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Lecture 9: Circoviridae and Anneloviridae

Russ Fraser, Fall 2023

Questions?

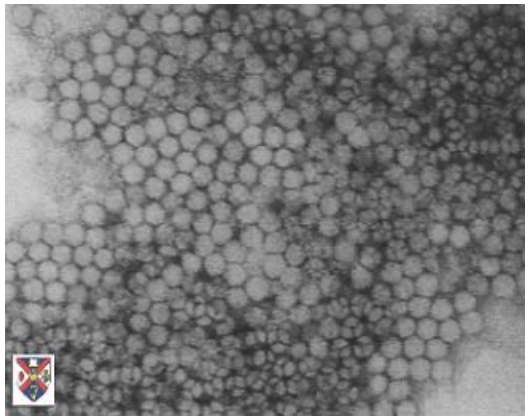


Circoviridae

Figure credit: Jamie Hanson / Newspix / Rex USA

Circoviridae: General characteristics (p 24)

Size	Smallest virus, 12 – 25 nm
Morphology	Naked
	Icosahedral
Genome	Circular, ssDNA
Site of Replication	• Nucleus • Requires S phase (active division)
Properties	• Very stable in the environment • Intranuclear AND intracytoplasmic bodies



Circoviridae: diseases (pages 24-25)

- Family Circoviridae contains 2 genera:
  - Genus: Circovirus
    - ★ Porcine circovirus (PCV) (types 1, 2, 3, & 4)
    - ★ (Psittacine) beak & feather disease virus (BFDV)
    - Pigeon circovirus
    - Canine circovirus
    - Mink circovirus, etc.
  - Genus: Cyclovirus (new)
    - Human, chimpanzee, bovine & goat associated cycloviruses, etc.
- The genus Gyrovirus (containing Chicken anaemia virus (CAV)) was moved to family Anelloviridae
  - Has similar viral characteristics (size, genome, etc)



(Psittacine) beak and feather disease virus

a.k.a. French molt, beak and feather disease virus

BFDV: host range and transmission

- Infects > 60 New World, Old World, and South Pacific psittacines
  - Cockatoos especially
  - Often young birds (most common during first feather formation)
- Transmitted via aerosolized viral particles, feco-oral, and ingestion of contaminated materials
- Is a chronic disease in most birds.
  - May live for years without feathers
  - Secondary infections become common
- Can be lethal in very young birds.



BFDV: pathogenesis & clinical signs

Tropism for rapidly dividing cells of the feather follicles, beak, and claw as well as lymphoid tissues.

↓

Necrosis & dystrophy of these tissues

↓

Symmetrical feather loss

± beak & claw deformities

↓

Chronically, lymphoid depletion occurs
→ secondary infections



Loss of powder down feathers

BFDV: pathogenesis & clinical signs

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Necrotic, dystrophic feathers which are symmetrically lost

BFDV: pathogenesis & clinical signs

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Beak deformities (not always present – depends on bird species involved)

BFDV: Diagnosis (pages 25-26)

- DDx: Avian polyomavirus, nutritional, behavioural, endocrinopathy
- Suspicious based on clinical signs
- Lab diagnosis:
  - PCR
  - Clinical samples:
    - powder-down feathers
    - Serum
  - Histopath

BFDV: Control (page 26)

- No treatment
  - control secondary infections
- Strict hygiene
  - disinfect with common household bleach
- Good screening protocols
- Lengthy quarantines are used in cockatoo breeding colonies to keep virus out.
- Maintain closed flock or purchase birds only from PBFDV-free flocks and breed birds in quarantine.
- Inactivated vaccine is available in Australia:
  - Vaccinate at 14 days of age, with booster after 1 month.
  - Breeding birds are vaccinated 1 month before breeding to generate maternal antibody in egg yolk.



Porcine circovirus 2

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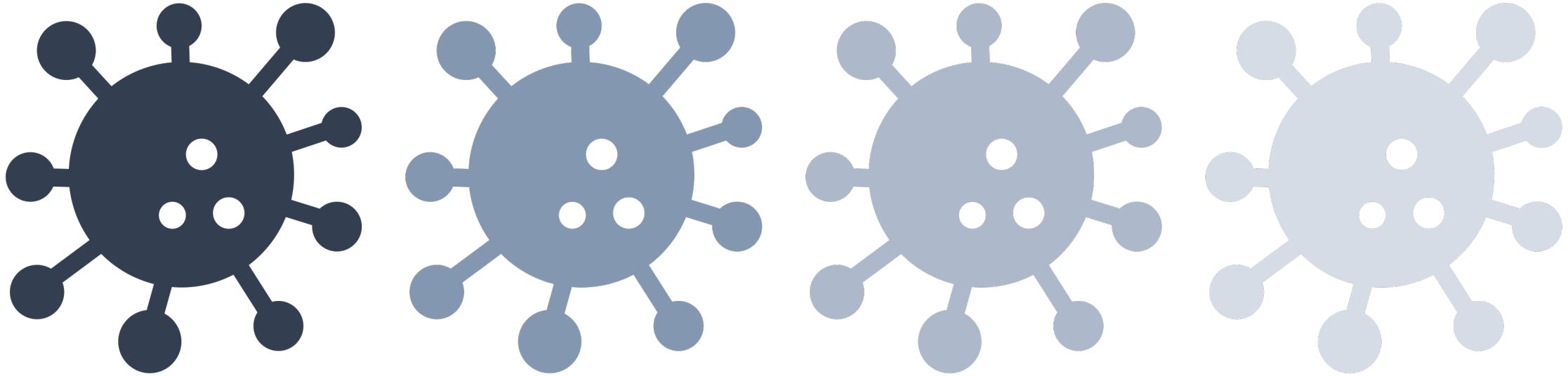
Porcine circovirus: general



PCV1

↓

Non pathogenic

PCV2

↓

Very important

PCV3

↓

Uncertain relevance

PCV4

↓

Uncertain relevance

PCV2: general (pages 26-28)

- Virus occurs in 8 subtypes (PCV-2a to PCV-2h)
  - Types PCV-2a, -2b, and -2d are the most common
- Virus is ubiquitous in global swine population – most herds have anti-PCV2 abs
- Infection >>> disease (disease incidence 4 – 30 %)
  - Other factors (stress, concurrent infection with other agents, e.g., PRRS or parvovirus) contribute to clinical disease.
- A very important disease of swine, causing significant economic loss

PCV2: transmission



- Horizontal via ingestion or inhalation of feces, urine, or other body fluids
- Vertical transmission also occurs

Survival in the environment?

PCV2: clinical presentation

- Often subclinical
- Presents in a variety of different ways:
  - Post-weaning multisystemic wasting syndrome (PMWS)
  - Porcine dermatitis and nephropathy syndrome
  - Reproductive loss
  - Porcine congenital tremor, type A-II (shaker pigs/dancing pigs/congenital trembles).

Porcine circovirus-associated disease (PCVAD)



PCV2: PMWS – clinical signs

- PMWS is systemic PCV-2 infection.
- Characterized by ↓ growth and wasting, respiratory disease, and LN enlargement
- Can cause significant losses in some herds, especially where crowding, poor ventilation & continuous flow management are practiced.
  - Often presents as chronic, insidious, & often protracted disease.
  - Most affected pigs are 6-16 weeks of age (late in nursery or early in grower/finisher).
  - Mortality rate may reach 10% in outbreaks.
  - Disease must be differentiated from Porcine Respiratory & Reproductive Syndrome (PRRS).

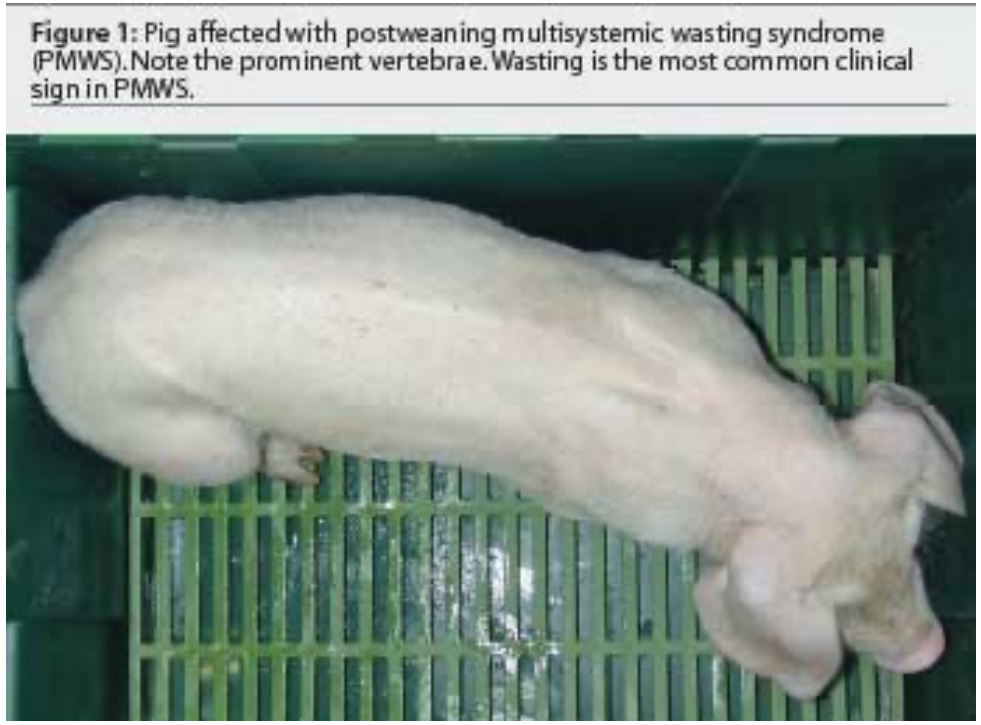


Figure 1: Pig affected with postweaning multisystemic wasting syndrome (PMWS). Note the prominent vertebrae. Wasting is the most common clinical sign in PMWS.

https://www.aasv.org/shap/issues/v10n6/v10n6p277.html

PCV2: PDNS

- Porcine dermatitis and nephropathy syndrome
  - Multifocal necrotizing dermatitis, particularly around the hind limbs and perianal region
  - Kidneys swollen with petechia
  - Histologically, systemic vasculitis and fibrinous glomerulonephritis

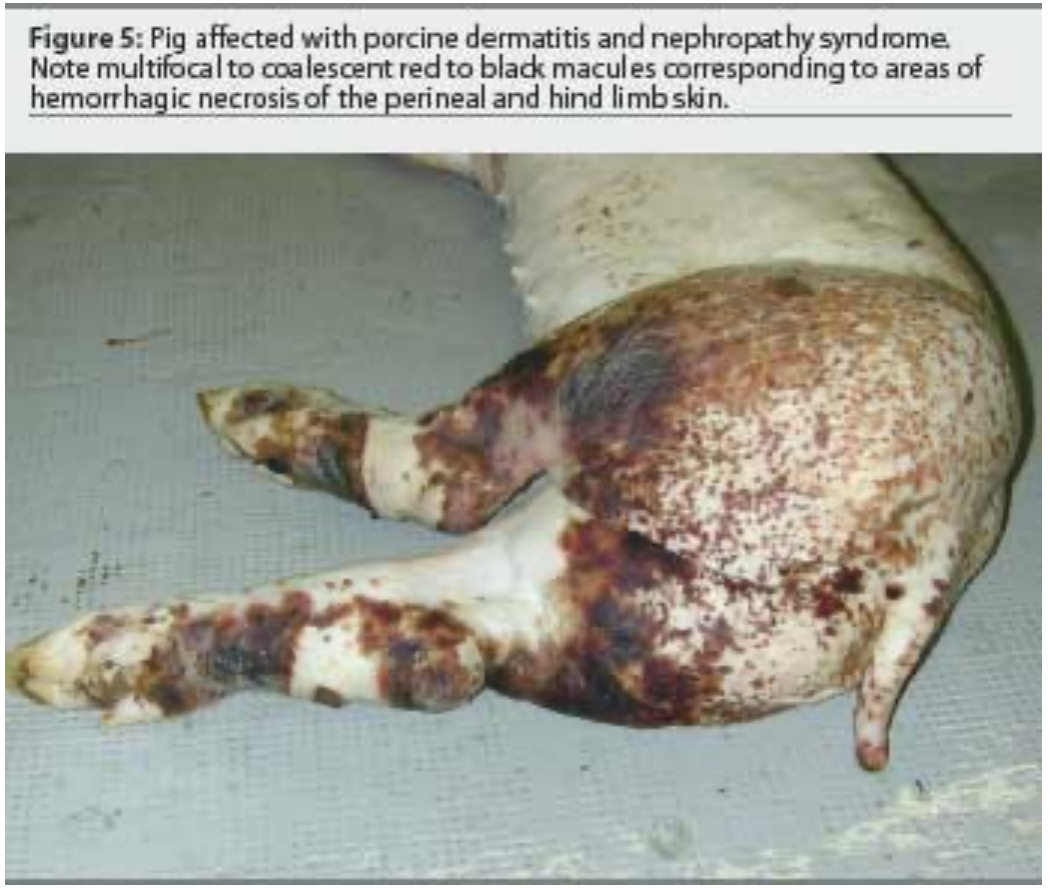


Figure 5: Pig affected with porcine dermatitis and nephropathy syndrome. Note multifocal to coalescent red to black macules corresponding to areas of hemorrhagic necrosis of the perineal and hind limb skin.

PCV2: Pathogenesis



PCV2: Diagnosis



If a virus is present in all animals – some who are clinically affected and others who are not – how do you decide whether the virus is responsible for the disease?

PCV-2: Diagnosis

- Clinical signs
- PCR
- Hallmark microscopic lesions of systemic PCV-2 infection (particularly PMWS):
  - Lymphoid depletion
  - Granulomatous lymphadenitis
  - Large multiple basophilic intracytoplasmic inclusion bodies in macrophages & giant cells.
- Co-infections are very common.

PCV-2: control

- Vaccines:
  - Inactivated PCV-2 vaccine is available; administered to piglets 4 weeks of age and older.
  - In 2013, Merck Animal Health released “Circumvent PCV G2” vaccine for use in as early as three days after birth & immunity lasts 5 months (25% longer than any other PCV2 vaccine on the market).





Anelloviridae

Chicken anaemia virus (CAV)

Anelloviridae: Characteristics (page 28)

- Only one virus of interest: chicken anemia virus

Size	22-30 nm
Morphology	Naked
	Icosahedral
Genome	Circular, ssDNA
Target cells	• Precursor cells of the bone marrow and thymus
Transmission	• Vertical • Feco-oral • Inhalation of feather dander • Direct contact
Species	• Chickens (and quails)

- This is a disease of very young chickens, 2-3 weeks old (mainly broilers 10-18 days old).

CAV: Pathogenesis

- Infection and necrosis of precursor cells → immunosuppression and anemia

- Concurrent infection (Marek's disease, IBDV, adenovirus) ↑ ↑ severity of disease
  - Infection with these viruses in older birds → ↑ susceptibility to CAV

CAV: Clinical signs (page 29)

- Depression, anorexia, marked pallor (anaemic)
- Multifocal hemorrhage
- Mortality by 3-4 weeks (10 – 60 %)
- Survivors show:
  - growth retardation (therefore take longer to reach market weight), &
  - gangrenous dermatitis (secondary bacterial infection due to immunosuppression), leading to high condemnation rates at slaughter.



This is an economically important disease to the broiler industry

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A marked difference in size and growth retardation is observed between healthy (left) and infected (right) birds.

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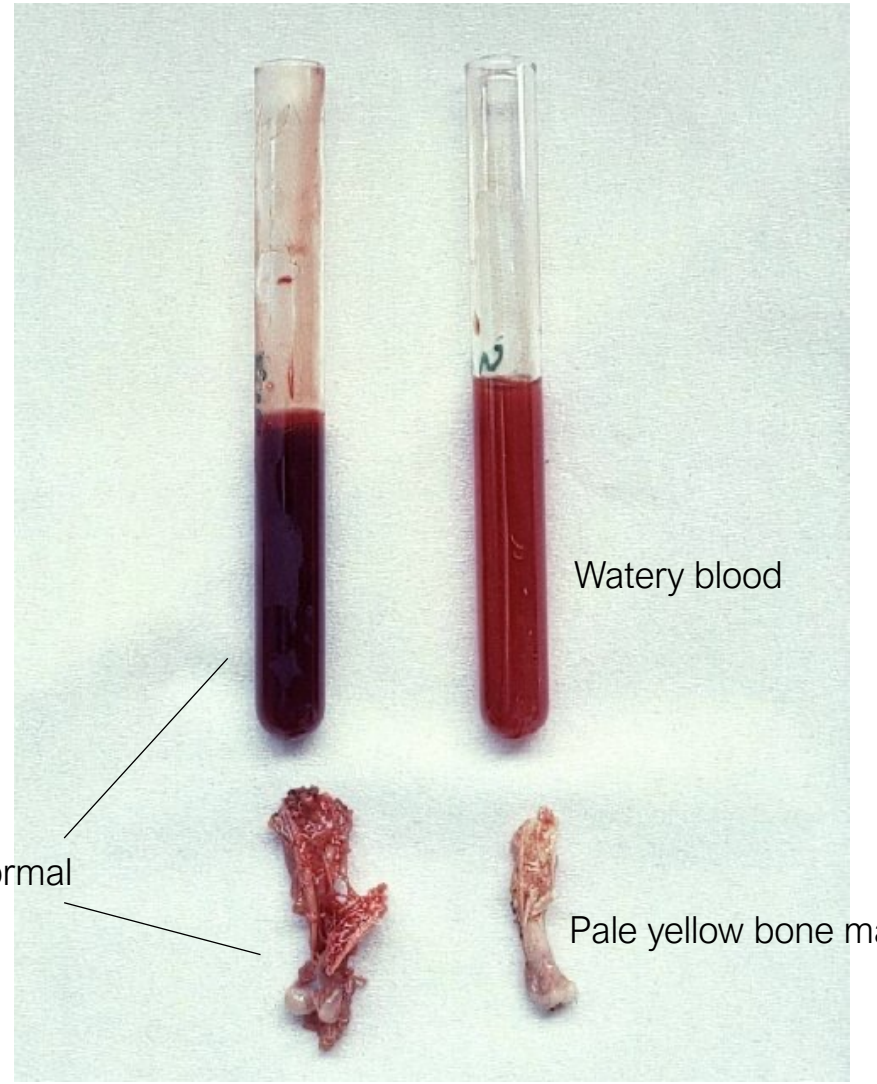
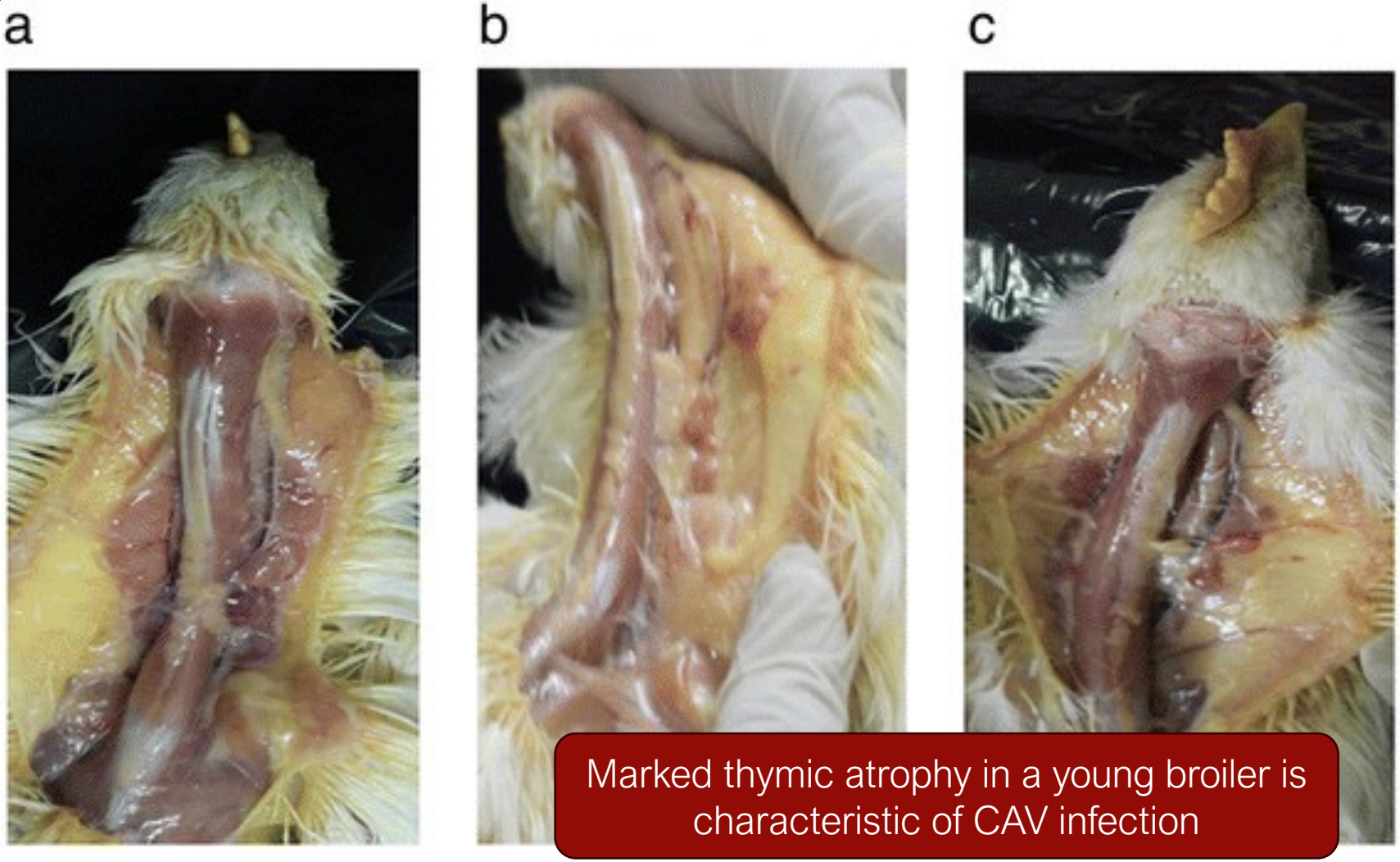


CAV: Gross lesions



Marked thymic atrophy in a young broiler is characteristic of CAV infection

Rimondi, Agustina & Pinto, Silvina & Olivera, Valeria & Dibárbora, Marina & Pérez-Filgueira, Mariano & Craig, Maria I & Pereda, Ariel. (2014). Comparative histopathological and immunological study of two field strains of chicken anemia virus. Veterinary research. 45. 102. 10.1186/PREACCEPT-1742080974130974.

CAV: Diagnosis (page 29)

- Disease is suspected based on history, clinical signs and PM lesions
- Confirmation of diagnosis is by:
  - Virus isolation
  - PCR
  - IHC
- Ideal samples are:
  - Liver
  - Spleen
  - Buffy coat
  - Serum

CAV: Control (pages 29-30)

- Vaccination of breeders with live attenuated CAV vaccine will induce high and uniform maternal antibodies and prevent vertical transmission.

- Young breeder hens may be infected deliberately with wild-type virus by adding crude homogenates of tissues from affected chickens to drinking water. This ensures infection and seroconversion before hens begin to lay eggs.
  - Disadvantage: maintains high levels of virus in the population.

CAV: Control (pages 29-30)

- Control is mainly by good hygienic practices to prevent horizontal transmission (virus is ubiquitous & very resistant to inactivation).

- Control (vaccinate) co-morbitities:
  - Marek's disease & IBDV (to reduce severity of infection & protect age-associated resistance of birds to CAV).