown that adding shade in the way of shade cloth over the hutches has benefits. Shade over the hutches decreased the rise in hutch temperature (Spain and Spiers, 1996) and shaded calves tended to have better feed efficiency (Coleman et al., 1996).

**Conclusions** - Shade can be a less expensive alternative to misting, spraying or fan cooling systems and is the first consideration when remediating heat stress in cattle, in addition to unlimited access to water. There are many configurations for shade structures for different operations but a couple of guidelines can be used. For feedlot cattle, a good resource is provided by Rinehart (2006). For dry lot dairies: Drainage comes first, shaded area must be 2 feet higher than the pen surface, with at least 600 square feet of dry lot space and 48 square feet of shade space per adult animal (Moore, 2010). For placing shade cloth over hutches for pre-weaned calves, the shade needs to be about 11 feet off the ground to provide for adequate air circulation above the hutch (Moore et al., 2012). Providing shade to cattle under heat stress is one way to improve their welfare, health and performance.

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## Beef: Wet Muddy Lots = Footrot

by Andy Allen, Field Disease Investigation Unit, WSU

Cattlemen in Washington State have been seeing a lot of footrot in the last couple of months. This article highlights what to look for, how to manage and how to prevent this condition in cattle.

**What is Footrot?** Footrot, also known as interdigital necrobacillosis or necrotic pododermatitis is a common lameness-causing infectious disease in both beef and dairy cattle. The disease is characterized by inflammation and tissue necrosis of the soft tissues between the toes. Footrot is caused by two anaerobic (grow in the absence of oxygen) bacteria *Fusobacterium necrophorum* and *Bacteroides melaninogenicus*. Both of these

bacteria are common in the environment where cattle have live. *Fusobacterium necrophorum* can also be present in the rumen and in feces of normal cattle.

**What are the clinical signs, and how do you diagnose footrot?** Footrot is characterized initially by soft tissue swelling between the toes spreading the claws, then swelling of the foot up to the level of the fetlock. Fissures or skin cracks become visible in the soft tissues between the toes, there is foul smelling discharge from the lesion, mild fever, and severe lameness. If left untreated the infection can involve tendons, ligaments and joints leading to possible surgical intervention with toe amputation and poorer prognosis. Diagnosis is typically made from observation of the clinical signs and a thorough examination of the foot. Ruling out other causes of lameness such as entrapped foreign bodies (sticks, rocks, etc.), sole abscesses, and hairy heel warts is important for appropriate diagnosis.



Figure 1. Examples of swelling and extreme soft tissue damage due to footrot.

**What are the economic impacts of footrot?** Even mild cases of footrot can cause significant production losses. Cattle with footrot will have decreased milk production, rate of gain, and fertility. There is also the cost of treating the animal with antibiotics and anti-inflammatory drugs. Footrot will also inhibit bulls from breeding cows and cows with footrot are hesitant to, or will not stand to be mounted. In one study performed in 2010 the average cost of a single case of footrot in a dairy cow would be \$120.70.

**What are the risk factors for footrot?** Not all cattle exposed to *F. necrophorum* or *B. melaninogenicus* will get footrot. First, the cow has to have some sort of disruption in the skin between the claws to allow the bacteria to invade. Continuous exposure of feet to very wet, muddy or manure-laden environments is one of the most common risk factors. Over time the continuous exposure to moisture breaks down the skin to allow the bacteria to invade the soft tissues. Other common risk factors are exposure to frozen mud, ice, dry hardened mud, mowed or cut plants with stiff stems, and sharp gravel or stones.

**How do you treat and prevent footrot?** Treatment of footrot is relatively easy if it is caught early in the disease process, when most animals will respond well to injectable antibiotics. Some animals may also require anti-inflammatory drugs such as flunixin meglumine, foot wraps, and topical wound care products to protect the damaged, exposed

tissues. And finally, some animals may require surgical intervention if the infection involves tendons, bones, and or joints. Antibiotics labeled for use on footrot include: LA 200® (and generic equivalent), Tetradure 300® (and generic equivalent), Tylan 200®, Excenel®, Excede®, Draxxin®, Nuflor® (and generic equivalent), and Albon® (and generic equivalent).

Prevention of footrot requires limiting exposure of cattle to the risk factors described above. Establishing appropriate drainage of lots, frequent scraping of manure to avoid buildup, allowing pastures to dry prior to turnout, covering ice and frozen mud with bedding, breaking up sharp hardened mud, and removal of sharp rocks. Evaluation of trace mineral supplementation specifically zinc and iodide appear to be beneficial for hoof health and prevention of footrot. There are vaccines commercially available for footrot, however to date their effectiveness is controversial.

**Summary** Footrot can be a very costly disease in cattle herds. Diagnosis and treatment of footrot is not difficult, however delay in treatment can lead to very expensive alternative treatments. Awareness of risk factors and preventing these risk factors is usually cost effective.

#### References

1. E. Cha, J.A. Hertl, D. Bar, Y.T. Grohn. The cost of different types of lameness in dairy cows calculated by dynamic programming. 2010 Pre Vet Med, 97:1-8.

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## What's New at WADDL?

### How to get the most out of an abortion work-up

by Chrissy Eckstrand, DVM, PhD, Dipl. ACVP, [ceckstrand@vetmed.wsu.edu](mailto:ceckstrand@vetmed.wsu.edu)

Fetal loss is both economically devastating and frustrating for cattle farmers and veterinarians, and determining the underlying cause often poses a significant challenge due to the array of different causes. The list includes numerous etiologies that encompass infectious, metabolic, genetic, toxic/deficiencies, and maternal diseases.

To optimize the success of identifying the underlying cause of fetal death, the Washington Animal Disease Diagnostic Laboratory (WADDL) has assembled a **bovine abortion panel** that aims to assist in ruling in or out a number of important etiologies. Submission of the appropriate samples for diagnostic testing is the lynchpin in receiving the most useful information from the abortion panel. A succinct and comprehensive list of samples to be collected and submitted from the fetus and dam can be found on the WADDL website: ([Accession Form](#)).

Important samples to collect include the dam's serum, fresh and formalin fixed placental and fetal tissues (complete list on WADDL website), and fetal fluids (abomasal fluid and fetal heart blood). From these samples WADDL will run quality controlled and validated diagnostic assays to assist in identifying important abortifacients/factors including numerous infectious causes such as *Campylobacter*, *Leptospira*, Bovine viral diarrhea virus (BVD), Bluetongue virus (BTV), Infectious bovine rhinotracheitis virus (IBR), and *Neospora*. Aerobic bacterial cultures of both the placental and fetal tissues are also performed to identify other pathogenic or opportunistic bacterial pathogens that may be significant.

Fresh samples for culture should ideally be collected using aseptic technique to ensure quality results. Full histological examination of fetal and placental tissues by a Veterinary Pathologist is included in the abortion panel, which can further guide more specific diagnostic testing depending on the nature of observed lesions. Lastly, fetal liver tissue is directed to the toxicology lab to evaluate selenium levels.

Don't have time to collect samples in the field? WADDL will accept the submission of an entire bovine fetus, placenta and dam's serum and collect the appropriate samples in the laboratory. Submission of a full fetus may also improve the detection of fetal malformations, as a Pathologist will perform a thorough necropsy of all organ systems. Providing information on the herd's health, vaccination status, environment, and recent losses on the accession form is also of great benefit to the WADDL diagnosticians as it may aid in identifying specific underlying etiologies.

Have specific questions? WADDL provides a telephone consultation service at no charge for clinicians to speak with a Pathologist or Microbiologist. Consultants are available to discuss specific disease questions and can provide advice on optimal sample collection and submission. (509) 335-9696

#### Reference

1. Anderson ML. Disorders of cattle. In: Njaa BL, ed. *Kirkbride's Diagnosis of Abortion and Neonatal Loss in Animals*. 4<sup>th</sup> ed. West Sussex, UK: John Wiley & Sons, LTD; 2012: 13-48.
2. Learn more about WADDL at: <https://waddl.vetmed.wsu.edu/>

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## WSDA Corner

### Dr. Brian Joseph Appointed New State Veterinarian



The Washington State Department of Agriculture (WSDA) has hired a veterinarian with 40 years of experience in veterinary medicine as its new state veterinarian. Dr. Brian Joseph has held senior veterinarian and animal health positions in zoos, aquariums and nature centers in the U.S. and Canada and was chief veterinarian at the Point Defiance Zoo and Aquarium in Tacoma for six years. Most recently, Dr. Joseph was senior director of animal management and conservation at the Assiniboine Park Zoo in Winnipeg, Manitoba. Dr. Joseph started with WSDA in December 2016. Dr. Joseph holds a bachelor of science in zoology from San Diego State University and a doctorate of veterinary medicine from the University of California, Davis. While serving in the US Army, he participated in missions focusing on health and disease control of animals such as cattle, chickens, pigs, sheep, goats and horses around the world, including the Middle East, Central America and Africa.

“Dr. Joseph comes to us with a firm understanding of regulating animal health, working in government and engaging the community,” WSDA Director Derek Sandison said. “Our interview panel included Animal Services Division staff and industry stakeholders. We’re excited about our selection and eager to support his work for animal welfare and our livestock industry.” As the new state veterinarian, Dr. Joseph reports to Sandison and is responsible for policies related to animal health. He also works with the agency’s partners

and livestock stakeholders. The broad goals of the State Veterinarian's Office and the Animal Services Division are to protect and enhance animal health and animal wellbeing, promote the economic vitality of the livestock industry, and safeguard Washington residents by identifying and limiting exposure to zoonotic diseases that could affect humans.

Dr. Scott Haskell, hired in August as assistant state veterinarian, will continue in that role, responsible for daily operations of the Animal Health program.

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## Mobile Application For Creation of Electronic Certificates of Veterinary Inspection (mCVI)

by CS McConel, Extension Veterinarian and David Hecimovich, Animal Disease Traceability Program Manager, Washington State Department of Agriculture



The mobile Certificate of Veterinary Inspection (mCVI) is available to Washington State accredited veterinarians as a free mobile “app” that works on iPads, iPhones, and Android devices and is available through the Apple App Store or Google Play. The mCVI creates both large and small animal health certificates, is easier to fill out, and is cheaper than paper certificates. The mCVI can help prevent errors through specific submission requirements and allows the addition of animals in groups or with individual ID's to save you from writing multiple CVIs for a single animal movement. Once completed the mCVI can be signed and printed without access to the internet. The app will automatically submit signed certificates to the state

veterinarian's office in both the origin and destination states the next time it connects to the internet. The app also allows you to either print or email copies to recipients and maintain an electronic CVI for your records. Currently, 32 states and over 1,200 veterinarian are now utilizing the mCVI. Although the mCVI is for both Apple and Android devices there is a slight flaw in the Android version that cuts off some pages of the CVI when printed. The Washington State Department of Agriculture's Animal Disease Traceability Program is therefore requesting veterinarians be aware of this while future enhancements are made.

There are four easy steps to get started:

1. Download the mCVI app
  - a. Search Google Play or the App Store for “mCVI.”
2. Create an account
  - a. Each accredited veterinarian must create an account for his or her use.
  - b. Veterinarians must be licensed and accredited to practice in Washington State.
3. Wait for approval
  - a. The state veterinarian will approve accounts within 2 business days.
4. Use the app
  - a. Using the app, veterinarians can create large and small animal CVIs.

- b. Completed certificates are automatically submitted to the Washington State Veterinarian's office and may be emailed to the client or printed directly from the app.
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## Continuing Education

### Veterinarians

**Bull Breeding Soundness Evaluation** – Drs. Tibary and Campbell -- The evaluation of bulls for their ability to perform as herd sires has unfortunately become commonly labeled as “semen testing” or “semen evaluation”. It is important to stress that semen evaluation is just a small part of the complete evaluation of bulls for breeding soundness. Understanding the guidelines for breeding soundness examination requires a thorough knowledge of the reproductive physiology as well as factors that may affect spermatogenesis and/or sperm delivery. In addition, the veterinarian has all the adequate training to recognize the lesions or symptoms of diseases that may be relevant for herd biosecurity. **This course provides 1 credit at a cost of \$50.00. Go to the following site to register:** <https://apps.vetmed.wsu.edu/CVME>

**Calf Care Audit** -- \$50 + 1 CE credit – Visit <https://apps.vetmed.wsu.edu/CVME/Event/Details/28> . Go through the course, complete a 10 question quiz and you will automatically be issued a CE Certificate. PowerPoint notes and the 5 reference documents are also available!

**CVM Spring Conference.** April 21-23, 2017. WSU College of Veterinary Medicine in Pullman, WA. This program will offer 11 clock hours of CE credit. For food animal veterinarians, there is a special **BOVINE ULTRASOUND** workshop. Website: <http://cvme.vetmed.wsu.edu/cvme-index/spring-conference> Agenda: <http://cvme.vetmed.wsu.edu/cvme-index/spring-conference/agenda> Veterinarian (\$275) and Veterinarian Technician (\$175).

**CVM Homecoming CE Event**, October 21, 2017, WSU Pullman. Veterinarians & veterinary technicians -- earn 3 free credit hours of CE prior to the CVM Alumni Barbeque and Homecoming football game. SAVE THE DATE! More details to come!

### Producers

**Washington State Shearing School**, Program Contact: Sarah Smith, Animal Sciences Regional Specialist, (509) 754-2011 [smithsm@wsu.edu](mailto:smithsm@wsu.edu) **Shearing School Dates:** April 3-7, 2017 (Beginners School), April 8, 2017 (Advanced Tune-up Session) [Shearing School Information](#) [Shearing School Application](#)

**Central Washington Junior Livestock Show**, April 30th - May 3rd 2017, Sale - May 3rd 2017. For more information: <http://www.centralwashingtonjuniorlivestockshow.com/>

Visit our website for information on current research projects and outreach materials for veterinarians and producers! <http://vetextension.wsu.edu/>

**Send newsletter comments to the Editor: *ag animal health***  
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