

Lecture 24 – Caliciviridae (and others)

Russ Fraser, Fall 2023

Housekeeping

- This our second to last lecture
- Friday will cover the same material as this afternoon's case

- Next Tuesday: Jeopardy! Style review?
- Next Friday: SOTS

- Final Exam:
  - Group B first
  - Group A second

Caliciviridae: general

Size	27 – 40 nm
Morphology	Naked (non-enveloped)
	• Icosahedral symmetry • “Cup-shaped” depressions (calici = chalice)
Genome	Linear, ssRNA, ~ 7.4 – 8.3 kb
Site of Replication	• Cytoplasm
Properties	• Very large family of viruses • Resistant to lipid-solvents/detergents • Susceptible to household bleach • Contains the genus *Norovirus*, very important cause of GI illness in humans • Culture and isolation of some caliciviruses has been difficult, making experimental studies challenging

Caliciviridae: important viruses

- Feline calicivirus
- Rabbit hemorrhagic disease virus 1 and 2
- Vesicular exanthema virus





Feline calicivirus

Feline calicivirus: general

- Widespread, many strains exist with marked variation in pathogenicity
  - Cross-protection is variable
- Infects Felidae
- Common cause of upper respiratory and oral disease
  - Less frequently, arthritis
  - Rarely, virulent systemic disease





Feline calicivirus: transmission

- Virus shed in **oral**, nasal, and ocular secretions
- Direct contact between cats is the primary mode of transmission
- ~10 % of animals become asymptomatic long-term carriers (and shedders) of the virus
  - Mechanism of persistence is unclear
  - No latent phase

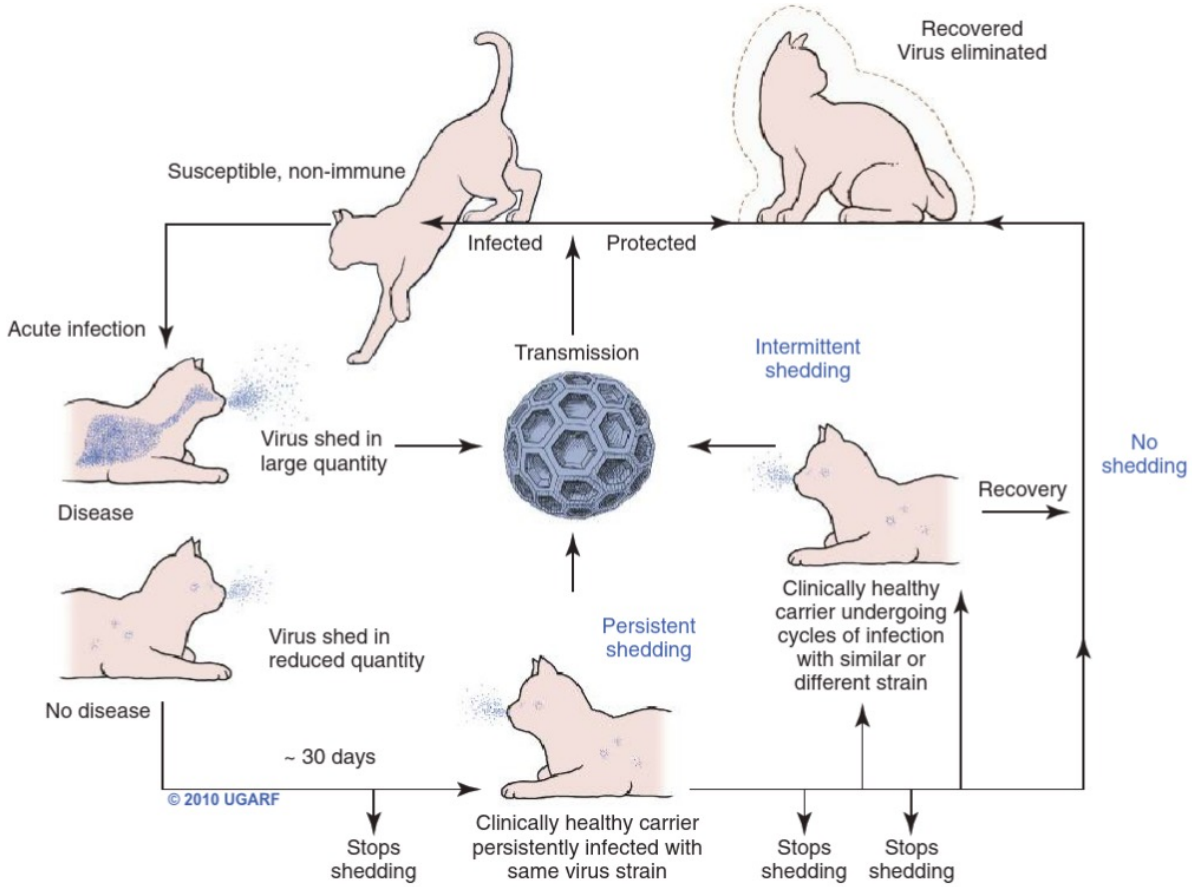

FIG. 14-2 FCV carrier state: epidemiology. (Art by Brad Gilleland © 2010, University of Georgia Research Foundation Inc.)

Feline calicivirus: transmission

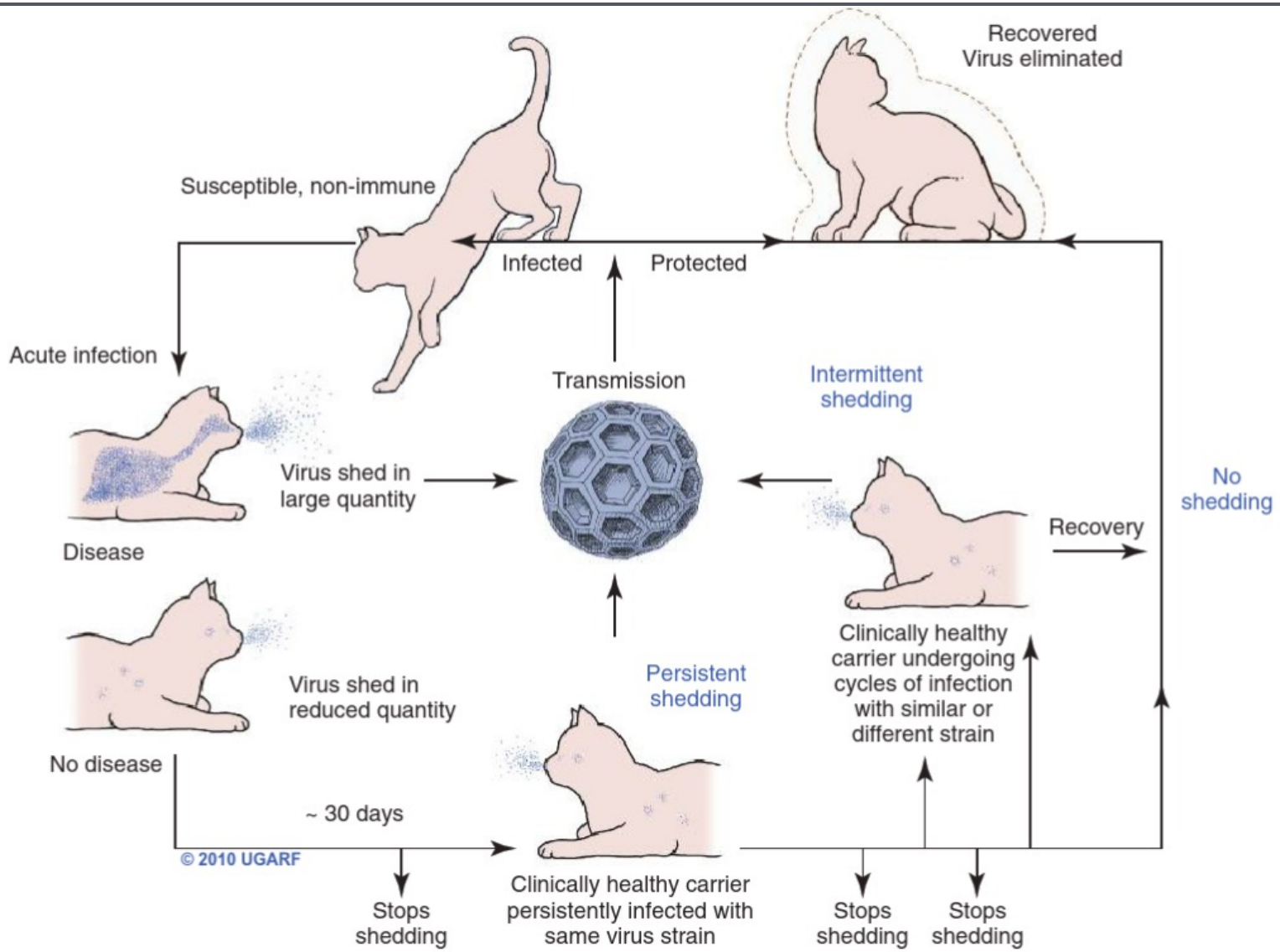

FIG. 14-2 FCV carrier state: epidemiology. (Art by Brad Gilleland © 2010, University of Georgia Research Foundation Inc.)

Feline calicivirus: pathogenesis

Oral, nasal, conjunctival exposure

↓

Local replication in epithelium

↓

Oral vesicles ← Rarely seen

↓

Ulcers (most characteristic lesion)

Feline calicivirus: clinical signs and gross lesions

- Vary depending on strain

- Oral, particularly lingual, ulcers are common
- Ocular and nasal discharge

- Dyspnea (rarely, associated with pneumonia)
- Lameness (rarely, associated with arthritis)

- Virulent systemic disease:
  - Pyrexia, cutaneous edema, ulcerative dermatitis, jaundice
  - Death in ~50 % of animals
  - Arises in an explosive, usually small (~ 100 cats) outbreak, then fades away



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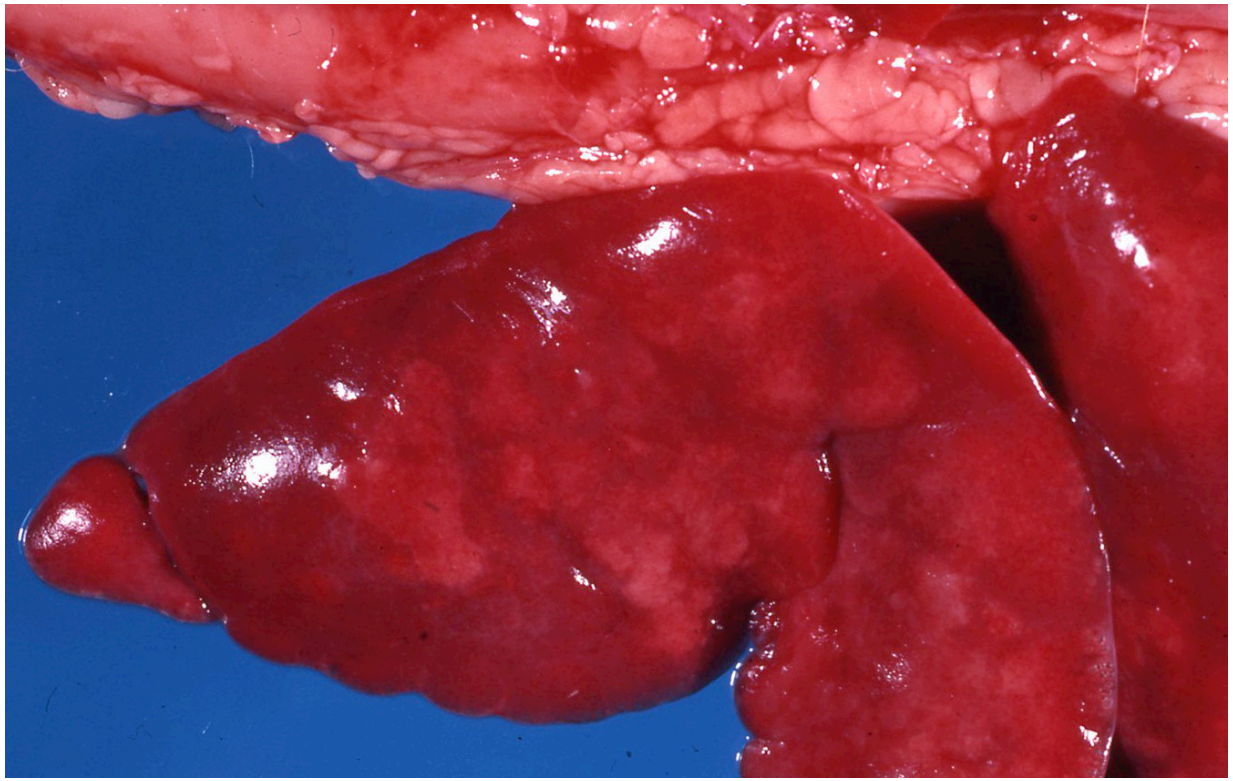

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Feline calicivirus: diagnosis and control

- DDx:
  - Feline herpesvirus, (*Bordatella*, *Chlamydia*)
- Diagnosis
  - History, PCR, virus isolation
- Control
  - Vaccination is widespread
  - Core vaccine
    - Protects against disease, but not against infection. Vaccinated animals can become long-term carriers.
    - Lameness is an uncommon side-effect of vaccination.

TABLE 14-1

Essential Features of Clinical Respiratory Disease[a] Related to the Pathogen Involved

Feature	FHV-1	FCV[b]	Bb	ChF
Lethargy	+++	+	+	+
Sneezing	+++	+	++	+
Conjunctivitis	++	+	–	+++[c]
Hypersalivation	++	–[d]	–	–
Ocular discharge	+++	+	(+)	+++
Nasal discharge	+++	+	++	+
Oral ulceration	(+)	+++	–	–
Keratitis	+	–	–	–
Coughing	(+)	–	++	–
Pneumonia	(+)	(+)	+	+/–
Lameness	–	+	–	–

+++, Marked; ++, moderate; +, mild; (+), less common; +/–, subclinical; –, absent; *Bb, Bordetella bronchiseptica; ChF, Chlamydophila felis*; *FCV*, feline calicivirus; *FHV-1*, feline herpesvirus.
[a]For FCV-associated virulent systemic disease signs, see text.
[b]Strain variation.
[c]Often persistent.
[d]Slight wetness may be seen around the mouth if ulcers present.
Adapted from Ref. 50. Used with permission.

Greene's Infectious Disease of the Dog and Cat

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FYI

Recommendations to Prevent the Spread of the Respiratory Viruses in a Boarding Cattery	Feline Respiratory Disease Control Program in a Breeding Cattery with Endemic Disease
Admit only fully vaccinated cats, preferably revaccinated within a year before admission. House cats individually with solid partitions between pens, unless cats are from same household. Ensure that frontages are at least 1 meter apart. Put known carriers or cats with history of respiratory disease in a separate section, or at least at one end of the cattery, and clean, feed, and water them last. Ensure that surfaces of pen are easily washable and that food and litter bowls can be easily removed without entering the pen (i.e., do not handle cats more than necessary). Feed cats in same order each day and attend to each pen completely before moving on to the next. Either disinfect hands between each pen or have individual rubber gloves for each pen, for use only with that pen. Disinfect gloves thoroughly before use with a new boarder. Alternatively, use disposable gloves. Wear rubber boots and step into a disinfectant bath if it is necessary to enter a pen. Either use disposable food trays or have two sets of food bowls used on alternate days. Soak used set in a 1:30 household bleach (5.25% sodium hypochlorite) solution with detergent for several hours and then rinse and allow to dry until reuse 24 hours later. Prepare food in a central area. Use similar system to food bowls for litter trays. When a cat goes home, thoroughly disinfect cage, allow to dry, and preferably leave empty for 2 days before reusing. Reduce concentration of virus in the environment by providing adequate ventilation, low relative humidity, and optimal environmental temperatures.	Provide regular vaccination programs. Give booster vaccinations to queens either before mating or during pregnancy (inactivated vaccine only, but only if licensed for this use). Keep cats as stress-free as possible, and use good management practices to reduce spread of viruses within the colony. Avoid breeding queens with a history of oral or respiratory disease in their kittens. Move queens into isolation at least 3 weeks before term so that the kittens are not exposed to carriers in the colony and that any shedding episode from the queen, as a result of the move, will end before parturition. Wean kittens into isolation away from the queen as soon as feasible (ideally at 4 to 5 weeks before MDAs wane, if it is likely she is a carrier). Vaccinate all kittens as soon as MDAs are at a noninterfering level (normally 9 or more weeks), and keep them in strict isolation until a week after the second dose (normally at 12 weeks, but in some cases after a third vaccination 3 to 4 weeks later). Earlier FHV-1 and FCV vaccination schedules with intranasal (if available) or parenteral vaccines may be used, although these are not always licensed for early use and should be used with care. Parenteral vaccines can be given from 6 weeks of age, at 3-week intervals until 12 weeks, and if indicated, up to 15 to 16 weeks of age. Intranasal vaccination should be carried out 7 to 10 days or so before disease has been occurring and then again at 12 weeks. Some evidence indicates that multiple doses are not necessary, although they were originally advocated.

Adapted from Ref. 50. Used with permission.

FCV, Feline calicivirus; *FHV-1*, feline herpesvirus-1; *MDAs*, maternally derived antibodies.



Rabbit hemorrhagic disease virus

RHDV: general

- RHDV-1 and 2
  - RHDV-2 is becoming predominant
  - RHDV-2 is the most recent form detected in NA
- Highly contagious
- Highly fatal
  - Lower fatality with RHDV-2?
- Wild and domestic European rabbits
  - Most commercial and domestic rabbits
  - RHDV-2 → at least some native NA rabbits
- Introduced into Australia to study its effectiveness as a biological control agent for wild rabbits (considered pests)
  - Escaped quarantine
  - Killed 60 % of rabbits nationwide



RHDV: transmission and pathogenesis

- Virus is present in all secretions and excretions
- Direct contact with live or dead rabbits and exposure of oral, nasal, conjunctival surfaces
- Insects/carnivores/raptors as mechanical vectors

- Animals < 30 days old do **NOT** develop disease
  - Not sure why – lack of appropriate cellular receptor for viral entry?
  - Differential innate immune response?

<u>Pathogenesis is poorly described</u>

Infection of liver

↓

Necrosis

↓

DIC

RHDV: clinical signs and gross lesions

- Peracute disease:
  - Fever, sudden death within 24-36 hrs
- Acute:
  - Dullness, anorexia, apathy, depression, death
  - ± neurological signs
  - ± dyspnea, cyanosis, epistaxis
  - ± death within days
- Subacute (uncommon):
  - Marked jaundice
  - Weight loss, lethargy
  - ± death within weeks



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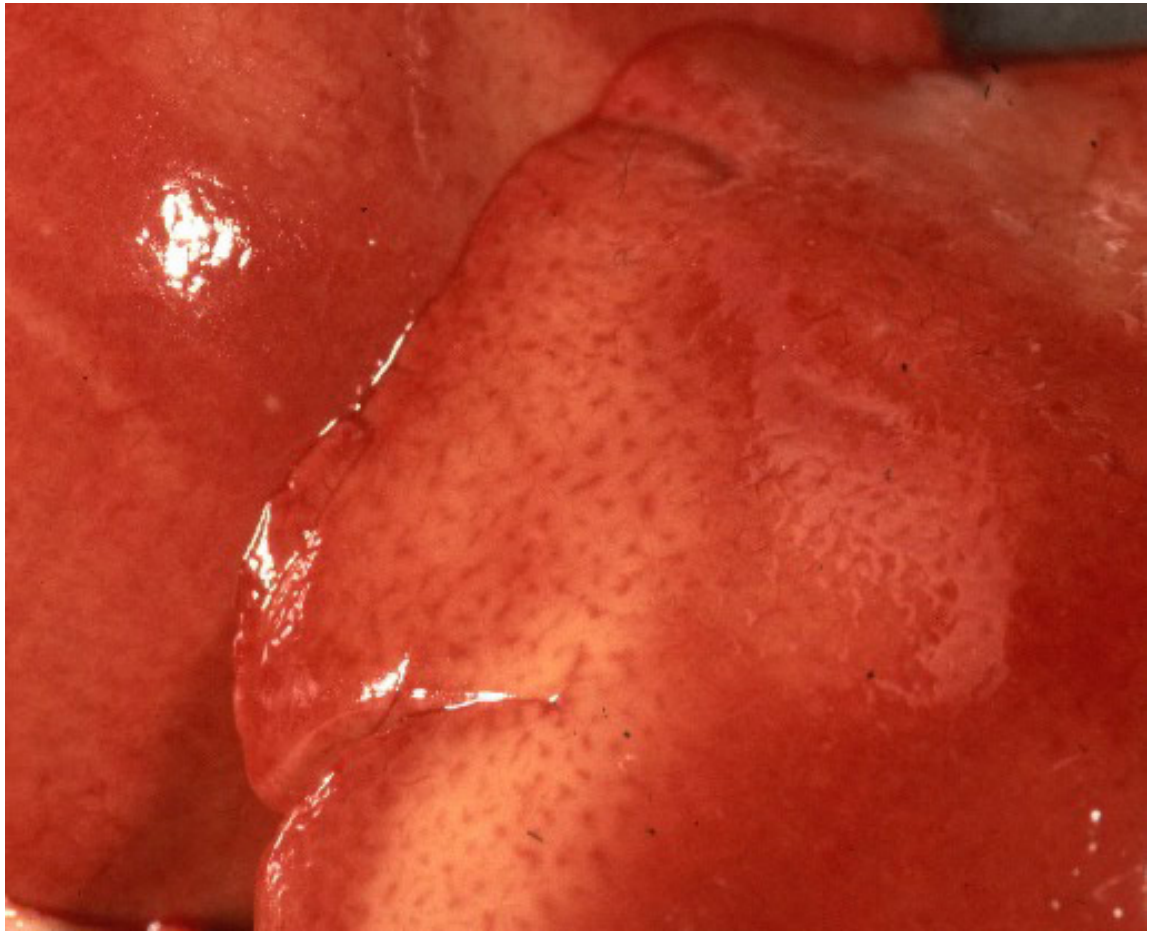


RHDV: diagnosis and control

- Diagnosis:
  - PCR, IHC on liver, spleen

- Control:
  - Good management
  - Vaccines are available
    - For RHDV-1 and -2
    - Infection with RHDV-1 does not protect against RHDV-2
  - Genetic diversity poses a challenge



Vesicular exanthema of swine (San Miguel sealion) virus

History of Vesicular exanthema of swine (VES) disease in USA:

- 1932 - A vesicular disease occurred in swine in Buena Park, California, – thought to be FMD: 19,000 pigs killed & buried (remember last confirmed outbreak of FMD in USA was in 1929).

- 1933 - Disease occurred in swine in San Diego County – shown not to be FMD; Disease was named Vesicular Exanthema of swine.

- 1932-1936 - 10 more outbreaks of VES occurred in California, and each was eradicated.

- 1936-1939 - No VES outbreaks.

- 1936-1952 - VES spread to all major swine-producing areas in USA. It is believed that spread from California occurred through garbage carried on a train running between California and Chicago, and by virus getting into a Hog Cholera antiserum production plant in Nebraska.

- 1954 - National VES eradication program was started; enforcement of cooking raw garbage fed to pigs.

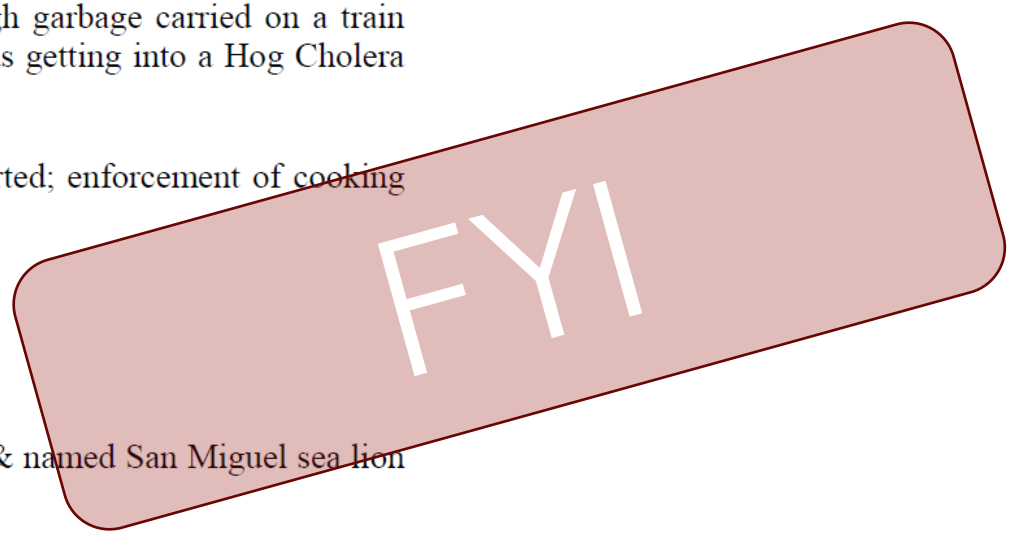
- 1956 - Last VES case recorded

- 1959 - USA declared free of VES.

- 1972 - Virus was recognized in marine mammals (& named San Miguel sea lion virus) and fish in Pacific Ocean.

- 1972-1995 At least 14 serotypes of San Miguel sea lion virus recognized.

In retrospect, the source of infection in the first recognized disease outbreak in Swine in 1932 in Southern California was marine mammals, California sea lions on San Miguel island (hence San Miguel sea lion virus).



Vesicular exanthema of swine (San Miguel sealion) virus

- VES virus (13 serotypes) & San Miguel sea lion virus (17 serotypes) are very closely related (San Miguel sea lion virus can cause lesions of VE).

- Mostly a 'historical curiosity'

- Disease was only seen in swine in continental USA, Hawaii, & Iceland.
  - It is now extinct (last case in USA was in 1956) but virus is still present in marine mammals (California sea lions) in Pacific Ocean
  - Eradication of the disease in pigs in 1959 was possible through:
    - Instituting a slaughter program, &
    - Enforcement of laws requiring cooking raw garbage prior to its being fed to pigs in 1954.

- <u>Disease is important because of clinical resemblance with FMD, VS, and SVD in swine</u>

Vesicular exanthema of swine (San Miguel sealion) virus

- Clinical signs in swine are same as for FMD
- Piglets & sea lions may develop encephalitis or myocarditis (as in SVD)
- Control methods would be similar to those instituted for other vesicular diseases

	Reportable	Disease in pigs	Disease in horses	Disease in cattle
Food and mouth disease	Yes	Yes	No	Yes
Vesicular stomatitis	Yes	Yes	Yes	Yes
Swine vesicular disease	Yes	Yes	No	No
Vesicular exanthema	No	Yes	No	No
Senecavalley A virus	No	Yes	No	No

Vesicular exanthema of swine (San Miguel sealion) virus

Vesicular Diseases Reference Chart

	Foot-and-Mouth Disease	Vesicular Stomatitis	Swine Vesicular Disease	Vesicular Exanthema of Swine
Etiology	Aphthovirus	Vesiculovirus	Enterovirus	Calicivirus
Geographic Distribution	Endemic in Asia, Africa, Middle East, parts of South America; U.S. free since 1929	North & Central America, northern South America	Many European countries	U.S. only (eradicated in 1956)
Transmission	Respiratory aerosols; oral consumption; direct and indirect (fomite) contact	Insect vectors (sand flies & black flies); contact, aerosol in humans	Ingestion of contaminated meat; contact with animals, feces	Ingestion of uncooked garbage contaminated with pork
Incubation Period	Ingestion 1-3 days, Exposure 3-5 days	Animals 3-5 (up to 21) days, Humans 24-48 hours	Ingestion 2-3 days, Exposure 2-7 days	18-72 hours
Clinical Signs by Species	**All vesicular diseases produce a fever with vesicles that progress to erosions in the mouth, nares, muzzle, teats, and feet. These 4 diseases are clinically indistiguishable from each other, particularly in swine.**			
Notification	**State & Federal Veterinarians should be contacted IMMEDIATELY and informed of suspicions**			
	Disease Indicators			

VESV/SMSLV: gross lesions in marine mammals





FYI


Astroviridae

Astroviruses

- A virus family that is starting to be increasingly examined.

- Gastrointestinal disease in young animals (differential for rotavirus, coronavirus)

- Neurologic disease
  - Cattle, pigs
  - Non-suppurative polioencephalomyelitis
  - Causation has not been demonstrated

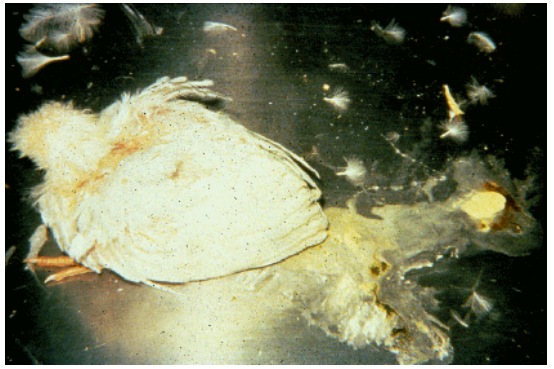




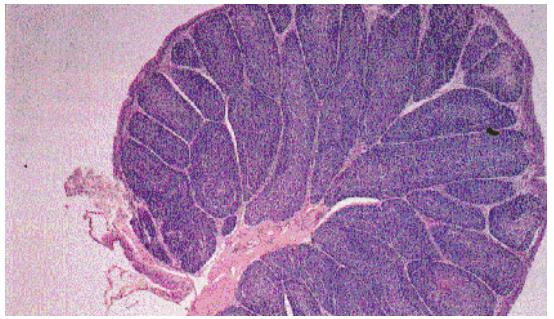
Birnaviridae

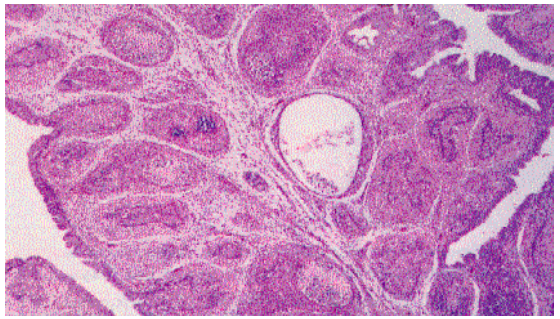
Infectious bursal disease virus

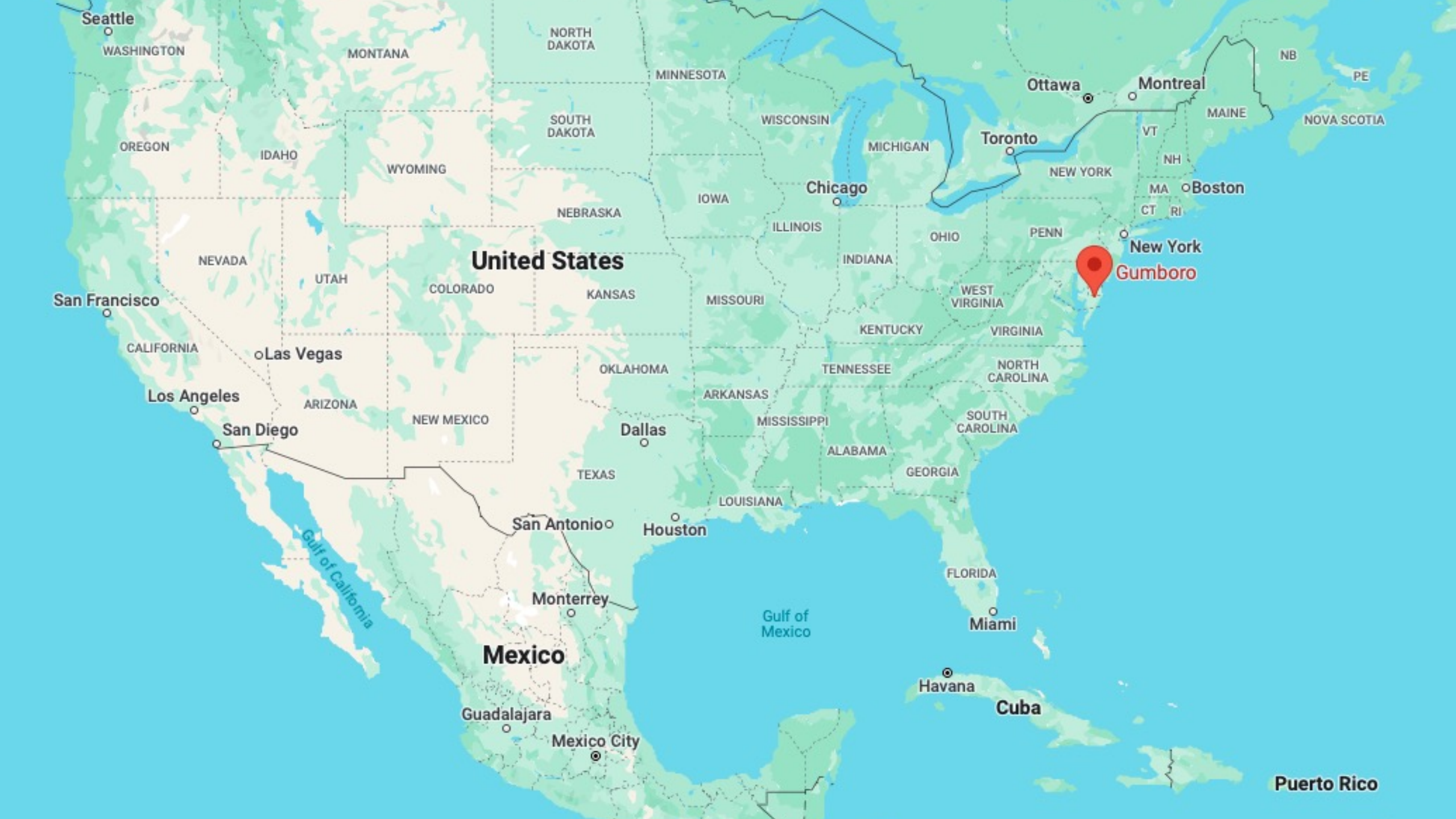
- Causes infectious bursal disease or Gumboro disease.
- Transmission: fecal-oral route.
- Target is the Bursa of Fabricius
  - Destruction of pre-B lymphocytes → immunosuppression
- Disease is most severe in chickens 3 – 6 weeks:
  - Distress, ruffled feathers
  - Diarrhea
  - Hemorrhage in skeletal muscles
  - Death (up to 30% mortality, but may be higher with very virulent strains).
- Grossly, bursal swelling followed by atrophy (lymphoid depletion)
- Animals that survive have ↑ susceptibility to other infectious agents
- Antibody production following disease is ↓







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FLORIDA
Monterrey
Gulf of Mexico
Miami
Mexico
Havana
Cuba
Guadalajara
Mexico City
Puerto Rico

Infectious bursal disease virus

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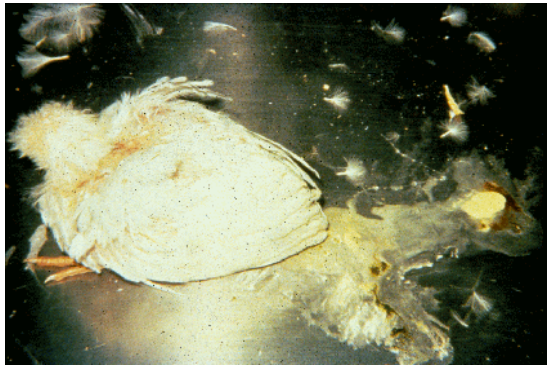
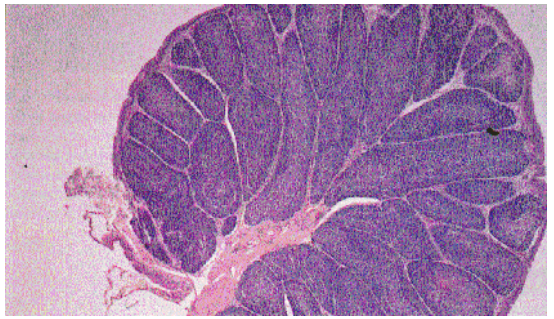
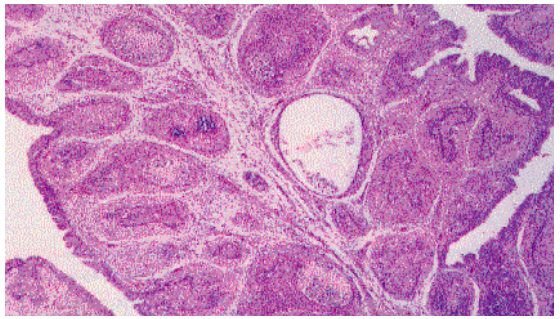


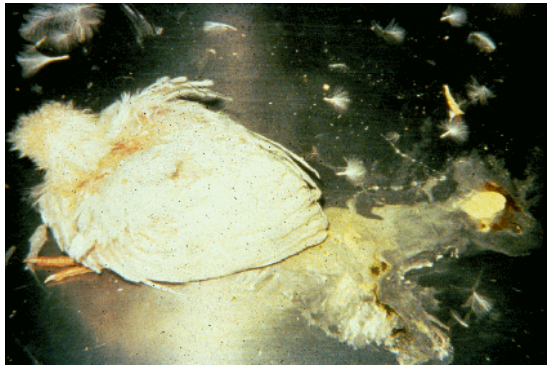




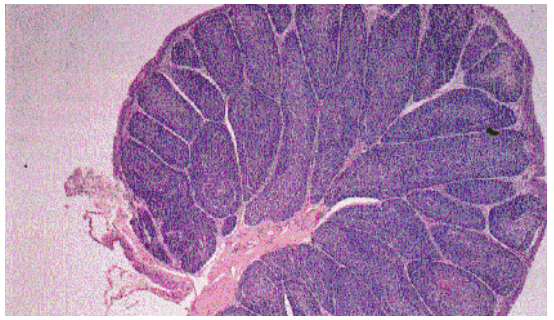


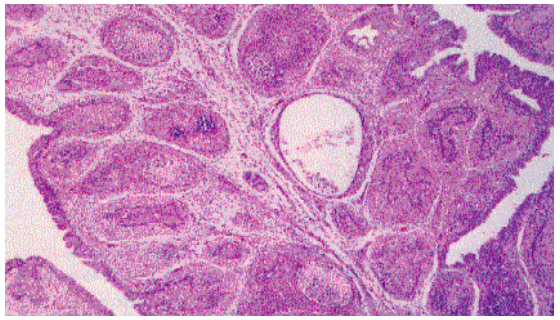
Infectious bursal disease virus

- Control:
  - Vaccination of breeding stock with attenuated vaccines
    - Provides maternal antibody
  - There is virus/strain variation and cross-protection is not complete










Peribunyaviridae

Peribunyaviridae

Foreign

Akabane virus
Schmallenburg virus

California encephalitis virus
Cache valley virus

In North America

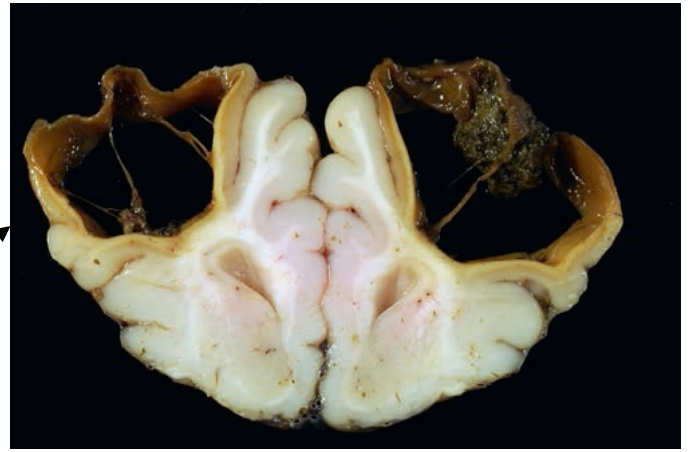
Arboviruses

Birth defects in ruminants

Arthrogryposis

Hydranencephaly

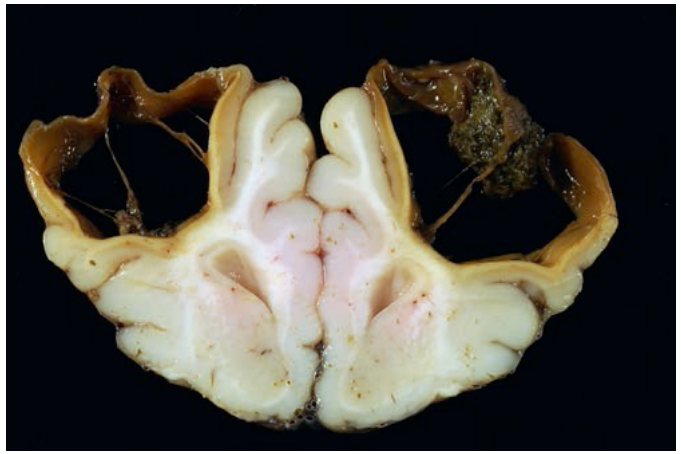




Porencephaly

Phenuiviridae

★Rift valley fever virus



Porencephaly

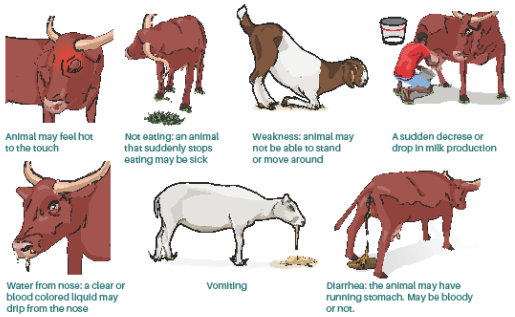




SIGNS OF A RIFT VALLEY FEVER OUTBREAK

- Many unexpected pregnancy losses in goats, sheep, and cattle
- Stillbirths and births of weak animals
- Illness and death of young livestock (less than one year of age)

SYMPTOMS OF RVF IN ANIMALS



Once animals recover from Rift Valley Fever, they are no longer able to infect people.

RVF IN PEOPLE

Those at greatest risk of getting Rift Valley Fever are people with contact with sick animals, including:

- Animal health workers
- Herders, other people who take care of sick animals
- Abattoir workers and people involved in slaughtering sick animals
- People who may touch the bodies of sick or dead animals
- Those who may handle uncooked meat or drink raw milk of sick animals

Typically, people infected with Rift Valley Fever recover 2-7 days after mild illness; however, a small number of people develop much more severe symptoms.

Signs of serious illness in people include: vomiting, diarrhea, muscle or joint pain, intense fatigue, abdominal pain, and unexplained bleeding.

One person cannot infect another person with Rift Valley Fever.

Seeking care early is an important way to help your chances of survival if you become seriously ill. Although there is no cure for RVF, there is supportive care, such as transfusions and IV fluids that can help save lives.

WHEN ANIMALS OR FAMILY MEMBERS ARE SICK:

If you notice an abnormal number of spontaneous abortions, stillbirths or increased numbers of of premature deaths in animals, report it to veterinary services for coordination and reporting of testing, and, if necessary, inform the Ministry of Health.

Contact: _____



When testing and drawing samples from animals, veterinarians should follow the recommendations below. To avoid contact live with bodily fluids sick or dead animals, or products of abortion, veterinarians should wear gloves, boots, long sleeves, and a face shield to protect against splashing.

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Phenuiviridae



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SYMPTOMS OF RVF IN ANIMALS



Animal may feel hot to the touch



Not eating: an animal that suddenly stops eating may be sick



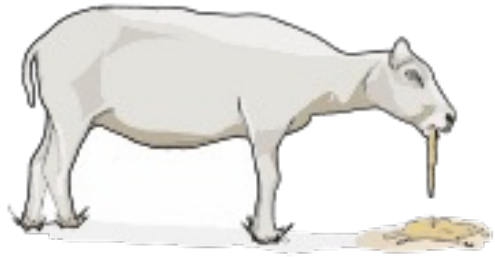
Weakness: animal may not be able to stand or move around



A sudden decrese or drop in milk production



Water from nose: a clear or blood colored liquid may drip from the nose



Vomiting



Diarrhea: the animal may have running stomach. May be bloody or not.

Once animals recover from Rift Valley Fever, they are no longer able to infect people.

WHEN ANIMALS OR FAMILY MEMBERS ARE SICK:

If you notice an abnormal number of spontaneous abortions, stillbirths or increased numbers of of premature deaths in animals, report it to veterinary services for coordination and reporting of testing, and, if necessary, inform the Ministry of Health.

Contact: _____



Once animals recover from Rift Valley Fever, they are no longer able to infect people.

RVF IN PEOPLE

Those at greatest risk of getting Rift Valley Fever are people with contact with sick animals, including:

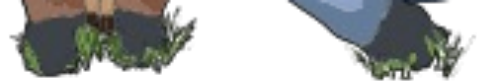
- Animal health workers
- Herders, other people who take care of sick animals
- Abattoir workers and people involved in slaughtering sick animals
- People who may touch the bodies of sick or dead animals
- Those who may handle uncooked meat or drink raw milk of sick animals

Typically, people infected with Rift Valley Fever recover 2-7 days after mild illness; however, a small number of people develop much more severe symptoms.

Signs of serious illness in people include: vomiting, diarrhea, muscle or joint pain, intense fatigue, abdominal pain, and unexplained bleeding.

One person cannot infect another person with Rift Valley Fever.

Seeking care early is an important way to help your chances of survival if you become seriously ill. Although there is no cure for RVF, there is supportive care, such as transfusions and IV fluids that can help save lives.



When testing and drawing samples from animals, veterinarians should follow the recommendations below. To avoid contact live with bodily fluids sick or dead animals, or products of abortion, veterinarians should wear gloves, boots, long sleeves, and a face shield to protect against splashing.

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

CS265393D



Bornaviridae

https://www.azuloxworkshops.com/wp-content/uploads/2018/02/Chasing-Macaws-Manuel-Antonio-002.jpg

Bornaviridae: PaBV

- Psittaciform bornavirus (PaBV), causative agent of proventricular dilation disease, is the most important virus in this family.
  - **Psittacines** and other bird species

- Epidemiology and transmission are poorly described
  - Natural host? Waterfowl?
  - Transmission? Feco-oral is considered most likely; vertical transmission considered possible

Psittaciform bornavirus: clinical signs

- Many birds will be infected (and have antibodies) but will not be clinically ill
- Clinical signs relate to progressive neurological and/or gastrointestinal tract dysfunction
  - Weight loss
  - Dysphagia
  - Regurgitation
  - Ataxia
  - Proprioceptive deficits
  - Death is inevitable
- Gross: emaciation with atrophy of pectoral muscles and dilatation of the crop, proventriculus, ventriculus, and small intestine



Hoppes SM, Shivaprasad HL. Update on Avian Bornavirus and Proventricular Dilatation Disease: Diagnostics, Pathology, Prevalence, and Control. Vet Clin North Am Exot Anim Pract. 2020 May;23(2):337-351. doi: 10.1016/j.cvex.2020.01.006. PMID: 32327040.

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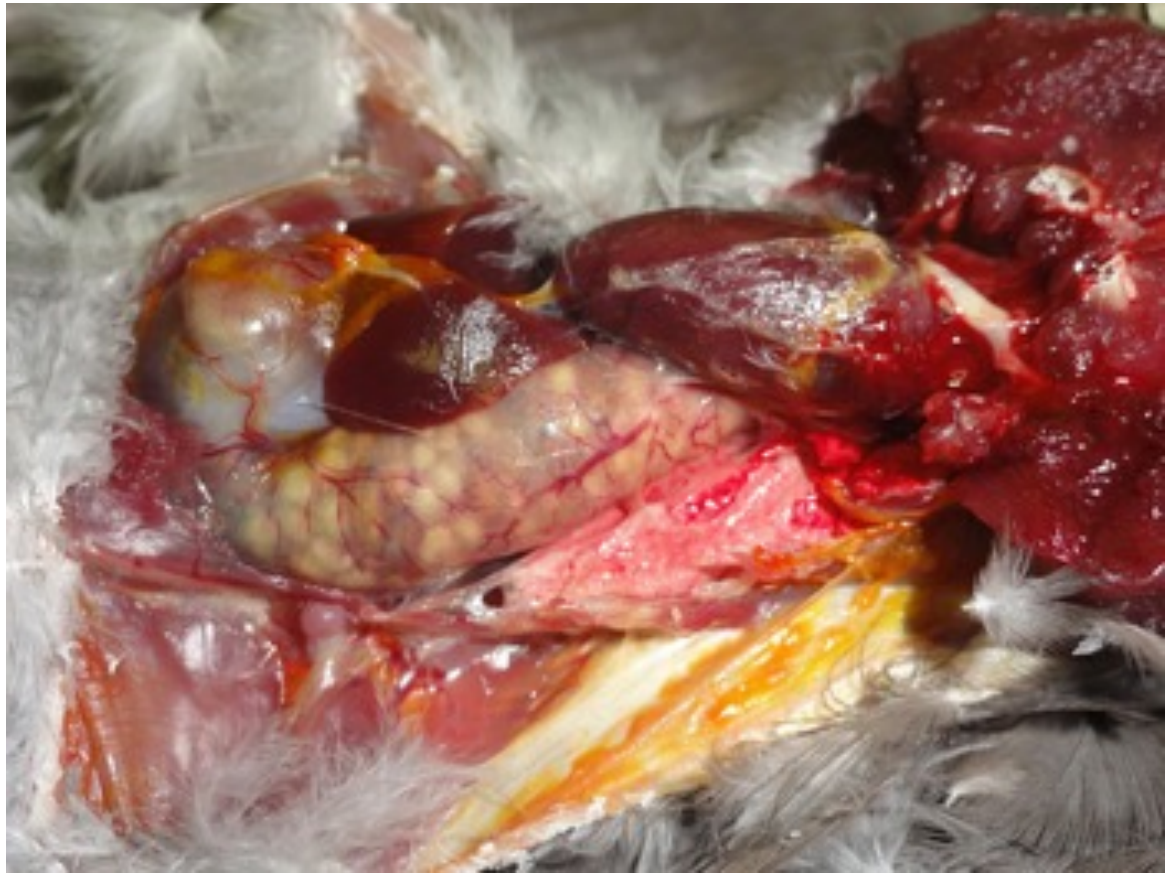
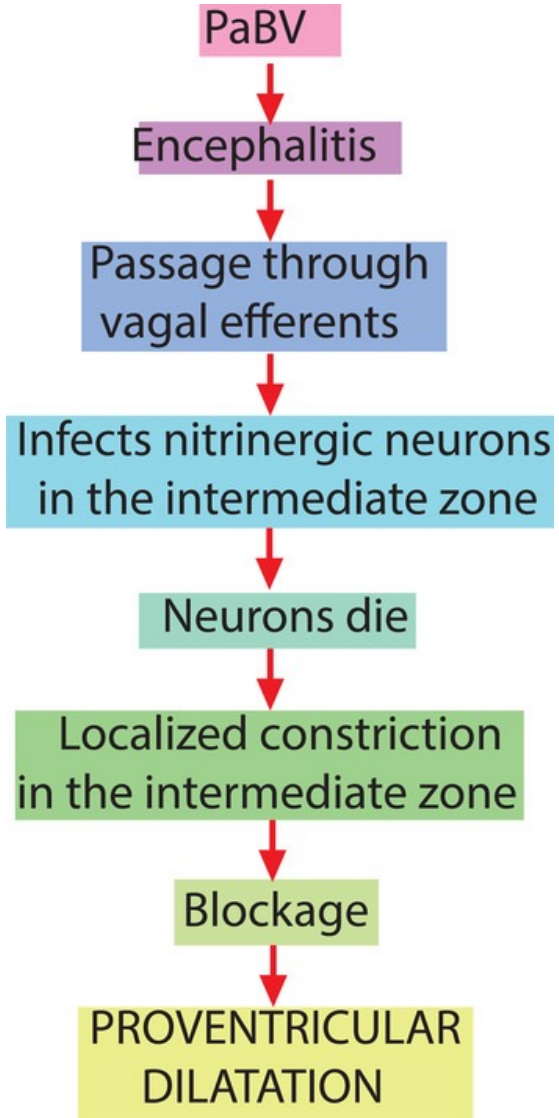


Tizard, I., Shivaprasad, H., Guo, J., Hameed, S., Ball, J., & Payne, S. (2016). The pathogenesis of proventricular dilatation disease. *Animal Health Research Reviews, 17*(2), 110-126. doi:10.1017/S1466252316000189

Psittaciform bornavirus : pathogenesis

- Complicated and controversial.

- Proposed pathogenesis:



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Psittaciform bornavirus : diagnosis

Recommended Diagnostics and Interpretation

In a clinical setting, the following tests are recommended[1,45]:

- A choanal and cloacal RT-PCR with a serologic assay offers the best combination to determine infection, recognizing that one negative test on all does not rule out infection.

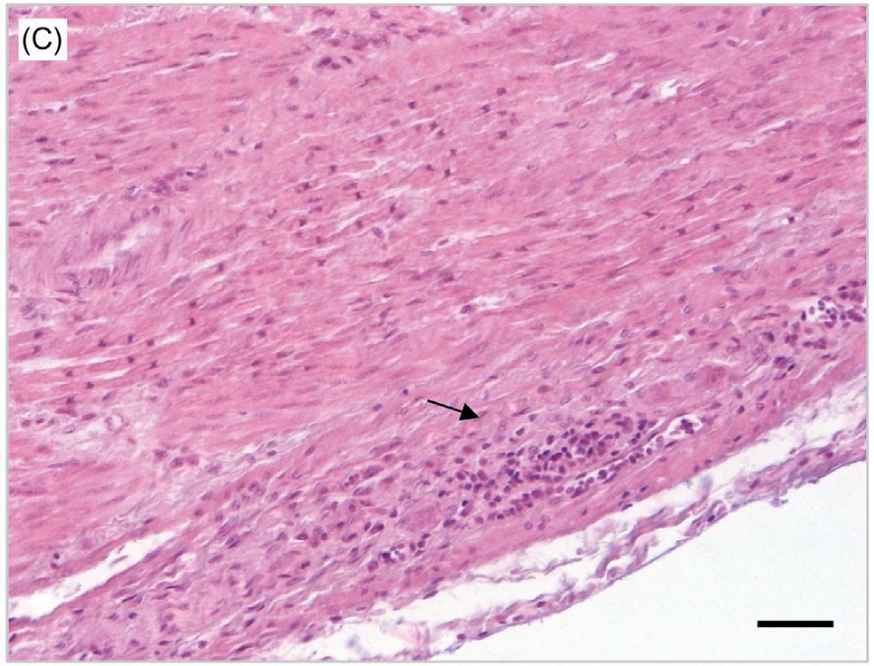
Interpretation of tests

- Birds positive for both PCR and serology should be considered positive and housed separate from negative birds.
- Birds positive for PCR only should be retested in 4 to 6 weeks. They should be housed separate from other birds until deemed negative (3 negative tests). If they seroconvert or test positive on additional tests they should be considered positive.
- Birds positive only on serology should be considered positive and housed separately from negative birds.
- Some birds are infected and will not test positive on any test, and infection is found at necropsy (Sharman Hoppes, personal experience, 2013).

As previously discussed, positive ABV PCR and serology results in a healthy bird do not predict that a bird will ever become clinically ill with PDD.

Hoppes SM, Shivaprasad HL. Update on Avian Bornavirus and Proventricular Dilatation Disease: Diagnostics, Pathology, Prevalence, and Control. Vet Clin North Am Exot Anim Pract. 2020 May;23(2):337-351. doi: 10.1016/j.cvex.2020.01.006. PMID: 32327040.

- Given the difficulty of obtaining a clinical diagnosis, a definitive diagnosis of PDD is often obtained only at necropsy.
  - Histology and IHC



Psittaciform bornavirus : control

- No vaccine

- Biosecurity and hygiene
  - Isolate incoming birds until confirmatory negative
  - Chicks from infected birds should be incubator hatched

- Do not euthanize healthy birds based solely on positive testing

Arenaviridae



Arenaviridae – Boid inclusion body disease-associated virus

- Relatively little known
- Associated with “inclusion body disease”
- Transmission?
  - Vertical?
  - Venereal?
  - Snake mite?
- Clinical signs:
  - Regurgitation
  - CNS: torticollis, paralysis, disequilibrium
  - Stomatitis
  - Pneumonia
- Gross lesions are not distinctive
- Histologically:
  - Eosinophilic cytoplasmic inclusions in a variety of organs

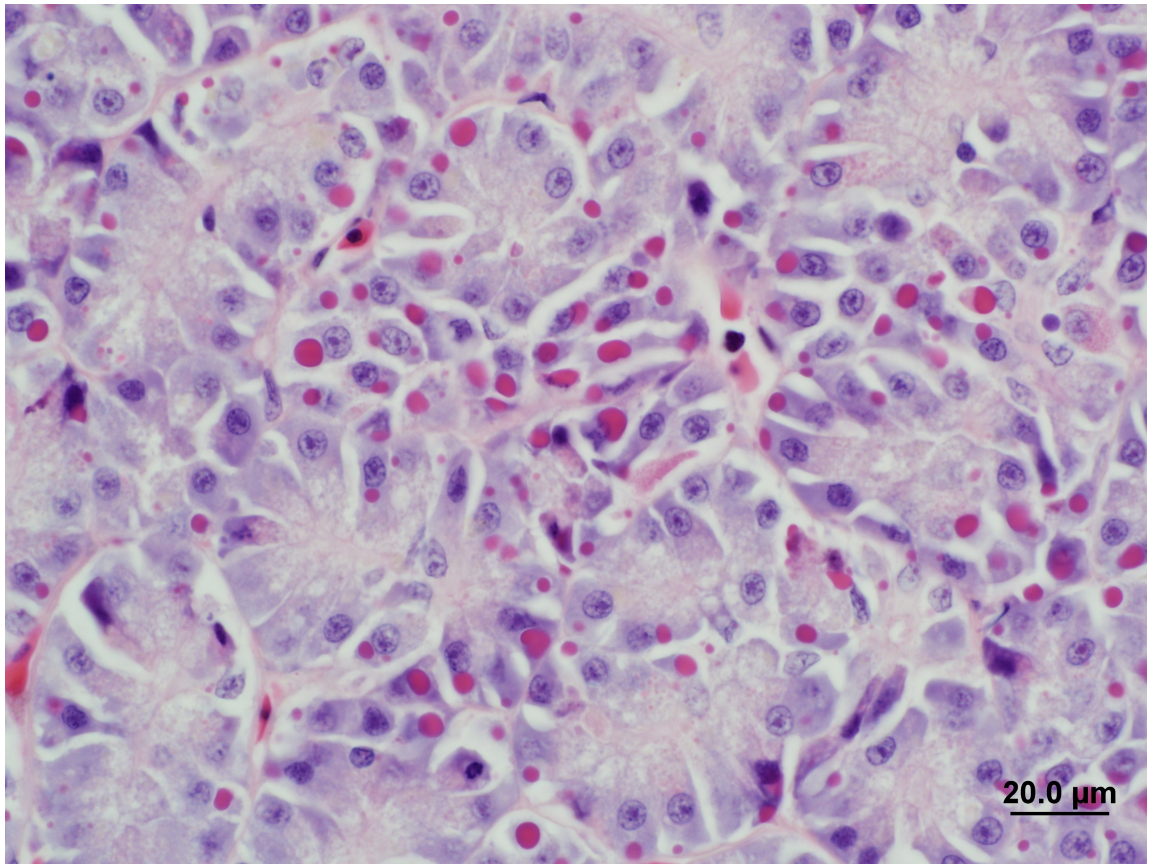


Photo: Shannon Martinson

Arenaviridae

- Lymphocytic choriomeningitis virus infection
  - Lifelong, usually asymptomatic infection of mice
  - Vertical transmission maintains the virus
    - Infection *in utero* results in a persistently infected mouse
  - Hamsters may develop persistent infections at any point
  - In humans:
    - Fever, headache, myalgia
    - Meningitis (uncommon)
    - Encephalomyelitis (rare)

- Several viruses in rodents that are zoonotic and cause highly fatal hemorrhagic fevers in humans