Bluetongue Disease in Camelids

What is Bluetongue Disease?

Bluetongue disease (BT) is a non-contagious, infectious viral hemorrhagic disease affecting primarily domestic and wild ruminants and camelids. Canines are also susceptible. It is considered non-zoonotic but there has been one documented case of human infection in a laboratory worker. It is caused by the Bluetongue virus (BTV), of which there are officially 26 identified serotypes, fifteen of which have been found in the USA, predominantly in the south (BTV-1, 2, 3, 5, 6, 9, 10, 11, 12, 13, 14, 17, 19, 22, 24) and four of those appear in the western states (BTV-10, 11, 13,17). Ten (BTV-1, 3, 5, 6, 9, 12, 14, 19, 22, 24) of the fifteen found in the USA appeared after 1997 as the serotypes have expanded their distribution. The virulence of BTV varies quite markedly; even strains with matching serotypes have variable virulence. The disease is considered non-contagious in that it cannot be transmitted between animals through casual contact, saliva, etc., but instead requires inoculation. Vectors include blood sucking insects capable of transporting the virus, oral ingestion of infected animal products (such as predator/prey transfer or fresh food products containing infected animal sources), or semen transfer from virulent animals. The primary reservoir of virus in the USA is cattle. The overall seroprevalence of BTV in cattle in the United States is >18%. In many regions virtually 100% of cattle have been infected by age 2. Aside from the common vectors, accidental infection has been reported in dogs in the USA following administration of modified live canine distemper vaccine that was unknowingly contaminated with BTV during manufacture.

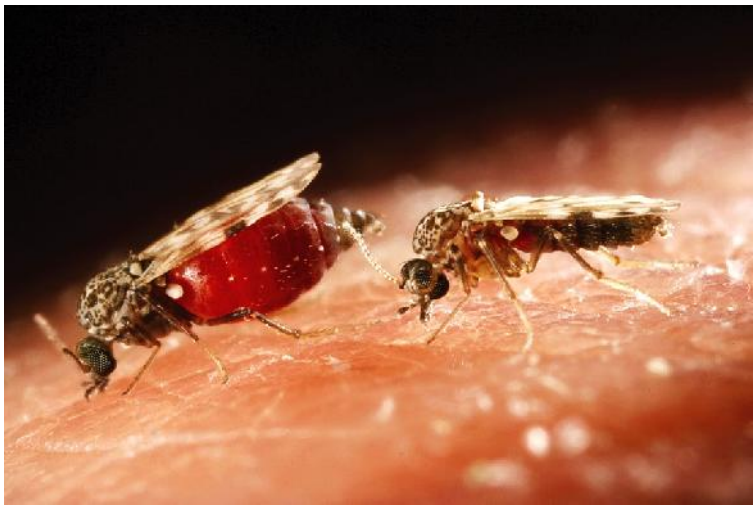
Where is Bluetongue Disease Found?

Bluetongue disease was first recognized in Africa, identified in South Africa over a hundred years ago concurrent with the introduction of Merino sheep that soon succumbed to the disease, but its existence likely dates to antiquity. In fact, most scholars and scientists believe the biblical account of the ten plagues to afflict Egypt as recorded primarily in the Book of Exodus and retold in the Ipuwer papyrus include swarms of culicoides midges spreading hemorrhagic diseases, specifically, African Horse Sickness (AHS) and Bluetongue Disease, rapidly killing the horses, camels, cattle and goats in the region.

It has since spread to other parts of Asia, Australia, Europe, the Middle East, and the United States. It is currently thought that Antarctica is the only continent still free from BTV. In the USA first identified in the southeast in 1923, it was later confirmed present in California in 1952, Michigan and New Jersey in 1955, and Washington State in 1959. Since 1998 it has been occurring with increasing frequency in more northerly latitudes in the U.S., particularly in Washington, Montana (ex. 30-50% population loss of antelope and deer in 2008, confirmed BTV-17), Minnesota and Michigan (ex. 15,000 whitetail deer killed in 2012), and in southern British Columbia. In warmer southerly states the disease is a continual threat, but is seasonal in northern climates, typically appearing later in the warm season the further north one gets; late summer and early fall in the most northerly states. It subsides during the winter, as adult midges cannot survive in the cold. The survival of the disease past the winter season is due either to midges that survive the winter in a dormant state or seasonal expansion from warmer climates. (See attachment 1 for a distribution map.)

The 1959 Washington State outbreak was of unknown serotype. Another outbreak occurred in 1969, again serotype unknown. BTV-17 was identified in Washington State in 1974. An outbreak of BTV-11 appeared in 1976 and 1977. Another widespread die-off occurred in 1980 due to BTV-10. The 2013 epidemic is identified as BTV-11. Each of these cases involved Washington, Oregon and Idaho, starting in July and ending in November with the arrival of vector-killing freezing temperatures. BTV-10 was first isolated in Montana in a 1967 outbreak. It's notable that the most severe BT seasons in the Pacific Northwest appear to occur about

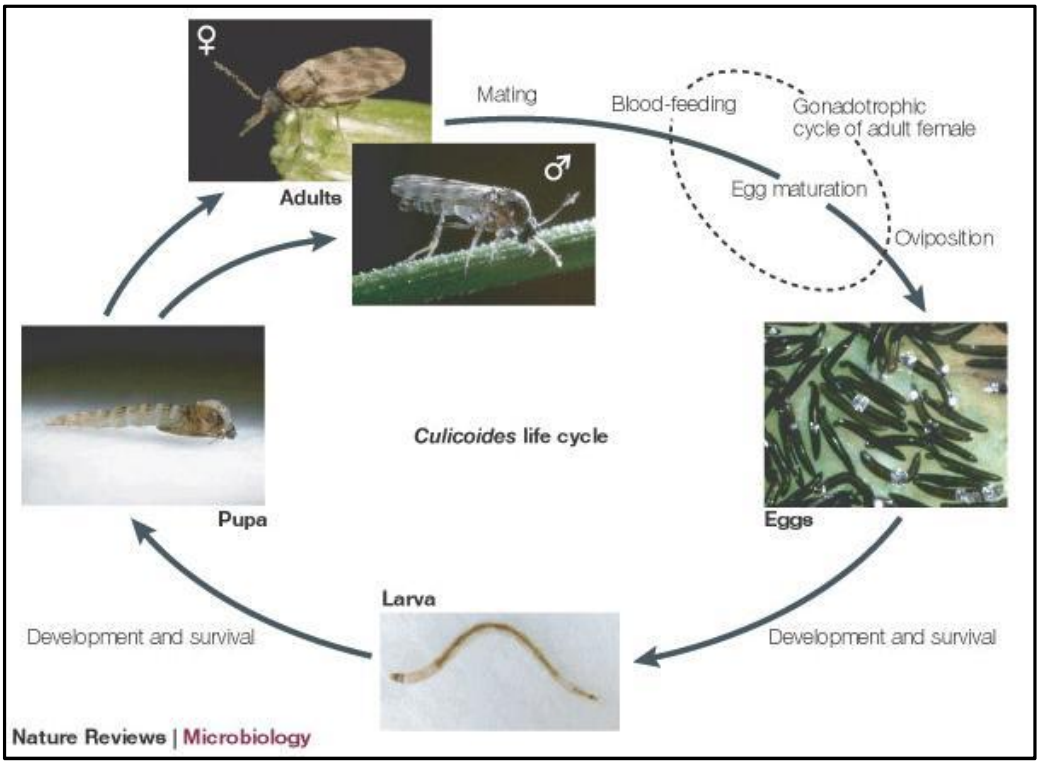
every 3-4 years, rotating through a different serotype each time. Contributing to this cycle may be the practice of beef cattle being slaughtered at age 3-4 and replaced with immunologically naïve calves.



How does Bluetongue Virus Spread?

BTV is in nearly all cases transmitted by biting midges of the genus *Culicoides*. These flies are small, sometimes referred to as gnats or no-see-ums, only 2-3mm in size prior to engorgement. In camelids they typically get into the ears where they reside for hours feeding on blood safe from harm. The skin is much thinner in the ears than elsewhere so the flies can easily reach the shallow blood vessels. Other hairless or nearly hairless areas are also vulnerable but exposed areas are not as attractive to the flies.

While there are over 1400 species of Culicoides around the world, only 30 or so have been implicated in the transmission of the virus. Culicoides sonorensis is the carrier species found throughout most of the USA and almost exclusively in the western states. BTV-2 relies exclusively upon Culicoides insignis to spread, which is found only in the southeast. Culicoides variipennis is found in the eastern USA and is a vector for various serotypes. The virus actually replicates inside certain species of the Culicoides midge, but there is no evidence that it replicates in other species or in mosquitoes, though can be mechanically transmitted by mosquitoes or other species. Animal to animal transmission is not capable of maintaining an endemic state and the virus is dependent upon suitable Culicoides vectors to persist in the environment. Culicoides ranges appear to be increasing in recent years, possibly due to warming climates, milder winters, and increased winds. Increased movement of infected animals or animal products may also contribute to wider distribution of various serotypes.



The midge life cycle involves an egg, four larval stages, a pupal stage, and an adult. Eggs are laid in a mass on moist surfaces and hatch in 2 to 7 days. The larval stage lives in moist habitats such as mud or shallow water; development into a pupa is complete in about 2 weeks. The pupal stage is formed in the same site as the last larval stage, and adults emerge in about 2 to 3 days. Adult midges usually live 10 to 20 days in warm weather.

Culicoides are not good flyers, ranging generally up to a mile and a half, though typically under a half mile, in search of a blood meal, unless winds happen to carry them farther. Females feed five times prior to laying eggs, potentially feeding on five different animals and spreading disease in the process. Once infected with bluetongue, a midge can transmit the virus the rest of its life. The flies are active day and night but often remain in the ears overnight. A single fly can easily spread virus from one viremic animal to four others. When these flies appear in an area they often do so in large numbers, spreading virus rapidly. Llamas typically flick their ears at the faint sound of these flies, and once in the ears can be very irritating and the llamas may be seen rubbing their ears on objects or the ground occasionally in a vain attempt at relief. A swipe of a finger or thumb in their ear may come out bloody, and the flies trying to escape may be seen.

Species of Culicoides at northern latitudes mostly survive the winter as larvae, so it might appear that the most likely mechanism for overwintering is the vertical transmission of virus from infected vectors to offspring via the developing egg ("transovarial" transmission). However, experiments designed to look for vertical transmission of BTV in Culicoides have consistently reported negative results. Although adult Culicoides are far less tolerant of sub-zero temperatures than Culex mosquitoes and are normally thought to survive no longer than 10–20 days, laboratory studies suggest that this lifespan may be extended by mild winter conditions, with individuals surviving for up to three months at 10 °C in the laboratory. In mild winters it is possible that a small fraction of the infected adult Culicoides population might survive long enough to bridge the gap between transmission seasons. Adult Culicoides may also be sheltered from the worst conditions of winter to some degree by their choice of resting place.

Infectious BTV can be isolated from the blood of cattle for much longer than from sheep and goats, and although the vast majority of infections in cattle endure for less than 60 days, a fraction may last for much longer. Such infections could permit the virus to persist for three to four months without infecting new hosts, and thereby survive short periods of vector absence. White-tailed deer and elk typically show a detectable viraemia for only 16 days or less. However, one study has suggested that viraemia in elk may resume up to three months post-infection in response to stress.

What other diseases are spread by this vector?

Culicoides transmit other arboviral diseases in the genus Orbivirus to animals, such as African horse sickness virus (AHSV), equine encephalosis virus (EEV), and epizootic hemorrhagic disease virus (EHDV). Each has been found in the USA though originated outside the USA. AHSV and EHDV can infect and afflict camelids. AHSV is mostly confined to Africa, Middle East and southern Europe and Asia and is as-yet uncommon in the USA. EHDV occurrence and distribution in the USA is similar to BTV and most visibly afflicts deer, but can strike all ruminants. There is a confirmed 2013 epidemic of EHDV in both eastern and western Montana – both sides of the continental divide. Hundreds of dead deer have been found, as happens periodically. It appears annually in Washington and Idaho, identified in cattle starting in the 1990's. EHDV is quite common in the Midwest, and even several Yak herds in Colorado have been hit by this disease. Southwest North Dakota, Montana and Wyoming experienced a large scale deer die-off in 2011 and 2013 from EHDV. EEV, as the name suggests, afflicts all equine species. Symptoms of these hemorrhagic diseases are similar to BTV, though mortality from EHDV in sheep is less than that for BTV.

As an aside, arboviral diseases in camelids are not limited to the Orbivirus genus. Eastern, Western and Venezuelan Equine Encephalomyelitis (EEEV, WEEV and VEEV) in the Alphavirus genus are spread by mosquitos, with distribution throughout most of South, Central and North America into Canada. These diseases are about 75-90% fatal in equine species while camelids appear to have a lesser but still very high

mortality rate. The first visible signs generally become apparent at four to five days post-infection. At that time, the animal generally has a fever and rapid heart rate and is showing signs of anorexia, depression, and variable other neurological signs. As the illness progresses, more consistent neurologic signs develop. Muscle weakness becomes apparent and there are behavioral changes and dementia. Some develop aggression, head pressing, wall leaning, compulsive circling, and blindness. Other signs might include uncontrolled twitching of the eyeball, facial muscle paralysis and seizures. As the disease progresses even further, a semi-comatose and convulsive state occurs. Death usually follows two or three days later. If the animal survives, residual nervous system problems are likely to result. There are vaccines available for EEEV, WEEV and VEEV.

West Nile Virus (WNV) is of the Flavivirus genus, spread by mosquitos and the culicoides midge, with similar symptoms to EEEV and WEEV, but with lower mortality rate, estimated at 10-15% in camelids and 35% in equine species. A characteristic symptom of WNV disease but not EEEV and WEEV is head and neck tremors. Distribution includes the entire United States. Vaccine is also available for WNV.

What are the Effects of Bluetongue Disease?

BTV can devastate livestock populations. For example, a case of 179,000 sheep dying in one region over a four month period has been documented. Sheep are the most severely affected, particularly certain breeds, as are white-tailed and mule deer and antelope. In these species the mortality rate is as high as 90%, and the most afflicted animals (regardless of species) can die within hours of showing visible symptoms. Recovery for those that survive is often a months-long process. Organ and muscle damage can be severe.

In all afflicted animal species, after a prepatent period of typically 3-8 days virus-mediated damage to endothelial cells ensues resulting in vascular thrombosis, tissue infarction, necrosis and hemorrhage. In five to twenty days symptoms include fever, serous to bloody nasal discharge, congestion, difficulty breathing, excessive salivation, severe pulmonary, intramuscular and subdermal edema, hydrothorax, hydropericardium, myocardial and intestinal hemorrhage, oral erosions and ulcers. The ulcers are typically painful and contribute to anorexia. Diarrhea is commonly seen. Animals become listless and anorexic. Late in the progression, typically 7-12 days following initial symptoms, lameness with hyperemia of the coronary band (in hooved animals) and torticollis may occur. Occasionally the hooves will eventually die and slough. Abortion and congenital malformations are typical of infection during pregnancy, canines included. (Pregnant dogs typically die 3-7 days following a stillbirth abortion due to BTV.) Bluetongue disease is so named because infected animals sometimes develop cyanosis, or blue coloration, of the tongue. Secondary bacterial pneumonia and other opportunistic complications can also develop, sometimes contributing to death in animals that don't immediately succumb to BTV. Direct cause of death in peracute cases is usually due to frothing from the lungs, sometimes visible in the nostrils, and the accompanying asphyxiation, often before other visible symptoms appear short of listlessness and difficulty breathing. In such cases death can occur in less than six hours of the first appearance of subtly visible unusual behavior such as increased recumbency. Due to the rapidity of such deaths the telltale signs may be missed and the animal(s) mysteriously discovered deceased with no outward causal indications. Such individuals are often disposed of with indeterminate cause of death, leaving caretakers unaware and the herd vulnerable to expanded suffering and death.

Some infected animals exhibit no outwardly obvious symptoms, apart from various degree of lethargy. Cattle are usually asymptomatic aside from reproductive effects (abortions, congenital defects) and reduced milk production in dairy cattle but can be virulent for weeks (up to 11, but 90% are clear in 7). Elk are also often asymptomatic. In other ruminant species and in camelids, symptom frequency, manifestation and severity varies somewhat relative to others, but in all susceptible species the disease can progress very quickly, striking

some individuals, even young, vibrant, healthy ones, with debilitating or fatal consequences. Severity is also dependent on the virility of the particular strain of BTV.

Multiple small scale studies have been performed indicating surviving camelids typically clear the virus more quickly, in less than 3 weeks. It has not yet been disproven that the virus may lay dormant in organ tissues and re-emerge during a period of extreme stress, but if not detectable in the bloodstream then it is not transmissible by the midges. Studies have also reported that the amount of detected virus in the bloodstream of virulent camelids is significantly less than in cattle.

Infected animals don't become viremic until at least four days following infection. A serologic response in ruminants can be detected 7–14 days after infection. In peracute cases death can occur prior to a detectable serologic response. In camelids blood viremia infectious to Culicoides is normally cleared within 21 days. Once the virology is cleared they possess long term, possibly lifelong though no studies have proven this, immunity for the homologous serotype following field infection. Because of this immunity, an animal exposed to BTV and already exhibiting a positive antibody titer in laboratory testing normally presents no risk of harboring or transporting the virus, particularly following a brief quarantine period.

How is Bluetongue Disease Controlled?

There is no treatment for Bluetongue disease, but it can be controlled through quarantine, vaccination (where available), and control of the midge vector. Complicating and secondary infections should be treated appropriately during the recovery period. Vaccinations are only available for some strains of BTV. Midges can be controlled by preventing midge breeding sites, often cow and horse dung heaps and moist soil, from proliferating, and by keeping animals sheltered during dusk through dawn, when midges are most active.

Vaccines

The only vaccine approved for use in the USA by the USDA is for BTV-10. California has approved vaccines for BTV-10, 11, 17 in-state only. The vaccines are inexpensive and effective, but these modified live (MLV) vaccines cannot be given to pregnant dams as it can cause teratogenic defects such as cavitating encephalopathy and retinal dysplasia, often blind, stillborn and/or dying soon after birth. As modified live vaccines these effects are simply milder cases of field infection with the live virus during pregnancy, which typically causes severe birth defects and abortion. More advanced vaccines are in development but lack urgency since the cattle industry is not very concerned about BTV.

Vector Control

Larval Stage – Controlling Culicoides sp. at the larval stages typically involves the removal of the breeding areas and/or treating those areas with various insecticides. Culicoides sp. breed and develop in areas of standing water, particularly shallow, damp areas such as mud, irrigated pasture and marshlands. These areas, depending on their structure, location and regulations surrounding them, may be drained or filled-in to potentially reduce the population of Culicoides sp. and subsequently, theoretically, reduce the bite risk to livestock.

Some insecticides have been experimented with in the control of breeding grounds and larva stages of Culicoides, and although some have had minimal effects, the need for widespread application, hazards to human health and environmental health, as well as the risk to the animals in the pasture makes this an unlikely option.

Some potential methods being looked at for control include the use of the bacteria, Bacillus thuringiensis, to treat insect breeding grounds and potentially reduce the number of insects. Thus far, research has shown that the levels of bacteria required to significantly reduce insects, including Culicoides sp., populations are too high to be practical in a field setting.

Adult Stage – There are various models of propane powered CO_2 traps as well as large fan traps. Both types get good ratings when they work but poor ratings for operational reliability and the former has high operating costs in addition to high purchase price. If expense is no object the combination CO_2, UV, Fan traps are the most effective, but check them frequently. I opted for three advanced UV zappers with oscillating light that simulates movement and chemical attractant (octenol), replacing one old zapper. The chemical attractant is designed primarily for mosquitoes but manufacturers claim it attracts all blood feeding insects at close range, as it mimics breath and sweat scent. The attractant cartridges last for about a month and emit their scent when heated by the lamp. Cost is low and reliability is high with minimal maintenance. Units with 40W blacklights will cover up to a half acre unobstructed. Place them about 6 – 7 feet off the ground. Multiple UV/chemical traps can be purchased for less than a single CO_2 trap. Try to place them between potential sources of midges and the animals. The limitation of these traps is that they are really only effective from dusk to dawn, but the midges are active all the time.

Animal Preventive Treatment

Using topical fly repellant significantly decreases the biting risk from Culicoides sp. and in addition the ears and face, the axilla (armpit) and inguinal (inside of the hind legs) regions may need to be included to cover thinner, more exposed skin. The inside ears are the primary target of the midges so are the most important areas to treat. Synthetic pyrethroid products, e.g., Permethrin + Piperonyl Butoxide, are the only ones proven effective against Culicoides. Spray on pyrethroid products need to be reapplied about every three days, though some claim to be effective for a week or more. Despite such claims, they are not effective beyond three days. These products are safe to use topically, as they are non-toxic to mammals.

Each year we applied a long lasting product of this type ("repels flies up to 14 days", per the label) while the flies were observed to be pestering the llamas, but were not diligent in applying it frequently enough, seeing the flies as just an irritant, not a risk of debilitation or death. The product does not appear to deter all flies for that long. It only takes one infected fly to spread the virus. An interviewed etymologist that began studying arbovirus transmission by midges beginning in 1959 (Texas) and developed a synthetic pyrethroid made the recommendation to apply at least every three days to be highly effective against culicoides, regardless of label claims.

High dosage Ivermectin at 400ug/kg (1cc of 1% injectable solution per 50 pounds of body weight) results in a partial kill of biting midges (roughly 80% effective while Ivermectin levels are high in the animal), but transmission of BTV can occur before the insect's demise. Lower "normal" dosages of Ivermectin (200ug/kg) effective for intestinal parasites have been found to be completely ineffective at killing Culicoides. Consequently, this high dose method can only be used in combination with others and issues of parasite resistance need to be considered. This method is most appropriate in meningeal worm regions where regular dosing with Ivermectin is already practiced.

Pour-on systemic pyrethroid fly repellent products may be effective at reducing BTV transmission and last longer than topical products, but as with Ivermectin the transmission may occur before the insect's demise. Also, there is evidence of adverse fetal effects of some systemic (pour-on) pyrethroids acting as an estrogen

antagonist. Topical products, if maintained, exhibit greater repellency and a more rapid kill. Note that pour-on products need to be applied more gradually on camelids than the label instructions for cattle, due to the more rapid absorption in camelids. Recommended is to split the dose and apply each a day or two apart.

Pyrethroid repellants technically are a pesticide (insect killer) moreso than a repellant. Flies will still land on the animal and try to get into the ears, but will then flee. In this way they are a contact repellant. They are still an irritant to the llama but won't bite. Actual repellants deter the flies from landing in the first place. Citronella and geraniol are examples of non-contact (and non-toxic) repellents. Products with citronella tend to last only a couple of hours so must be applied very frequently. A geraniol product such as BugBand can be effective up to a day if applied liberally. In stable/shelter areas with little air movement there is a BugBand portable diffuser that emits the volatile vapors for 4 days to keep the flies out. They both have a citrus odor. A new spray-on product on the market called EcoVet can repel the flies for two days. It has a pungent odor that some find irritating, but it is effective and non-toxic. The llamas don't seem to mind it if kept off the face.

Repellents containing DEET (diethytoluamide) provide only limited protection. Note: Some camelids have been observed to exhibit pronounced adverse reaction to DEET. Its use is not advised.

We have tried a fly mask for llamas (custom made), but the couple of llamas that wore it didn't like to have it on despite the relief it provided from the midges and each managed to eventually remove it. Contact us for details if you are interested in trying this method.
llamas@rattlesnakeridgeranch.com

Avoid shearing llamas late so coats grow out prior to the appearance of infected midges.

Conclusion

No single defensive strategy can be relied upon to prevent infection from BTV. All reasonable measures need to be taken to control Culicoides breeding areas, trap adult flies with modern traps, and diligently apply topical synthetic pyrethroid fly repellents throughout BTV season each year. Recommended is to apply topical Permethrin + Piperonyl Butoxide inside the outer ears every three days. It is not effective to just spray the top or back of the head as UV exposure will quickly break it down and the flies will not be sufficiently deterred from finding their way into the ear. Alternately or in addition, topical application of EcoVet will keep the flies away but in dry, hot sunny conditions it will evaporate more rapidly and not last as long. Llamas will initially take offense at sprays and owners may prefer to wipe it on, but llamas will soon adjust to the routine and sprays will be tolerated and even welcomed in time.

Even in areas that often see outbreaks of this disease, veterinarians may actually rarely see it and hence may not recognize it when seen. Many animals, cattle particularly, don't show clear symptoms, usually just reproductive and productive, which vets are not called in for and can be easily dismissed. When other animals such as goats or camelids are found dead of mysterious causes it's too late to call the vet so they don't get contacted. Necropsies are rarely performed and the hemorrhagic diseases have to be specifically tested for. The disease can spread silently while veterinarians grossly underestimate the risk and can fail to correctly diagnose when encountered.

References

See Attachment 1

WSU Farm Inspection Report

Following the confirmed BTV-11 death of King Asher, WSU dispatched a Field Disease Investigative Unit to the property.

See Attachment 2

This article is dedicated to:

King Asher, Master Pack Llama (PLTA) and devoted hiking and travel companion. Cut down in his prime at age 9 from peracute BTV in a matter of hours.

Asher means "happy" or "blessed" in Hebrew. He loved adventure and was happiest when on a trip. It was I that was blessed with his companionship.

Santa Fe, a very gentle and easy to handle llama rescued in 2013 from owner abandonment, suffering from infection with West Nile Virus, progressing into poliomyelitis resulting in paralysis of the right rear leg and weakness in the left rear leg. Surviving that, he was subsequently hit with BTV infection and died at age 10.

Wild Bill, rescued in 2013 as an orphaned feral llama, born in the wild from parents released into open desert rangeland seven years before. The dam was killed by a pack of dogs. Initially fearful of close contact and able to leap 4' to nearly 6' fences effortlessly (out and then back in elsewhere), was really a mild-mannered sweetheart appreciative of those who cared for him. With just a short period of training was halter and lead trained and tolerated (nervously) unrestrained foot pickup on command for nail trimming, and was about to begin pack training. A smart, vibrant and spirited presence snuffed out at age 5 by BTV.

Dazy May, herd matriarch. This classic gal was tough as nails and extremely intelligent, diagnosed with kidney failure at WSU in 2004 at age 14 (positive leptospirosis titer) and a follow-up in 2006 with never before seen creatinine levels in a live llama (>30) yet acted completely normal aside from loss of appetite at the time of admission. Kidney biopsies found 100% scar tissue. She was predicted to die within a couple weeks of discharge each time but somehow managed to maintain herself. February 2013 she again went off feed and returned to WSU only to find that kidney function appeared normal per blood test but liver infection was evident. She recovered with antibiotics. End of July she suddenly developed diarrhea with no parasites or coccidia, a symptom of BTV; still possessing the pasterns of a youth, dying less than two days later at age 23, the first to succumb to BTV (unconfirmed), initially attributed to old age.

Five other llamas in our herd were confirmed infected but survived.

And to all the many other innocent lives lost in the 2013 BTV epidemic in the PNW.

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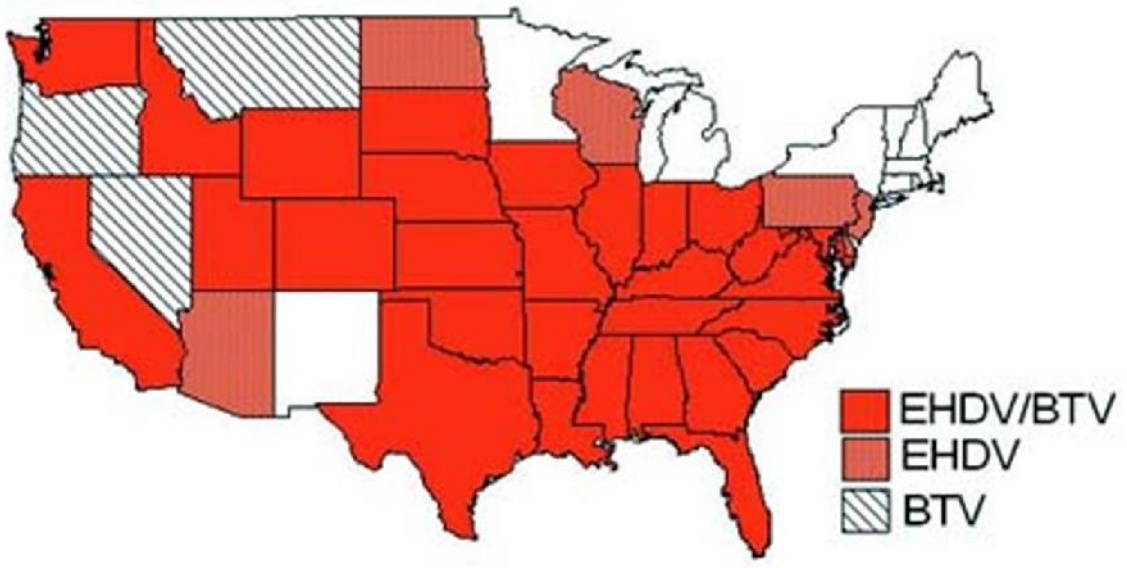

Confirmed Distribution in 2007

Attachment 2

WSU Farm Inspection Report Observations

1. Background:

The farm has been in operation for 12 years and sets out to rescue llamas and place llamas into homes. The property consists of around 5 acres of flat, well irrigated pasture with a large pumper pond serving a block of properties that continually varies in water level and the amount of exposed shoreline sandy mud (from zero to about two feet). A house and large shop is on the property and the remaining area is divided into 2 main fenced pastures (one with the pond in it) with two small pastures and catch pens and five small to medium canvas and wood shelters with large fans running in hot weather.

The pond is tested and sprayed for mosquitos through the summer and is generally drained by late November. "Mosquito eating fish" are added to the pond annually for insect control. Diatomaceous earth is periodically added to manure piles. Fly traps (four different types, seven total) are placed around the property annually Spring through Fall and 3 new blacklight bug zappers have been recently added at locations near the llama shelters, replacing a single old zapper located near the house. Landowner reported conditions are that during seasonal warm weather mosquitoes are very rarely observed but the culicoides midges are frequently observed and bothersome to the llamas.

A canvas shelter and fan

1 of 2 ground insect traps

The pumper pond

20 llamas, 3 dogs, 3 cats and numerous chickens were on the property. The dogs, cats and chickens had free run of the property. The llamas are divided based on gender between the pastures. The females (13/20) are in the pasture with the pond. The males (1/20 intact, 5/20 gelded) were in the adjoining pasture. The first deceased gelded male resided in this pasture. A male (1/20 intact) had a small pasture to himself adjacent to the other males' pasture. The second deceased gelded male also resided in this pasture. The third deceased intact male resided in a small pasture to himself adjacent to the females.

1 of 2 biting fly traps

All the llamas are fed Llama Supplement Plus (Nutritional Services, Inc.) in addition to their pasture in the summer and the supplement and hay through the winter months. Each llama is vaccinated for Clostridium C, D and Tetani annually. Each llama is occasionally treated with ENDURE pyrethroid fly spray during the summer and fall, mainly in the ears where biting flies congregate. Most llamas present on the property in the early Spring were treated with Cylence pyrethroid pour-on for treatment of lice at shearing time.

2. Presenting Case:

A 9 year old intact male llama, King Asher, was referred to the Washington State University Veterinary Teaching Hospital on 08/25/2013 (WSU VTH) for a <1 day history of anorexia and very brief history of reluctance to rise, coughing, foaming at the mouth, lung crackles and severe dyspnea ("wet" lung sounds) with open-mouth breathing. Suspecting choke, the referring DVM had easily passed an orogastric tube, air escaped from the C1 compartment and the rDVM administered Banamine and Draxxin prior to transport to WSU VTH. Upon arrival the patient was found deceased. A necropsy was performed and revealed King Asher died from acute, severe, diffuse pulmonary edema and congestion. This was accompanied by acute, severe, submucosal, segmental trachea edema and acute, multifocal hemorrhage of aorta and pancreas, and liver congestion. Bluetongue virus serotype 11 was isolated via RT-PCR from lung tissue and serology from serum samples revealed no Bluetongue antibodies; death occurring prior to detectable immunological response. West Nile Virus test via Direct PCR negative.

Bill, an approximately 5 year old Male, castrated llama became acutely lethargic, reluctant to rise and anorexic. Bill developed very pronounced edema in his limbs and ventral midline as well as became dyspnic and had "wet" lung sounds. Watery diarrhea was present and fecal exam revealed a bloom of Nematodirus and Strongyle. Excessive coccidia was not observed. He had pale mucous membranes with slow refill, indicating anemia, with a creatinine >20 and BUN >180. He also exhibited a small penile hemorrhagic lesion. Bill did urinate. No necropsy performed.

Concurrent with Bill's illness Santa Fe, an approximately 11 year old Male, castrated llama became acutely lethargic and anorexic. This male had been rescued from Kennewick, WA in the Spring, exhibiting very mild unsteady gait, soon progressing to severe right hindquarter lameness (video sent to WSU VTH). Following multiple visits and laboratory testing in the Spring, the rDVM diagnosed his condition as West Nile Virus induced poliomyelitis. Results and confirmatory blood tests were forwarded to the WA State Vet. office. In Kennewick Santa Fe had been housed next to an irrigation canal, along with a horse. No necropsy performed.