Vector-Borne Diseases of Sheep and Goats

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Definitions
- A disease that is transmitted to humans, plants, or animals by any agent, arthropod, or fomite is a vector-borne disease
- A vector carries and transfers an infectious agent

Examples
- Rabies – vectors are raccoons, skunks, foxes, bats
- Lyme disease – vector is deer tick
- Anaplasmosis – vector is tick
- Cache Valley virus – vectors are mosquitoes and biting midges
- Meningeal worm / Deer worm – vector is snail or slug

Lyme disease
- Deer ticks (Ixodes) carry the bacterium, Borrelia burgdorferi.
- Tick needs to be attached for at least a day to spread the disease
- Very serious in humans - rash, often in a bull's-eye pattern; flu-like symptoms; joint pain and weakness in the limbs
- Treatment is doxycycline in people, dogs and horses

Lyme disease in sheep and goats?
- Described by veterinarians in Lyme CT
- In regions where Lyme disease affects other animals.
- Many have antibodies, few show signs such as lameness, unable to culture from goats
- May be footrot or CAE instead
- Prevent with permethrin every 2 weeks

Anaplasmosis
- Bacterium Anaplasma phagocytophilum
- Anaplasmosis
- Vectored by ticks (Ixodes deer tick)
- Live in white blood cells (“granulocytic”)”
- Dogs show lameness, joint pain, fever, lethargy, and lack of appetite
- High fevers in horses in the fall
- People get fever, headache, chills, and muscle aches after tick bite
- Treat with doxycycline
Anaplasmosis in sheep and goats?

- common in Europe
- tick-borne fever
- sheep may abort
- susceptible to other infections such as Staph (“tick pyemia”) because not enough white blood cells – septic arthritis
- not seeing locally
- maybe don’t have right strains

Asian long-horned ticks

- first found in US in 2017 – widespread now
- *Haemaphysalis longicornis*
- may spread Rocky Mountain spotted fever, less apt to spread Lyme or anaplasmosis
- females can reproduce without males
- SR can bleed to death
- very small

Probably won’t spread Lyme Disease


Cache Valley virus

- named for a valley in Utah and Idaho where trappers hid their trading goods – first isolated from mosquitoes there in 1956
- identified as the cause of malformations in lambs in Texas in 1987-1989
- later found to infect deer, cattle, horses – these animals can multiply and spread the virus, but don’t see malformed fetuses
- rare human infections, but no spread from people

Some big words to describe malformed lambs

- arthrogryposis – joints don’t bend properly, fused (muscle contractures)
- hydrocephalus – fluid accumulates in brain
- hydranencephaly – cerebral hemispheres replaced by sacs of cerebral spinal fluid
- scoliosis – sideways curvature of the spine
- kyphosis – upward curvature – hunched back

Lambs with arthrogryposis frequently cause dystocia
This lamb has hydranencephaly and the brain has collapsed – skull has been opened.

Cache Valley virus in USA
- Bunyavirus
- Akabane, Schmallenberg virus elsewhere - cattle, sheep, goats affected
- mosquito vectors, some *Culicoides* midges
- virus crosses placenta at 30-40 days
- abortions, mummification
- arthrogryposis, hydrocephalus, hydranencephaly
- fetal and maternal serology to diagnose

Cache Valley virus in NY sheep
- traditionally saw in January lambing in Cornell flock – would have been 30 days pregnant in early September
- next lambing was March – did not see (mosquitoes killed by frost by early Nov)
- global warming is changing this!

Lamb delivered February 1, 2021 in Newark Valley

Lamb delivered March 2, 2021 in Ithaca NY
Boer goat farm
- upstate New York, USA
- established herd
- 31 Boer and Boer cross does, 1 purchased buck
- 3 unexplained abortions in November and December 2015
- negative lab tests (Q fever, toxoplasmosis, chlamydia, leptospirosis)

Dystocias - #1
- Dec 24 – 4 feet presenting – neighbor pulled the kid straight out – 2.8 kg, male cryptorchid, ankylosis, short hind limbs, scoliosis, kyphosis, hydranencephaly

Dystocias - #2a
- December 25 twins
  - 2.0 kg dead deformed female; scoliosis, cervical kyphosis, arthrogryposis, short lower jaw, hydranencephaly, cerebellar hypoplasia, short hind limbs

Dystocias - #2b
- December 25 twins
  - 3.9 kg male with head tilt and nystagmus, euthanized; mild torticollis, kyphosis, mild hydranencephaly
Kid 1
Kid 2a

Dystocias - #3a
- December 28 twins
- 2.6 kg cryptorchid male with arthrogryposis, "seal flippers", hydranencephaly, atrial septal defect

Dystocias - #3b
- December 28 twins
- 2.6 kg female with severe scoliosis, arthrogryposis, hydranencephaly

Dystocias - #4
- January 5 single
- 4.7 kg male very similar to #1, front limbs fixed along body
- hydranencephaly, no cerebellum

Bunyavirus suspected
- Cache Valley viruses detected previously in sheep in New York (January lambing)
- kids thawed and examined
- CVV serogroup antibodies detected in serum using dam blood and kid blood or thoracic fluid in this outbreak
- two does that aborted had CVV antibodies
- Schmallenberg, bluetongue, bovine virus diarrhea virus types 1 and 2 ruled out

Why this outbreak?
- arbovirus surveillance data in New York State NYSDOH, obtained in 2017
  - 2014 - no Cache Valley virus detected
  - 2015 – total of 67 positive samples
  - 2016 – no Cache Valley virus
- other viruses detected during these 3 years:
  133 total of FLAV, HJC, JCV, POTV, TVTV
Data from New York State mosquito pools

https://www.wadsworth.org/programs/id/arbovirology/services

Cache Valley virus is green, West Nile virus is dark blue

How about CVV cases in 2022?

We identified a total of 12 positive mosquito pools this year, ... an average year in terms of prevalence. Importantly, there was also evidence of 2 human infections, which is quite unique. We have a manuscript coming out soon showing that the general increase in activity since 2010 may be driven by the emergence of lineage II CVV in NYS, which we experimentally demonstrated is more transmissible by mosquitoes. Whether or not this translates to increases in infection or disease in lambs is not clear but something to consider.

Alexander T. Ciota, Ph.D., Director, Arbovirus Lab.

Cache Valley Virus Summary

- goats as well as sheep can be affected
- mosquito-borne
- teratogenic
- enzootic to much of North America
- white-tail deer may be host
- infected does expected to be immune for next pregnancy
- delay breeding until end of the vector season

Parelaphostrongylus tenuis

dereer worm
meningeal worm

- nonpathogenic in the white-tailed deer, Odocoileus virginianus

Life cycle in deer

- slug or snail ingested – this is the vector
- digested in abomasum
- larvae migrate through abomasal wall to peritoneal cavity
- migrate along nerves to spinal cord in 10 days
- develop in spinal cord
Life cycle in deer
- return to surface of cord at 40 days
- mature and migrate to cranium
- eggs laid into blood vessels
- hatch into first-stage larvae in lungs
- enter bronchial tree, coughed up, swallowed

Prevalence of *P. tenuis* in White-tailed Deer
- 41% of 172 adults - Ontario
- 58% of adults, 33% of juveniles - NY
- 59% of deer > 1 year - MN
- 0 to 100% in counties in S.E. USA (soil type?)

First-stage larvae of *P. tenuis*
- pass larvae 90+ days after infected
- in mucous coat of deer fecal pellet
- killed by drying, solar radiation
- resist freezing

Intermediate hosts
- land snails and slugs crawl over feces
- larvae penetrate gastropod’s foot
- develop to third stage, encyst (3-4 wk)
- persist for life of snail or slug
- 0.04% infected on summer range, 0.16% in winter deer yards
- mean of 3 larvae each

Reported Distribution - *P. tenuis*
### Aberrant hosts of *P. tenuis*
- moose
- caribou
- wapiti
- red deer
- black-tailed deer
- mule deer
- antelope
- llamas
- alpacas
- sheep
- goats
- fallow deer
- (cattle)
- (horses)

### Infection of Aberrant Hosts
- stay longer in spinal cord
- worms active, coil on themselves
- may reinvade cord or brain
- immune response
- usually not patent (don’t shed larvae into the environment)

### Experimental Incubation Period
- Very high doses used
- llamas: 45 - 53 d
- goat kids: 11 - 52 d
- lambs: 11 - 27 d
- fallow deer: 54 - 67 d
- signs common in fall, early winter

### Clinical signs of *P. tenuis*
- ataxia, hypermetria
- stiffness, lameness
- hindlimb weakness or paralysis
- progression to forelimbs
- circling, head tilt, twisted neck
- depression, blindness, nystagmus
- seizures, death
Differential Diagnoses
Sheep and Goats

- CAE, OPP
- fracture, vertebral body abscess
- copper deficiency
- tail docking infection
- foot rot, white muscle disease

Differential Diagnoses
Sheep and Goats

- listeriosis
- polioencephalomalacia (thiamine deficiency)
- brain abscess
- rabies
- scrapie

Cerebrospinal fluid with *P. tenuis*

- lumbosacral tap
- increased cells, usually eosinophils (not as common in sheep)
- increased protein
- llama normals: 30-70 mg/dl protein, 0-20 cells/ul
- goats: <40 mg/dl protein, 0-4 cells/ul

Treatment of *P. tenuis* in White-tailed Deer

- ivermectin
  - kills larvae penetrating abomasum
  - ineffective after day 6 (in spinal nerves)
  - transient decrease in larval output
- albendazole 25 mg/kg for 2 weeks kills adult worms
Treatment of *P. tenuis* in aberrant hosts

- often better to slaughter sheep
- no controlled studies published
- escalation of drug dosages
- ivermectin 0.2 to 1.0 mg/kg for 1-5 d
- fenbendazole 10 to 50 mg/kg for 1-5 d
- usually both simultaneously

Treatment of *P. tenuis* in aberrant hosts

- anti-inflammatory drugs important
- corticosteroids if not pregnant:
  - dexamethasone 0.1 mg/kg daily for 3-5 d
  - flunixin 1 mg/kg daily or twice a day for 3 d
  - [phenylbutazone 2-4 mg/kg q 2 d]

Physical therapy

- deep straw bedding
- roll from side to side
- avoid urine scald, maggots
- flotation tank, cart
- sling? Don’t hold up by tail!
- weight bearing, range of motion, muscle massage

Prognosis

- depends on number of larvae ingested
- poor if recumbent (10 to 20% of down llamas recover)
- fair to good if can stand unaided (75 to 85% improve or recover)
- improvement over weeks to months
- some recover without therapy

Necropsy Findings

- exclude other diseases
- sections of parasite in cord or brain
- linear migration tracks; swollen axons, demyelination
- perivascular infiltrate - lymphocytes, plasma cells, eosinophils
Prevention of exposure – control the host and vector
- exclude deer with double fencing
  - 6-strand electric slopes outward
  - inner netting, 8 ft high
- exclude mollusks
  - cordon sanitaire between fences
  - gravel, limestone, or plowed regularly
  - molluskicides (contaminate water)

Prevention of exposure
- do not pasture at edge of woods
- avoid low-lying poorly drained fields
- fence off deer watering spots
- use fields deer prefer for hay, not grazing
- guardian dog may help
- take advantage of hunting season!

Prophylaxis in abnormal host
- no controlled studies
- optimism prevalent
- injectable ivermectin q 4-6 wk to llamas
- pour-ons? (not sheep)
- resistant GI worms will kill S&G instead
- newer avermectins with longer duration?