# Pathology of Swine

C. L. Davis Gross Pathology Review Course
U. of Minnesota, St. Paul, July 8-12, 2013

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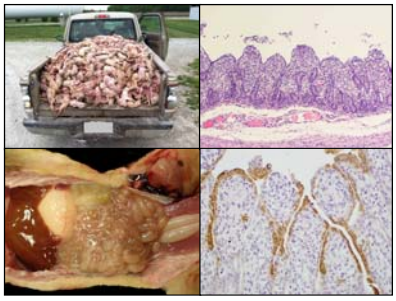
Veterinary Diagnostic Laboratory
College of Veterinary Medicine
Iowa State University



## Porcine Epidemic Diarrhea U.S. Outbreak





## Management Systems → Disease

- Traditional Technologies
  - Small farms: 50-100 sows, outside
  - Group farrowing: 2-4 groups/year
  - Weaning age: 4-8 weeks-of-age
  - Continuous-flow rearing
- Evolution
  - Large farms: 2000-6000 sows, inside
  - Reduced weaning ages: 3 weeks
  - Age-segregated rearing: AI/AO
  - Site-segregated rearing: 2- or 3-site



## Ages: Stages

| | Conventional | SEW |
|---|---|---|
| Suckling | < 3 weeks | < 2 weeks |
| Nursery | 3-8 weeks | 3-8 weeks |
| Grower | 2-6 months | 2-5 months |
| Breeding | > 6 months | > 6 months |

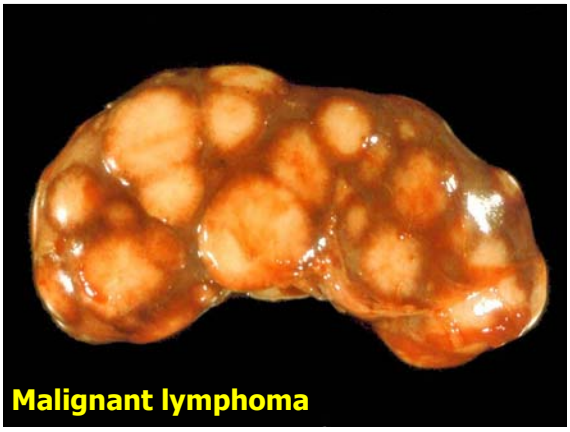


## Lecture Outline

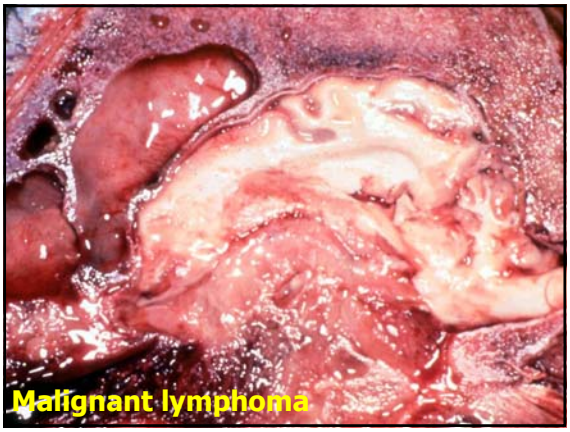
- Neoplastic Diseases
- Congental – Heriditary Diseases
- Generalized Diseases
- Organ Systems
  - Gastrointestinal Diseases
  - Respiratory Diseases
  - Cardiovascular Diseases
  - Integumentary Diseases
  - Nervous and Musculoskeletal Diseases
  - Urogenital Diseases

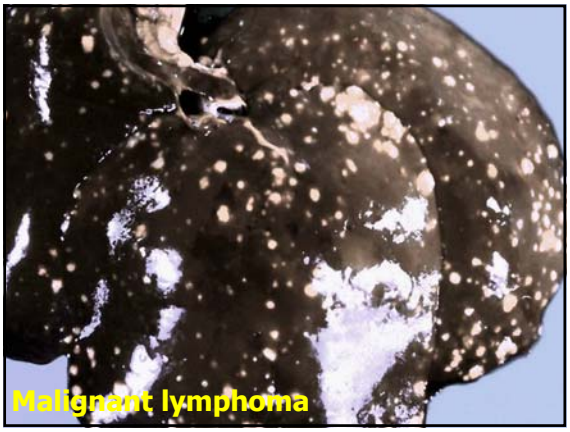


## Neoplastic Diseases


Malignant lymphoma


Malignant lymphoma


Malignant lymphoma


Malignant lymphoma

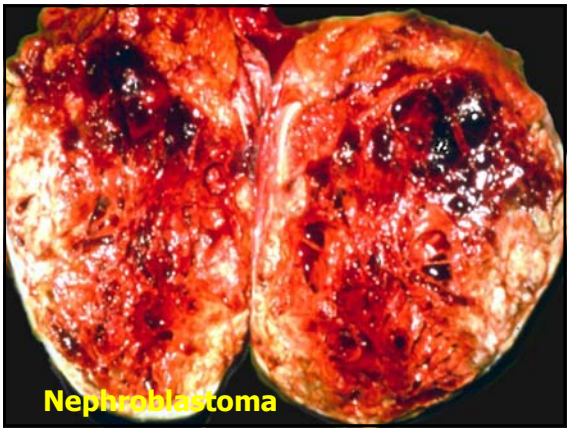

Malignant lymphoma


Malignant lymphoma


Melanoma


Malignant melanoma


Nephroblastoma


Nephroblastoma

## Congenital – Heriditary Diseases

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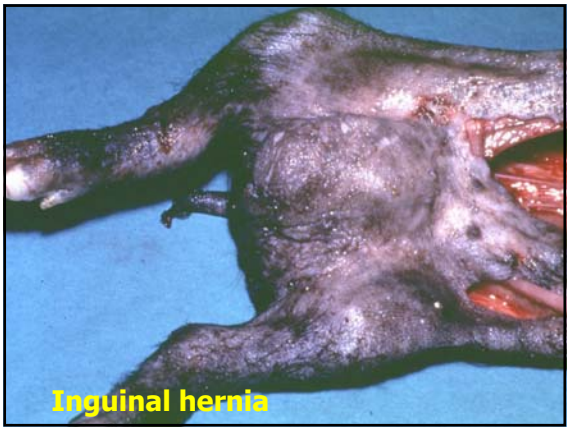
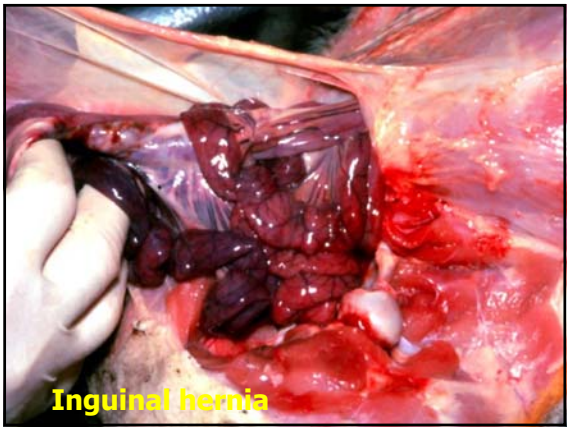

Inguinal hernia


Inguinal hernia


Umbilical hernia


Myofibrillar hypoplasia

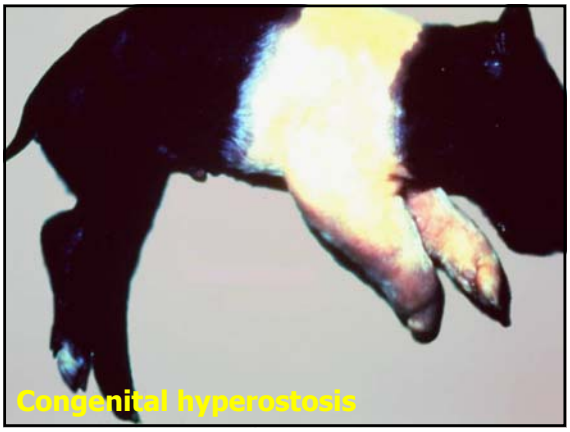

Myofibrillar hypoplasia


Arthrogryposis

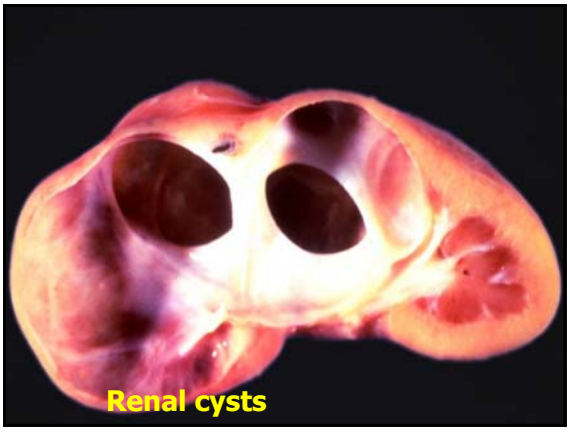

Microphthalmia


Aplasia chiasma opticum

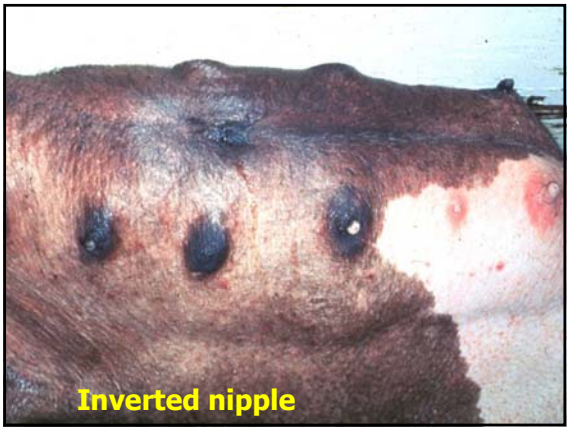

Conjoined twins


Congenital hyperostosis

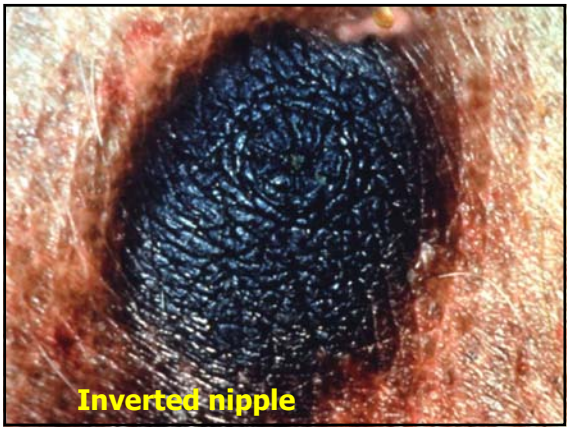

Congenital hyperostosis


Renal cysts


Hydronephrosis



Inverted nipple



Inverted nipple

Porcine juvenile pustular psoriasiform dermaititis (Pityriasis rosea)

Porcine juvenile pustular psoriasiform dermaititis (Pityriasis rosea)

Thrombocytopenia purpura

Thrombocytopenia purpura


Dermatosis vegetans

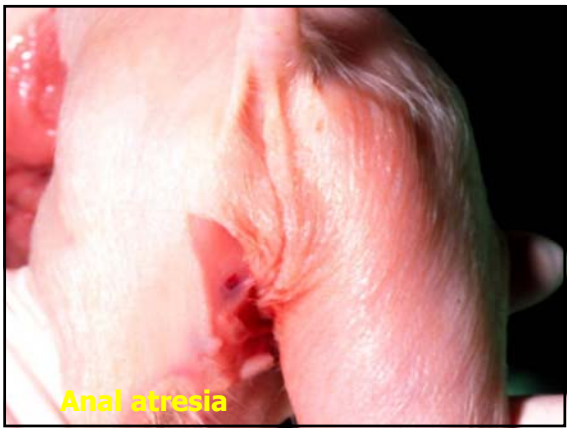

Dermatosis vegetans


Dermatosis vegetans


Dermatosis vegetans


Epitheliogenesis imperfecta


Syndactly

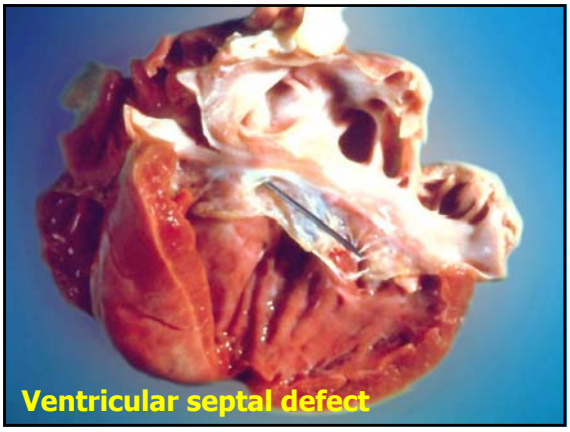

Anal atresia


Anal atresia


Palatoschisis

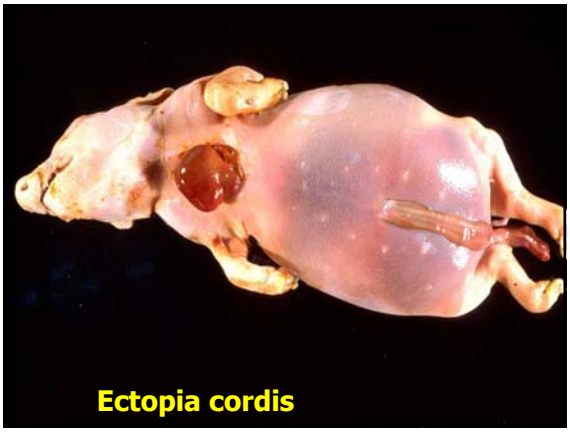

Menigoencephalocoele

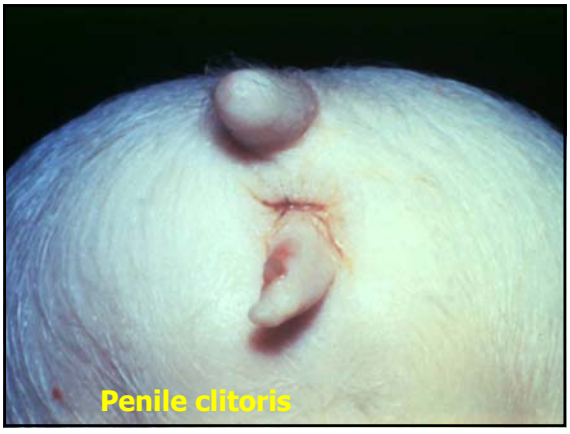

Cranioschisis

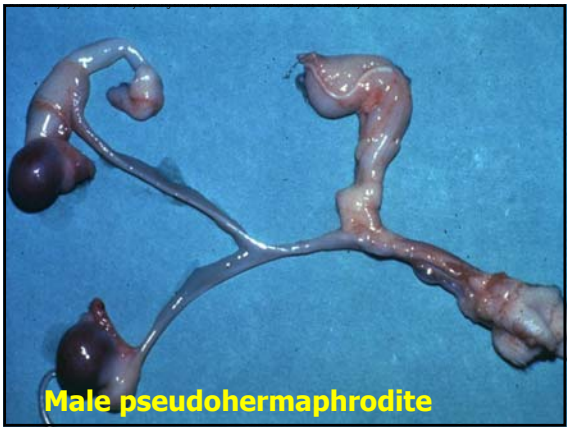

Ventricular septal defect


Persistent Meckel's diverticulum


Biliary cyst


Ectopia cordis


Penile clitoris


Male pseudohermaphrodite

## Generalized Diseases

## *Salmonella choleraesuis*

- Severe septicemia in weaned pigs
- +/- concurrent pneumonia or enterocolitis
- Multifocal ehpatic necrosis (paratyphoid nodules)
- Replicates in macrophages and extracellularly in lymphoid tissues
- Systemic endotoxins → vascular damage:
  - Hemorrhage, interstitial pneumonia with edema, glomerulonephritis, gastric mucosa venous thrombosis, arterial thrombosis → infarcts in skin of extremities and in colon (mucosal ulcers)

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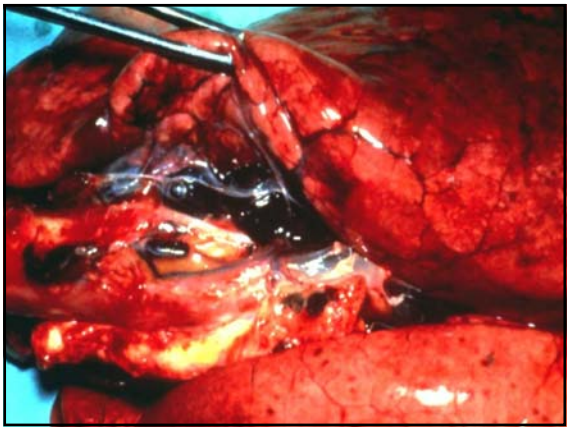
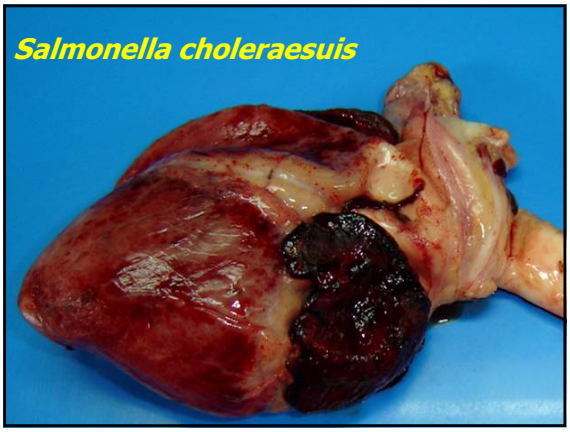
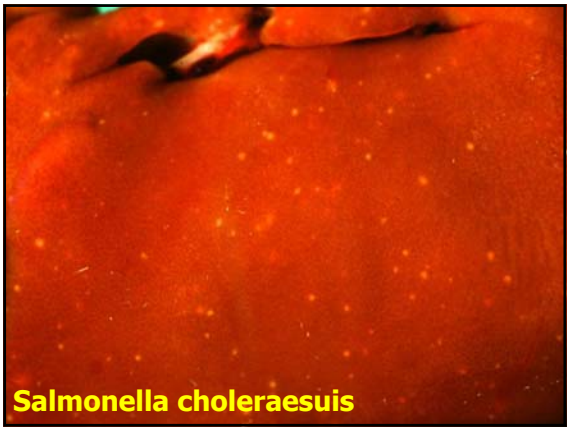


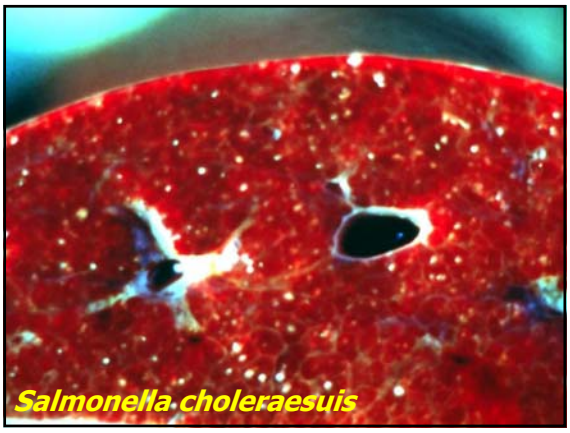

Salmonella choleraesuis


Salmonella choleraesuis


Salmonella choleraesuis

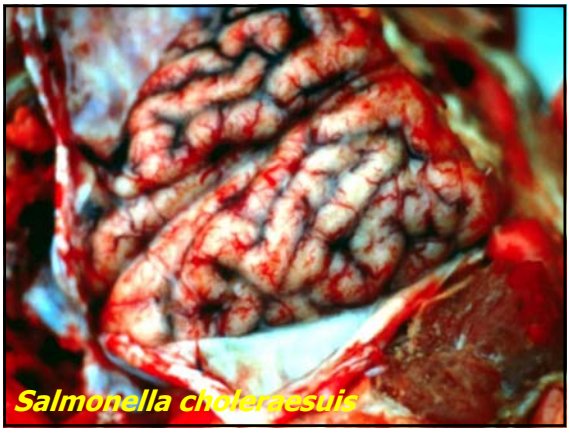

Salmonella choleraesuis


Salmonella choleraesuis


Salmonella choleraesuis


Salmonella choleraesuis


Salmonella choleraesuis


Salmonella choleraesuis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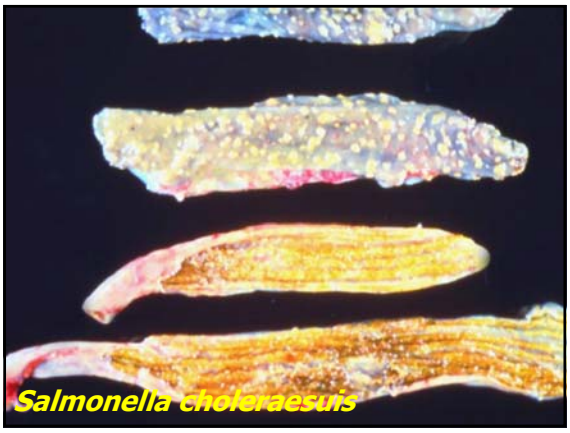
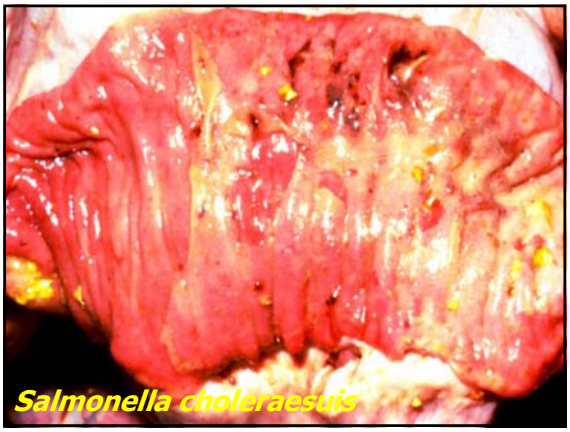




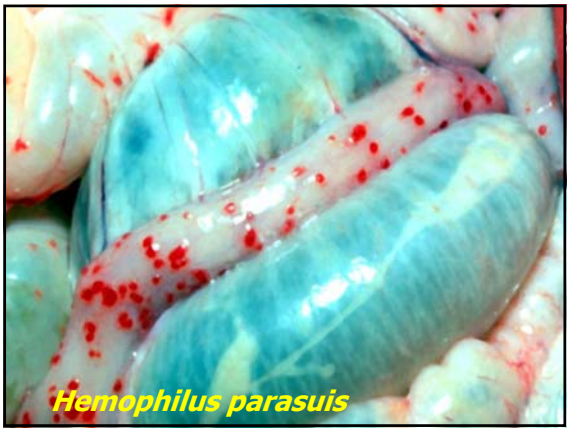

## *Hemophilus parasuis*

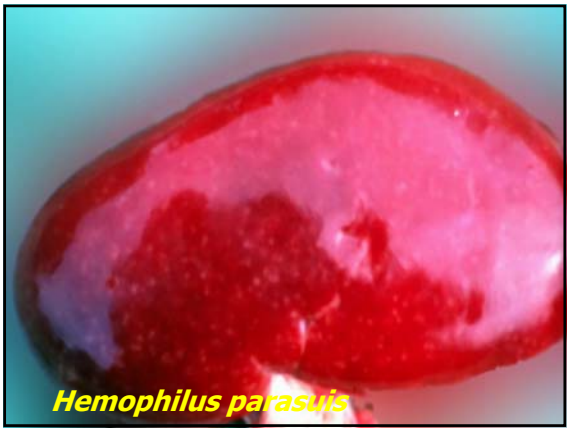
- Acute septicemia
- Resembles septicemic salmonellosis
- More commonly causes polyserositis, polyarthritis, and meningitis (Glässer's disease) in weaned pigs
- Neurological clinical signs are uncommon
- Occasional acute outbreaks of highly fatal fibrinosuppurative leptomeningitis in young adult replacement breeding stock shortly after entry into recipient herds
- Eustachitis and temporary otitis media predisposing to secondary pyogenic otitis media

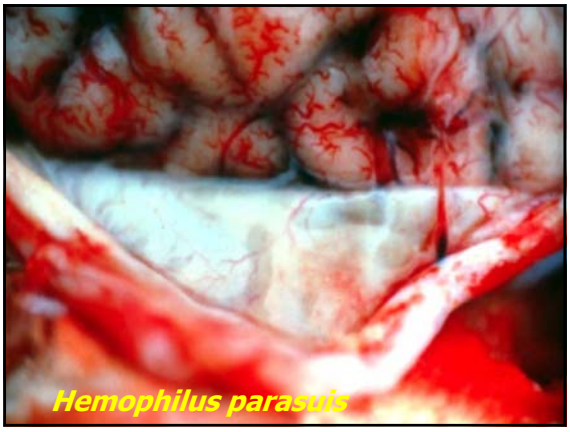


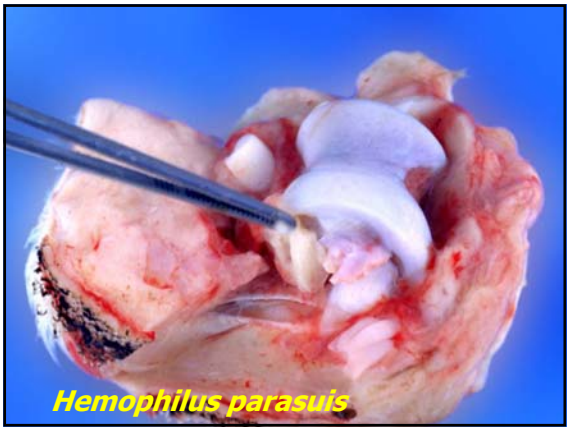

Hemophilus parasuis

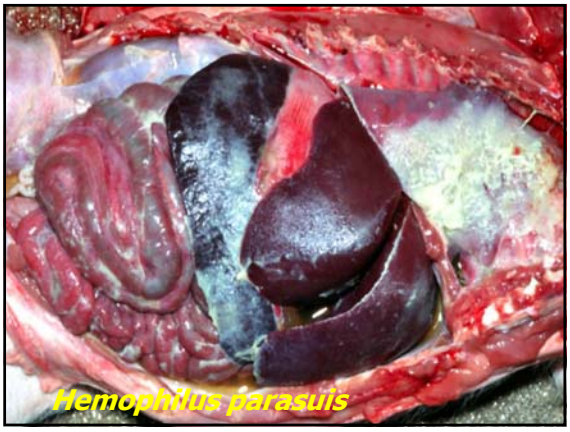

Hemophilus parasuis


Hemophilus parasuis


Hemophilus parasuis


Hemophilus parasuis


Hemophilus parasuis


Hemophilus parasuis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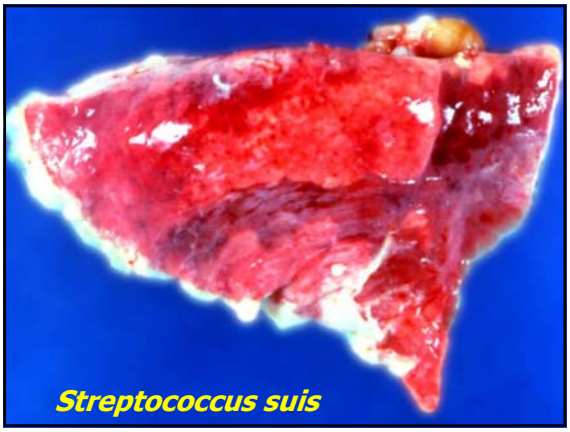
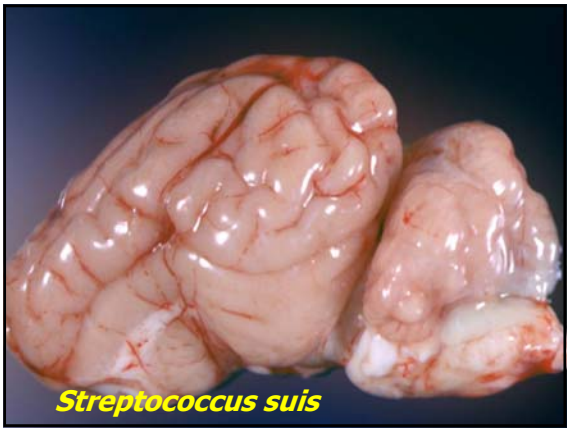
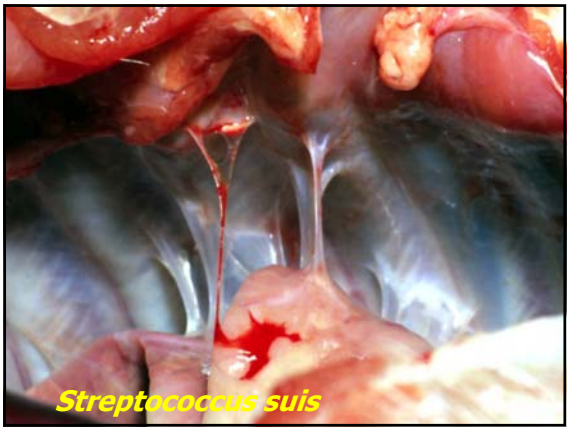

Streptococcus suis

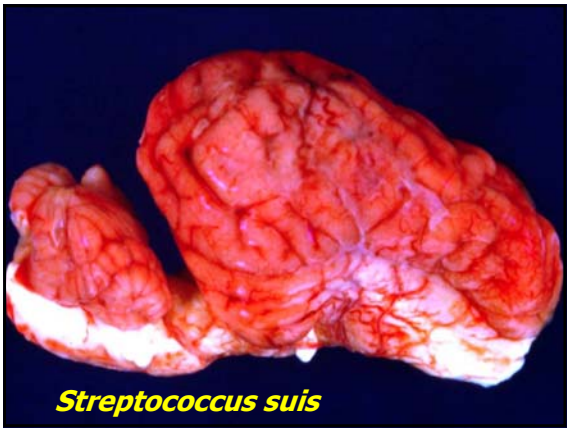

Streptococcus suis


Streptococcus suis

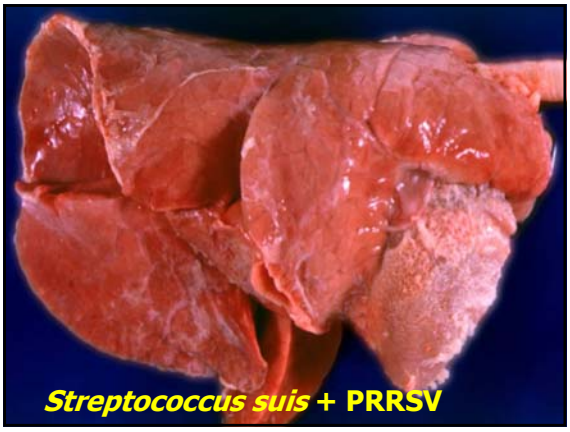

Streptococcus suis

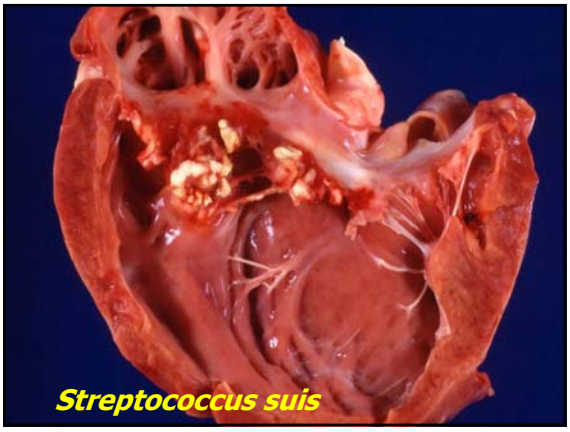
## *Streptococcus suis*

- 35 serotypes: 1-34 and 1/2, disease: 2, 1/2, 3, 4, 7, 8 and 9
- Commensal of tonsil and nasal mucosa, antibiotics will not clear, MEW and SEW will not eliminate
- Herds and individuals often carry multiple serotypes
- Healthy pigs - nasal cavities: 94% of 4-8 week-old pigs, 71% of these were serotypes 17, 18, 19 and 21
- Outbreaks of disease: Nursery-age >> all ages; horizontal transmission of a single serotype
- 3 disease forms: septicemia, pneumonia, reproductive
- Septicemia: splenomegaly, mild interstitial pneumonia, fibrinous polyserositis, polyarthritis and leptomeningitis, vegetative valvular endocarditis
- Bronchopneumonia: suppurative, fibrinohemorrhagic
- Reproductive: abortion, vaginitis







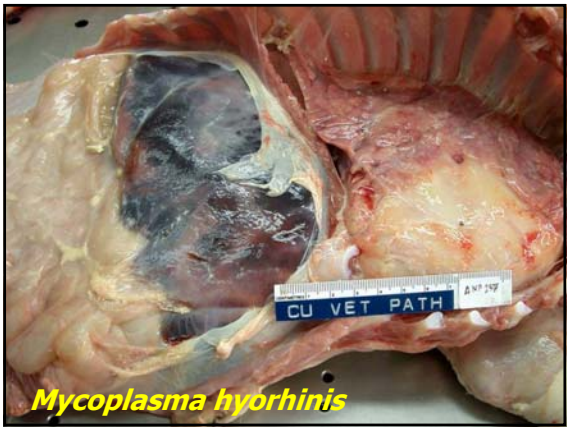
## *Streptococcus suis* : an emerging zoonotic pathogen

- Human infection with *S. suis* occurs mainly among risk groups that have frequent exposure to pigs or pork
- First case in Denmark in 1968, worldwide more than 200 cases before 2005, most from Europe and Asia
- Large outbreak in July 2005 in Sichuan province, China (third outbreak, two earlier outbreaks in 1998 and 1999)
- In past 8 years in China, at least 237 people infected with *S. suis* and 53 of them died
- All human *S. suis* infections attributed to type 2; except for 2 cases caused by type 1, and 1 case of septicemia caused by type 14
- Manifested as purulent meningitis, less common septic shock with multiple organ failure, endocarditis, pneumonia, arthritis, and peritonitis

Lun et al. Lancet Infect Dis. 7, 201-9, 2007

## *Mycoplasma hyorhinis*

- **Fastidious, pleomorphic - can be isolated from nasal cavities of ≈ 40% of weaners**
- **Polyserositis in 3-10 week-old pigs**
- **Role in pneumonia is controversial**
  - **primary pathogen - mild lesions like *Mycoplasma hyopneumoniae***
  - **secondary pathogen?**
    - **↑ proportion of pneumonic lung**
    - **↑ localized pleuritis**
- **Diagnosis: culture, FA, IHC, PCR**



*Mycoplasma hyorhinis*


*Mycoplasma hyorhinis*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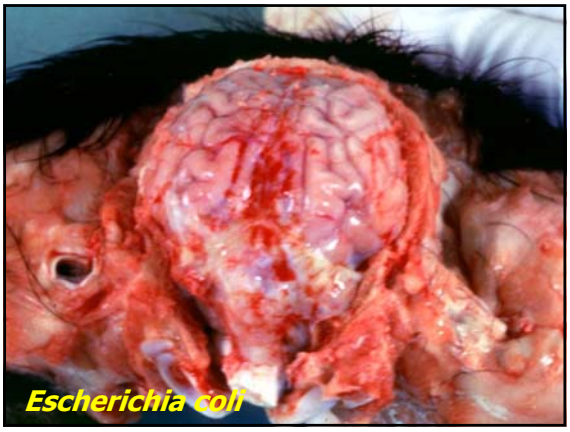
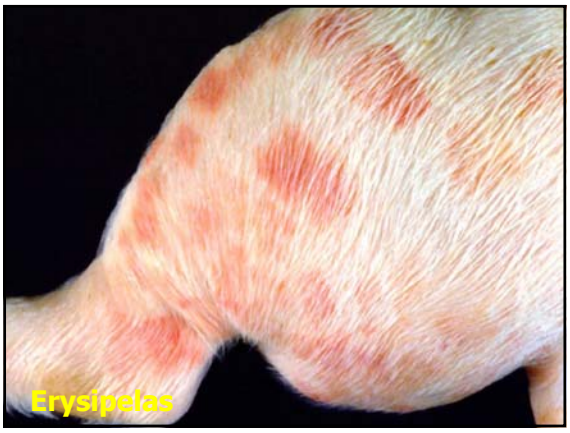
*Mycoplasma hyorhinis*

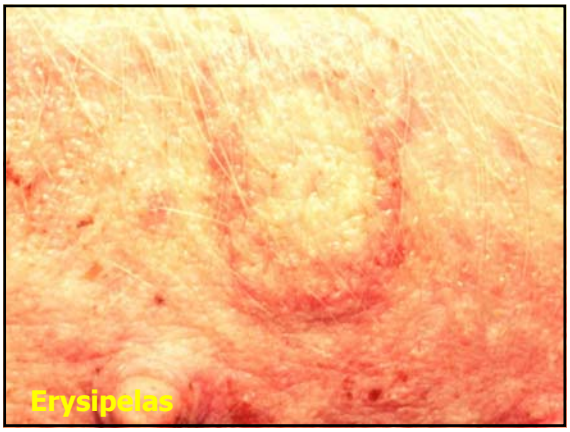

*Escherichia coli*

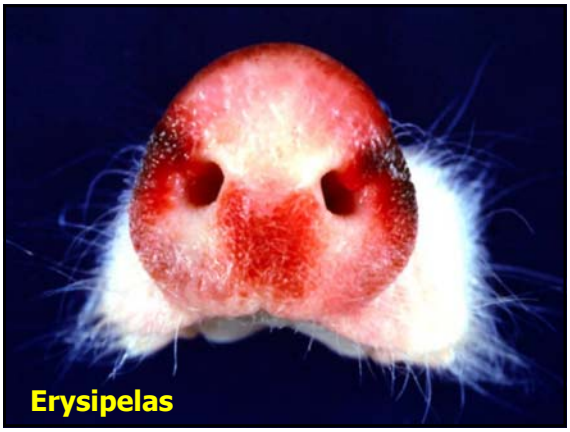
## Erysipelas

- Still occurs in swine raised entirely in environmentally regulated buildings
- *E. rhusiopathiae* causes disease in all ages
- Mortality is highest and lesions are most extensive and severe in suckling and recently weaned pigs
- In growing and finishing pigs; pigs may be found dead with few gross lesions
- Renal cortical petechiae, enlarged spleen, lameness with proliferative synovitis and fibrous periarthritis, pyrexia, anorexia, few cutaneous infarcts and occasional abortions

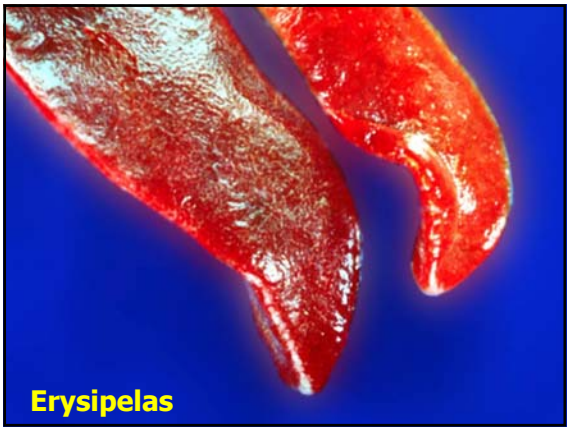

Erysipelas


Erysipelas

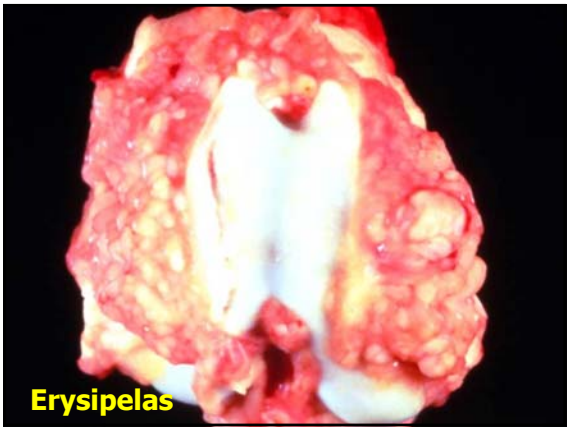

Erysipelas


Erysipelas


Erysipelas

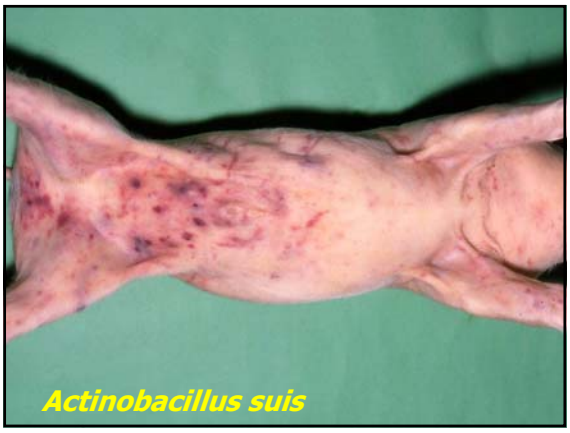

Erysipelas

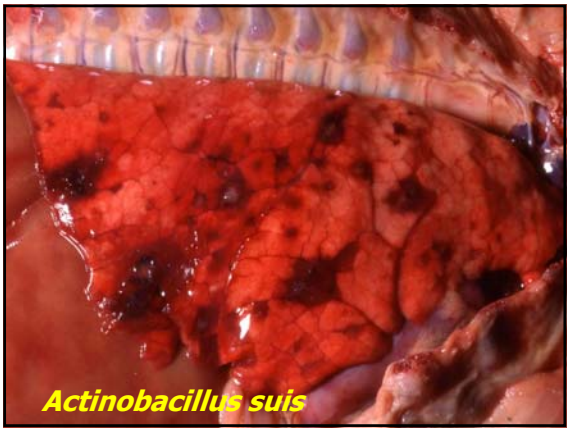

Erysipelas


Erysipelas

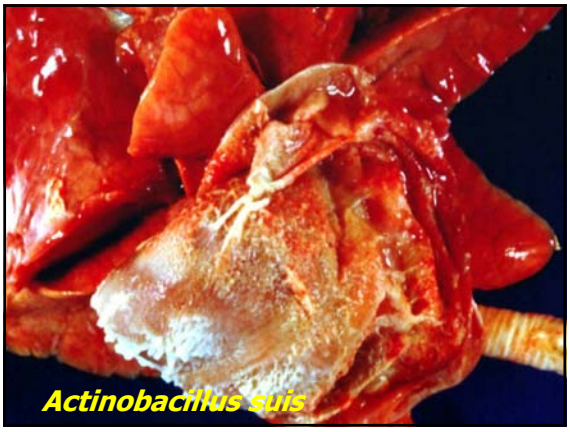
## *Actinobacillus suis*

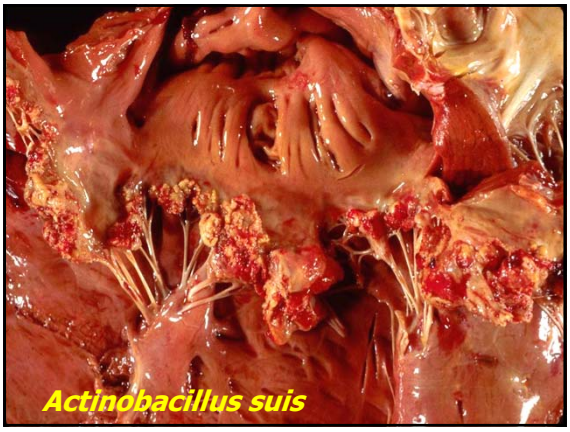
- Prevalence in herds in unknown
- Disease: high health herds, 2-28 days of age
- Virulence factors: exotoxins similar to APP
- Septic-embolic disease: multifocal or diffuse fibrinonecrotic pleuropneumonia
  - widespread hemorrhages – meningitis
  - vegetative endocarditis – skin infarcts
  - fibrinous pericarditis – fibrinous arthritis
- Diagnosis: lesions, culture

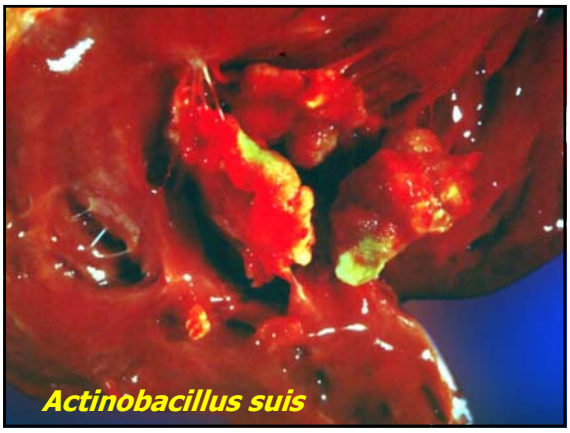


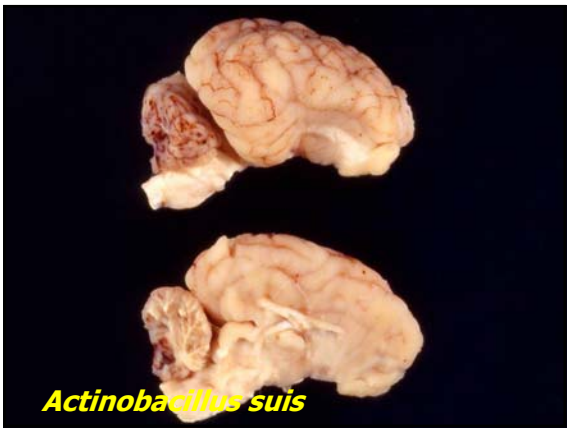



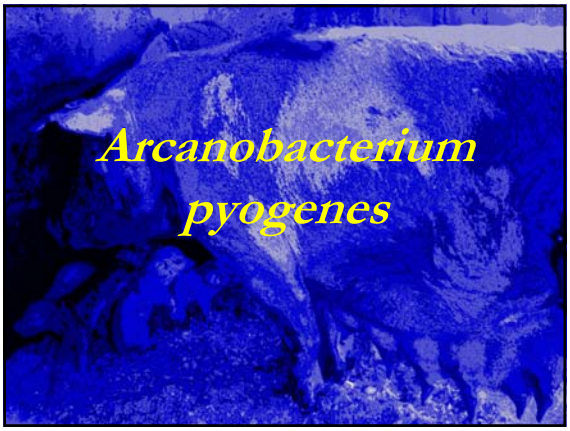


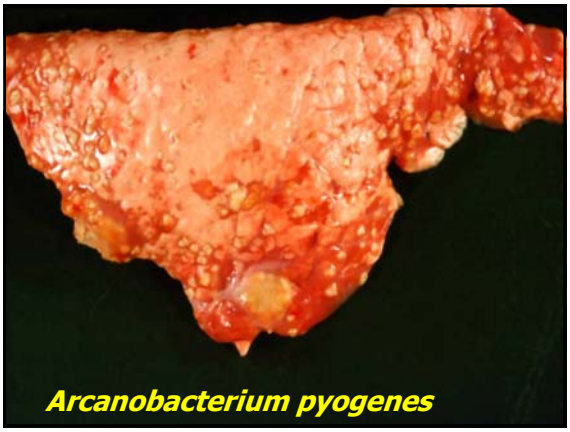



Actinobacillus suis


Actinobacillus suis


Arcanobacterium
pyogenes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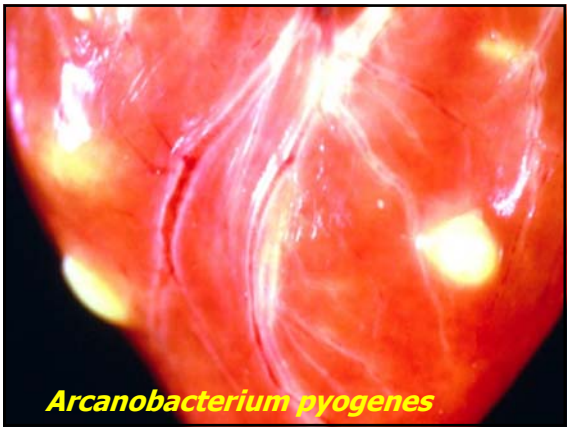
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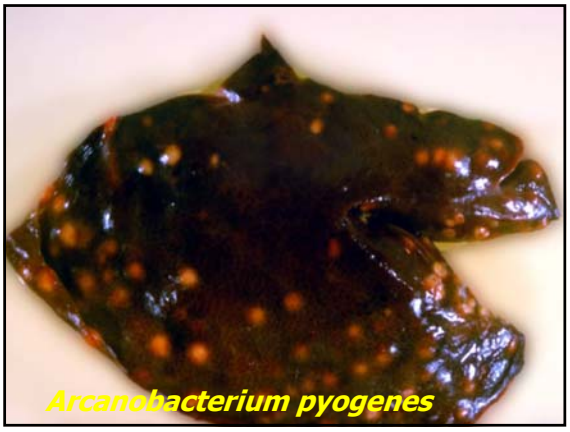
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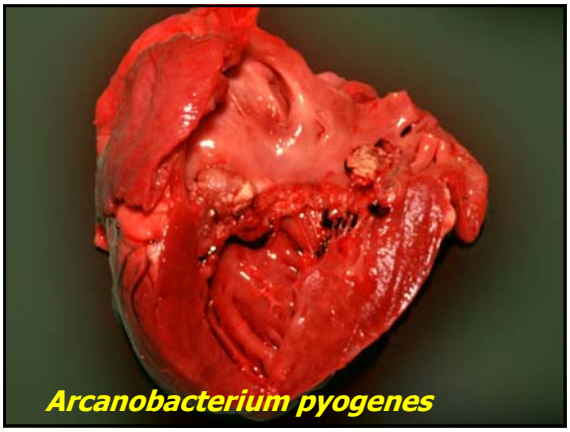
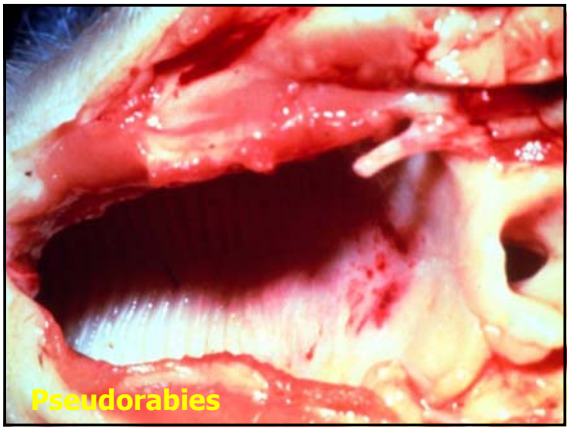
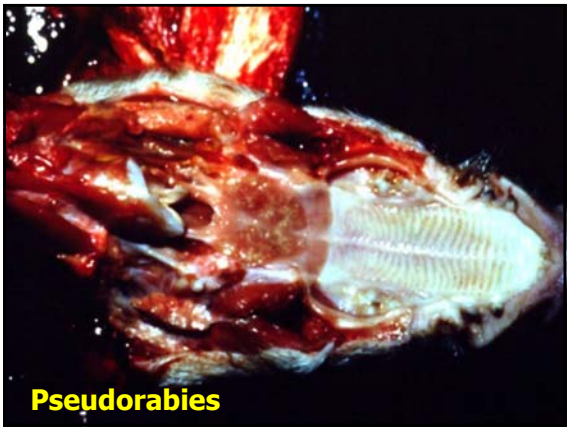



## *Pseudorabies*


Pseudorabies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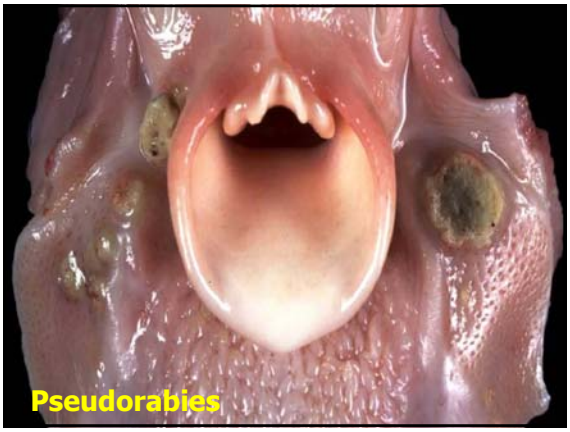
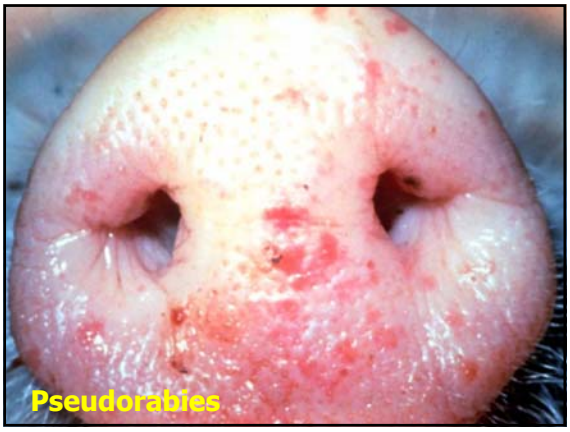

Pseudorabies

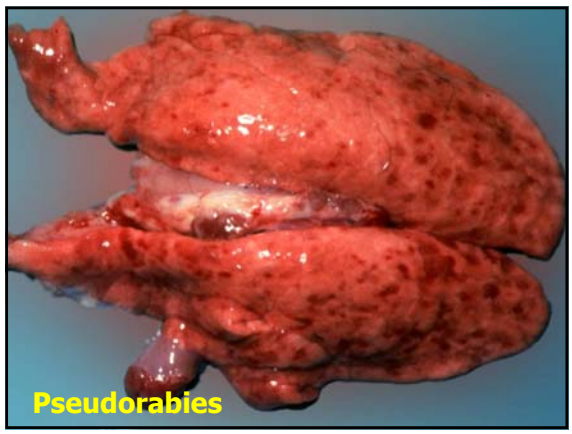

Pseudorabies

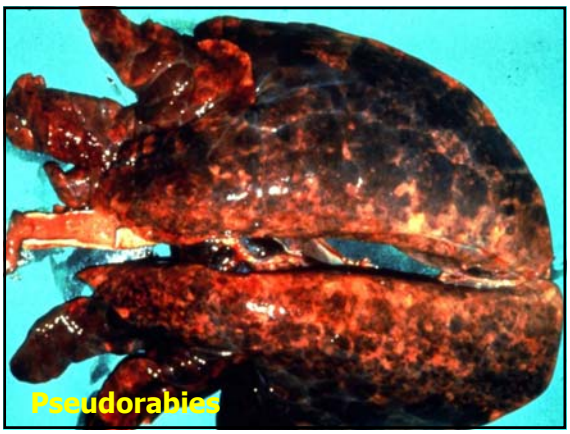

Pseudorabies

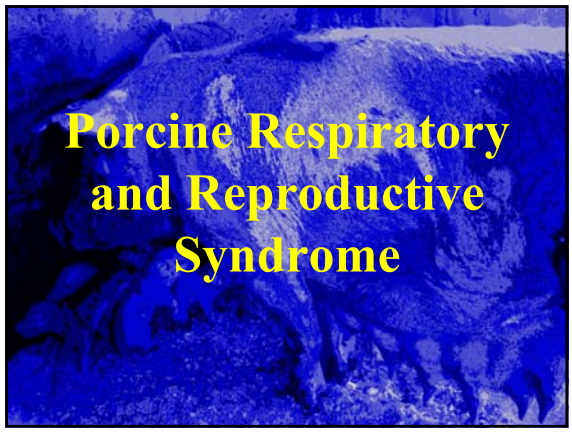

Pseudorabies


Pseudorabies

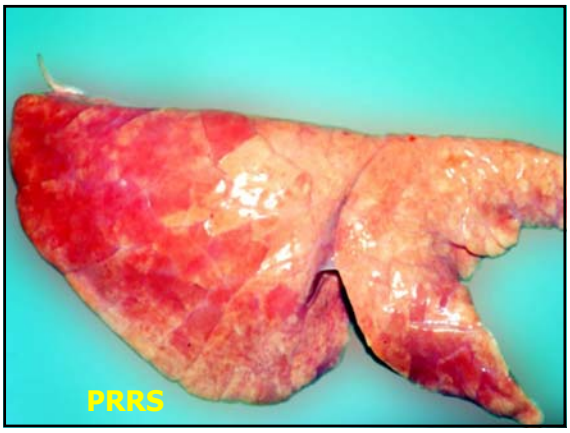

Pseudorabies


Pseudorabies


Pseudorabies


Porcine Respiratory and Reproductive Syndrome


PRRS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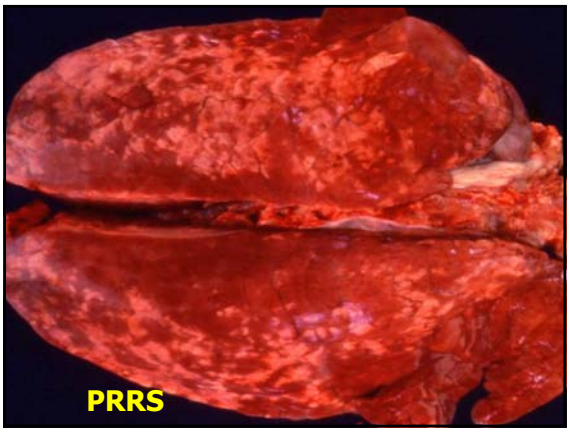
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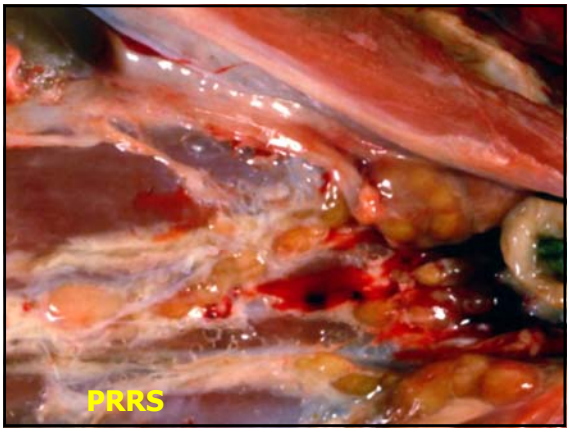
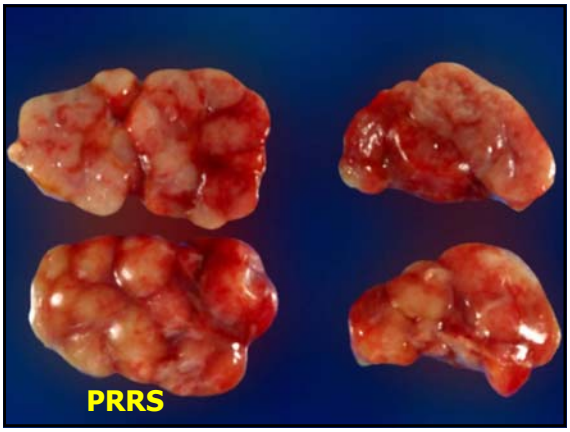

PRRS


PRRS

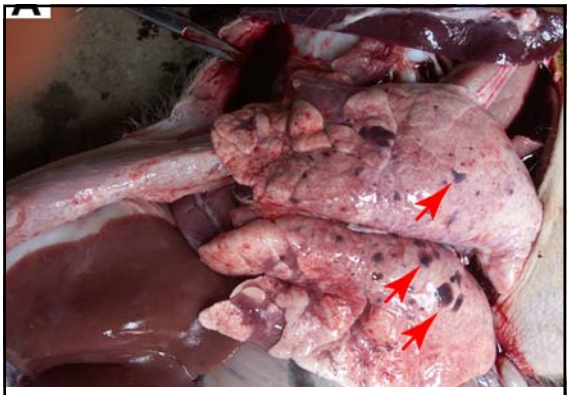

PRRS


PRRS

## Atypical PRRS in Asia

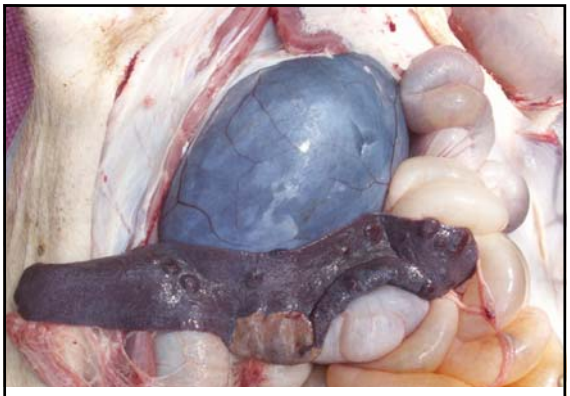
- "Outbreak" in China and Vietnam, 2004-2007
- 2006 in Jiangxi Province
- High fever and elevated mortality in grow/finish pigs, progressed to high mortality, CNS signs, swollen joints and eyelids, and late-term abortions
- Experimental reproduction by Zhou et el., 2008
- According to Chinese sources
  - Affected more than 2 million pigs
  - Genetic homogeneity of strains isolated in outbreak, single and 29 AA deletion in Nsp2, highly virulent NA strain
  - Development of an effective vaccine

Tian et al. Plos One Vol. 6, 526, 2007

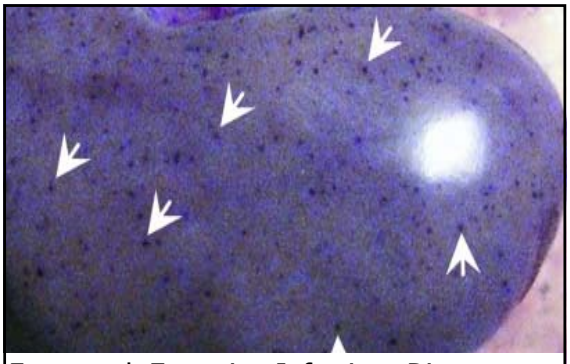


Tian et al. Plos One Vol. 6, 526, 2007

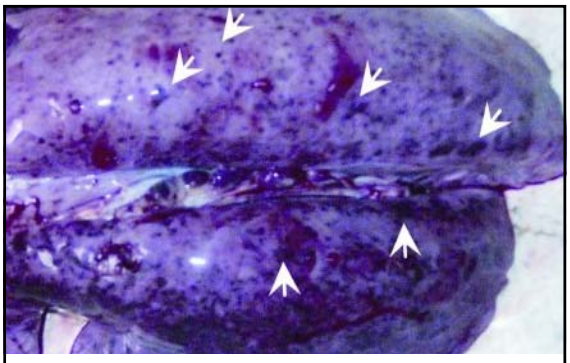
Tian et al. Plos One Vol. 6, 526, 2007



Tian et al. Plos One Vol. 6, 526, 2007



Feng et al. Emerging Infectious Diseases Vol. 14, No. 11, November 2008



Feng et al. Emerging Infectious Diseases Vol. 14, No. 11, November 2008

## Atypical PRRS in Asia

- Skepticism by PRRS researchers:
  - Of 1500 "sources" sampled, 30% were PCR positive to the new PRRS variant
  - *Streptococcus suis* was isolated from 1/3 of the cases and *Haemophilus parasuis* from another third
  - Samples are negative for PRV
  - Classical Swine Fever is endemic in China's pig herds
  - Producers routinely vaccinate for CSF as well as for Foot and Mouth Disease and Pseudorabies
  - Prior to this most recent outbreak, some herds were being vaccinated against PRRS using a Chinese vaccine
  - Did not appear to prevent infection with the latest pathogen

# PCVAD

## Porcine Circovirus Associated Diseases

## The Role of PCV in Swine Diseases

**Field isolates of PCV from pigs have been associated with:**

- Postweaning Multisystemic Wasting Syndrome (PMWS)
- Porcine Respiratory Disease Complex (PRDC)
- Abortions
- Porcine Dermatitis Nephropathy Syndrome (PDNS)
- Proliferative and Necrotizing Pneumonia (PNP)

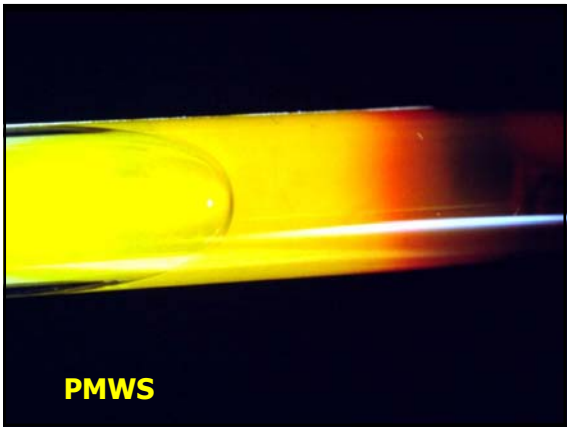
**Two types of PCV have been identified:**

- PCV1 (similar to virus from PK-15 cells)
- PCV2 (isolates from pigs with PMWS)

# PCVAD

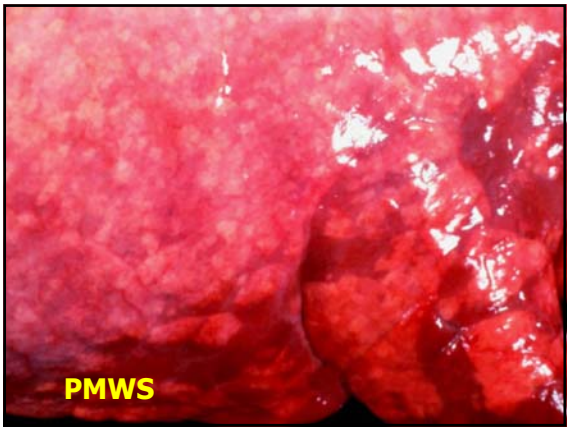
## Postweaning Multisystemic Wasting Syndrome


PMWS

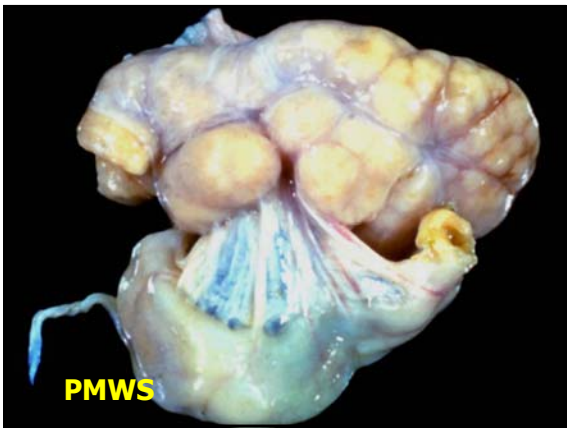

PMWS


PMWS


PMWS


PMWS

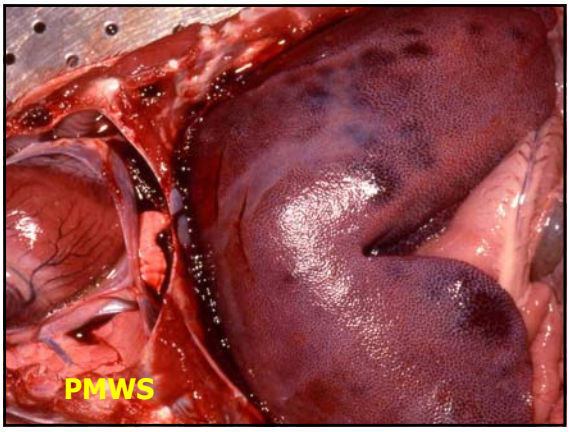

PMWS

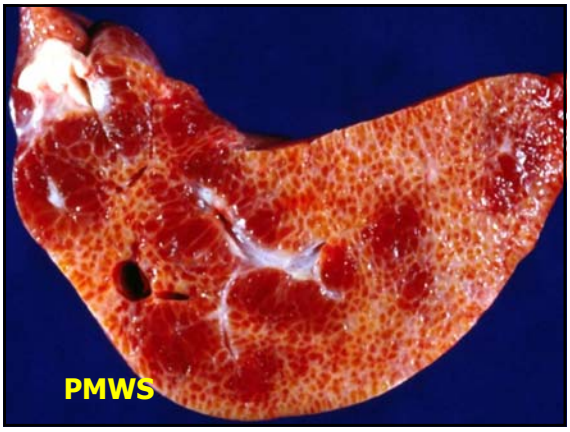

PMWS

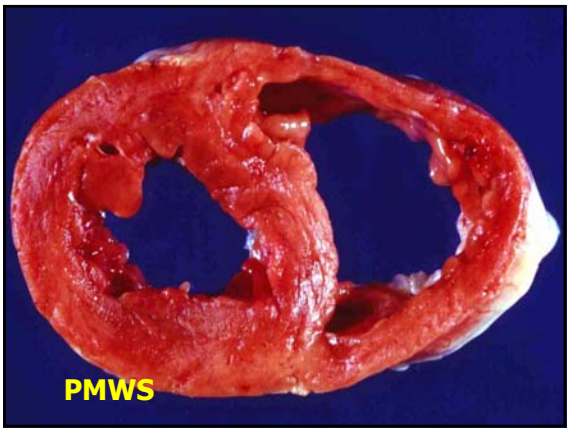

PMWS


PMWS


PMWS


PMWS

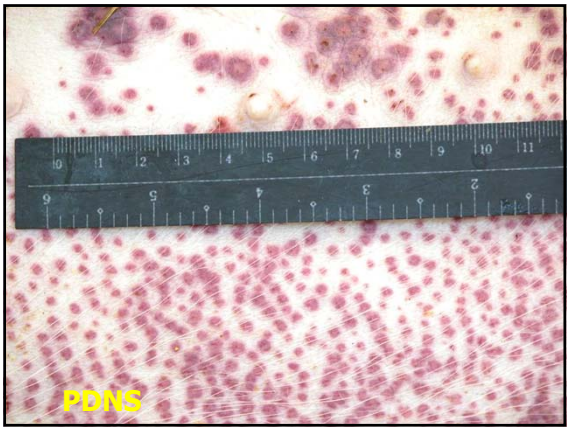

PMWS


PMWS

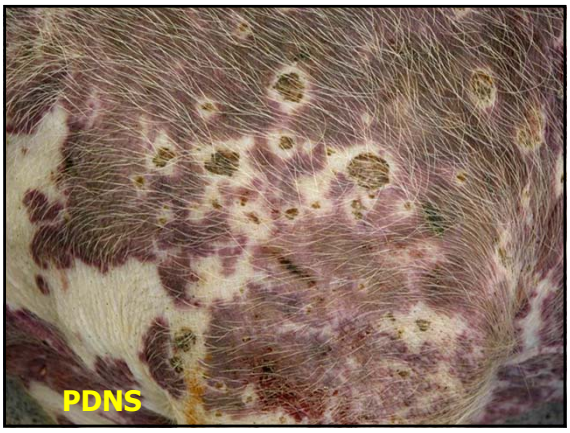

PDNS

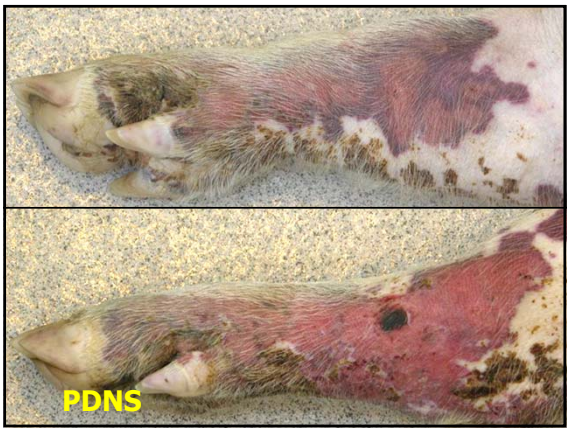

PDNS

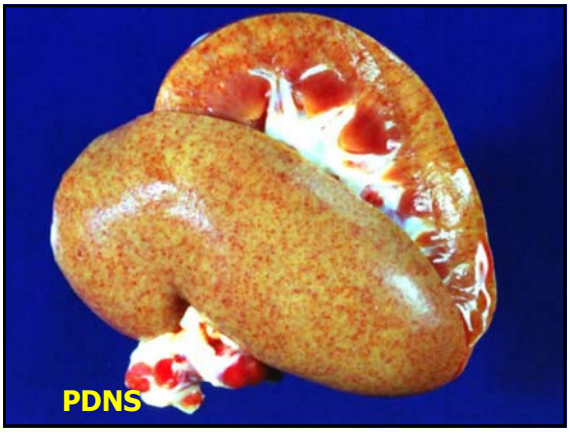

PDNS


PDNS


PDNS





## Classical Swine Fever

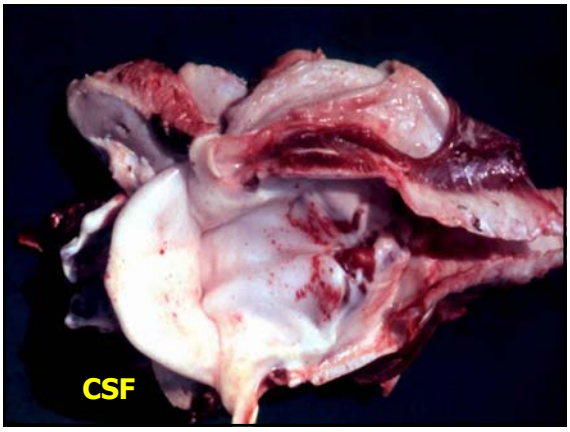
- Forms: acute, subacute, reproductive
- Pyrexia, cutaneous cyanosis, conjunctivitis, anorexia, constipation followed by severe diarrhea ("cholera"), convulsions and death
- Peripheral hemorrhages of lymph nodes, generalized vasculitis, tonsillar necrosis, splenic infarcts, serosal hemorrhages, button ulcers in colon
- Mummified, stillborn and weakborn pigs, congenital tremors, cerebellar hypo- or aplasia, limb deformation, arthrogryposis


CSF


CSF


CSF


CSF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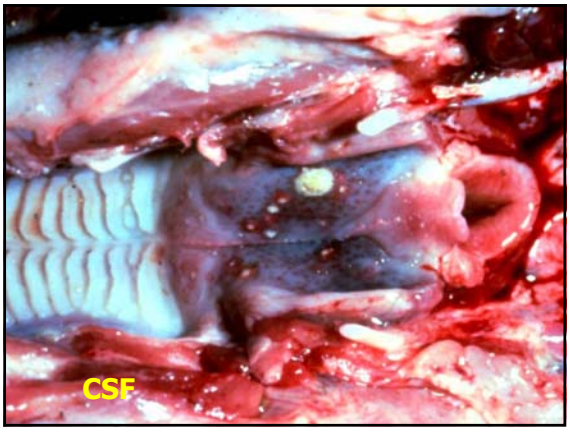
CSF


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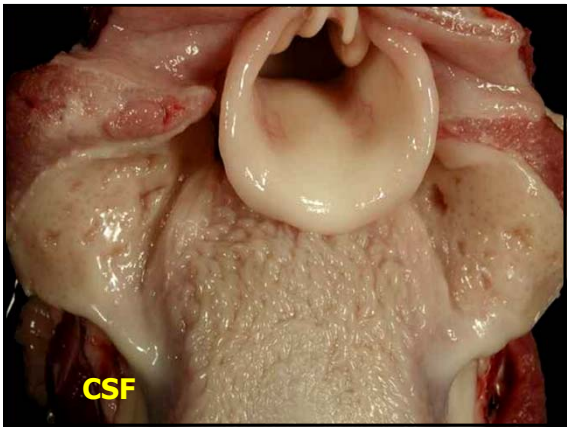
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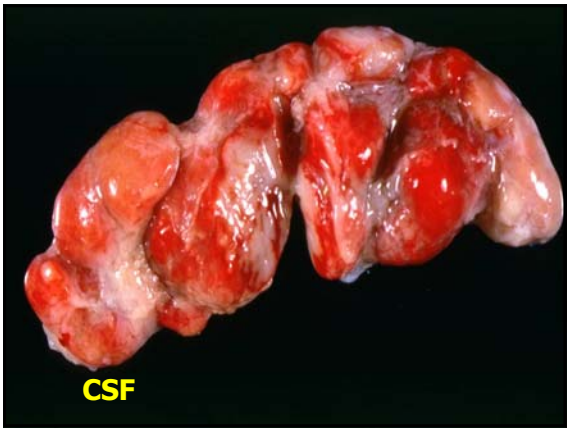
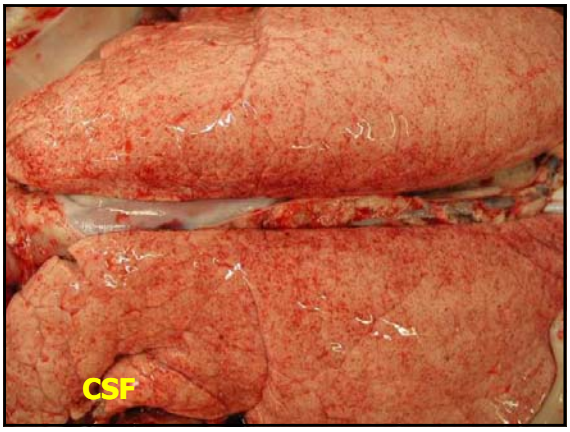
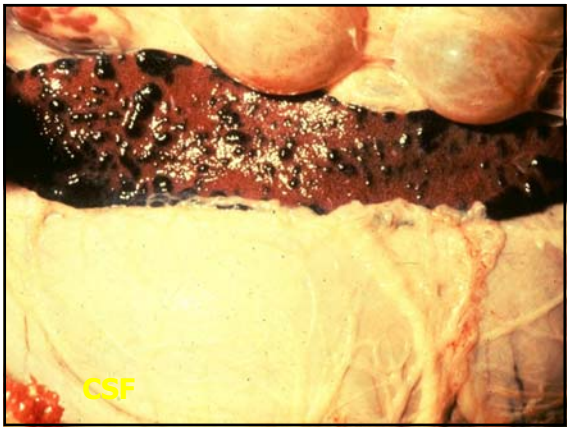

CSF


CSF


CSF


CSF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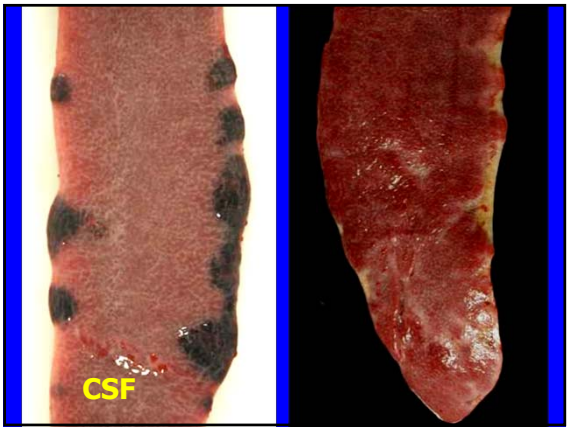
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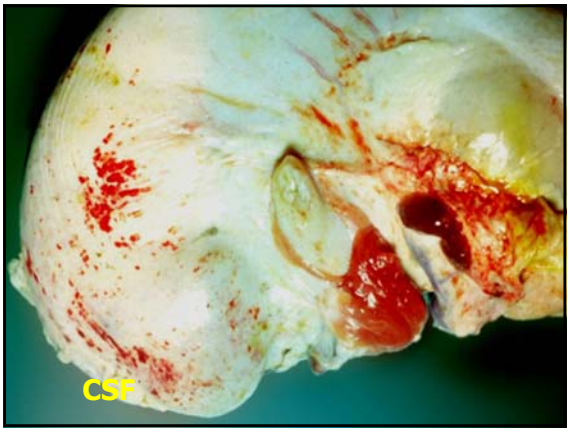
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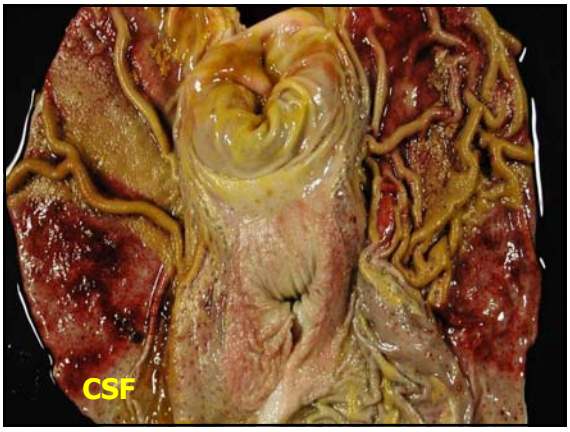
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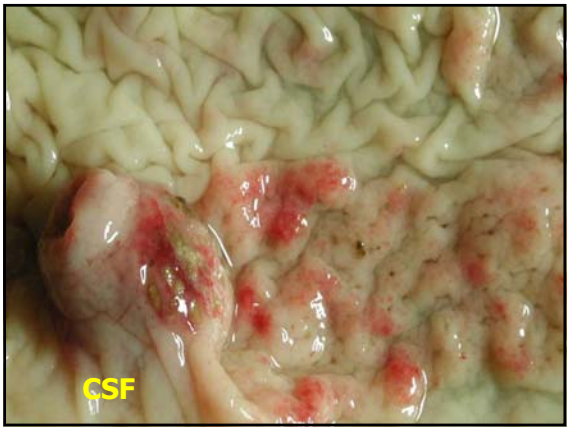
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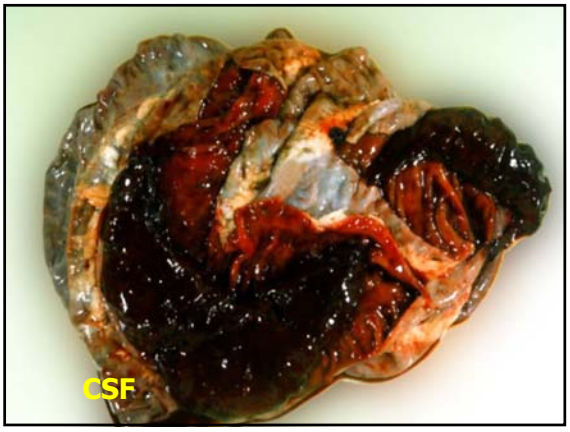
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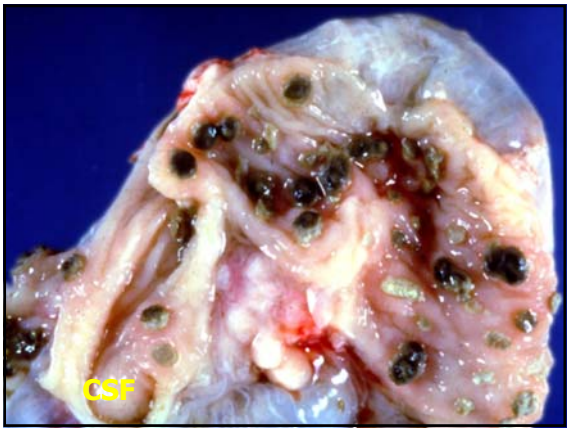
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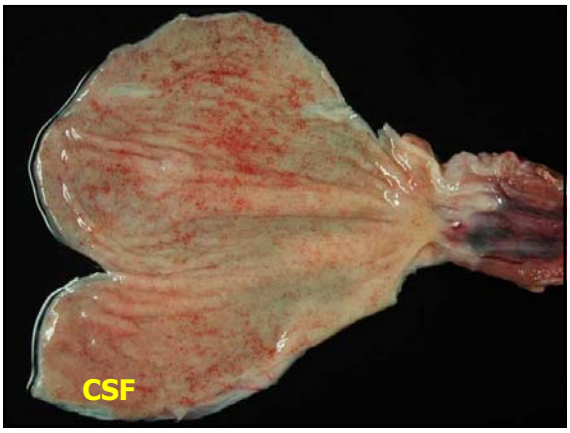
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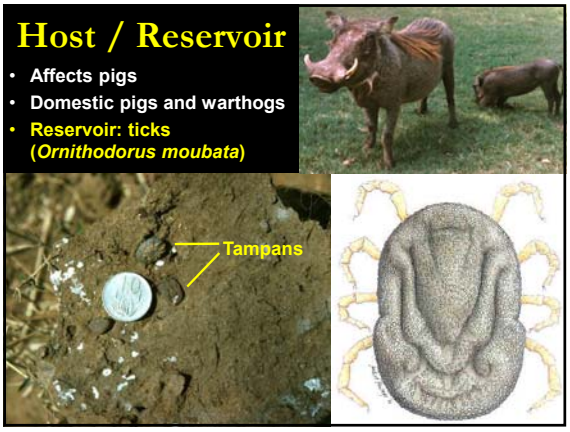
African Swine Fever


Host / Reservoir
• Affects pigs
• Domestic pigs and warthogs
• Reservoir: ticks (*Ornithodorus moubata*)
Tampans

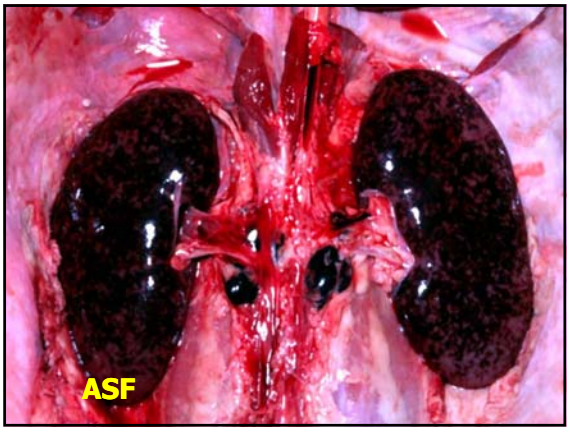

Transmission
Pig 'khola'


ASF


ASF


ASF


ASF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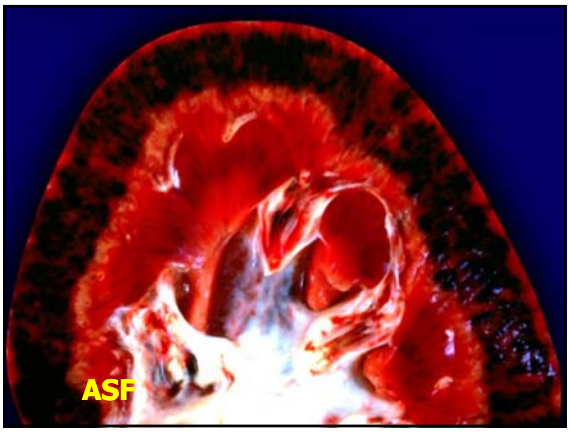
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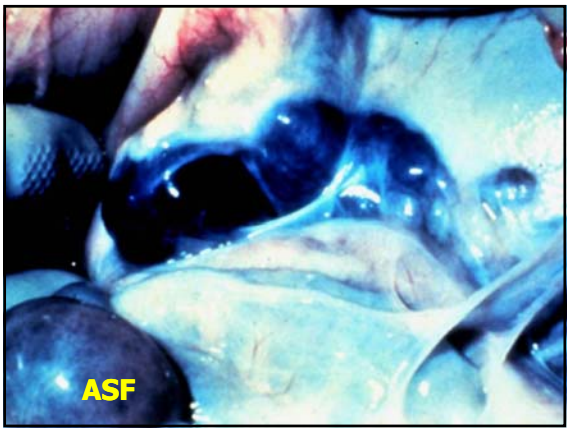
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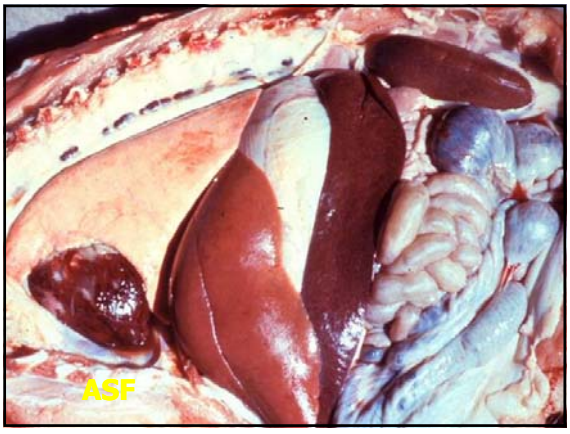
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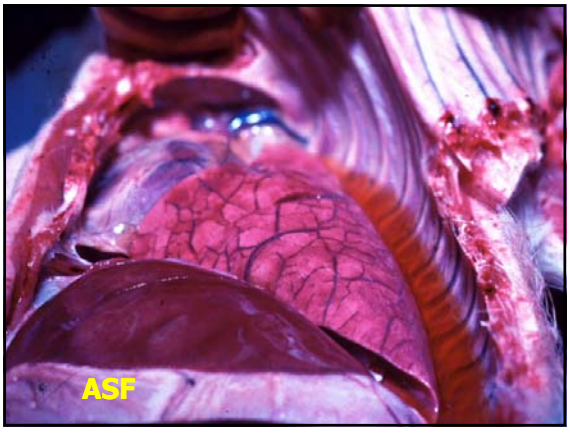
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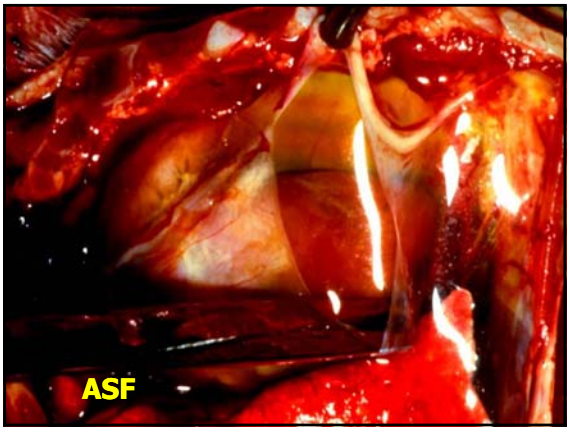
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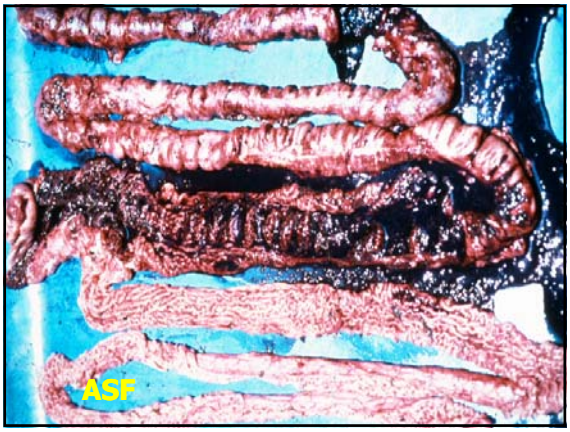
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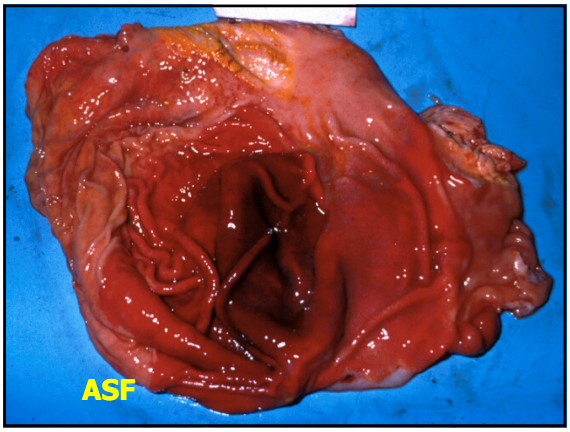
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ASF


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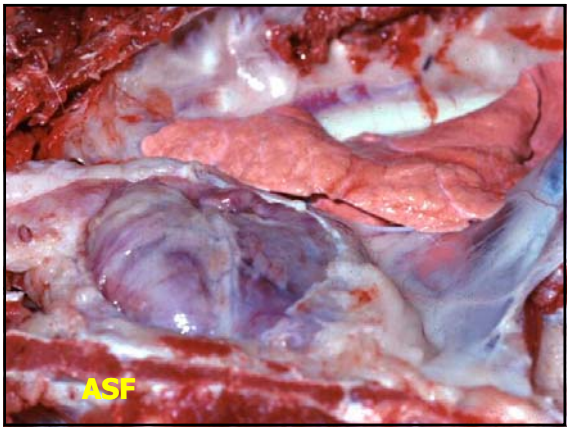
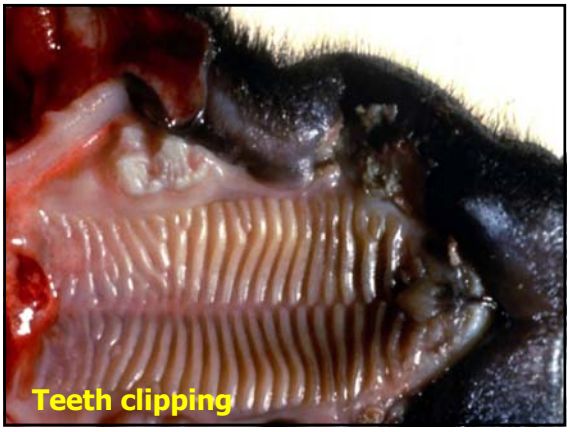


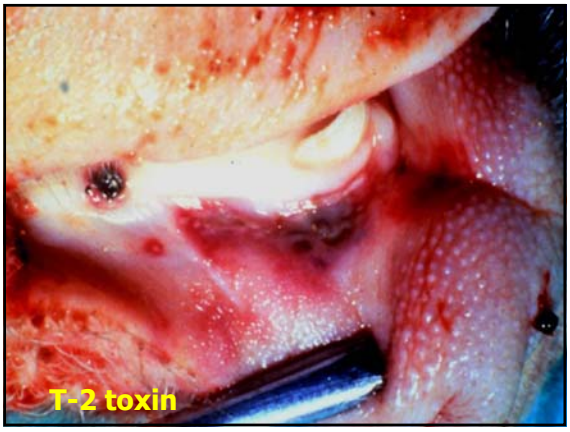


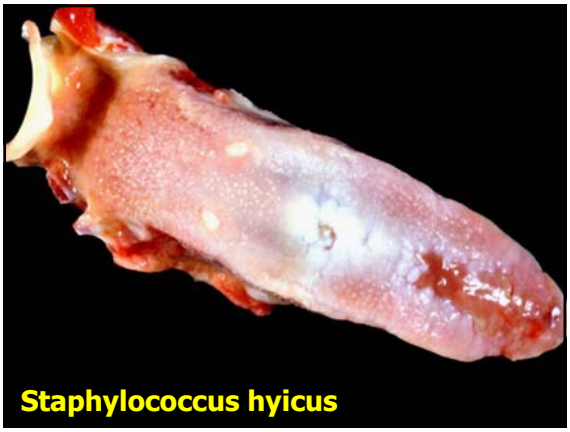

## Eperythrozoonosis

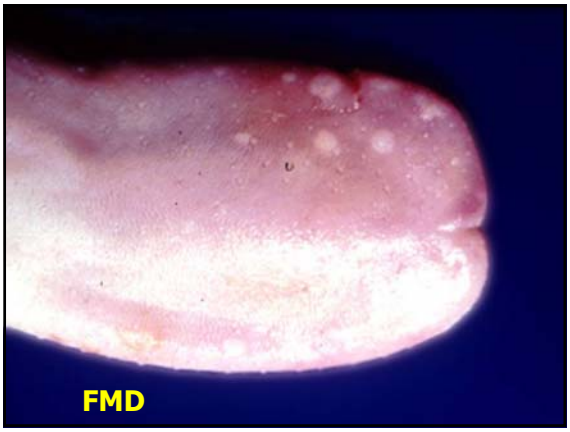
- Haemoplasma
- *Mycoplasma suis*
- Targets red blood cells
- Bacteria without cell wall
- Consistently pathogenic, but chronic or latent infections
- Worldwide distribution
- Anemia and jaundice in piglets
- Enlarged spleen and icterus
- Giemsa stain of blood smear
- Tetracycline

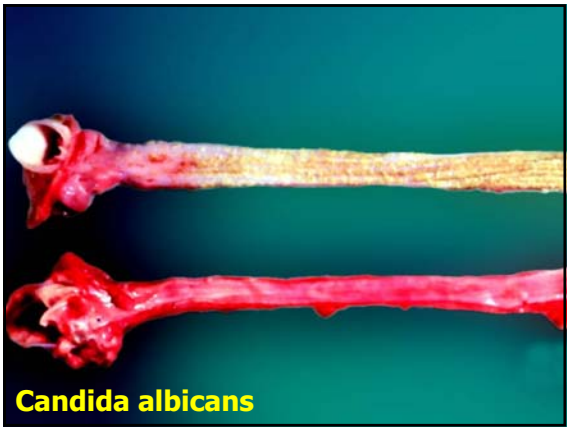

# Gastrointestinal System


Teeth clipping


T-2 toxin


Staphylococcus hyicus

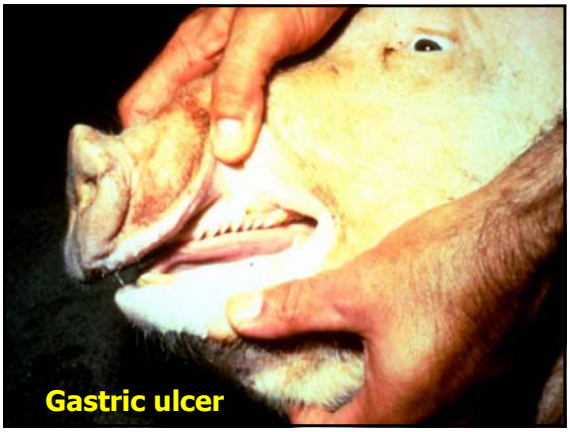

FMD


Candida albicans

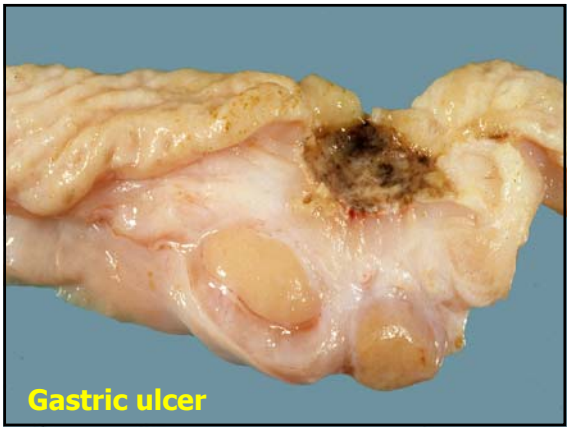

Candida albicans


## Ulceration of the pars esophagea

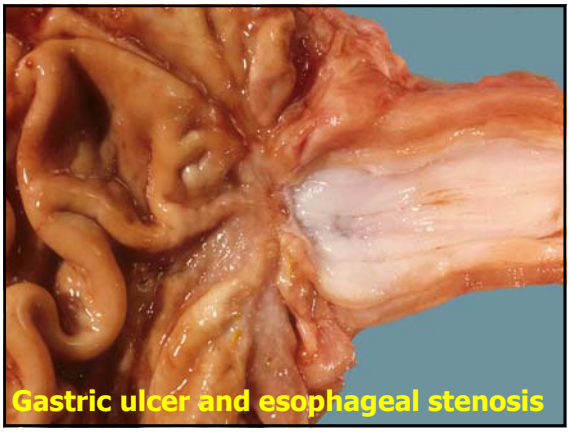
- Risk Factors
  - Gender (barrows)
  - Genotype
  - Season (summer)
  - Fine grind of feed (fine or pelleted)
  - Anorexia (concurrent disease)
- *Helicobacter sp.* ??
- ↑ carbohydrate diet + fermenting bacteria

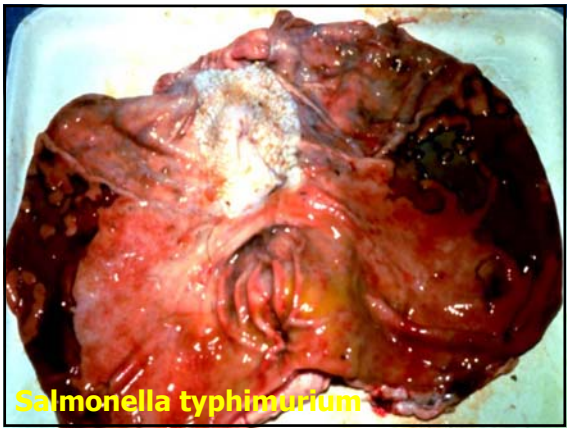






Gastric ulcer


Esophageal perforation


Gastric ulcer and esophageal stenosis


Salmonella typhimurium


Aspergillus fumigatus


Hyostrongylus rubidus


Anisakiasis


HEV-WVD

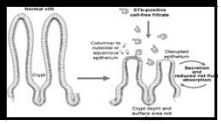

HEV-WVD


## *E. coli*

| Virotype | Virulence factor | Disease |
|---|---|---|
| Enterotoxic | pili, LT, $ST_{a,b}$ | Diarrhea |
| Enteroaggregative | bundle forming pili, EAST | Diarrhea |
| Enteropathogenic | effacing enteroadherence | Diarrhea |
| Enterohemorrhagic | SLT, effacing enteroadherence | Diarrhea, dysentery, HUS |
| Enteroinvasive | invasins, no SLT | Diarrhea, dysentery |
| Necrotoxic | fimbria, CNF-1, -2 | Diarrhea, septicemia |

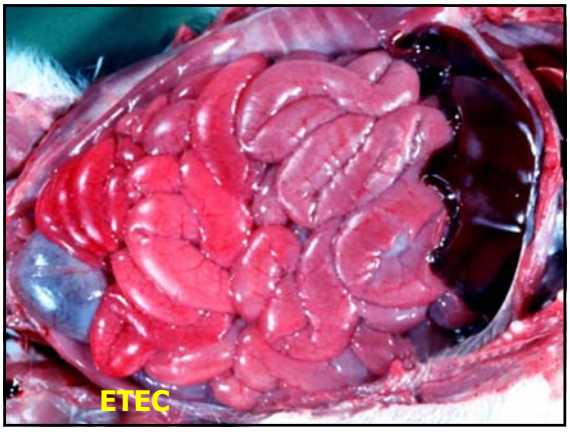
## ETEC

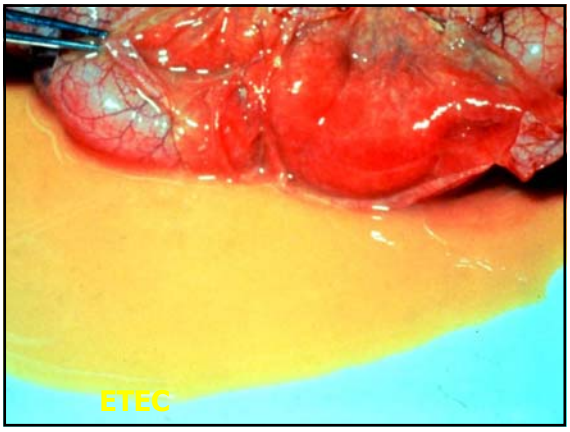
- Hemolytic or non-hemolytic
- Small intestine only
- Colonize via fimbria
  - suckling: K88, 987P, K99, F41
  - weaned: K88, F18ac (2134P)
- Secrete enterotoxin(s)
  - LT adenylate cyclase - cAMP
  - Sta guanylate cyclase - cGMP
  - STb cytotoxic, but causes villus stunting, enterocyte damage at villus tips

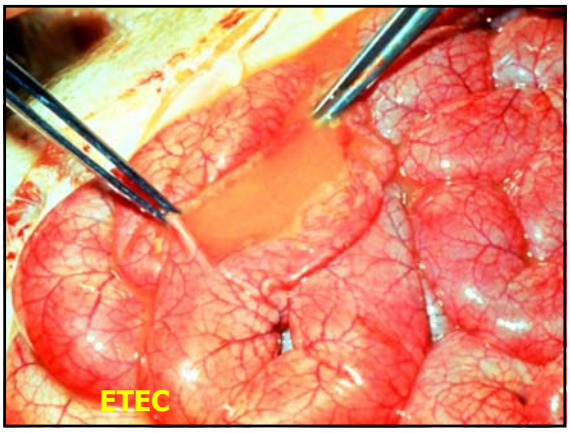


## EPEC (AAEC)

- 1-6 weeks of age, uncommon
- Small and large intestine
- Classic AE lesion
- *eae* gene product 94 kd protein “intimin”
- verotoxin negative



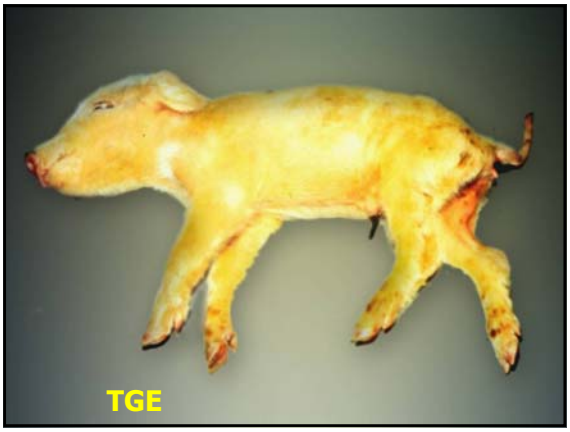



## Atrophic Enteritis

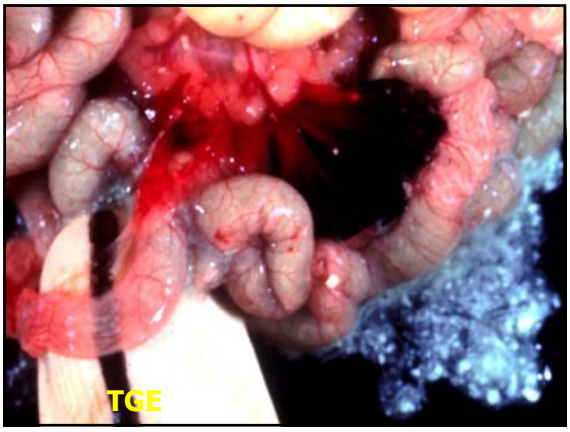
- TGE virus
- PED virus
- Rotavirus groups A, B, C
- Coccidia (Isospora suis)
- Chlamydophila
- Porcine enteric calicivirus
- Norovirus
- Sapovirus
- Astrovirus
- Parvovirus (crypt cells)

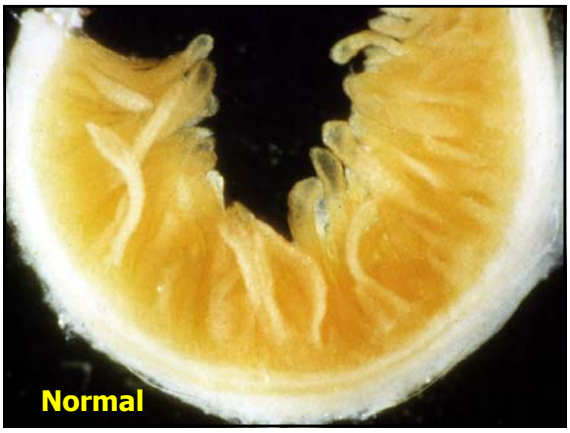
## Transmissble Gastroenteritis Porcine Epidemic Diarrhea

- Coronaviruses; infect all ages of pigs
- Replicate in small intestinal villous epithelial cells
- Cells swell and rupture or slough; 18-24 hours!
- Viral shedding in feces
  - Most shed in first 36 hours
  - Shedding usually ceases in 2 weeks (TGE)
  - Reported up to 104 days P.I. (TGE)











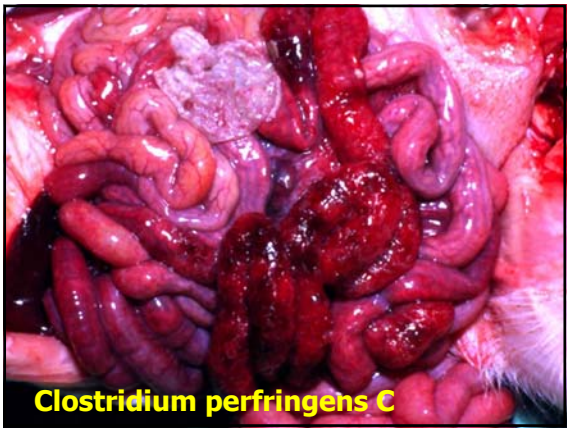
## Isospora suis

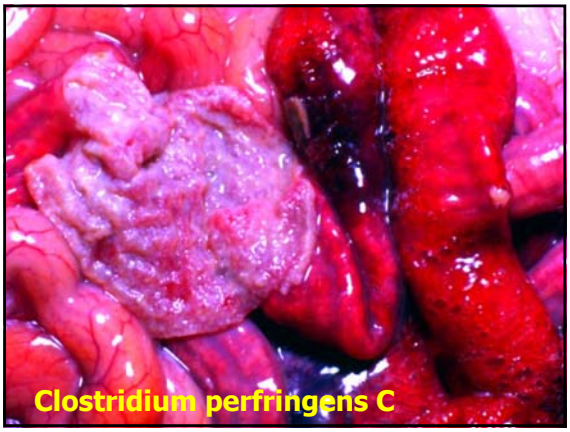
- most common in pigs from 5 days to 4 weeks-of-age
- I. suis replicates through 2 sequential asexual cycles (schizogeny) and 1 sexual cycle (gametogeny) in the cytoplasm of the epithelial cells in the small intestine
- moderate to severe atrophic enteritis
- bright yellow fibrinous mucosal pseudomembrane, can be removed with gentle scraping to reveal a glistening mucosa beneath
- rarely occurs in older pigs (Eimeria debliecki, spinosum)

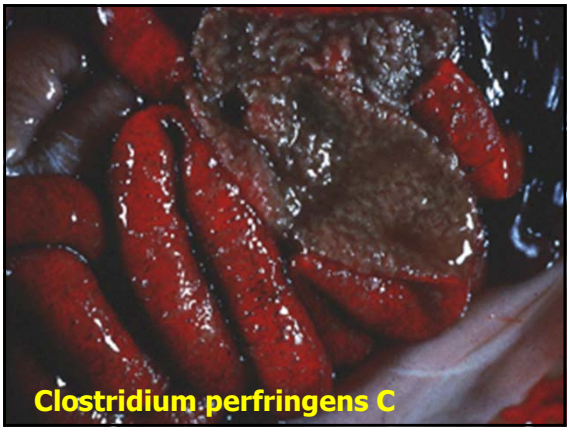


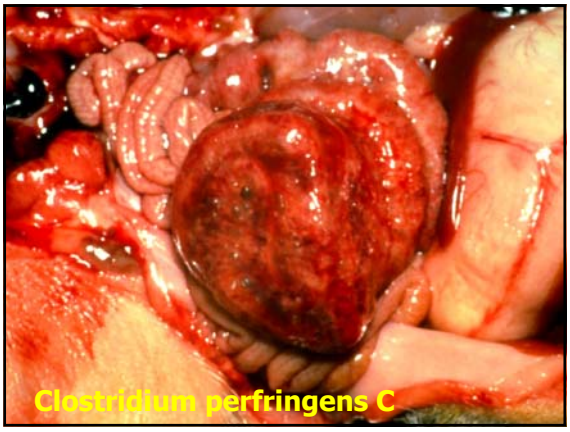
## Clostridium perfringens type C

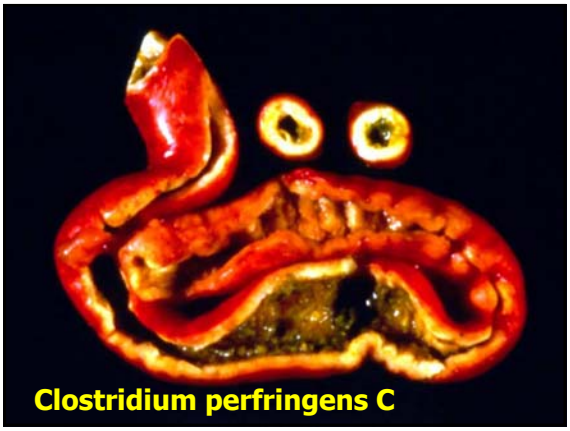
- less than 1 week of age
- some pigs may survive initially, but tend to grow poorly and die by 2 – 3 weeks-of-age
- present in small numbers in sow feces
- out-compete “normal flora” C. perfringens strains in gut
- segmental transmural necrohemorrhagic enteritis with subserosal and intramural emphysema

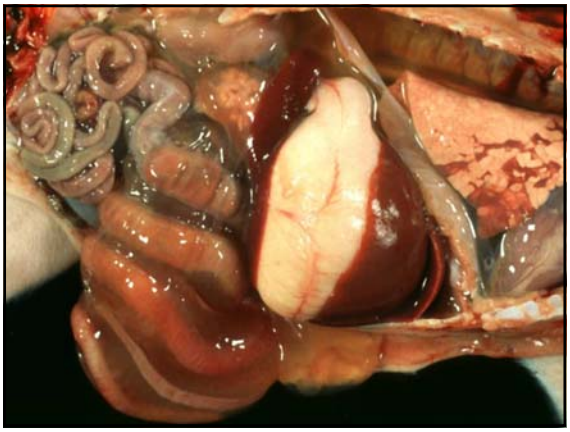

Clostridium perfringens C

## Clostridium perfringens type A

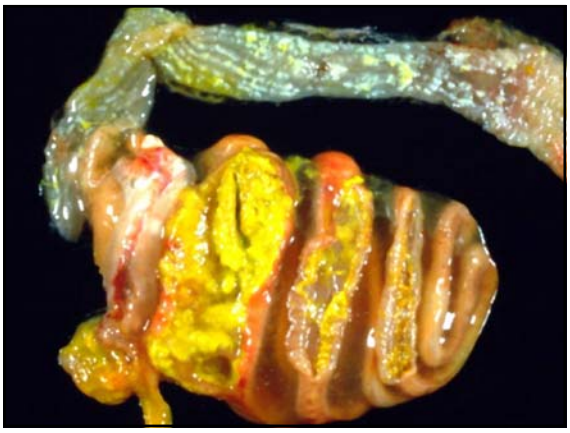
- 1 - 4 days of age
- High morbidity, low mortality
- Overgrowth of organisms in lumen
- Enterotoxin beta2
- No gross lesions
- Neutrophilic infiltrate and volcano-like eruption in small intestines?

## Clostridium difficile

- neonates; startup herds, low parity dams
- high morbidity
- average 10% loss of condition at weaning, not recovered in grow-out period
- pasty, yellow colonic contents; constipation, obstipation
- gross lesions
  - ascities, subcutaneous edema
  - mesocolonic edema, necrotizing colitis
- microscopic lesions
  - erosive colitis w/ “volcanic” exudation

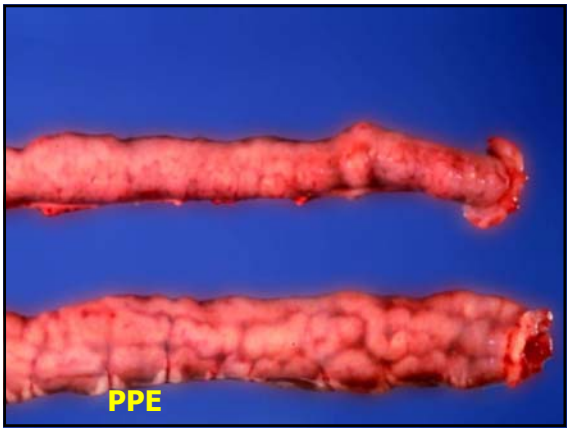


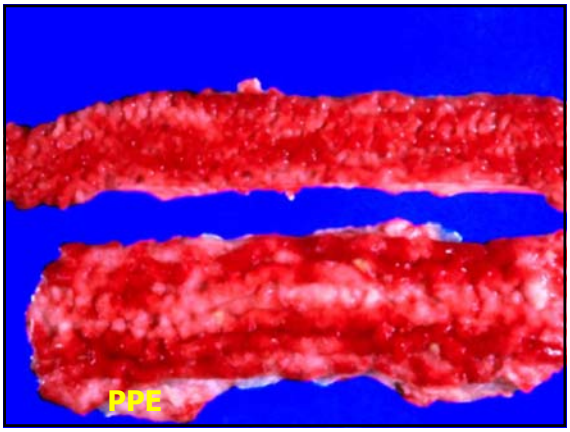

Clostridium difficile



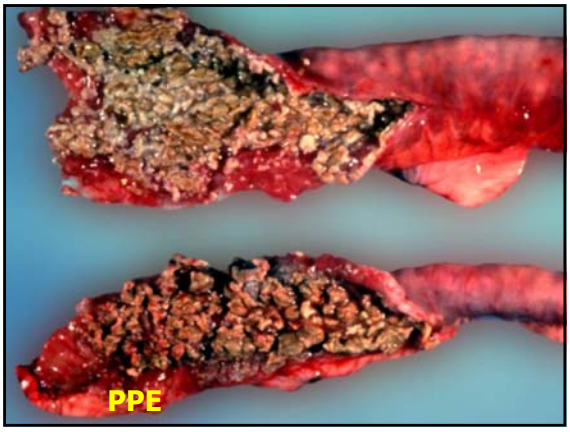
## Proliferative Enteropathy

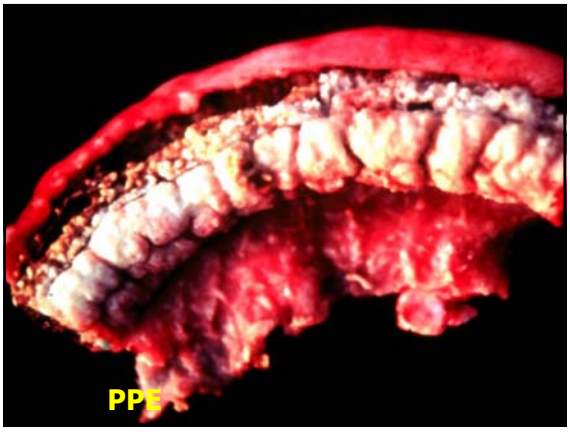
- *Lawsonia intracellularis*
- pigs, foals, foxes, ferrets, hamsters, rabbits, guinea pigs, dogs
- Proliferative enteritis, ileitis, adenomatosis, necrotic ileitis and/or typhylocolitis, hemorrhagic enteritis
- Obligate intracellular bacterium
- Koch's postulates fulfilled:
  - in SPF pigs
  - in gnotobiotic pigs with addition of *Bacteroides vulgaris* and *E. coli*



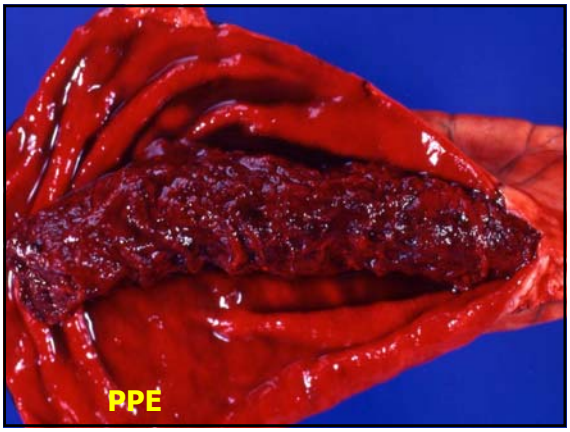




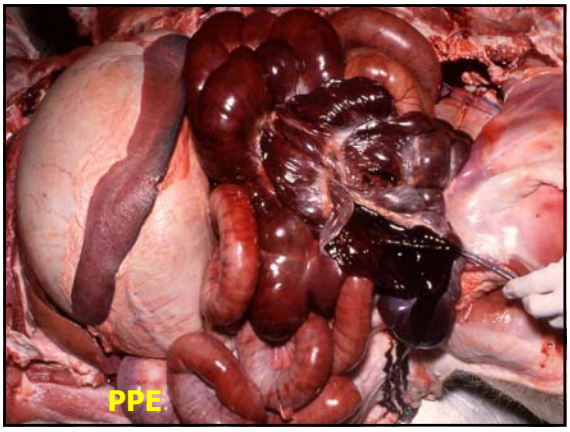

PPE

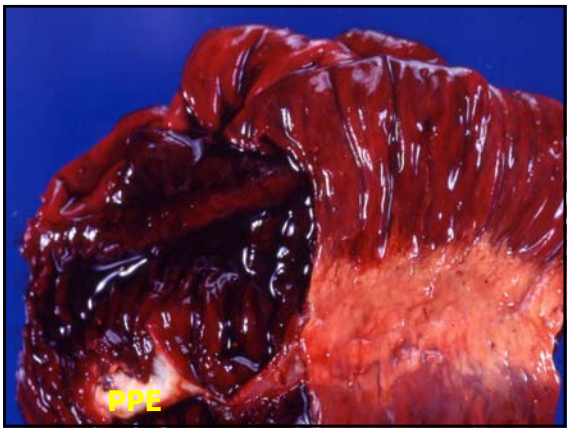

PPE


PPE


PPE


PPE

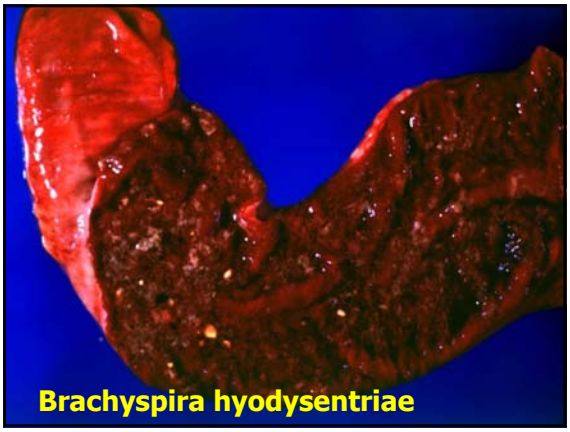

PPE

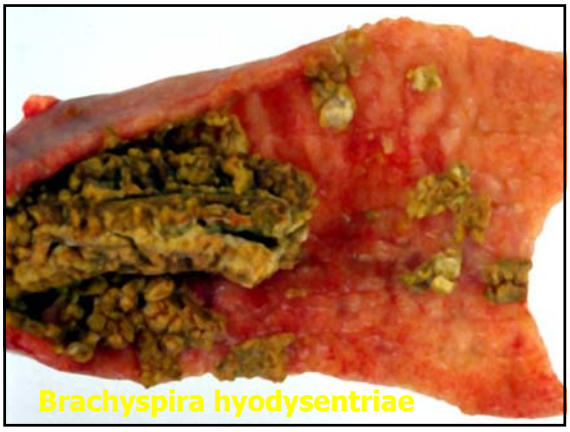



## Brachyspira sp. in Swine

| Organism | ß Hem. | # A.F. | Disease |
|---|---|---|---|
| B. hyodysenteriae | S | 7-14 | Swine Dysentery |
| B. hamsonii | S | 7-14 | Swine Dysentery |
| B. intermedia | W | 7-14 | Nonpathogenic |
| B. innocens | W | 7-14 | Nonpathogenic |
| B. murdochii | W | 7-14 | Nonpathogenic* |
| B. pilosicoli | W | 4-6 | Intestinal Spirochetosis |

Chander et al., JVDI 24:903-10
Burrough et al., JVDI 24:1025-34
Rubin et al., PloS One 8:e57146


## Colonic Spirochetosis

- weaned to adult pigs
- affects humans (potentially zoonotic), non-human primates, pigs, dogs, guinea pigs, opossums, wild and domesticated fowl
- *Serpulina pilosicoli (Anguillina coli)*
- genetically distinct from *B. hyodysenteriae* and *B. innocens*
- weak beta hemolysis, indole negative; hydrolyze hippurate
- 4-6 axial fibrils (vs. 7-14)
- mild fibrinous colitis
- colonize surface of mature colonic epithelium
  - heavy perpendicular growth “brush border”


Brachyspira pilosicoli


Brachyspira pilosicoli

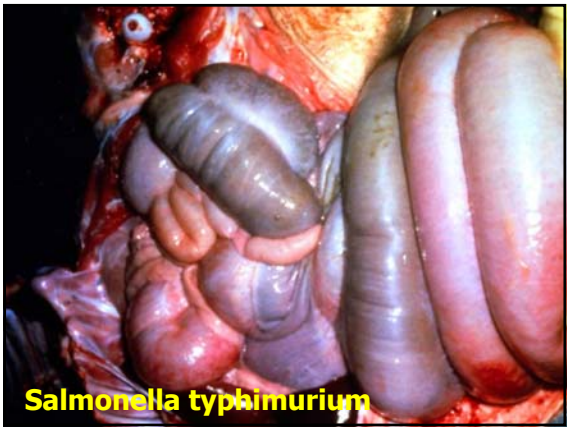

Colitis cystica

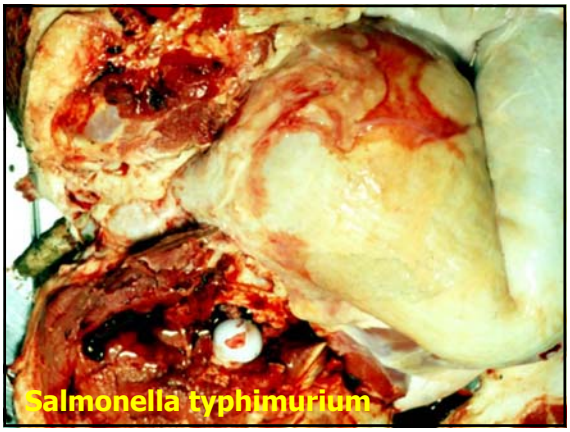

Salmonella typhimurium


Salmonella typhimurium


Salmonella typhimurium


Salmonella typhimurium

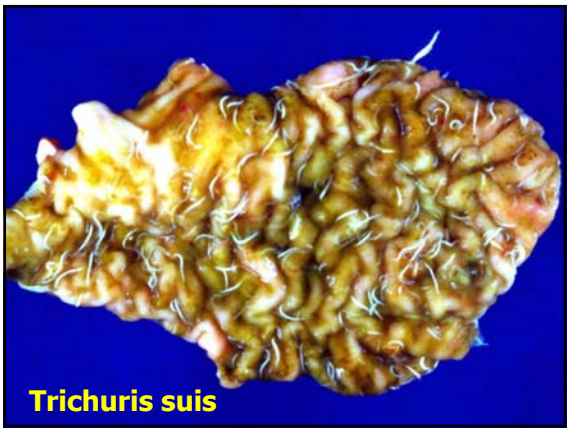

Salmonella typhimurium

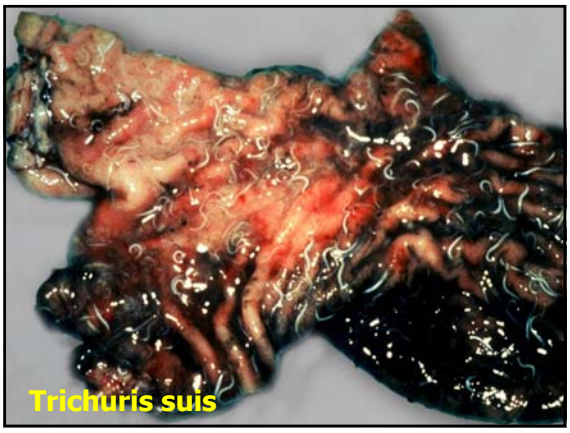

Salmonella typhimurium

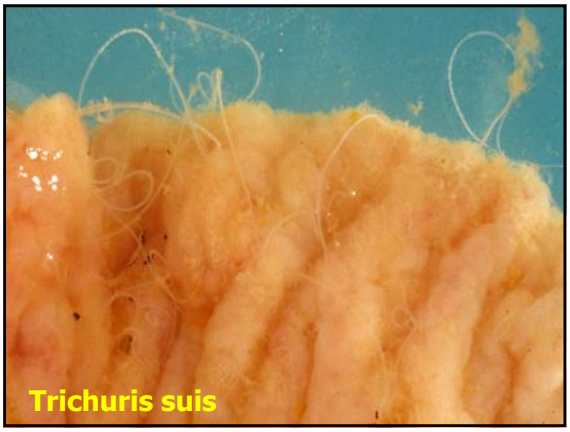

Salmonella typhimurium


Prolaps


Salmonella typhisuis

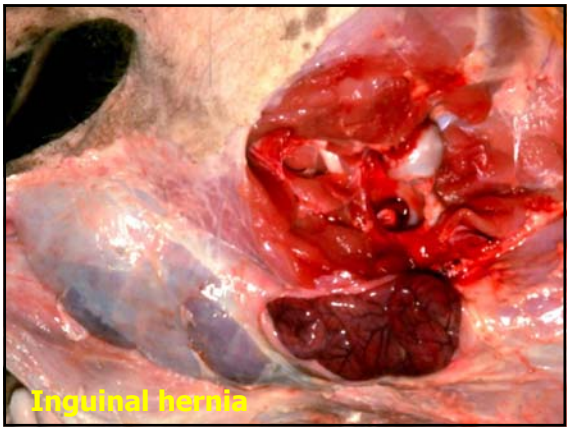

Trichuris suis

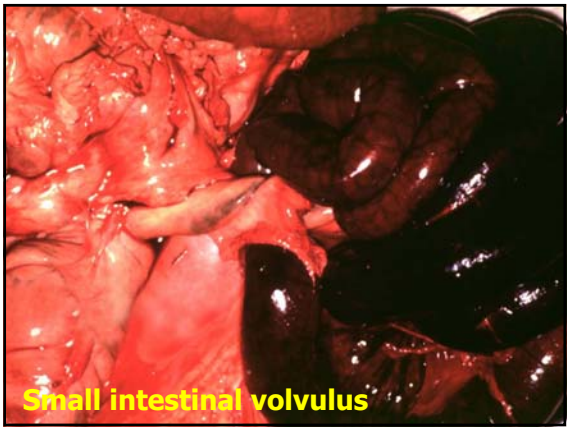

Trichuris suis

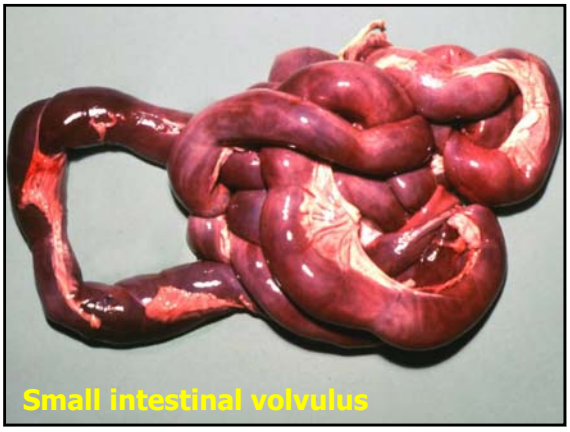

Trichuris suis


Warfarin


Warfarin

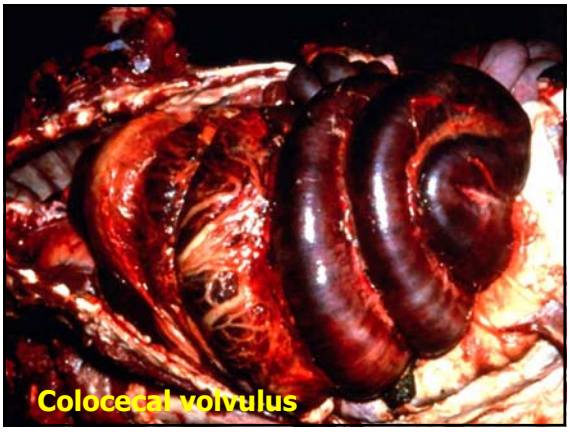

Inguinal hernia

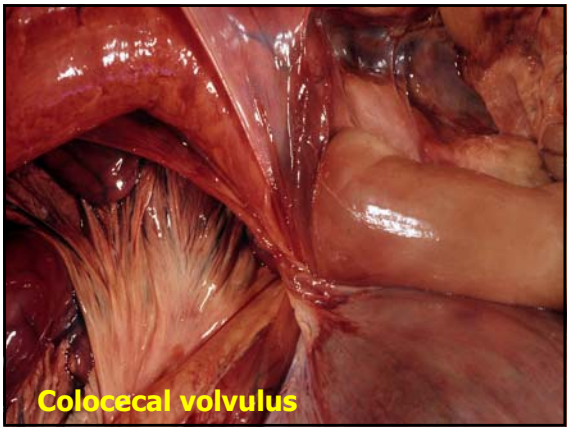

Small intestinal volvulus

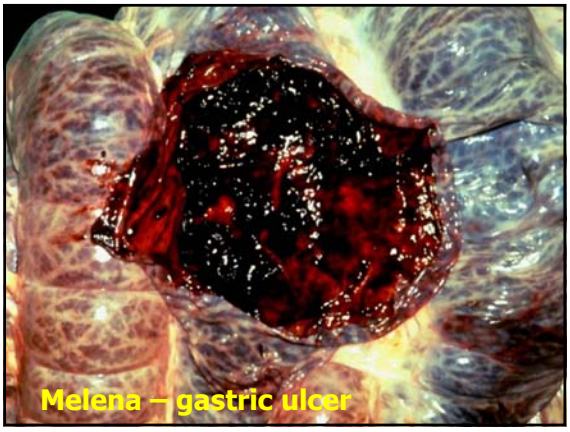

Small intestinal volvulus

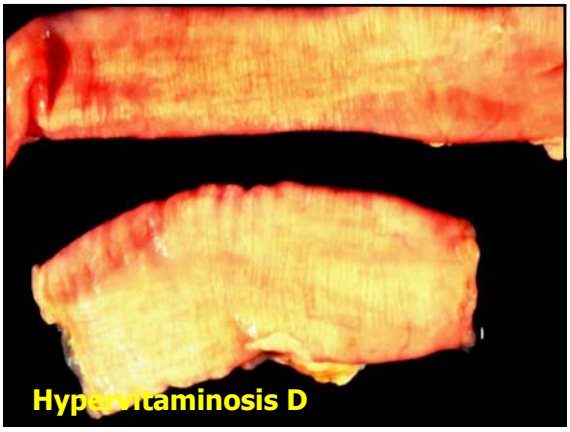

Hemorrhagic Bowel Syndrome

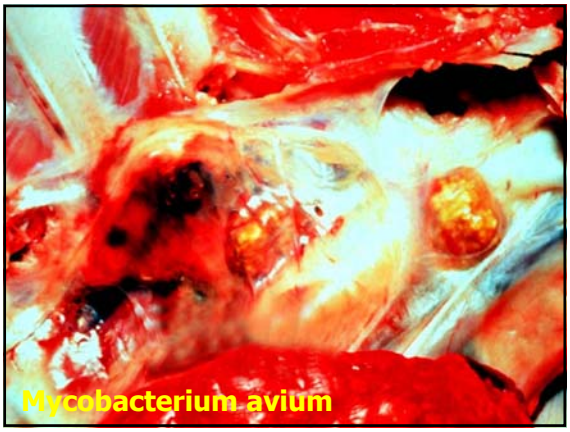

Colocecal volvulus


Colocecal volvulus


Colocecal volvulus


Melena – gastric ulcer

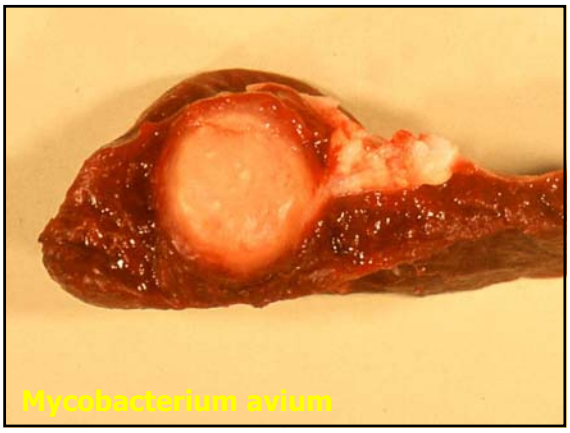

Hypervitaminosis D


Mycobacterium avium


Mycobacterium avium


Mycobacterium avium


Mycobacterium avium

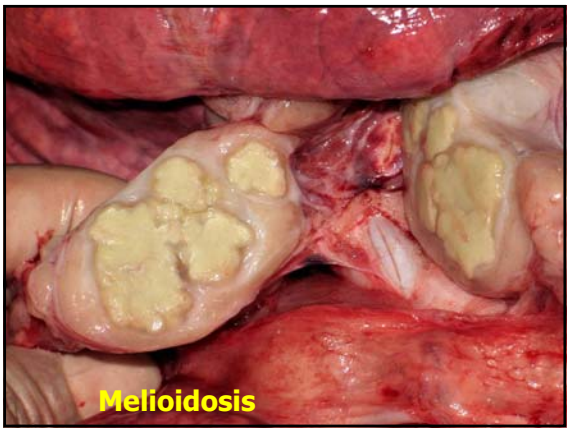




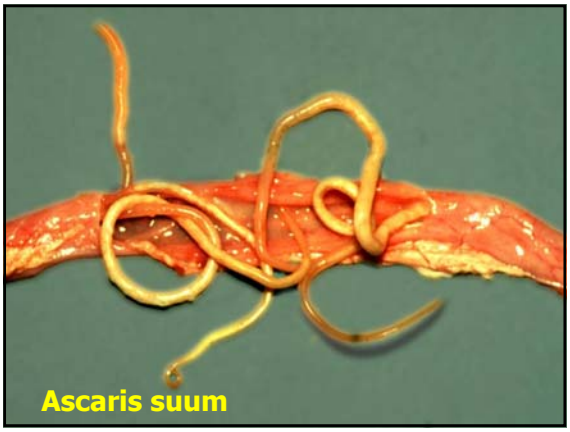
## Melioidosis

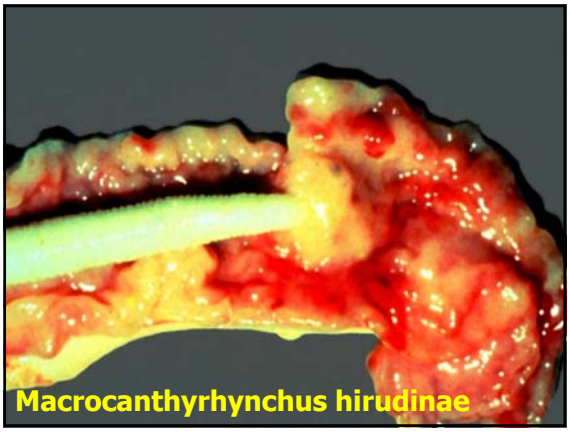
- Zoonotic infection caused by the Gram negative bacteria, *Burkholderia pseudomallei*
- South-east Asia and North Australia, South pacific
- In humans, infection is spread via direct contact with broken skin, inhalation, or by ingestion, occurs mainly in severely immune compromised patients
- Both domestic and wild animals
- Variation in host susceptibility
- Location of lesions: associated with route of infection
- Multiple abscesses, orchitis
- Non specific clinical signs
- Public health problem
- Definite diagnosis of melioidosis: bacterial culture



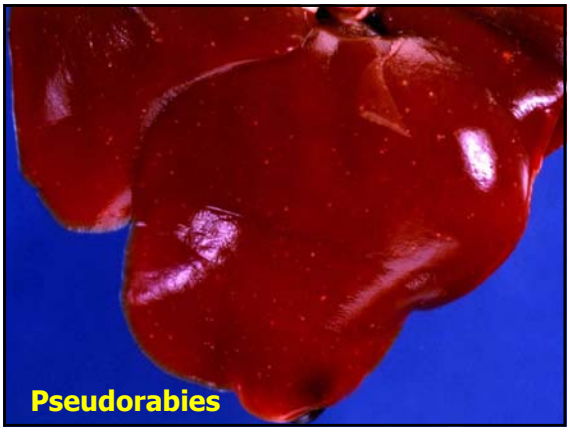

Melioidosis


Ascaris suum


Ascaris suum


Macrocanthyrhynchus hirudinae

# Liver

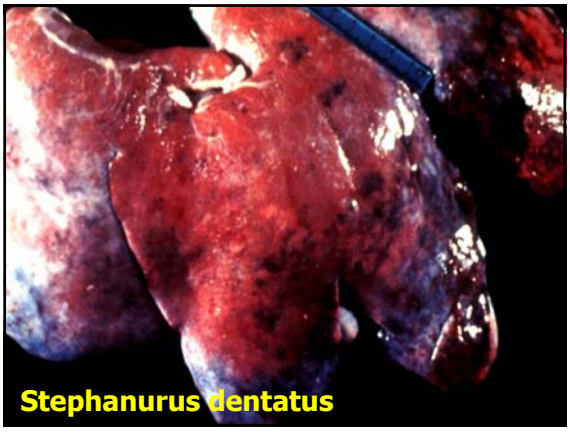

Pseudorabies


Salmonella choleraesuis


Listeria monocytogenes


Ascaris suum

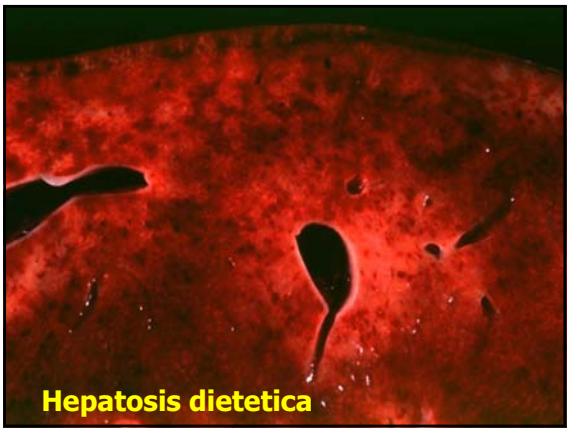

Ascaris suum

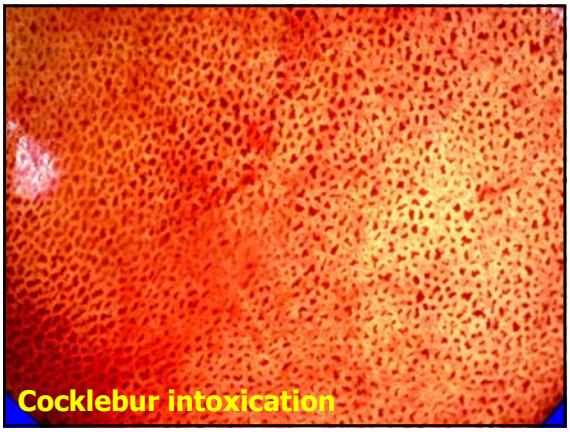

Stephanurus dentatus

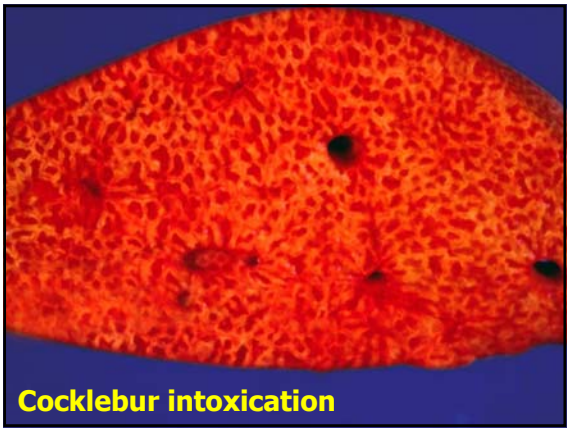
## Toxic Hepatopathy

- Hepatosis dietetica  Vit. E/Se deficiency
- Xanthium toxicosis (Cocklebur)
- Aflatoxicosis (>1200ppm)
- Gossypol toxicosis
- Coal Tar toxicity
- Fumonosin toxicosis (>80ppm)


Hepatosis dietetica

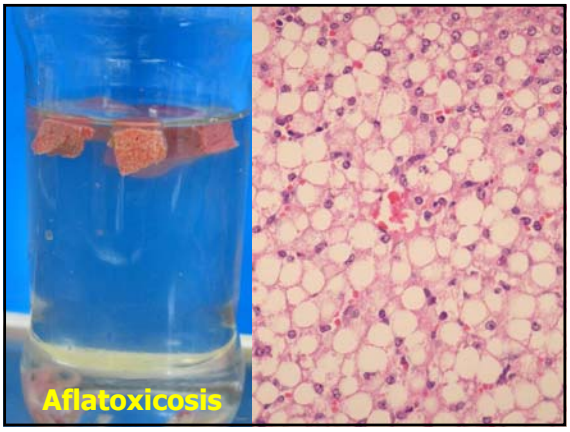

Hepatosis dietetica


Cocklebur intoxication


Cocklebur intoxication


Aflatoxicosis


Aflatoxicosis


Aflatoxicosis

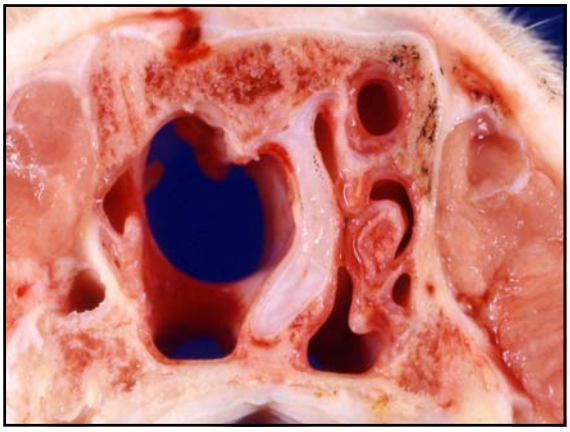

PMWS

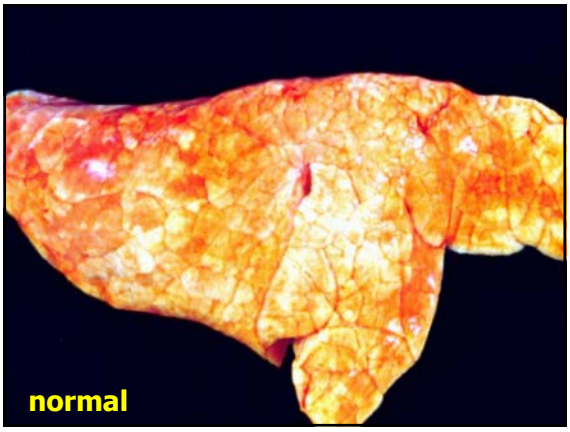

PMWS

# Respiratory System

## Progressive atrophic rhinitis

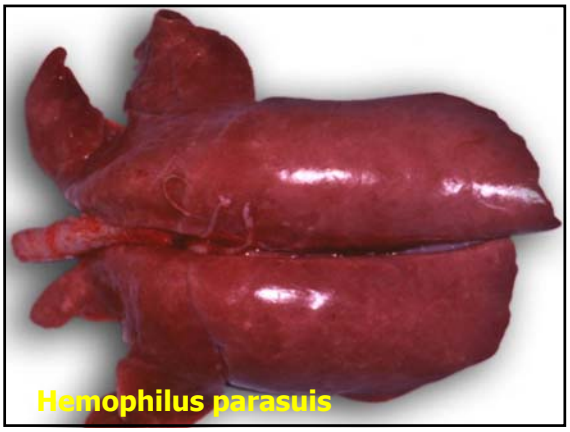
- Toxigenic *P. multocida* (usually type D) +/- co-infection with *B. bronchiseptica*
- *P. multocida* produces dermonecrotoxin > demise of osteoblasts > enhanced osteoclast activity > turbinate atrophy > distortion of nasal septum > possibly shortening and twisting of upper jaw
- Clinical signs: Sneezing (1-8 weeks of age) +/- epistaxis, blockage of lachrymal ducts with tear staining, mucopurulent nasal discharge, shortening of upper jaw and corrugation of the skin of snout
- Pathology: Rhinitis, turbinate atrophy and nasal distortion
- Diagnosis: Clinical signs confirmed by culture of nasal swab for toxigenic *P. multocida*





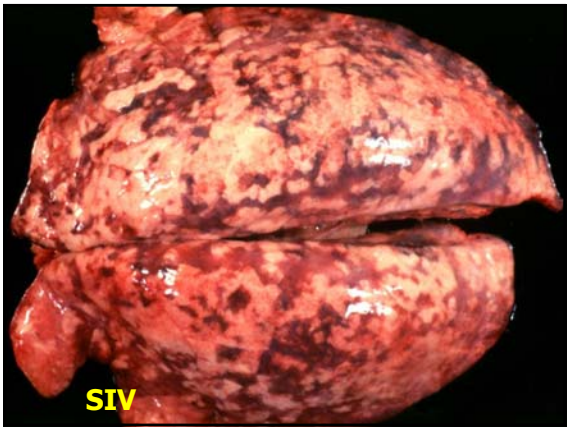
## Interstitial Pneumonia in Swine

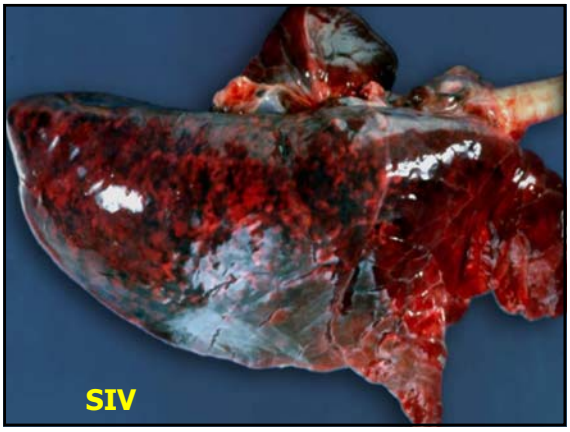
- Viral
  - Swine influenza
  - Pseudorabies
  - PRRSV
  - PRCV
  - PCV2
- Septicemia
  - S. cholerasuis
  - H. parasuis
  - S. suis
  - Other
- Allergic
  - Ascarid larval migration


## Swine Influenza

- Epizootic and endemic form
- Replicates in:
  - Epithelium of small airways within 2 h
  - Epithelium of nasal cavity, trachea, alveoli by 24 h > alveolar macrophages
- Small bronchi blocked by neutrophil-rich exudate
- Alveolar necrosis/bronchial epithelial hyperplasia causes clinical signs
- Type A
  - H1N1 later H3N2
  - also H1N2, H4N6

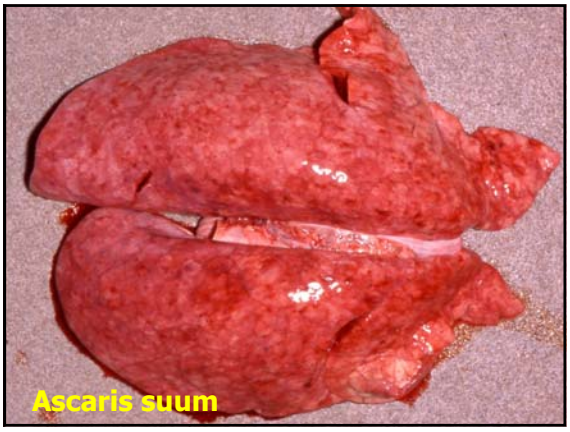



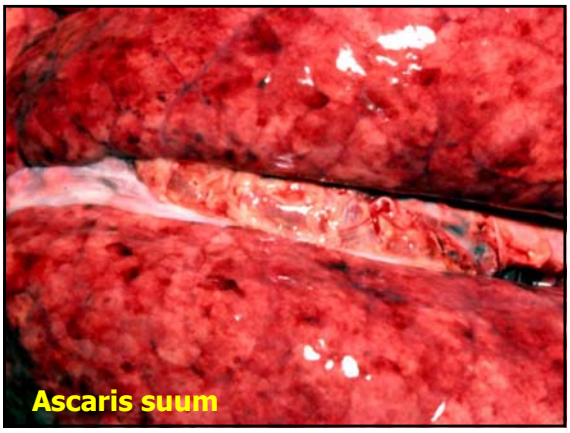




## Swine Influenza in Humans

- Pigs are important role in inter-species transmission, because they have receptors to both avian and human influenza virus strains: "mixing vessel"
- Outbreaks and sporadic human infection with swine influenza have been occasionally reported
- Influenza outbreak caused by swine H1N1 virus in Fort Dix, New Jersey in 1974
- Outbreak in Wisconsin in 1988 resulted in multiple human to human infections
- People in contact with swine have higher antibody levels
- Swine influenza viruses have been isolated from turkeys, indicating transmission between pigs and avian species
- Pigs can be infected with the highly pathogenic avian influenza (HPAI) H5N1 virus





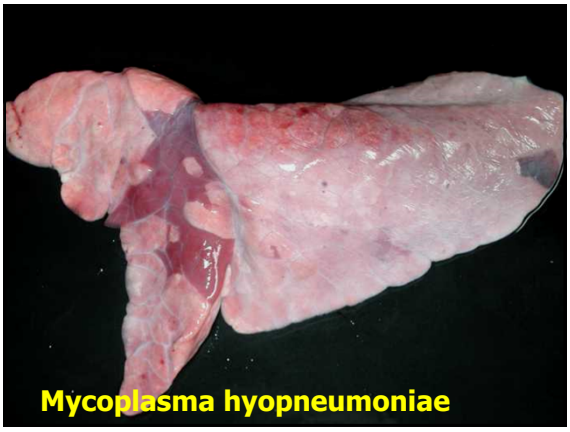

Ascaris suum


A. suis


Mycobacterium avium

## Mycoplasma hyopneumoniae

- Colonizes cilia in respiratory epithelium
- Clinical signs
  - Slowly spreading nonproductive cough
  - Depression in growth rate
  - Dyspnea, anorexia, death
- Lesions: Catarrhal bronchopneumonia
- Confirmation:
  - Antigen: Tissue FA, IHC
  - Nucleic Acid: PCR
  - *Antibodies: CF, ELISA


Mycoplasma hyopneumoniae


Mycoplasma hyopneumoniae

## Secondary Inhaled Pathogens
### Purulent Bronchopneumonia

- upper respiratory commensals
- follow Mycoplasma (enzootic pneumonia) or viral infections
- most common
  - P. multocida
  - S. suis
  - T. pyogenes
  - H. parasuis
- diagnosis: culture

## Pasteurella multocida

- common in nearly all swine herds
- non-toxigenic and toxigenic strains
- most common bacterial isolate from pneumonic lungs in slaughter swine
- lung isolates
  - most are capsular type A
  - most are serotypes 3 or 5 (of 16 total)
  - toxin as a virulence factor??
  - some strains: pleuritis, abscessation



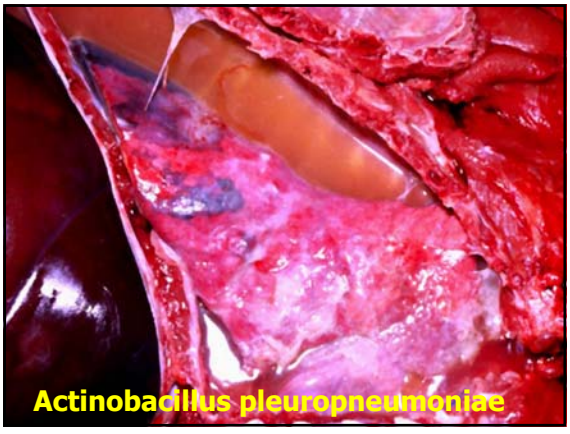

## Bordetella bronchiseptica

- primary or secondary inhaled pathogen
- pathogenesis: Colonisation and destruction of cilia in upper respiratory tract, may colonize lung causing broncho-pneumonia (cranial and middle lobes)
- primary pathogen: first few weeks of life
- lobular necrohemorrhagic bronchopneumonia
  - coughing, sneezing +/- epistaxis and mucopurulent nasal discharge, mild (reversible) turbinate atrophy (regressive atrophic rhinitis), death
- chronic progressive bronchopneumonia
  - coughing and poor growth



## Actinobacillus pleuropneumoniae

- fastidious encapsulated coccobacillus found only in swine
- biovar 1: require NAD for growth
- biovar 2: NAD not required for growth
- 15 serotypes
  - predominant serotypes vary by region
  - serotypes and strains vary in virulence

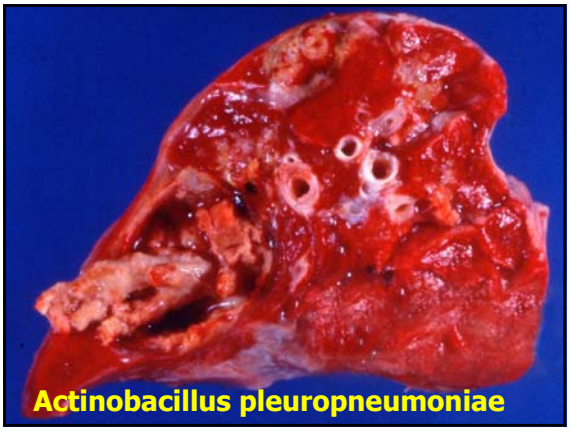


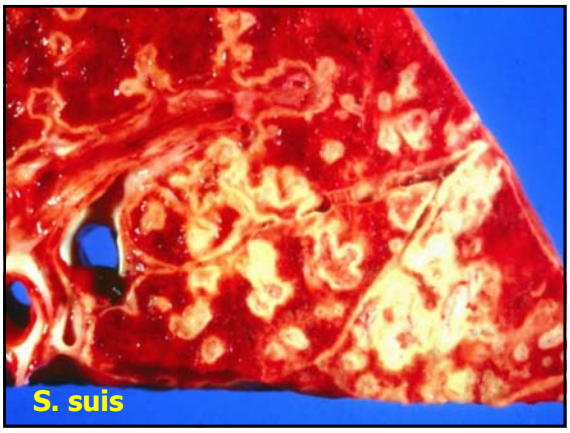
Actinobacillus pleuropneumoniae


Actinobacillus pleuropneumoniae


Actinobacillus pleuropneumoniae


Actinobacillus pleuropneumoniae

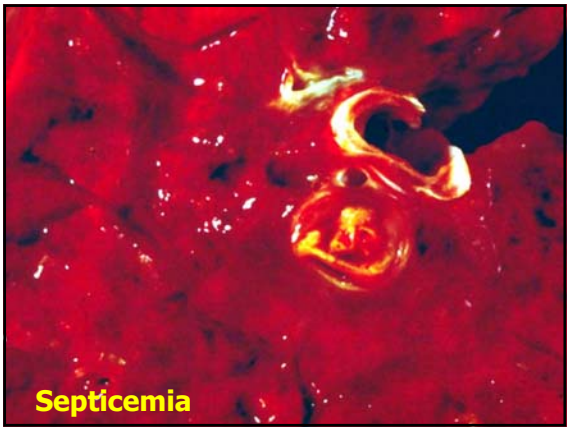

Actinobacillus pleuropneumoniae


S. suis


Fumonisin intoxication

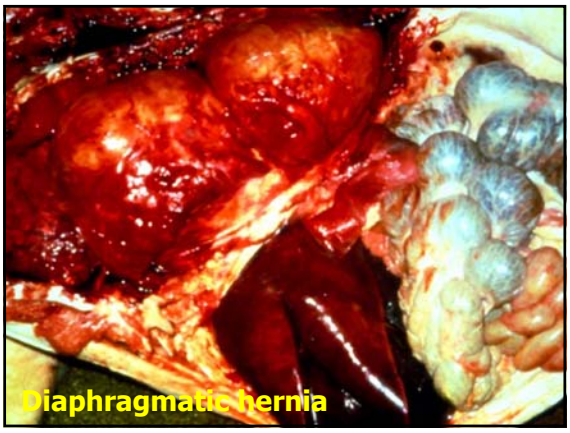

Fumonisin intoxication

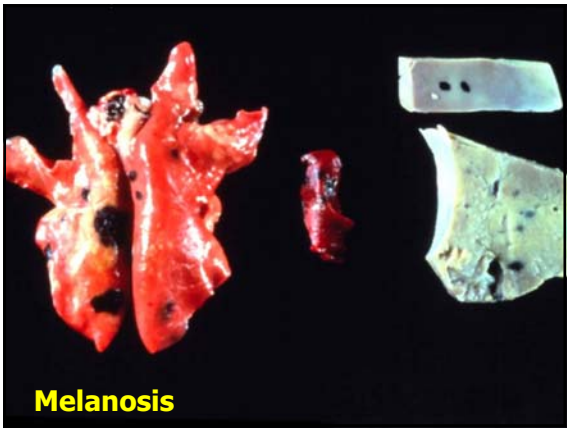

Septicemia

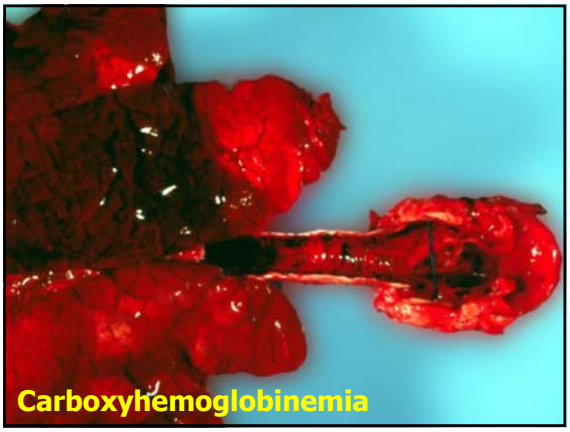

Septicemia


Metastrongylus elongatus


Metastrongylus elongatus


Diaphragmatic hernia

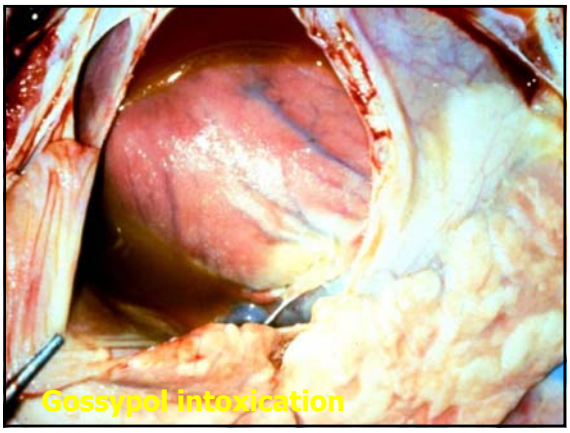

Melanosis


Carboxyhemoglobinemia

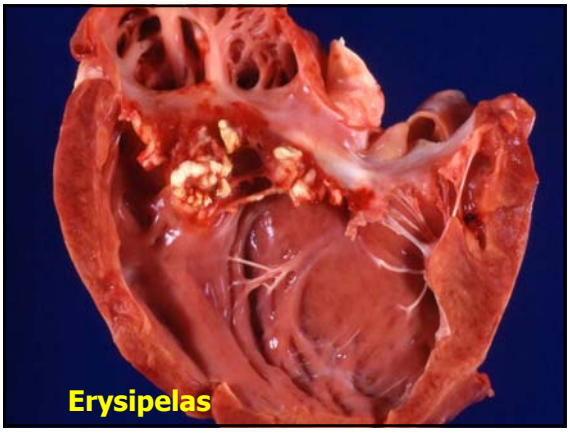
# Cardiovascular System

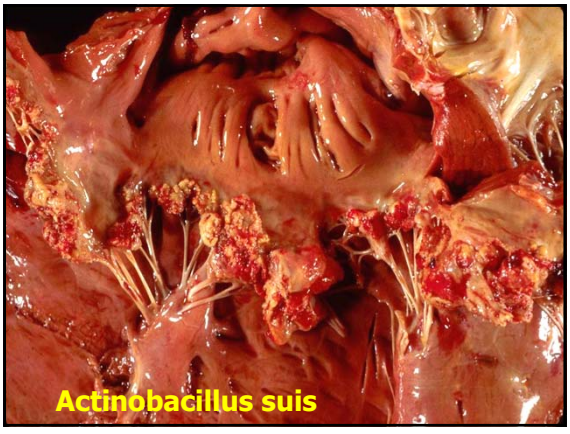

Mulberry heart disease

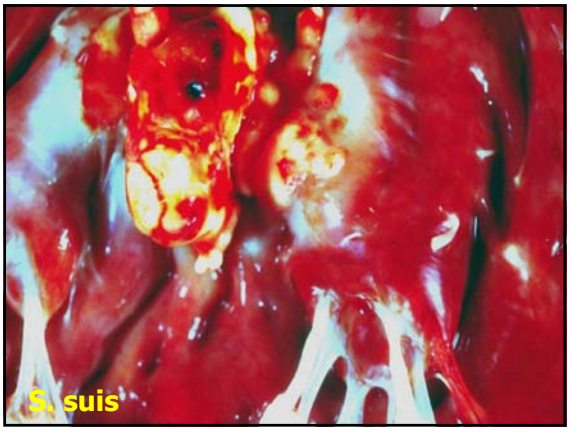

Mulberry heart disease

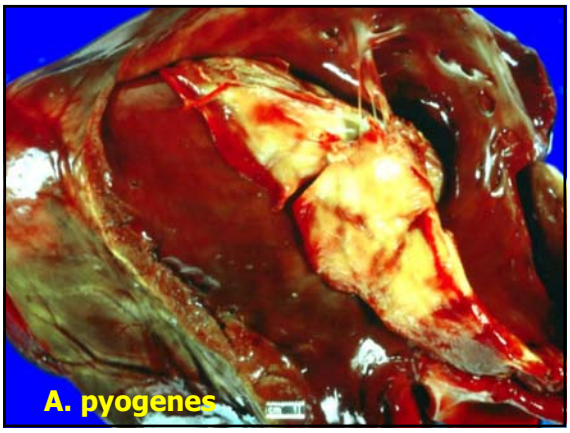

Gossypol intoxication

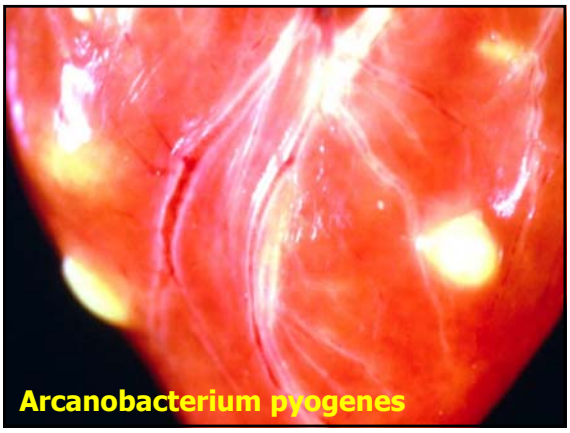

S. suis

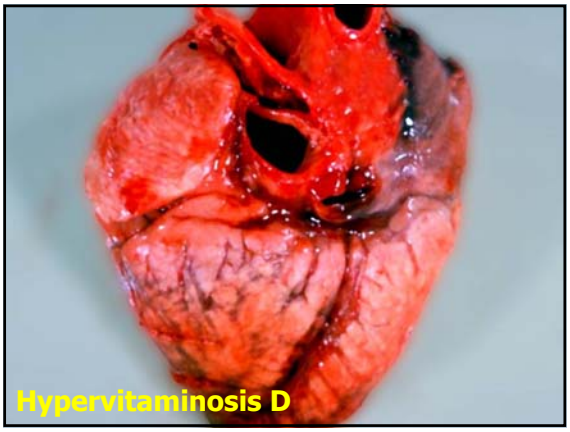

Erysipelas


Actinobacillus suis


S. suis

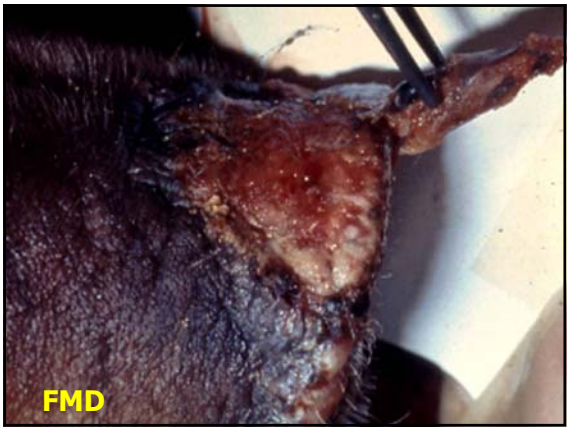

A. pyogenes

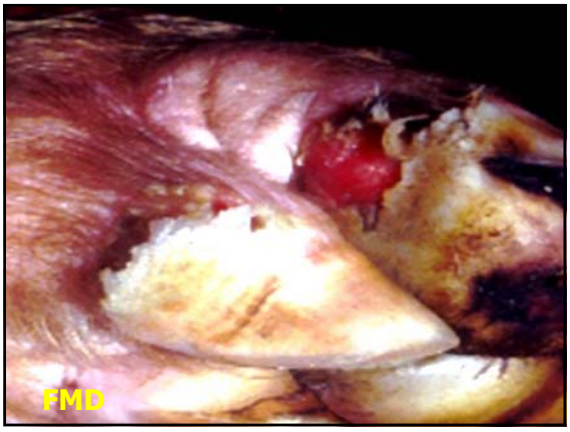

Arcanobacterium pyogenes


Hypervitaminosis D

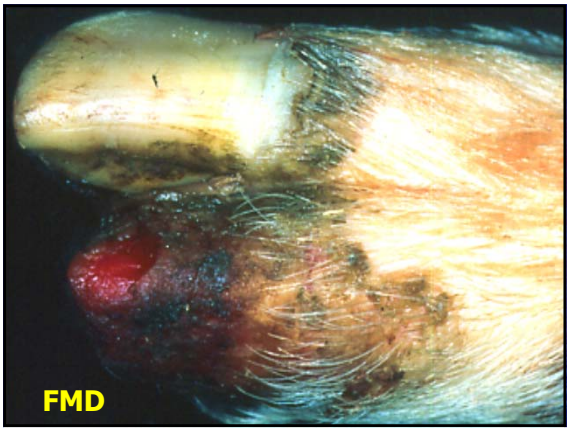
# Integumentary System


FMD


FMD


FMD


FMD

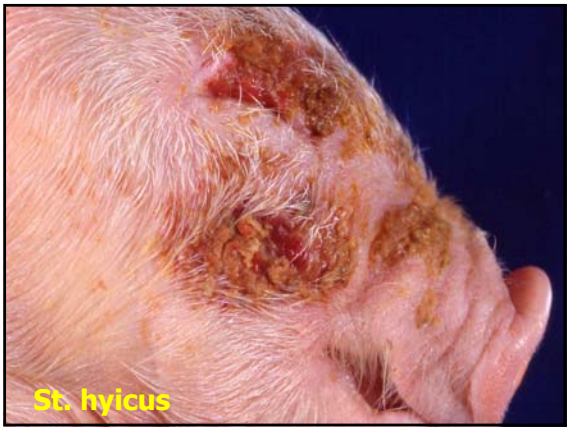

FMD


FMD


VSV


VSV

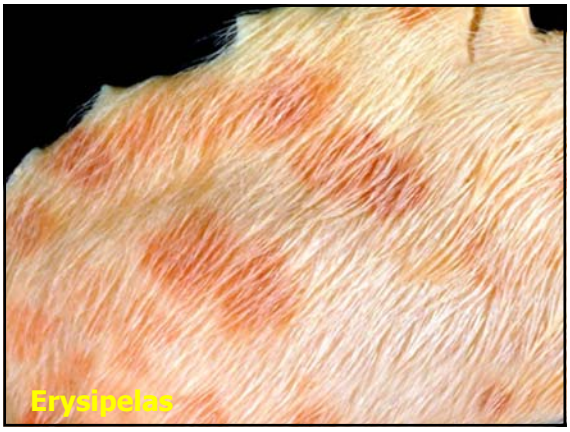

Fusobacterium necrophorum

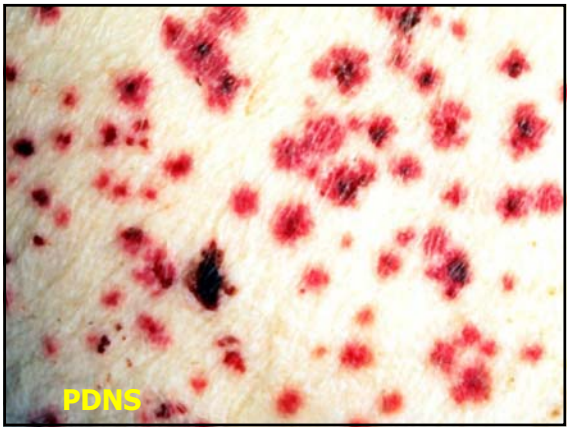

St. hyicus


St. hyicus

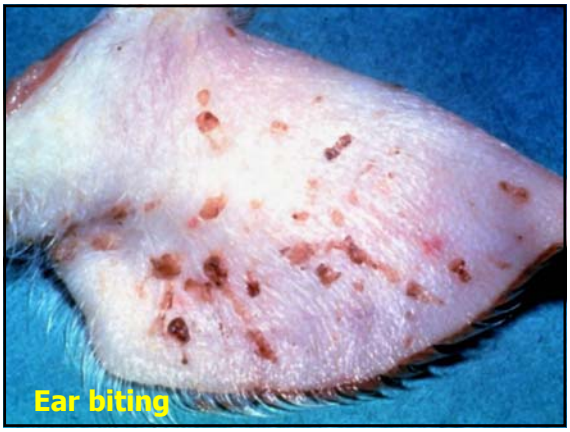

St. hyicus

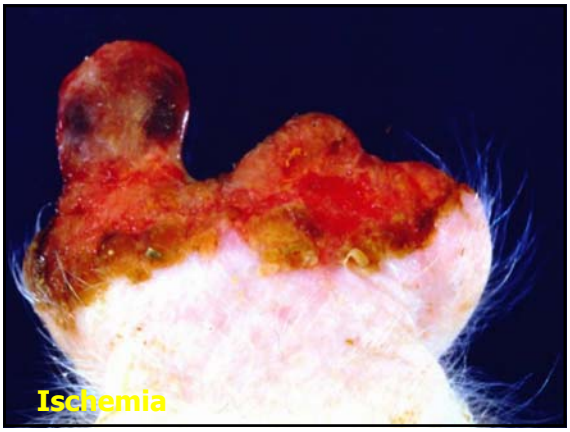

St. hyicus

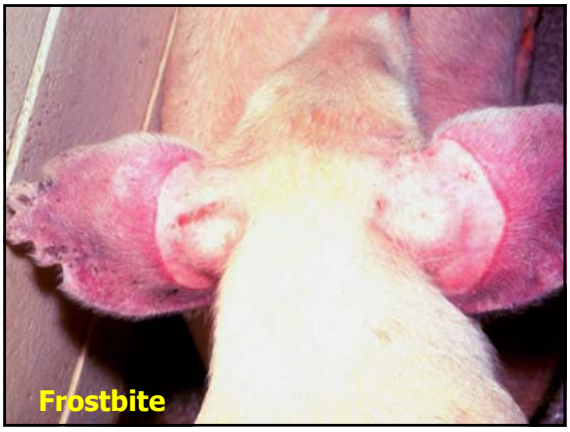

Erysipelas


PDNS


PDNS


Ear biting

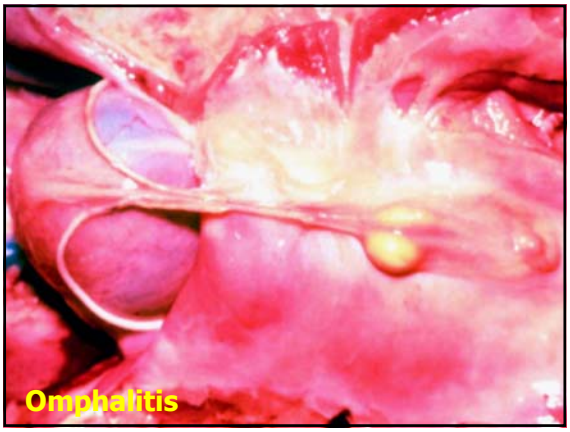

Ischemia

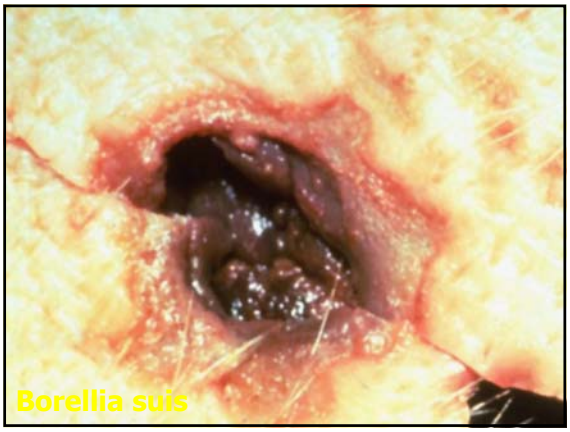

Frostbite

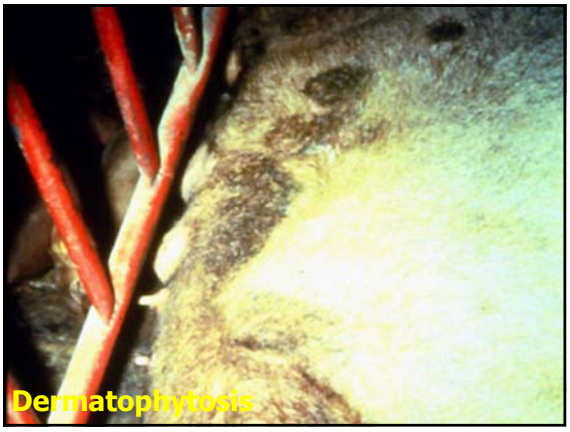

Frostbite

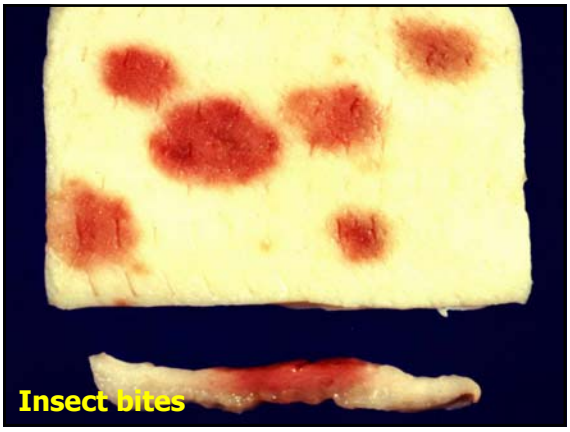

Tail biting


Navel ill


Omphalitis


Borellia suis


Dermatophytosis


Insect bites


Mange


Swinepox


Swinepox


Swinepox


Swinepox

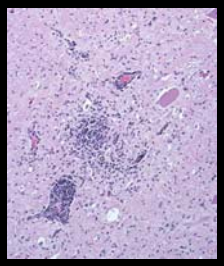

Swinepox

Swinepox

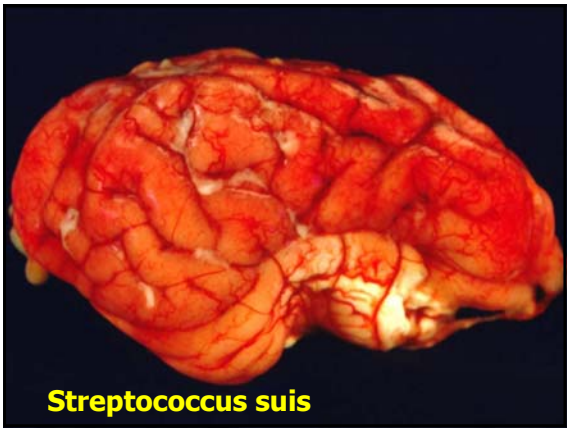
Zinc deficiency

# Nervous & Musculoskeletal Systems

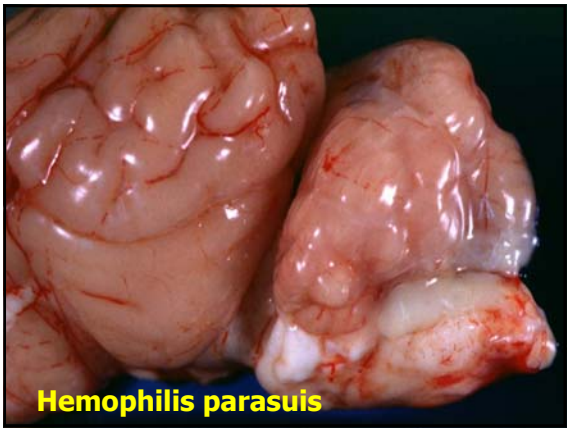
## Viral Encephalitis in Swine



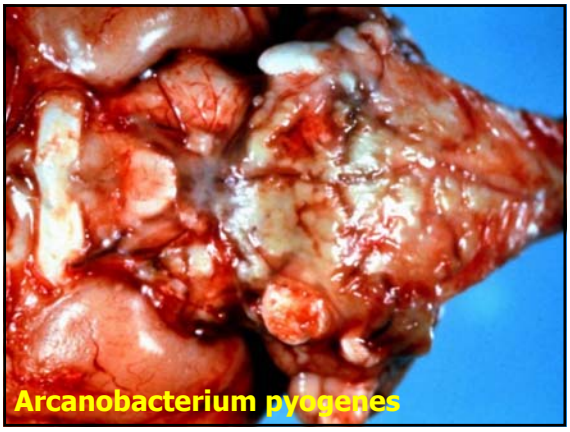
- Pseudorabies virus
- Teschovirus
- H.E.V. (coronavirus)
- E.E.E.V.
- Paramyxovirus (B.E.)
- Rabies virus
- PRRS virus
- Classical swine fever virus
- EMC virus
- Cytomegalovirus
- Nipah virus
- Japanese encephalitis virus



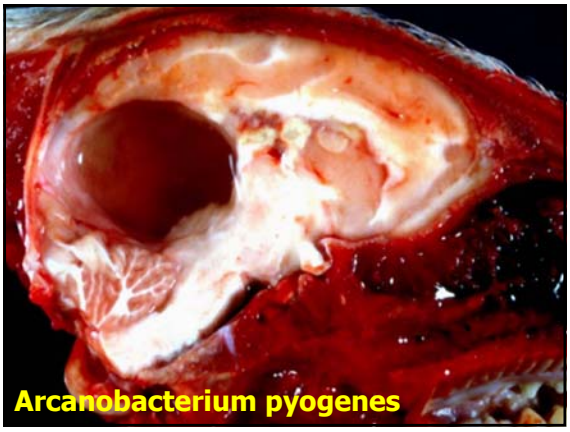
Streptococcus suis



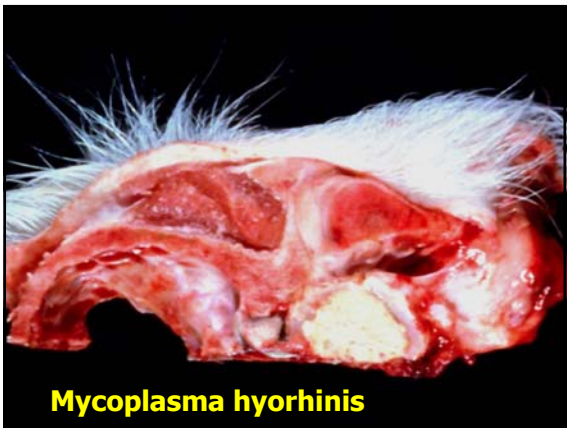
Hemophilis parasuis

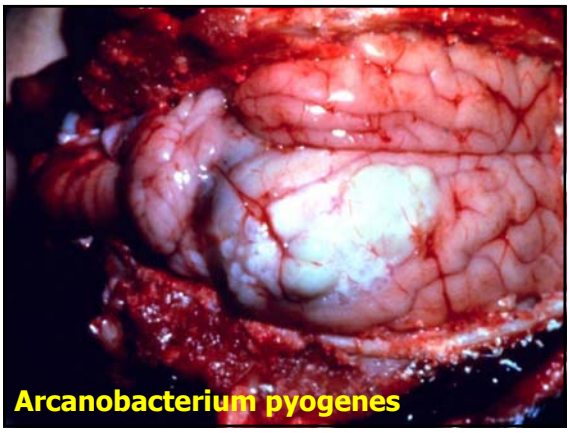


Arcanobacterium pyogenes



Arcanobacterium pyogenes


Mycoplasma hyorhinis


Arcanobacterium pyogenes

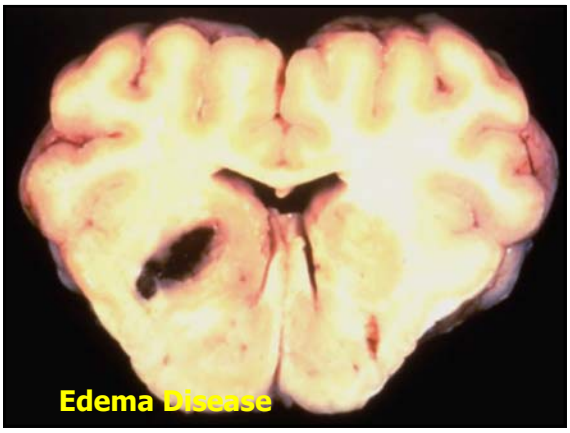
## ETEEC: Edema Disease

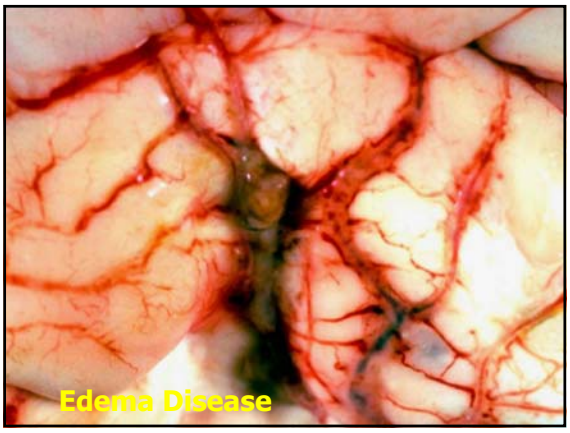
- Post-weaning, sporadic epizootics
- Hemolytic; O138, O139, O141
- F18ab (F107) fimbria, S.I. Only
- SLTv induces angiopathy, +/- Sta, STb
- Delay between colonization and clinical disease of 7-9 days
- Edema: eyelids, subcutis of face, mesocolon, gastric wall, brain


Edema Disease


Edema Disease

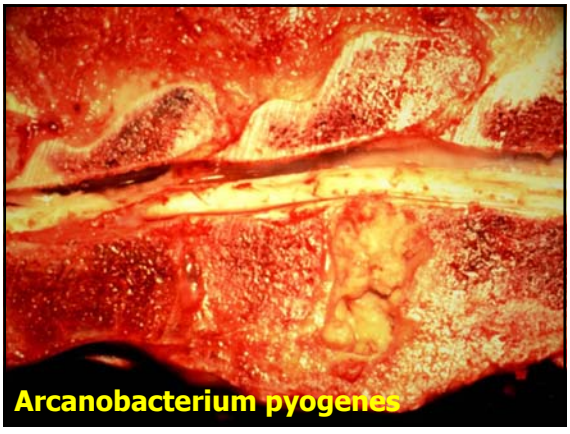

Edema Disease

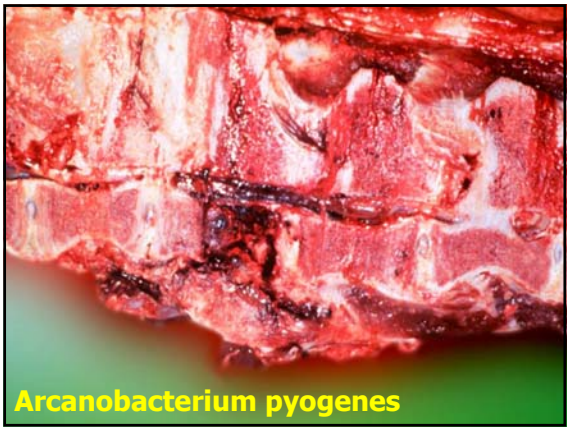



## Posterior Paralysis/Paresis in Swine

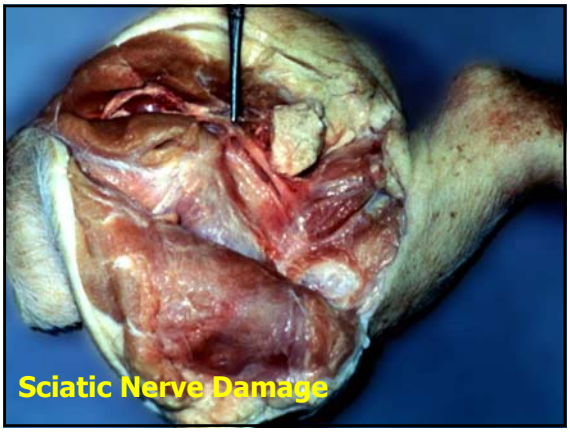
- Spinal cord
  - enterovirus
  - selenium toxicity
  - ruptured disk
  - trauma
  - lymphosarcoma
- Vertebral column
  - osteomyelitis
  - osteomalacia
- Bones and Muscles
  - Ischial epiphysiolysis
  - Torn "hamstring"
  - Osteomalacia/osteo-myelitis ➲ fracture
- Nerves
  - Organic arsenicals
  - Trauma

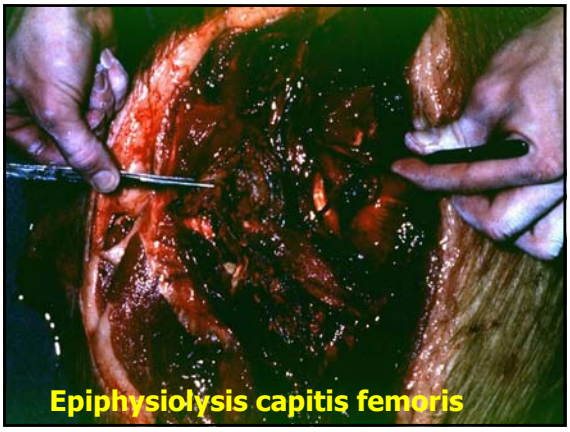




Degenerative Disc Disease

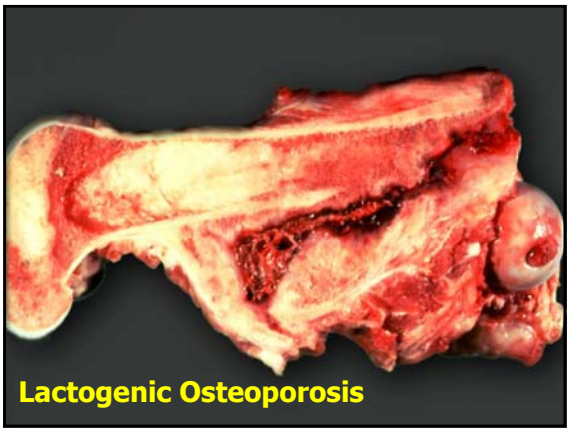

Sciatic Nerve Damage

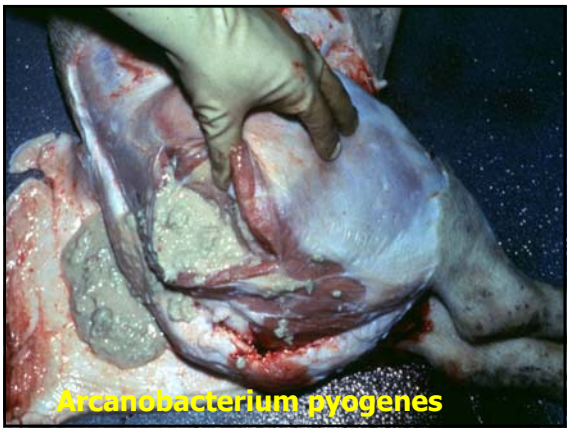

Sciatic Nerve Damage


Apophysiolysis tuberis ischii


Epiphysiolysis capitis femoris


Compartment Syndrome


Lactogenic Osteoporosis

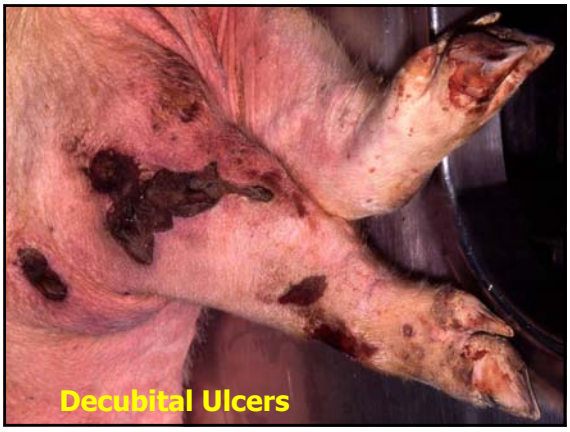

Arcanobacterium pyogenes

## Lameness in Swine

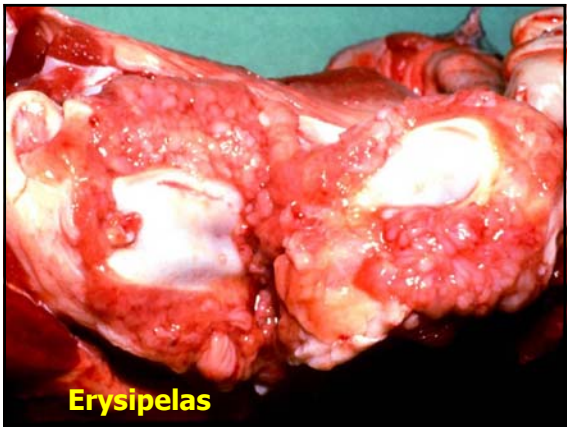
- Bacterial arthritis
  - S. suis
  - H. parasuis
  - M. hyorhinis
  - S. equisimilis
  - E. rhusiopathiae
  - M. hyosynoviae
  - A. pyogenes
- Degenerative arthritis
  - OCD
- Other
  - Fractures
  - Ruptured cruciates
  - Overgrown hooves
  - Hoof and heel abscesses


Pododermal Abrasions

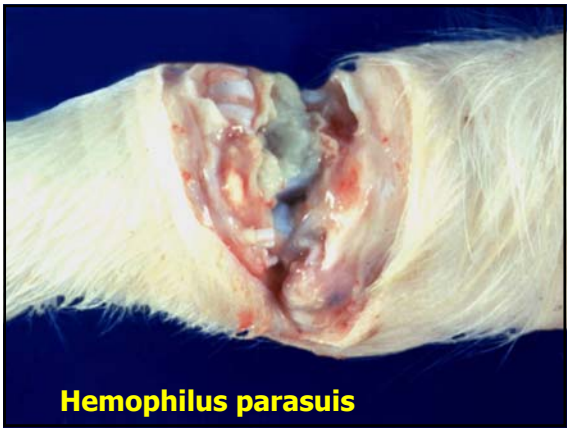

Phalangeal Arthritis

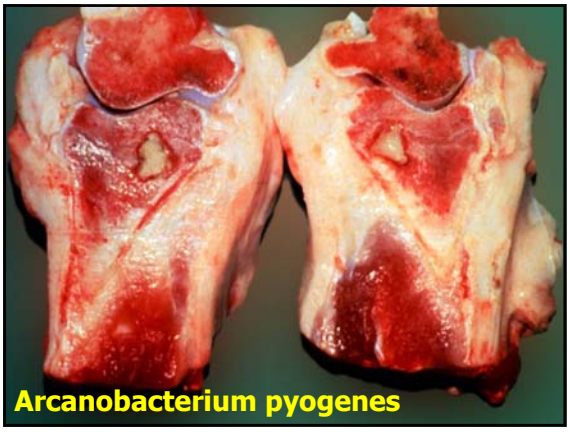

Decubital Ulcers

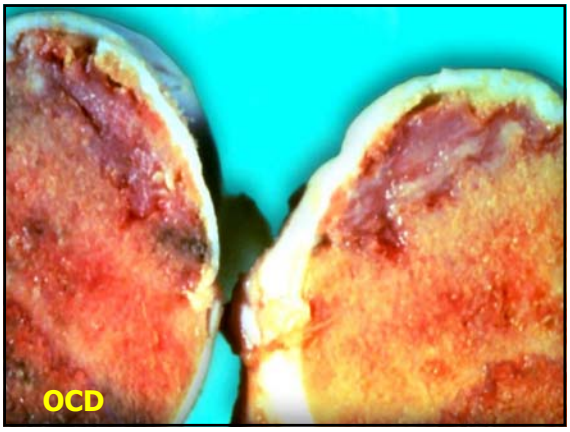

Mycoplasma hyosynoviae

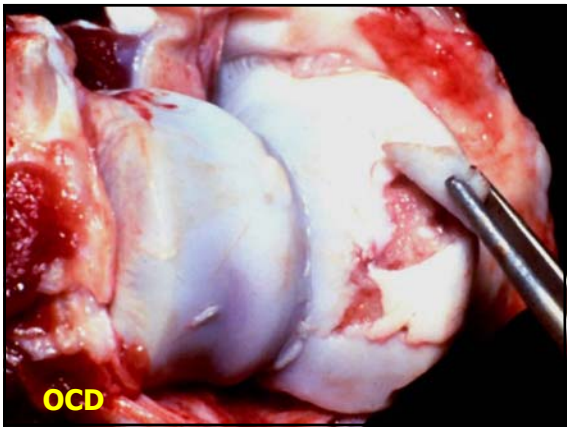

Erysipelas


Streptococcus equisimilis

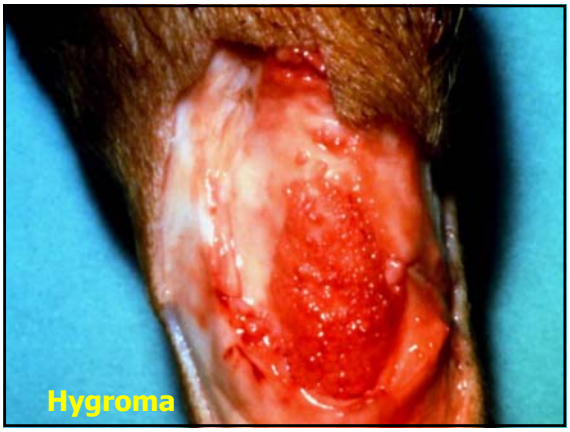

Hemophilus parasuis


Arcanobacterium pyogenes


OCD


OCD


Ruptured cruciate Ligaments

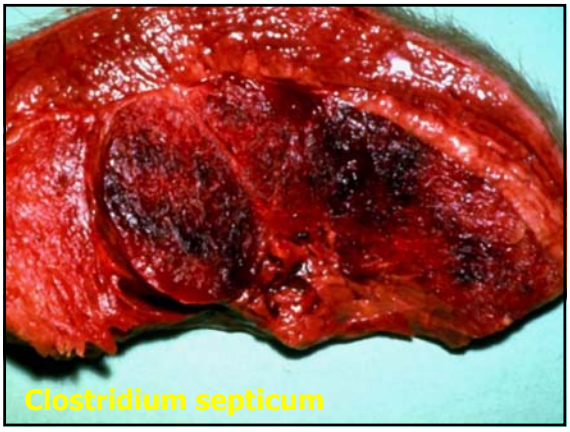

Hygroma


Anthrax


Clostridium septicum

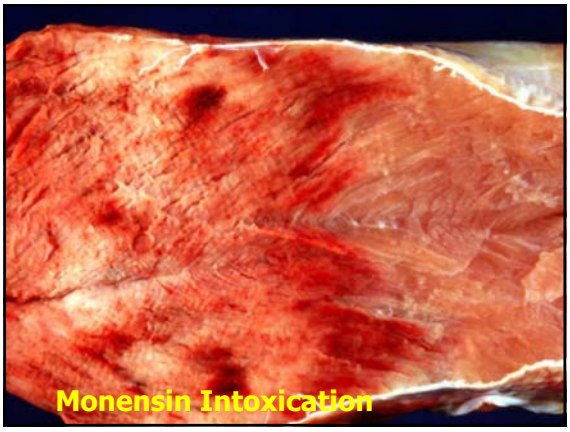

Clostridium septicum


Clostridium septicum


Clostridium septicum

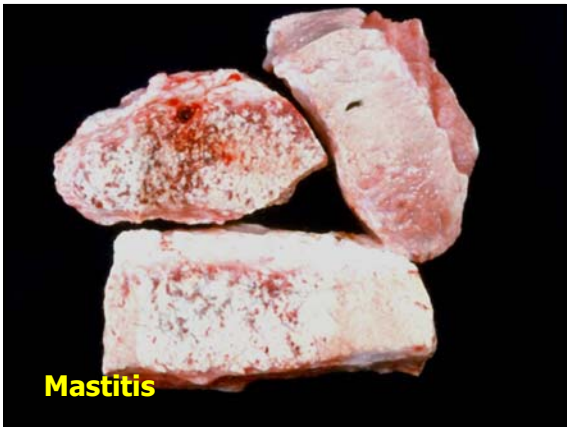

Porcine Stress Syndrome


Porcine Stress Syndrome

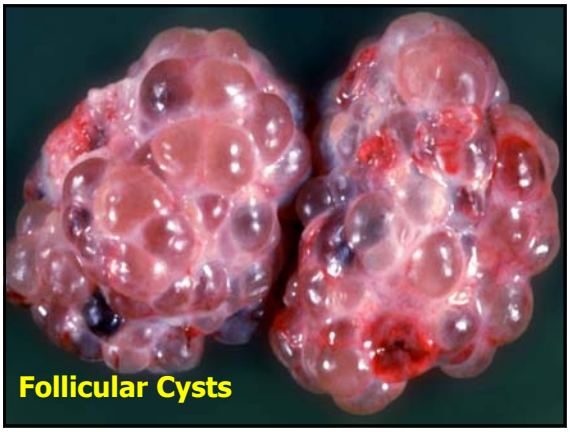

Monensin Intoxication

## Genital System


Mammary Edema


Mastitis


Active Ovary

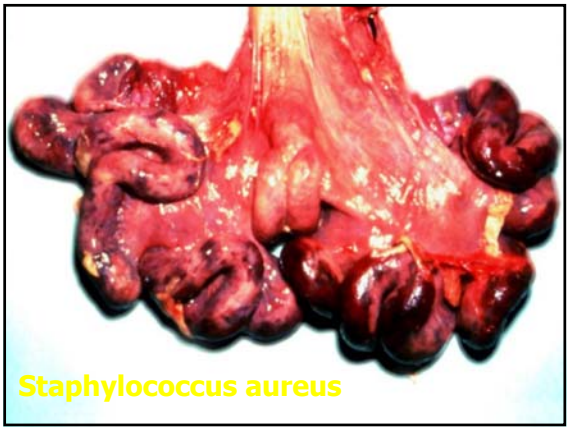

Follicular Cysts

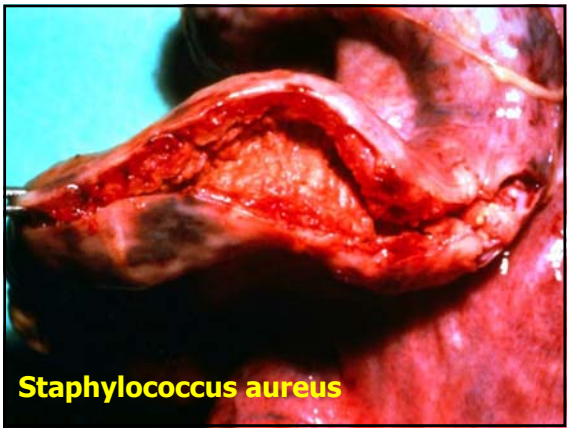
## Zearalenone

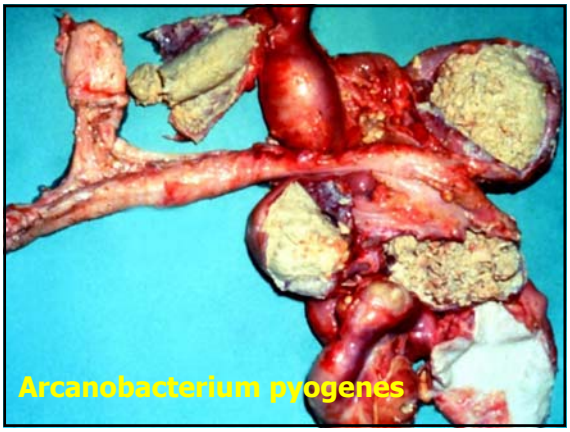
- *Fusarium*
- estrogenic effect of toxin ("false pregnancy"?)
- affects implantation - return to heat
- occasional weak litters
- swollen vulva and rectum
- Increased incidence of vulvar or rectal prolapses

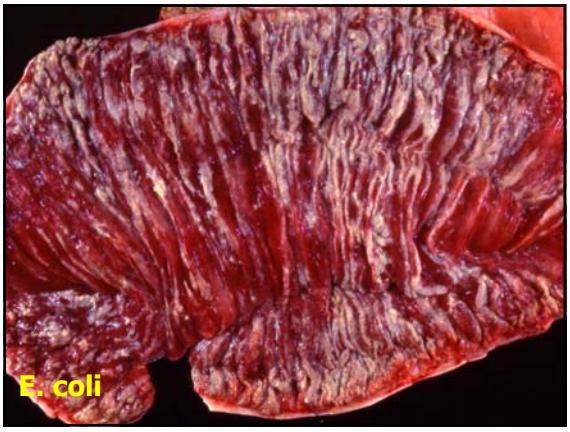

Normal Vulva

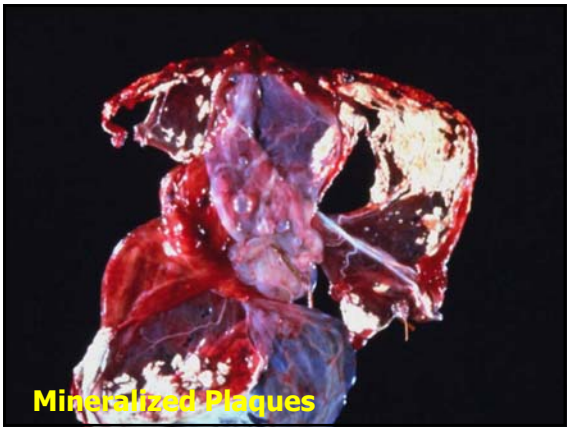

Zeralenone


Staphylococcus aureus

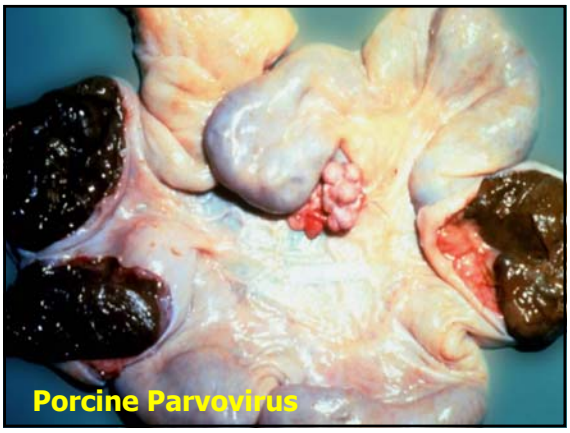

Staphylococcus aureus


Arcanobacterium pyogenes


E. coli

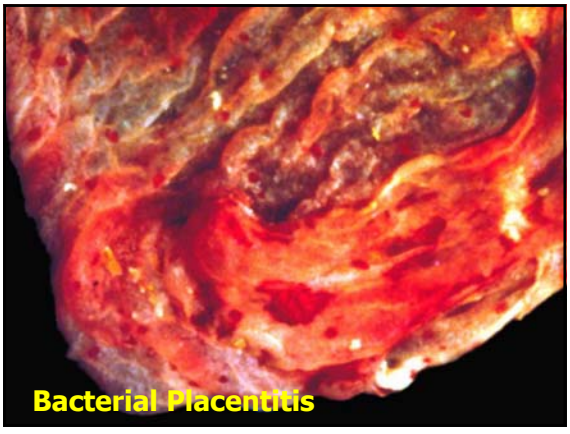

Mineralized Plaques

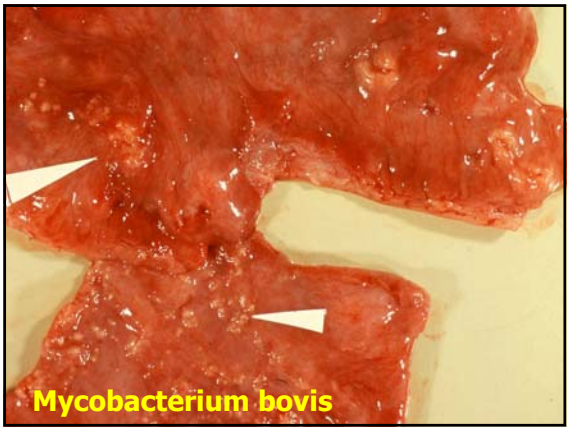

Normal Placentome


Porcine Parvovirus

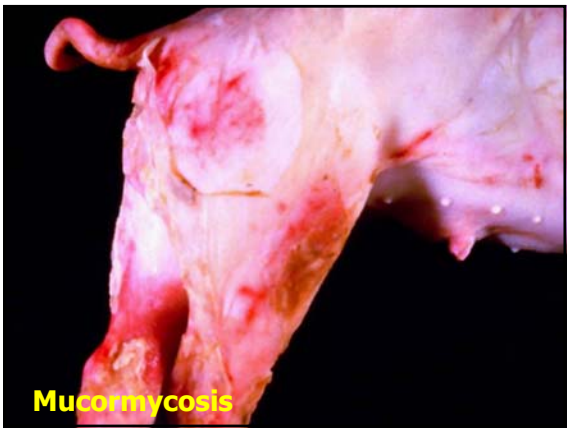

SMEDI


PRRSV

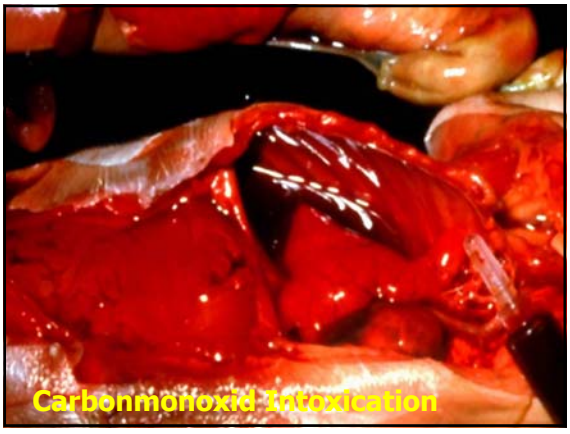

Bacterial Placentitis


Mycobacterium bovis

Nocardia asteroides



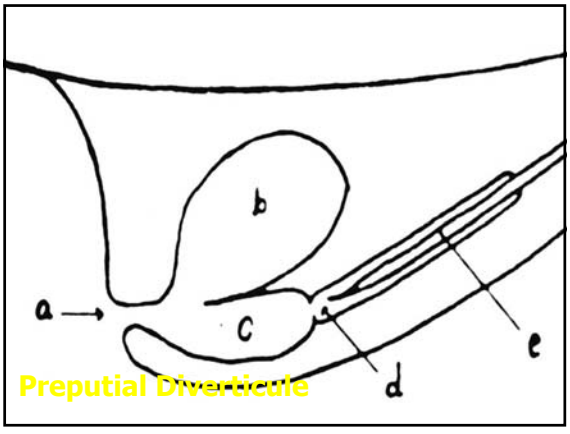
Mucormycosis



Carbonmonoxid Intoxication

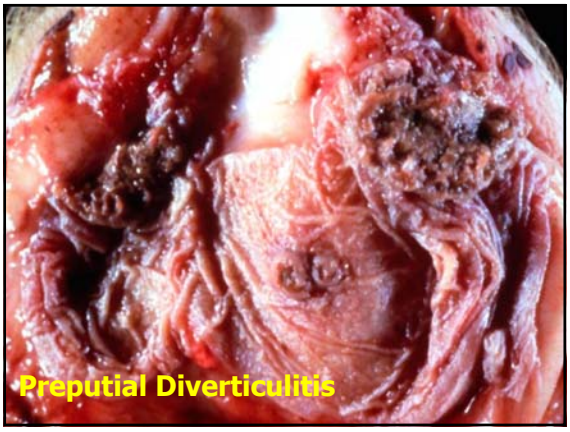
Paraphimosis

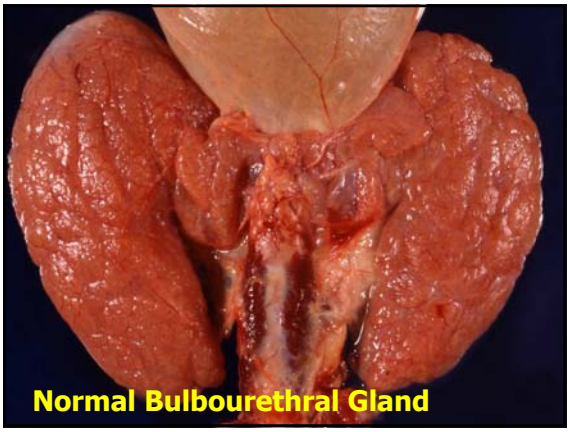
Fibrinonecrotic Posthitis

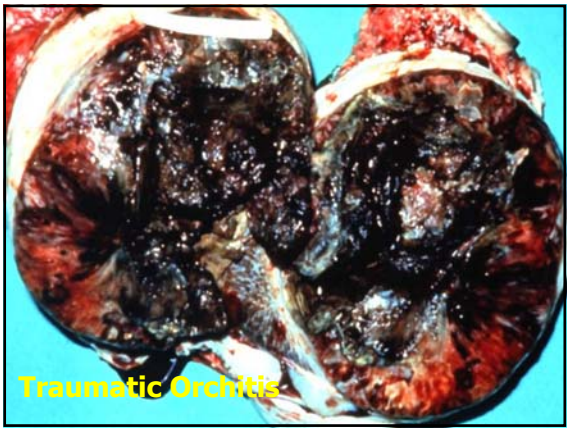



Preputial Diverticule


Preputial Diverticulitis

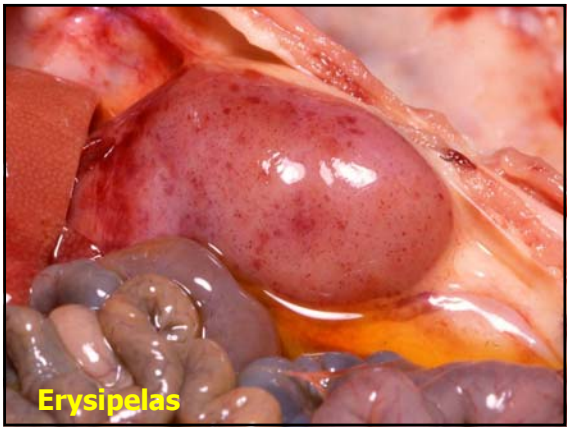

Preputial Diverticulitis


Normal Bulbourethral Gland

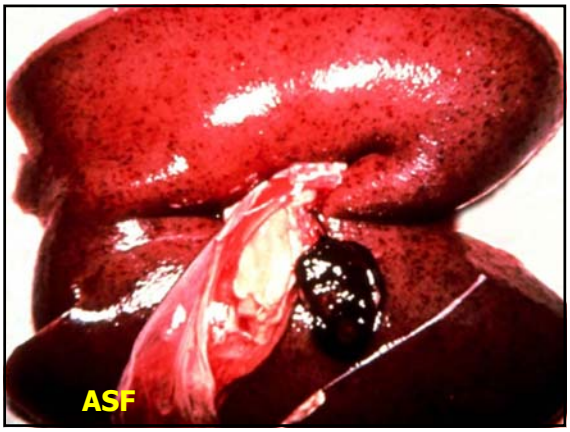

Traumatic Orchitis

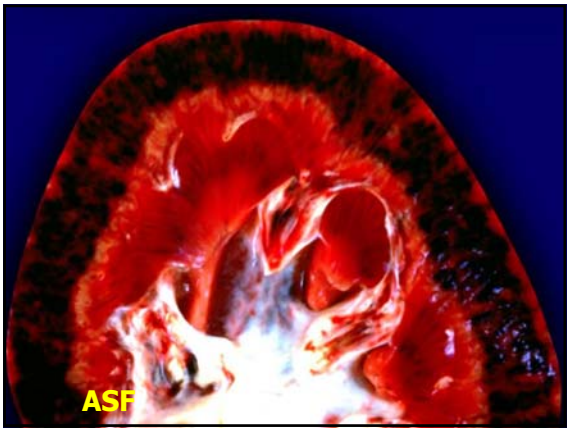
# Urinary System

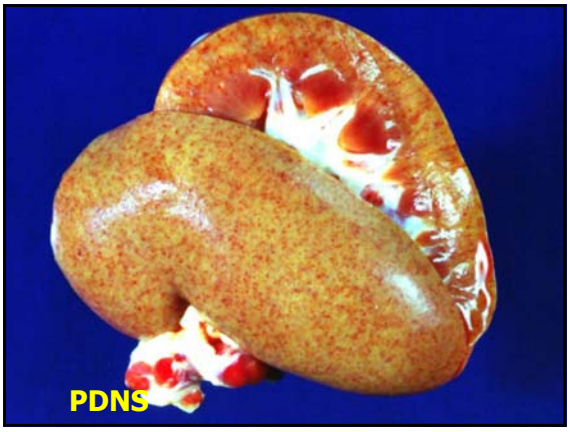

Salmonella choleraesuis

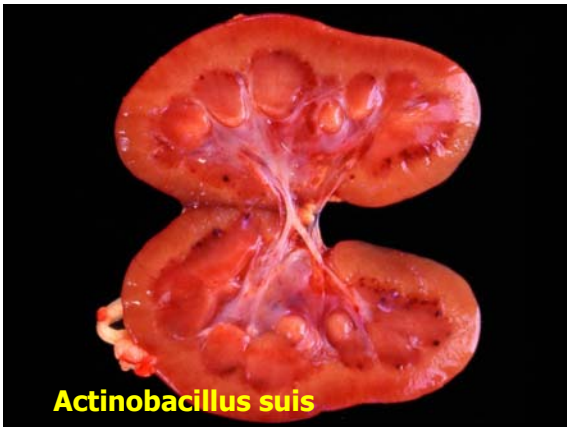

Erysipelas


CSF


ASF


ASF

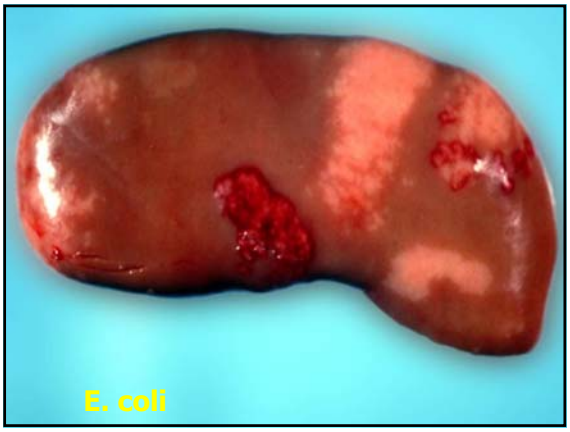

PDNS


Actinobacillus suis


Actinobacillus suis


Streptococcus suis

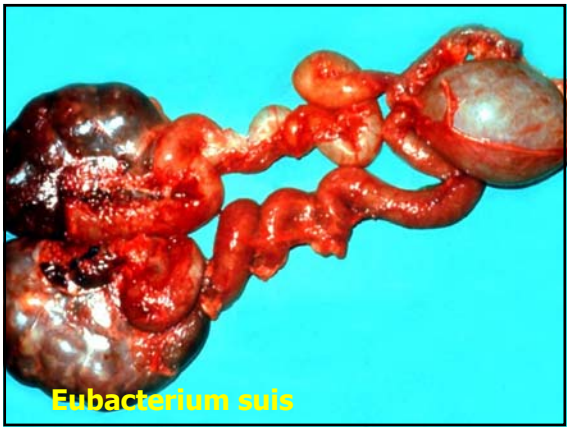

Arcanobacterium pyogenes


E. coli

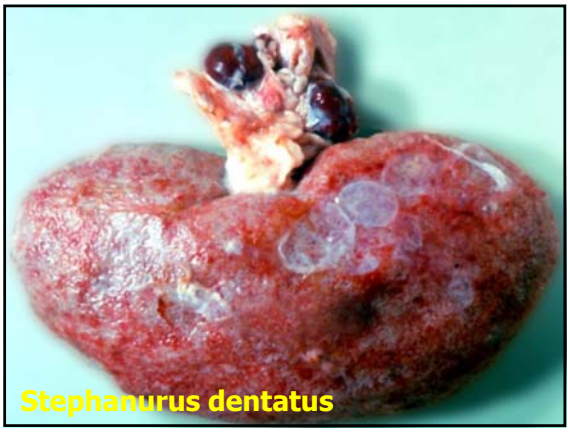

E. coli

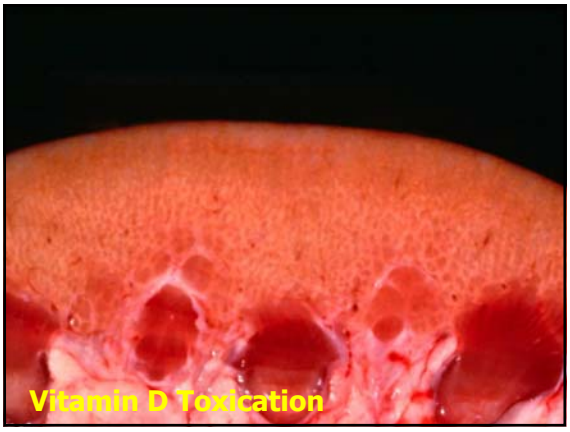

Leptospira pomona


Leptospira pomona


Eubacterium suis

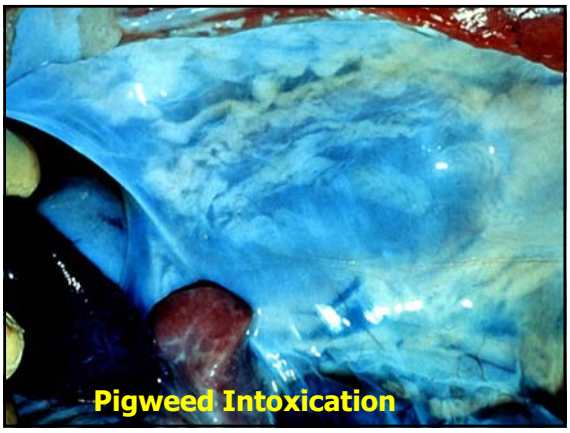

Eubacterium suis


Stephanurus dentatus


Vitamin D Toxication


Ochratoxin


Ochratoxin


Pigweed Intoxication


Pigweed Intoxication

**Gross Morbid Pathology of Swine**

C. L. Davis Foundation's "Gross Morbid Anatomy of Diseases of Animals"

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Senior Diagnostic Pathologist, Veterinary Diagnostic Laboratory
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Acknowledgements: William G. Van Alstine DVM, MS, PhD, DACVP Purdue University
Matti Kiupel, BS, MS, PhD, DACVP Michigan State University
Ingeborg M. Langohr DVM, PhD, DACVP Michigan State University

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| | | **File 1** | | |
| 1 | Title Slide | | | |
| 2 | PEDv U.S. Outbreak | | | |
| 3 | Word slide | **Changes in Management Technologies ⇒ Changes in Disease**<br>**Traditional Technologies:** / **Evolution in Management Technologies:**<br>Small farms; 50-100 sows / Large farms; 1000-5000 sows<br>Group farrowing; 2-4 gps/yr / Reduced weaning age; 10-21 days-of-age<br>Weaning age: 4-8 weeks-of-age / Age-segregated rearing; AI/AO, SEW<br>Continuous-flow rearing / Site-segregated rearing; 2- or 3-site | | |
| 4 | Word slide | **Ages/stages:** Suckling < 2-3 weeks; Nursery ≈1-2 mo.; Grow/Fin. 3-6 mo.; Breeding/Adult > 6 mo. | | |
| 5 | Word slide | Outline | | |
| 6 | | **NEOPLASTIC DISEASES** | | |
| 7 | Kidney | Malignant Lymphoma | Multifocal renal lymphoma | All ages and sexes; multicentric or thymic **(Alsop, 2005, J Swine Health Prod 13: 31-33)** |
| 8 | Kidney, renal ln. | Malignant Lymphoma | Multifocal to coelescing renal lymphoma | LN's, thymus > liver, spleen, kidney > other, leukemia in terminal stage |
| 9 | Head, sagittal | Malignant Lymphoma | Lymphoma; mesencephalon and frontal sinuses | Space occupying mass; homogeneous white |
| 10 | Liver | Malignant Lymphoma | Multifocal hepatic lymphoma | DDX: Multifocal granulomatous hepatitis – M. avian |
| 11 | Stomach | Malignant Lymphoma | Intramural gstric lymphoma | can be limited to digestive tract |
| 12 | Rib cage | Malignant Lymphoma | Multifocal lymphoma, lymph nodes | DDX: thymic or multicentric form |
| 13 | Skin | Benign melanoma | Multiple cutaneous melanomas | Duroc, usually young pigs, heavily pigmented, may be malignant. Cutaneous melanocytic tumors of Duroc and Iberian swine have been characterized by IHC. The local cellular immune response may play a crucial role in the regression of these tumors. **(Perez et al., Vet Pathol, 2002, 39: 445-451)** |
| 14 | Thoracic vertebral body | Metastatic melanoma | Metastatic melanoma; thoracic vertebrae and spinal canal | High incidence in Sinclair strain of minuature swine |
| 15 | Kidney | Nephroblastoma | Focal renal nephroblastoma | Young - < 1 yr old; females > males, 4 types: nephroblastic and epithelial (most common), mesenchymal and mixed (less common) |
| 16 | Cut section | Nephroblastoma | Nephroblastoma | Occassional metastasis to liver and lungs |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| Other neoplasms: Hepatocellular carcinoma, splenic hemangiosarcoma in a potbellied pig (Morrow 2002, Can Vet J 43: 466-468), oral squamous cell carcinoma (Kleinschmidt et al., 2006, Vet Pathol 43:569-57), scrotal and testicular hemangiomas (Teankum et al., J Comp Pathol, 2008, 139: 177-186), rhabdomyomas (Jacobson et al., Vet Pathol, 2010, 47:738-740) | | | | |
| 17 | CONGENITAL – HERIDITARY DISEASES | | | |
| 18 | Inguinal canal | Genetic, polygenic | Inguinal/scrotal hernia | Male>female, left>right (unilateral), Weakness of tunica vaginalis, occasionally associated with freemartinism **(Tiranti et al., J Agricult Sci, 2002, 138: 333-340)** |
| 19 | Inguinal canal | Genetic, polygenic | Inguinal/scrotal hernia | |
| 20 | Ventral abdomen | Genetic, polygenic | Umbilical hernia | DDX: Sequel to omphalitis<br>less common than inguinal hernia |
| 21 | Pig-sitting | Genetic, polygenic, Landrace | Myofibrillar hypoplasia | Syn.: Splayleg, Spraddleleg; deltoids and semitendonosis mm. most involved, 1-4 pigs per litter affected |
| 22 | Musc. legs | Genetic, polygenic | Myofibrillar hypoplasia | Male piglets more susceptible |
| 23 | Whole body | Genetic, autosomal recessive, Yorkshire pigs,<br>deficiencies,<br>teratogens | Arthrogryposis | DDX: In-utero Vit. A or manganese def., Classical swine fever (hog cholera), exposure to tobacco stalk, jimsonweed (thorn apple), wild black cherry (bark) or poison hemlock |
| 24 | Head | In-utero vitamine A deficiency | Microphthalmia | DDX: Classical swine fever (hog cholera), heritable |
| 25 | Brain | In-utero vitamine A deficiency | Aplasia of chiasma opticum | DDX: Classical swine fever (hog cholera), heritable |
| 26 | Fetus | Sporadic | Conjoined twins (Siamese twins) | Uncommon, but lethal, single pigs |
| 27 | Front legs | Genetic, autosomal recessive | Congenital hyperostosis | Syn.: Congenital thick foreleg |
| 28 | Front legs, c/s | Genetic, autosomal recessive | Congenital hyperostosis | Fatal in first few weeks of life. |
| 29 | Kidney | Hereditary | Multiple renal cysts | Mainly in pol of kidney, can become confluent |
| 30 | Kidney | Hereditary | Hydronephrosis | DDX: aquired |
| 31 | Mammary glands | Hereditary | Inverted nipple | Common condition, single inverted nipples are of no greater concern |
| 32 | Mammary glands | Hereditary | Inverted nipple | |
| 33 | Skin | Uncertain,<br>esp. Landrace | Porcine juvenile pustular psoriasiform dermaititis (Pityriasis rosea) | Genetic predisposition in Landrace |
| 34 | Skin | Uncertain,<br>esp. Landrace | Porcine juvenile pustular psoriasiform dermaititis (Pityriasis rosea) | Usually ventral abdomen, benign, self-limiting |
| 35 | Whole body | Type II hypersensitivity (cytotoxic-type) | Thrombocytopenia purpura | Piglets die between 1 and 3 weeks of age from haemorrhagic diathesis, a similar syndrome has been described in sexually mature Göttingen minipigs **(Carrasco et al., J Comp Pathol, 2003, 128: 25-32)** |
| 36 | Head | Type II hypersensitivity (cytotoxic-type) | Thrombocytopenia purpura | Passive antiplatelet antibody transfer, DDX: bacterial septicemias |
| 37 | Hind legs | Genetic, autosomal recessive | Dermatosis vegetans | Very infrequent, all carrier originated from 1 danish landrace sow |
| 38 | Abdomen | Genetic, autosomal recessive | Dermatosis vegetans | Thick papillomatous crusts |
| 39 | Abdomen | Genetic, autosomal recessive | Dermatosis vegetans | Thick papillomatous crusts, associated with fatal giant cell pneumonia |
| 40 | Feet | Genetic, autosomal recessive | Dermatosis vegetans | Hyperkeratotic pododermatitis |
| 41 | Skin- | Genetic, autosomal | Epitheliogenesis imperfecta (aplasia | May also affect tongue, concurrent |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| | forelimb | recessive | cutis) | hydroureter and hydronephrosis **(Benoit-Biancamano et al., JVDI 2006, 18: 573-579)** |
| 42 | Foot | | Syndactly | Polydactly is also rarely reported |
| 43 | Perineum | Genetic, unknown mode | anal atresia | Male > females, fatal in 2-3 weeks, rectum ends blindly **(Shobhamani et al., Indian Vet J, 2002, 79: 358-360)** |
| 44 | Abdominal cavity | Genetic, unknown mode | anal atresia | Secondary megacolon |
| 45 | Hard palate | Genetic, Poland China | Palatoschisis | DDX: Teratogenic event mid-gestation |
| 46 | Head | Neural tube defects; | Meningoencephalocoele | Insult at day 12-14 of gestation |
| 47 | Cranium | Congenital | Cranioschisis | Associated with meningoencephalocoele |
| 48 | Heart | Congenital | Interventricular septal defect | Septal defects: male > females<br>A relatively high incidence of persistent foramen ovale was observed in Large White and Landrace breeds, 15 and 13.4%, respectively **(Bajan et al., Magyar Allator Lapja, 2002, 124: 415-418)** |
| 49 | Small intestine | Congenital | Persistent Meckel's diverticulum | Residual omphalomesenteric duct |
| 50 | Liver | Congenital | Biliary cysts | **Komine et al., J Comp Pathol, 2008, 139: 202-207** |
| 51 | Heart | Congenital | Ectopia cordis | |
| 52 | Vulva | Male pseudohermaphrodite | Penile clitoris, clitoral enlargement | |
| 53 | Genital tract | XX karyotype | Male pseudohermaphrodite, abdominal testes w/ uterus masculinus | |
| A new infertility syndrome: hereditary sterilizing short-tail sperm defect in Yorkshire boars was recently described in Finnland: **Sukura et al., JVDI 2002, 14: 382-388**. A fibroepithelial hamartoma has been described in a single case (**Sipos et al., Vet Pathol, 2007, 44:411-413**). | | | | |
| 54 | **GENERALIZED DISEASES** | | | |
| 55 | **Salmonella choleraesuis:** Causes severe septicemia +/- concurrent pneumonia or enterocolitis in weaned and grower pigs **(Wolf et al., Vet Quart, 23: 3, 116-121, 2001, Cote et al., 2004, Can J Vet Res Oct 68: 241-248).** Multifocal hepatic necrosis (paratyphoid nodules) is a fairly consistent lesion. S. choleraesuis replicates in macrophages as well as extracellularly in lymphoid tissues (causing necrosis) and elsewhere. Large amounts of systemic endotoxin activate cytokines and induce vascular damage (hemorrhage, interstitial pneumonia with edema, glomerulonephritis, gastric mucosal venous thrombosis and arterial thrombosis (skin of extremities and colon → ulcers). Ochratoxins may increase the susceptibility of pigs to infection with S. choleraesuis **(Stoev et al., Exp Tox Path, 52: 287-296, 2000)**. The mechanisms of early cellular invasion were recently reviewed in the light of *Salmonella* serovar–host specificity **(Meyerholz and Stabel, Vet Pathol, 40: 371-375, 2003).** Mutations in the cAMP-receptor protein (CRP) gene affect the secretory function of the Salmonella pathogenicity island 1 (SPI-1) encoded type III secretion system (T3SS) and the resulting ability to invade the host intestinal epithelium **(Chen et al., Vet Res, 41: Epub, 2010).** Recent Review **Boyen et al., Vet Microbiol, 130:1-19, 2008.** | | | | |
| 56 | Sick pigs | | | |
| 57 | Pinna | | | |
| 58 | Lung | Salmonella choleraesuis | Congestion, cyanosis of skin on extremities | |
| 59 | Lung | Salmonella choleraesuis | Congestion, cyanosis, ischemic necrosis; skin of ears | |
| 60 | Lung | Salmonella choleraesuis | Hemorrhagic interstitial pneumonia with interlobular edema | |
| 61 | Bronchial lymph node | Salmonella choleraesuis | Hemorrhagic interstitial pneumonia with interlobular edema | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 62 | Lymph node | Salmonella choleraesuis | Suppurative bronchopneumonia, hemorrhagic interstitial pneumonia | |
| 63 | Heart | Salmonella choleraesuis | Hemorrhage, bronchial lymph node Hemorrhagic interstitial pneumonia | |
| 64 | Tonsil | Salmonella choleraesuis | Congestion and edema, lymph node (Lymphadenitis) | |
| 65 | Gall bladder Gastro-hepatic l.n. Liver | Salmonella choleraesuis | Atrial hemorrhage | |
| 66 | Liver | Salmonella choleraesuis | Multifocal tonsillar abscesses | DDX: Abscess - S. suis, A. pyogenes; Necrosis - PRV, CSF |
| 67 | Liver c/s | Salmonella choleraesuis | 1. Cholecystitis, cholangitis<br>2. Lymphadenitis, gastrohepatic lymph nodes<br>3. Multifocal hepatic necrosis, hepatic congestion | Case report of cholecystitis with vascular compromise and seondary infection with Clostridium perfringens type A **(Starost et al., JVDI, 2008, 20: 527-530)** |
| 68 | Spleen | Salmonella choleraesuis | Multifocal hepatic necrosis | "paratyphoid nodules": multiple foci of hepatocyte necrosis first replaced by hemorrhage and fibrin, later aggre-gates of macrophages and neutrophils |
| 69 | Kidney | Salmonella choleraesuis | Multifocal hepatic necrosis | |
| 70 | Brain | Salmonella choleraesuis | Splenomegally, bronchopneumonia, renal cortical petechiae | Infarcts are VERY rare; spleen is soft and purple – red pulp engorged with blood and fibrin +/- necrosis |
| 71 | Stomach | Salmonella choleraesuis | Multifocal renal cortical petechiae and ecchymoses | fibrinous glomerulonephritis |
| 72 | Stomach | Salmonella choleraesuis | Diffuse purulent leptomeningitis | |
| 73 | Ileum | Salmonella choleraesuis | Focally extensive fibrinohemorrhagic gastritis, fundus | Note: mucosal folds help in identification of fundus |
| 74 | Ileum, Colon | Salmonella choleraesuis | Focally extensive fibrinohemorrhagic gastritis, fundus | Note: mucosal folds help in identification of fundus |
| 75 | Colon | Salmonella choleraesuis | Severe diffuse fibrinonecrotic enterocolitis | |
| 76 | Funny Picture | Salmonella choleraesuis | Multifocal infarcts | DDX: CSF |
| 77 | | Salmonella choleraesuis | Severe chronic multifocal fibrinonecrotic ulcerative colitis (button ulcers) | Button ulcers are caused by ischemic necrosis secondary to vasculitis DDX: *S. typhimurium, S. typhisuis*, classical swine fever (Hog cholera) |
| 78 | *H. parasuis* can cause an acute septicemia that resembles septicemic Salmonellosis. H. parasuis more commonly causes polyserositis, polyarthritis and meningitis (Glasser's disease) in weaned pigs (**Oliveira et al., J Swine Health Prod, 2002, 10: 221-225**). Neurological clinical signs are uncommon in weaned pigs with Glasser's disease. Occasionally, *H. parasuis* causes acute outbreaks of highly fatal fibrinosuppurative leptomeningitis in young adult replacement breeding stock shortly after entry into recipient herds. *H. parasuis* also causes eustachitis and temporary otitis media is suggested as predisposing to ascending secondary pyogenic bacterial otitis media (**Morita et al., 1998 & 19999, AJVR, 59: 869 – 873 & Vet Pathol, 36: 174 – 178**) | | | |
| 79 | Whole pig | | Hemophilus parasuis | Cutaneous cyanosis and ischemic necrosis |
| 80 | Thorax, Abdomen | | Hemophilus parasuis | Diffuse, fibrinous pericarditis, epicarditis, hydropericardium |
| 81 | Peritoneal cavity | | Hemophilus parasuis | Multifocal serosal hemorrhages |
| 82 | Kidney | Hemophilus parasuis | Glomerulonephritis – Fibrinopurulent | Glomeruli are filled with fibrinopurulent exudate |
| 83 | Brain | Hemophilus parasuis | Purulent leptomeningitis - Fibrinopurulent | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 84 | Hock joint | Hemophilus parasuis | Acute fibrinopurulent arthritis | |
| 85 | Thorax, Abdomen | Hemophilus parasuis | Fibrinous polyserositis | **Fibrinous polyserositis:** In suckling pigs: E. coli is a sporadic cause when there is inadequate intake of colostrum. In weaned pigs: Differentials include H. parasuis (Glasser's disease), S. suis and M. hyorhinis. Although all 3 can cause meningitis in weaned pigs, clinical CNS disease is usually a consistent feature in only S. suis infections |
| 86 | Heart, Lungs | Hemophilus parasuis | Diffuse fibrinous pericarditis/epicarditis and pleuritis | DDX: S. suis, M. hyorhinis |
| 87 | Lungs | Streptococcus suis | Fibrinous pleuritis, bronchopneumonia | DDX: H. parasuis, M. hyorhinis |
| 88 | Brain | Streptococcus suis | Purulent leptomeningitis - Fibrinopurulent | DDX: H. parasuis |
| 89 | Hock joint | Streptococcus suis | Acute fibrinopurulent arthritis | DDX: H. parasuis, M. hyosynoviae |
| 90 | Thorax | Streptococcus suis | Fibrous adhesions, lung and parietal pleura | DDX: H. parasuis, M. hyorhinis, E. coli |
| 91 | **Streptococcus suis:** There are now 35 capsular serotypes that are described in pigs. Disease is most common in suckling and recently weaned pigs, but can occur in any age. Fibrinopurulent leptomeningitis causing CNS clinical signs and high mortality is common. Septicemia with or without fibrinous polyserositis or leptomeningitis is also common. When fibrinous polyserositis predominates, S. suis septicemia is difficult to differentiate from Glasser's disease. In general, the amount of fibrin and the severity of peritonitis are greater with Glasser's disease than with S. suis septicemia. Has assumed greater importance since PRRSV has become ubiquitious in swine populations. Acute PRRS predisposes to S. suis induced septicemic diseases **(Galina et al., 1994, Vet Rec 134: 60 – 64, Thanawongnuwech et al., Vet Pathol, 2000, 37: 143-152)**. Like H. parasuis, S. suis also may cause acute highly fatal outbreaks of leptomeningitis in young replacement breeding swine shortly after introduction into recipient herds. Recent pathogenesis studies point towards the tonsils as possible portals of entry for S. suis serotype 2. **(Madsen et al., J Vet Med Series B, 2002, 49: 211-215)**. Suilysin is considered an important S. suis virulence-associated factor **(Zheng et al., 2009, 46: 531-535)**. Furthermore, S. suis serotype 2 bacteria were frequently identified immunohistochemically in the regional lymph nodes of the upper respiratory tract, possibly reflecting primary lymphogenous spread from the tonsils **(Madsen et al., J Comp Path, 126: 57-65)**. There is participation of the innate immunity in the early phase of S. suis infection, represented by neutrophils, macrophages and likely epithelial cells, and there is a potential for the initiation of both humoral and cellular responses against S. suis within the crypt epithelium of the palatine tonsil. **(Salles et al., Vet Immunol Immunopath, 2002, 87: 51-63).** Fibrinogen-binding protein played a role in the colonization of organs involved in an S. suis infection **(Greeff et al., Infect Immun, 2002, 70: 1319-1325).** | | | |
| 92 | Thorax, Abdomen | Streptococcus suis | Fibrinous polyserositis | DDX: H. parasuis, M. hyorhinis |
| 93 | Brain | Streptococcus suis | Purulent leptomeningitis | |
| 94 | Lung, heart | Streptococcus suis | Fibrinous pericarditis/epicarditis, fibrinous pleuritis, lobular pneumonia | Interstitial pneumonia, when present with Streptococcal septicemia, is mild. |
| 95 | Lung, heart | Streptococcus suis PRRS virus | Fibrinous epicarditis, fibrinous pleuritis, interstitial pneumonia | |
| 96 | Carpus | Streptococcus suis | Fibrinopurulent arthritis | |
| 97 | Heart; AV valve | Streptococcus suis | Vegetative valvular endocarditis | |
| 98 | Whole pig | Streptococcus suis | Multifocal cutaneous macules | |
| 99 | ***Streptococcus suis*: an emerging zoonotic pathogen** | | | |
| 100 | Lun et al. Lancet Infect Dis. 7, 201-9, 2007 | | | |
| 101 | **Mycoplasma hyorhinis:** Common commmensal of the upper and lower respiratory tract of weaned pigs. Uncommon cause of polyserositis. | | | |
| 102 | Snout | Mycoplasma hyorhinis | Severe diffuse fibrinous rhinitis | |
| 103 | Thorax, Abdomen | Mycoplasma hyorhinis | Fibrinous polyserositis | DDX: H. parasuis, S. suis |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 104 | Lung, heart | Mycoplasma hyorhinis | Severe diffuse fibrinous pleuritis and pericarditis | DDX: S. suis, H. parasuis |
| 105 | Heart | Mycoplasma hyorhinis | Severe diffuse fibrinous epicarditis and pericarditits | DDX: S. suis, H. parasuis |
| 106 | Carpus | Mycoplasma hyorhinis | Acute fibrinopurulent arthritis | DDX: H. parasuis, M. hyosynoviae |
| 107 | Brain | E. coli | Severe purulent meningitis | **E. coli:** Ubiquitious fecal flora. Opportunistic cause of septicimia with or without polyserositis especially in colostrum–deficient pigs. Fibrin is typically abundant and exudate is sometimes fibrinopurulent. |
| 108 | **Erysipelas** still occurs in swine raised entirely in environmentally regulated buildings. E. rhusiopathiae causes disease in all ages. Mortality is highest and lesions are most extensive and severe in suckling and recently weaned pigs. In growing and finishing pigs, pigs may be found dead with few gross lesions – typically sparse renal cortical petechiae and a slightly enlarged speen that is firm and red or lameness may predominate with proliferative synovitis and fibrous periarthritis. Outbreaks in sows are typically associated with pyrexia, anorexia, few cutaneous infarcts and occasional abortions. Erysipelothrix has zoonotic potential and may cause endocarditis in humans **(Romney et al., Can. J Infect Dis 12: 254-256, 2001)**. Erysipelas may have a potential involvement in urogenital disease of the sow. **(Gertenbach et al., J Swine Health Prod, 2002, 10: 205-207)** | | | |
| 109 | Skin | E. rhusiopathiae | Multifocal cutaneous infarcts | |
| 110 | Skin | E. rhusiopathiae | Multifocal cutaneous infarcts | |
| 111 | Skin | E. rhusiopathiae | Cutaneous infarct | |
| 112 | Snout | E. rhusiopathiae | Ischemic necrosis | |
| 113 | Digit | E. rhusiopathiae | Ischemic necrosis, coronary band | |
| 114 | Spleen | E. rhusiopathiae | Splenomegally | Typically, 1.5-2X normal size, red firm |
| 115 | Joint | E. rhusiopathiae | Acute purulent arthritis and periarticular edema | |
| 116 | Joint | E. rhusiopathiae | Chronic proliferative synovitis and purulent arthtitis | |
| | | | **File 2** | |
| 1 | **Actinobacillus suis** causes sporadic oubreaks of fulminant embolic septicemia in all ages of pigs. In suckling and recently weaned pigs, most present as acute death. Those with clinical signs have fever and multifocal cutaneous hemorrhages. Occasionally, pigs may be lame, exhibit dyspnea or have nervous signs. In finishing-age pigs, most pigs are also found dead; however, in sick pigs the primary clinical manifestation is respiratory disease characterized by pyrexia, dyspnea and cyanosis. In adults (and sometimes younger animals), disease is less often fatal and resembles erysipelas. Sick adults typically are pyretic, anorectic and depressed with raised red rhomboid skin lesions typical of erysipelas. Some adults are found dead and occasionally sows abort. Lesions in all ages are the consequence of septicemia with septic embolism. Petechial hemorrhages are diffusely distributed on serosal surfaces and a wide variety of organs including lungs, kidneys, spleen and skin. Common lesions also include necrohemorrhagic pneumonia and serofibrinous pericarditis, pleuritis and peritonitis. Less common lesions include fibrinous arthritis, rhomboid cutaneous infarcts, meningitis and myocarditis. In pneumonic lungs, affected areas of necrosis, hemorrhage and fibrin deposition are multifocal and randomly distributed, suggesting a hematogenous origin. However, these pneumonic foci may coalesce until lung lesions are grossly indistinguishable from those caused by APP. **Yaeger, J Vet Diag Invest, 8:381-383, 1996; Odin, Can Vet J, 34:634, 1993, Sanford et al., Can Vet J, 31:443-447, 1990; Sanford and Miniats, Can Vet J, 29:595, 1988.** | | | |
| 2 | Whole pig | Actinobacillus suis | Multifocal cutaneous infarcts | DDX: Erysipelothrix |
| 3 | Lung | Actinobacillus suis | Multifocal embolic fibrinonecrotic pneumonia | Lung lesions may coalesce and be identical to APP |
| 4 | Kidney | Actinobacillus suis | Multifocal renal cortical petechiae and ecchymoses | septicemic lesions differentiate from APP – serosal hemorrhages, cutaneous infarcts, renal hemorrhages, pericarditis, meningitis |
| 5 | Heart | Actinobacillus suis | Fibrinous pericarditis and epicarditis | Lung lesions may coalesce and be identical to APP |
| 6 | Heart | Actinobacillus suis | Vegetative valvular endocarditis, tricuspid valve | DDX: S. suis, E. rhusiopathiae, E. coli, A. pyogenes, A. suis, S. equisimilis |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 7 | Heart | Actinobacillus suis | Vegetative valvular endocarditis, tricuspid valve | DDX: S. suis, E. rhusiopathiae, E. coli, A. pyogenes, A. suis, S. equisimilis |
| 8 | Brain | Actinobacillus suis | Multifocal embolic encephalitis, cerebellum | DDX: S. suis, E. rhusiopathiae, E. coli, A. pyogenes, A. suis, S. equisimilis |
| 9 | **Arcanobacterium pyogenes:** Common isolate from swine. Usually is an environmental contaminant of wounds, causing a localized purulent infection followed by bacteremia resulting in vegetative valvular endocarditis, purulent arthritis, embolic abscessing pneumonia, ascending urogenital infections or other localized pyogenic infection. It is also a common opportunistic secondary pulmonary pathogen. Neuraminidases of A. pyogenes play a role in adhesion of this organism to host epithelial cells **(Jost et al., Infect Immun, 2002, 70: 1106-1112).** | | | |
| 10 | Lung | Arcanobacterium pyogenes | Diffuse embolic abscessing pneumonia | DDX: S. suis, E. rhusiopathiae, E. coli, A. pyogenes, A. suis, S. equisimilis |
| 11 | Heart | Arcanobacterium pyogenes | Multifocal mural abscesses, heart | Also: vegetative valvular endocarditis, aortic and mitral valves |
| 12 | Liver | Arcanobacterium pyogenes | Multifocal hepatic abscesses | DDX: Lymphoma, Mycobacterium avium (poor choices |
| 13 | Kidney | Arcanobacterium pyogenes | Multifocal embolic nephritis and infarction | DDX: E. coli |
| 14 | Brain | Arcanobacterium pyogenes | Focally extensive cerebral abscessation | DDX: E. coli, maybe S. suis |
| 15 | Mandible | Arcanobacterium pyogenes | Multifocal abscessing osteomyelitis | DDX: E. coli |
| 16 | Heart | Arcanobacterium pyogenes | Vegetative valvular endocarditis, tricuspid valve | DDX: S. suis, Erysipelas |
| 17 | **Pseudorabies (Aujeszky's Disease):** Clinical signs and lesions vary according to age. In suckling pigs, mortality is high and is associated with nervous clinical signs (nonsuppurative encephalomyelitis) and multifocal necrosis in the parenchyma of organs (tonsil, lung, liver, spleen). In nursery, growing, finishing and adult swine, mortality is lower and is associated with respiratory clinical disease and lesions (rhinitis, laryngotracheitis, interstitial pneumonia). CNS clinical signs are less common, although microscopic lesions in the CNS are common. Late-term abortions occasionally in epizootics. Lesions uncommon in fetuses – same as for neonate. PRV has been shown to block apoptosis of infected trigeminal ganglionic neurons during acute infection and to induce apoptosis of infiltrating inflammatory cells as an important viral mechanism of immune evasion. **(Aleman et al., J Virol, 75: 469-479, 2001).** The glycoprotein B (gB) of Aujeszky's disease virus (ADV) has a role in the initial attachment of virus to the surface of susceptible cells, as well as virus entry and cell-to-cell spread. **(Vrublevskaya et al., Virus Res, 2002, 86: 7-19)** | | | |
| 18 | Neonatal pigs | Pseudorabies (Porcine Herpes virus) | Tremors, head tilt, ataxia, sternal recumbancy | nonsuppurative meningoencephalitis |
| 19 | Palantine tonsil | Pseudorabies (Porcine Herpes virus) | Tonsillar hemorrhage | |
| 20 | Palantine tonsil | Pseudorabies (Porcine Herpes virus) | Tonsillar necrosis | |
| 21 | Palantine tonsil | Pseudorabies (Porcine Herpes virus) | Tonsillar necrosis | |
| 22 | Liver | Pseudorabies (Porcine Herpes virus) | Multifocal hepatic necrosis or miliary hepatic necrosis | |
| 23 | Spleen | Pseudorabies (Porcine Herpes virus) | Multifocal spleenic necrosis | |
| 24 | Snout | Pseudorabies (Porcine Herpes virus) | Multifocal vesiculoulcerative nasal dermatitis | |
| 25 | Nasal cavity | Pseudorabies (Porcine Herpes virus) | Diffuse fibrinonecrotic rhinitis | |
| 26 | Larynx | Pseudorabies (Porcine Herpes virus) | Diffuse fibrinonecrotic laryngotracheitis | |
| 27 | Lung | Pseudorabies (Porcine Herpes virus) | Interstitial pneumonia with multifocal hemorrhage | |
| 28 | Lung | Pseudorabies (Porcine Herpes virus) | Interstitial pneumonia with multifocal hemorrhage | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 29 | **PRRS:** Review article: Rossow, 1998, Vet Pathol 35:1-20; Nodelijk, 2002, Vet Quart 24: 95-100; Consistent gross lesions are in lungs and lymph nodes only. Microscopic lesions: interstitial pneumonia with aggregates of necrotic alveolar macrophages in alveoli, lymphoid necrosis followed by nodular lymphoid hyperplasia in lymphoid organs, lympho-plasmacytic myocarditis and mild nonsuppurative encephalitis. Syncytial cells are more likely the result of PCV2 infection. Differentials for lympho-plasmacytic myocarditis in aborted or suckling pigs include porcine parvovirus (Bolt et al. 1997, J Comp Path 117:107-118) and porcine circovirus (West et. al., 1999, J Vet Diagn Invest 11: 530-532, Sanchez et al., Vet Microbiol 83: 169-176, 2001). Recently a proliferative vasculopathy has been described in aborted piglets (Scruggs et al., Vet Path., 38: 339-342, 2001). Many recent papers are available on various aspects of pathogenesis (Bierk et al., Can J Vet Res, 65: 261-266, 2001). PRRSV induces apoptosis in infected and bystander cells including macrophages (histiocytes, tingible body macrophages and pulmonary intravacular macrophages), alveolar pneumocytes and epithelial germ cells in the seminiferous tubules (Sur et al., 1997 & 1998, J Virol, 71: 9170 – 9179 & Vet Pathol, 35: 506 – 514; Sirinarumitr et al., 1998, J Gen Virol, 79: 2989 – 2995, Kim et al., 2002, Vir Res 85: 133-140). PRRS viral contamination of semen is due to PRRSV- infected epithelial germ cells, spermatocytes, macrophages and PRRS viral contamination of the cell-free fraction (Sur et al., 1997, J Virol 71: 9170 – 9179; Christopher Hennings et al., 1998, Vet Pathol 35: 260 – 267). Insemination with PRRSV-contaminated semen does not cause reduced conception, but PRRS does infect embryos and cause early embryonic death (Prieto et al., 1996, Theriogenology, 47: 647 – 654). Aerosol transmission of PRRSV has not been demonstrated under controlled field conditions. (Otake et al., Vet Rec, 2002, 150: 804-808). In contrast, mechanical transmission of PRRSV by mosquitoes, Aedes vexans has been shown experimentally (Otake et al., Can J Vet Res, 2002, 66: 191-195). PRRSV infects pulmonary intravascular macrophages (PIM) and infection of PIMs reduces uptake of intravenously administered copper, suggesting reduced phagocytic capacity (Thanawongnuwech et al., 1998, Vet Pathol 35: 398 – 406) resulting in an increase in susceptebility to S. suis infections (Thanawongnuwech et al., 2000, Vet Pathol 37: 143-152) and S. cholerasuis infections (Wills et al. 2000, Vet Microbiol 71: 177-192). Exposure of pigs to PRRSV and bacterial lipopolysaccharides resulted in severe respiratory signs upon LPS exposure, characterized by tachypnoea, abdominal breathing and dyspnoea. These pigs also showed enhanced general signs, such as fever and depression demonstrating a clear synergism between PRRSV and LPS in the induction of respiratory signs in conventional pigs (Labarque et al., Vet Microbiol, 2002, 88: 1-12). In vitro infection of PIMs caused significantly reduced bactericidal ability, decreased production of superoxide anion and reduced the myeloperoxidase-H2O2-halide product (Thanawongnuwech et al., Vet Immun Immunopath, 1997, 59: 323 – 335). Alveolar macrophages are more permissive for PRRS membrane binding and replication than are peripheral blood monocytes or peritoneal macrophages and cultivation for 1 day of PAM and PBM but not PM increases permissiveness. Results suggest differences in permissiveness are due to expression of membrane receptor for PRRSV (Duan et al., 1997, Arch Virol, 142: 2483 – 2497). The immunology of PRRSV infections has recently been reviewed. (Thacker et al., Vet Clin North-Am, Food Anim Pract 17: 551-565, 2001) as has the expression of cytokines by PRRSV infected pulmonary macrophages (Gomez et al., 2010, 142: 51-60). The capability of host animal to clear PRRSV from the systemic circulation appears to be related to the viral activity in the thymus and brain stem (Shin et al., J Vet Sci, 2002, 3: 75-85). Recent research has investigated nonstructural proteins encoded by ORF1 and identified a helicase (Bautista et al., Virology. 2002, 298: 258-270). An overview of PRRSV diagnostics, its interpretation and limitations was recently published (Christopher-Hennings et al., J Swine Health Prod, 2002, 10: 213-218). Tonsil biopsies can be used for detection of persistently PRRSV-infected breeding age gilts by PCR (Fairbanks et al., J Swine Health Prod, 2002, 10: 87-88). | | | |
| 30 | Whole pig | PRRS (Arterivirus) | Cutaneous hyperemia | |
| 31 | Lung | PRRS (Arterivirus) | Mild interstitial pneumonia | |
| 32 | Lung | PRRS (Arterivirus) | Severe interstitial pneumonia | |
| 33 | Caudal abdomen | PRRS (Arterivirus) | Enlarged iliac lymph nodes | |
| 34 | Lymph nodes | PRRS (Arterivirus) | Nodular lymphoid hyperplasia | |
| 35 | Sow head | PRRS (Arterivirus) | Cutaneous cyanosis, ears and snout | |
| 36 | Fetuses | PRRS (Arterivirus) | Stillborn fetuses | Note the characteristic but inconsistent umbilical lesion in the center fetus: necrotizing vasculitis causing umbilical edema and hemorrhage. |
| 37 | Atypical PRRS: “Outbreaks” in China and Vietnam, 2004-2007, 2006 in Jiangxi Province characterized by high fever and elevated mortality in grow/finish pigs, progressed to high mortality, CNS signs, swollen joints and eyelids, and late-term abortions. The disease was experimentally reproduced by Zhou et el., 2008. According to Chinese sources more than 2 million pigs were affected, there was genetic homogeneity of the strains isolated in the outbreaks (single and 29 AA deletion in Nsp2, highly virulent NA strain) and an effective vaccine was developed. | | | |
| 38 | Whole pig | PRRS (Arterivirus) | Cutaneous infarcts | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 39 | Lung | “Atypical PRRS” | Hemorrhagic interstitial pneumonia | |
| 40 | Lymph nodes | “Atypical PRRS” | Hemorrhage | . |
| 41 | Spleen | “Atypical PRRS” | Multifocal infarcts | |
| 42 | Kidney | “Atypical PRRS” | Hemorrhagic glomerulonephritis | |
| 43 | Lung | “Atypical PRRS” | Hemorrhagic interstitial pneumonia | |
| 44 | Atypical PRRS in Asia: Skepticism by PRRS researchers: Of 1500 "sources" sampled, 30% were PCR positive to the new PRRS variant, but *Streptococcus suis* was isolated from 1/3 of the cases and *Haemophilus parasuis* from another third. The samples are negative for PRV, but classical swine fever is endemic in China's pig herds and producers routinely vaccinate for CSF as well as for foot and mouth disease and pseudorabies. Prior to this most recent outbreak, some herds were being vaccinated against PRRS using a Chinese vaccine. This did not appear to prevent infection with the latest pathogen. | | | |
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| | Funny Picture | | | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 45 & 46 | **PCV2-Associated Diseases in Pigs:** Porcine circovirus (PCV) is one of the smallest (17 nm in diameter) animal viruses with a non-enveloped capsid and a single-stranded, circular genome (**Tischer I et al., Nature 295:64-66, 1982**) It is a member of the *Circoviridae* family, which includes the genera *Circovirus*, containing several avian circoviruses in addition to PCV, and *Gyrovirus*, comprising the chicken anemia virus (**Todd D et al. In: Virus taxonomy, eds. Fauquet C, Mayo M, Maniloff J, et al., pp. 327-334, 2005**) There are currently two types of PCV, porcine circovirus type 1 (PCV1) and type 2 (PCV2). PCV1 was first described in 1974 as a contaminant of the PK-15 porcine kidney cell line (**Tischer I et al., Zentralbl Bakteriol 226:153-167, 1974**) and later recognized as a potential cause of congenital tremors in newborn piglets. PCV2 was identified in the mid to late 1990s in weaned pigs from North America and Europe with a postweaning multisystemic wasting syndrome (PMWS, **Allan et al. J Vet Diagn Invest 10: 3-10, 1998, Clark E. Proc Am Assoc Swine Pract, pp. 499-501, 1997, Daft B. et al. Proc Annu Meet Am Assoc Vet Lab Diagn, p. 32. 1996, Ellis J et al. Can Vet J 39: 44-51, 1998**). Interestingly, a retrospective serologic study determined that PCV2 was already circulating in the pig population at least 10 years before PMWS was first reported (**Magar R et al., Can J Vet Res 64: 184-186, 2000**). Since its original implication as the primary cause of PMWS, PCV2 has also been associated with a number of other diseases in pigs, including respiratory, reproductive, enteric, and cardiovascular disorders. Furthermore, PCV2 has been discussed as a cause of porcine dermatitis and nephropathy syndrome (PDNS). Thus, an all-inclusive term was recently suggested to more accurately acknowledge the broad spectrum of diseases associated with PCV2 infections: porcine circovirus-associated diseases (PCVAD). A significant increase in both incidence and severity of PCVAD, affecting mainly 10 to 15-week-old pigs rather than recently weaned pigs, with a mortality up to 50%, has been observed in North America since fall 2004 (**Horlen KP et al. J Swine Health Prod 15:270–278, 2007**). Affected pigs have unique gross and histologic lesions, including cavitary effusions, marked pulmonary and mesenteric edema, splenic infarcts, typical circoviral intracytoplasmic inclusions in renal tubular and bronchial glandular epithelial cells, and vasculitis (**Carman S. et al., Can J Vet Res 72: 259-268, 2008,, Huang Y et al. Vet Pathol 45:640-644, 2008**). The increased incidence and severity of PCVAD has been attributed to the occurrence of a new subtype of PCV2 (PCV2b), not reported earlier in North America (**Cheung AK et al. Arch Virol 152: 1035-1044, 2007, DeLay J et al. AHL Newsletter 9:22, 2005, Lager KM et al. Vet Rec 161: 428-429, 2007**). PCV2b may be more virulent or may escape existing herd immunity stimulated by the previously circulating subtype of PCV2 (PCV2a). In-situ has been developed to differentiate the two types in tissue sections **(Kim et al., JVDI, 2010, 22: 231-233).** Vasculitis has been recently described as a hallmark lesion of the severe form of systemic PCVAD and experimental infections with PCV2b directly caused acute vasculitis, whereas chronic vasculitis may be in part be mediated by the immune system **(Langohr et al. Vet Pathol 2010, 47: 140-147)**. Another hypothesis is that the introduction of a co-factor led to increased replication of a previously unrecognized type of PCV2 (**Opriessnig T et al., J Vet Diagn Invest 19: 591-615, 2007, Segalés Jet al., Allen D. Leman Swine Conference, pp. 121-125, 2006**). | | |
| 47 | **Post-weaning Multisystemic Wasting Syndrome (PMWS):** A wasting syndrome affecting 5-15% of weaned pigs associated with porcine circovirus type 2 (PCV2). The most characteristic lesion in PMWS is granulomatous inflammation with or without unique globular intracytoplasmic viral inclusion bodies in macrophages. Inclusion bodies have also been described in the cytoplasm of bronchial and renal tubular epithelial cells **(Huang et al., Vet Pathol, 2008, 45: 640-644)**. Less consistent lesions include interstitial pneumonia, interstitial nephritis, myocarditis, hepatitis (with hepatic atrophy and/or icterus) and perivasculitis in a number of tissues **(Ellis et al., 1998, Can Vet J 39:44-51; Morozov et al., 1998, J Clin Micro 36: 2535 – 2541; Kiupel et al., 1998, Vet Pathol 35:303-307 Rosell et al., 1999, J Comp Path 120: 59-78, Krakowka et al., JVDI 17: 213-222, 2005)**. Liver lesions have been identified as a frequent finding and are the most likely cause of icterus and wasting **(Rosell et al., 2000, Vet Path 37: 687-692, Krakowka et al., Vet Pathol 41: 471-481, 2004)**. Ultrastructure of PCV in cell culture **(Stevenson et al., 1999, Vet Pathol, 36: 368-378)** and lymph nodes **(Rodriguez et al., Vet Pathol 2009, 46: 729-735)**. is described PMWS has been associated and reproduced with combined PCV2 and porcine parvovirus inoculation **(Allan et al., 1999 & 2000, J Vet Diagn Invest 11: 3-14 & 12: 1-11, Krakowka et al., Vet Path 37: 254-263, Kim et al., 2006 Vet Pathol 43: 718-725),** combined PCV2 and PRRSV infection **(Harms et al., Vet Path, 38: 528-539) and M. hyopneumoniae (Opriessnig et al., Vet Pathol 41: 599-711, 2004),** prenatal PCV2 infection and postnatal porcine parvovirus infection **(Ha et al., Vet Pathol, 2008, 45: 842-848)** and dual infections with g1-TTV and PCV2 **(Ellis et al., Am J Vet Res, 2008 69: 1608-1614)**. PMWS been reproduced in gnotobiotic pigs with PCV2 alone following administration of keyhole limpet hemocyanin in incomplete Freund's adjuvant **(Krakowka et al., 2001, Vet Path 38: 31-42)**. and has been reproduced with PCV2 alone in cd/cd pigs **(Bolin et. al., J Vet Diagn Invest, 13: 185-194, 2001, Okuda et al., J Vet Diagn Invest, 15: 107-114, 2003)**. Intramuscular injection of pigs with a vaccine against Mycoplasma hyopneumoniae or a nonspecific immunomodulating drug (Baypamun) caused clinical signs, moderate to severe gross and histopathological lesions of PMWS **(Kyriakis et al., J Comp Pathol, 2002, 126: 38-46).** There are also breed-dependent differences to PCV2 associated disease and lesions **(Oppriesnig et al., Vet Pathol 2006, 43:281-293).** It has been shown that MCP-1 expression, but not Il-8 may play a role in the pathogenesis of granulomatous inflammation in pigs with PMWS **(Kim and Chae, Vet Pathol, 40: 181-186, 2003).** The load of PCV2 is associated with lymphoplasmacytic, but not granulomatous inflammation in interstitial nephritis **(Sarli et al., Vet Pathol, 2008, 45: 12-18)**. Vaccination with selective bacterins increased the severity of lesions in conventional pigs infected with | | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| | PCV-2 **(Opriessnig et al., Vet Pathol, 40: 521-529, 2003)**. PCV2 was also associated with transplacental infection of fetuses that were aborted and had myocarditis **(West et al., 1999, J Vet Diagn Invest, 11: 530-532, O'-Connor et al., Can Vet J, 42: 7, 551-553, 2001, Sanchez et al. JVDI 16: 175-185, 2004).** Transplacental transmission of PCV2 has been shown experimentally and virus concentration were highest in the heart **(Sanchez et al., Vet Microbiol 83: 169-176, 2001)**. Porcine circoviruses have been reviewed **(Allan and Ellis, 2000, J Vet Diagn Invest, 12: 3-14)**. Although previous reports indicated that PCV2 capsid proteins localized predominantly in the nuclei of infected cells, abundant amounts of PCV2 capsid proteins were observed in the cytoplasm of many cells of the infected cultures **(Cheung et al., Arch Virol, 2002, 147: 43-58).** PMWS has been detected throughout North America, Europe and Asia **(Kiatipattanasakul et al., J Vet Med Sci, 2002, 64: 449-452, Allan et al., JVDI, 15: 553-560, 2003)).** Idiopathic thymic granulomas may occur **(Baba et al., 2006, Vet Pathol 43: 1037-40).** | | | |
| 48 | Whole pig | PMWS | Icterus | Differentiates from PRRS, inconsistent |
| 49 | Skin | PMWS | Icterus | Differentiates from PRRS, inconsistent |
| 50 | Serum | PMWS | Icterus | Differentiates from PRRS, inconsistent. |
| 51 | Iliac lymph node | PMWS | Nodular lymphadenopathy | |
| 52 | Lung | PMWS | Interstitial pneumonia | |
| 53 | Lung | PMWS | Interstitial pneumonia with lobular atalectasis | |
| 54 | Lung, lymph node | PMWS | Interstitial pneumonia, nodular lymphadenopathy | |
| 55 | Colonic lymph node | PMWS | Nodular lymphadenopathy, colonic lymph nodes | |
| 56 | Head | PMWS | Nodular lymphadenopathy | |
| 57 | Kidney, renal l.n. | PMWS | Renomegally, severe diffuse interstitial nephritis, lymphadenopathy | Differentiates from PRRS, inconsistent. |
| 58 | Liver | PMWS | Icterus, lobular hemorrhage and hepatic atrophy/necrosis | Differentiates from PRRS, inconsistent. |
| 59 | Liver | PMWS | Atrophy/necrosis, fibrosis, regeneration | Differentiates from PRRS, inconsistent. |
| 60 | Liver | PMWS | Atrophy/necrosis, fibrosis, regeneration | Differentiates from PRRS, inconsistent. |
| 61 | Heart | PMWS | Necrosis | . |
| 62 | Whole pig | Porcine Dermatitis and Nephropathy Syndrome | Multifocal cutraneous macules | Cutaneus infarcts (necrotizing vasculitis)<br>Recently dual infection with PRRSV and Torque Teno virus (g1-TTV) *purportedly* resulted in lesions of PDNS. Experimental studies suggest that PDNS is a manifestation of DIC rather than an antibody-antigen complex mediated disease**(Krakowka et al., AM J Vet Res, 2008, 69: 1615-1622)** |
| 63 | Whole pig | Porcine Dermatitis and Nephropathy Syndrome | Multifocal cutraneous macules | |
| 64 | Skin | Porcine Dermatitis and Nephropathy Syndrome | Multifocal cutraneous macules | |
| 65 | Skin | Porcine Dermatitis and Nephropathy Syndrome | Multifocal cutraneous macules | |
| 66 | Skin | Porcine Dermatitis and Nephropathy Syndrome | Multifocal cutraneous macules | |
| 67 | Skin | Porcine Dermatitis and Nephropathy Syndrome | Multifocal cutraneous macules | |
| 68 | Legs | Porcine Dermatitis and Nephropathy Syndrome | Multifocal cutraneous macules | |
| 69 | Kidney, renal l.n. | Porcine Dermatitis and Nephropathy Syndrome | 1. Severe diffuse glomerulonephritis<br>2. Lymphadenopathy | necrotizing vasculitis |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 70 | Kidney | Porcine Dermatitis and Nephropathy Syndrome | Severe diffuse glomerulonephritis | necrotizing vasculitis |
| 71 | **Classical Swine Fever (Hog Cholera, European Swine Fever), (Ruiz-Villamor et al., J Comp Path 124: 246-254, 2001): Acute virulent form**: Pigs are pyretic with cutaneous cyanosis, conjunctivitis, anorexia, constipation followed by severe diarrhea ("cholera"), convulsions and death. Lesions include peripheral hemorrhage of lymph nodes, generalized vasculitis, tonsillar necrosis, splenic infarcts, serosal hemorrhages, button ulcers in colon. **Subacute form**: pyrexia, diarrhea, low mortality with few gross lesions. **Reproductive form**: mummified, stillborn and weakborn pigs, congenital tremors, cerebellar hypo- or aplasia, limb deformation, arthrogryposis (underlined are lesions that help differentiate from existing US diseases). **Not currently in North America. (Dewulf et al., J Vet Med B, 48: 8, 583-591, 2001).** Allmost complete loss of lymphocytes associated with follicular necrosis of lymphoid tissues in pigs infected with highly virulent strains. B lymphocyte loss not prominent in pigs infected with less virulent strains. Increased activity of T lymphocytes with all strains **(Summerfield et al., Vet Immun Immunopath, 78: 3-19, 2001)**. CSF antigen detected in tonsillar epithelial cells, macrophages, endothelial cells in lymphoid tissues **(Narita et al., 2000, Vet Path 37: 402-408)**. Pulmonary intravascular macrophages have also been shown as target cells for CSFV infection (**Carrasco et al., J Comp Path, 125: 1-7, 2001)** and atypical cilia were observed in the bronchiolar epithelium **(Carrasco et al., J Comp Path, 124: 1, 29-35, 2001)**. CSFV infects bone marrow haematopoietic cells, especially myelomonocytic precursors, and causes apoptosis. **(Summerfield et al., J Gen Virol, 82: 1309-1318, 2001, Sanchez et al., Vet Pathol 42: 477-488, 2005).** Thrombocytopenia is caused by massive activation and subsequent phagocytosis of platelets secondary to the release of platelet-activating factors by activated macrophages infected with CSFV **(Bautista et al., Vet Pathol, 2002, 39: 84-91)**, for more pathology of CSFV in bone marrow: **Gómez-Villamandos et al., Vet Pathol, 40: 157-163, 2003.** The pathogenesis of CSFV in intestinal disease has recently been reviewed **(Sánchez-Cordón et al., Vet Pathol, 40: 254-262, 2003, Handel et al., JVDI 16: 132-138, 2004)** as has the pathogenesis of brain lesions: **Gómez-Villamandos et al., Vet Pathol, 43: 530-540, 2006.** In a recent study, perivascular cuffing of mononuclear cells in the gray and white matter of the brain was the most consisten microscopic lesion. Viral antigen has been detected consistently by IHC and in-situ in mononuclear cells of lymphoid tissues **(Cho et Chae, Vet Pathol, 2003, 40: 107-113).** A recent review: **(Gregg, J Swine Health Prod, 2002, 10: 33-37).** Pigs infected with BVDV-2 might develop antibodies that cross-react in tests for antibodies against classical swine fever virus. In addition, pigs developed leucopenia and thrombocytopenia after infection with BVDV-2 **(Makoschey et al., Dts Tierarztl Wochens, 2002, 109: 225-230).** | | | |
| 72 | Whole animal | Classical swine fever (Pestivirus) | Cutaneous infarcts | DDX: bacterial septicemia |
| 73 | Whole animal | Classical swine fever (Pestivirus) | Cutaneous infarcts | DDX: bacterial septicemia |
| 74 | Pinna | Classical swine fever (Pestivirus) | Cutaneous infarcts | DDX: bacterial septicemia |
| 75 | Leg | Classical swine fever (Pestivirus) | Cutaneous infarcts | DDX: bacterial septicemia |
| 76 | Head/eye | Classical swine fever (Pestivirus) | Conjunctivitis | DDX: Chlamydia suis, Porcine paramyxovirus |
| **Conjunctivitis in swine:** Chlamydia suis causes subclinical conjunctivitis in pigs (Rogers and Anderson, 1999, J Vet Diagn Invest, 11: 341-344), Porcine Paramyxovirus (Blue Eye Disease) causes conjunctivitis, encephalitis, pneumonia and reproductive failure review: (Ramirez-Herrera et al., 1997, J Vet Med B 44: 461-476, Corona, 2000, Pig J 45: 115-118, Hernandez-Jauregui et al., 2004, J Comp Pathol 130: 1-6,), in Mexico. | | | | |
| 77 | Larynx | Classical swine fever (Pestivirus) | Multifocal laryngeal hemorrhages | DDX: Pseudorabies |
| 78 | Tonsil | Classical swine fever (Pestivirus) | Multifocal tonsillar necrosis | DDX: Pseudorabies |
| 79 | Tonsil | Classical swine fever (Pestivirus) | Multifocal tonsillar necrosis | DDX: Pseudorabies |
| 80 | Tonsil | Classical swine fever (Pestivirus) | Multifocal tonsillar necrosis | DDX: Pseudorabies |
| 81 | Mesenteric lymph node | Classical swine fever (Pestivirus) | Peripheral hemorrhagic lymphadenitis | DDX: Salmonellosis |
| 82 | Heart | Classical swine fever (Pestivirus) | Multifocal atrial petechia | |
| 83 | Lung | Classical swine fever (Pestivirus) | Interstitial pneumonia | |
| 84 | Lung | Classical swine fever (Pestivirus) | Interstitial hemorrhagic pneumonia | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 85 | Spleen | Classical swine fever (Pestivirus) | Multifocal splenic infarcts | Hallmark lesion of CSF |
| 86 | Spleen | Classical swine fever (Pestivirus) | Multifocal splenic infarcts | Hallmark lesion of CSF |
| 87 | Spleen c/s | Classical swine fever (Pestivirus) | Spleenic infarct | |
| 88 | Stomach | Classical swine fever (Pestivirus) | Multifocal serosal hemorrhages | |
| 89 | Stomach | Classical swine fever (Pestivirus) | Venous infarction | DDX: S. choleraesuis, typhimurium or typhisuis |
| 90 | Colon | Classical swine fever (Pestivirus) | Multifocal serosal hemorrhages | Lesions in pigs recently affected with classical swine fever in Northern Europe have been mild and difficult to distinguish from septicemia: follicular depletion of lymphocytes, histiocytic hyperplasia, hematopoiesis in spleen; antigen widespread, but not associated with lesions (current marker vaccine trials: **(Uttenthal et al. and Deppner et al., Vet Microbiol, 83: 85-106 and 107-120, 2001)** |
| 91 | Colon | Classical swine fever (Pestivirus) | Ulcerative colitis | |
| 92 | Colon | Classical swine fever (Pestivirus) | Diffuse necrohemorrhagic colitis | |
| 93 | Colon | Classical swine fever (Pestivirus) | Multifocal ulcerative colitis (button ulcers) | DDX: S. choleraesuis, typhimurium or typhisuis |
| 94 | Colon | Classical swine fever (Pestivirus) | Multifocal ulcerative colitis (button ulcers) | DDX: S. choleraesuis, typhimurium or typhisuis |
| 95 | Kidney | Classical swine fever (Pestivirus) | Multifocal renal cortical petechia | DDX: S. choleraesuis, Erysipelas, PDNS, ASF |
| 96 | Kidney | Classical swine fever (Pestivirus) | Multifocal renal cortical petechia | DDX: S. choleraesuis, Erysipelas, PDNS, ASF |
| 97 | Urinary bladder | Classical swine fever (Pestivirus) | Multifocal serosal hemorrhages | |
| 98 | Fetuses | Classical swine fever (Pestivirus) | Fresh and autolyzed stillborn fetuses | |
| 99 | Brain | Classical swine fever (Pestivirus) | Cerebellar aplasia | Hallmark lesion of CSF |
| 100 | Piglet | Classical swine fever (Pestivirus) | Congenital tremors | |
| 101 | **African Swine Fever: Acute form**: High fever, terminal bloody diarrhea and death. Consistant lesions include hemorrhage and necrosis of lymphoid organs, hemorrhages on serosal surfaces, renal cortical, medullary and pelvic hemorrhage and hydropericardium and hydrothorax. **Subacute form**: Less fatal with hemorrhagic lymph nodes, spleen and kidneys. **Chronic form**: lymphoid hyperplasia, fibrous pleuritis and pericarditis, and pneumonia. **Not currently in North America.** The pathogenesis **has recently been reviewed: Gomez-Villamandos et al., Rec Res Devel Virol, 1: 7-17, 1999, Alonso et al., Rec Res Devel Virol,1: 277-285, 1999,** the role of NF kappa B was recently studied **(Palgrave et al., J Virol. 2011 epub.)** | | | |
| 102 | Host reservoir | | | |
| 103 | Transmission | | | |
| 104 | Whole animal | African Swine Fever | Hyperemia | DDX: bacterial septicemia |
| 105 | Whole animal | African Swine Fever | Cutaneous infarcts | DDX: bacterial septicemia |
| 106 | Head | African Swine Fever | Conjunctival hemorrhages | DDX: bacterial septicemia |
| 107 | Kidney, renal l.n. | African swine fever | Perirenal edema, renal cortical petechiae, hemorrhage of renal l.n. | (unclassified virus) |
| 108 | Kidney | African Swine Fever | Multifocal coelescing renal cortical and medullary hemorrhage | |
| 109 | Kidney c/s | African Swine Fever | Multifocal coelescing renal cortical and medullary hemorrhage | Hemorrhage more diffuse and deeper in cortex than with CSF, Salmonellosis |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 110 | Renal l.n. | African Swine Fever | Hemorrhagic and necrotic renal lymphnodes | African swine fever virus inhibits caspase activation and promotes cell survival in mammalian cells. (**Nogal et al., J Virol, 75: 6, 2535-2543, 2001)** |
| 111 | Spleen | African Swine Fever | Splenic hemorrhage and necrosis | DDX: Mycoplasma haemosuis (Eperythrozoon suis), **Ritzmann et al., 2002, Tierarztl Praxis 30: 72-74; Ha et al., 2006 J Comp Pathol. 133: 294-297)** |
| 112 | Abdomen, thorax | African Swine Fever | Myocardial hemorrhage and anemia | |
| 113 | Lung | African Swine Fever | Pulmonary edema | DDX: Bacterial septicemia, fumonosin intoxication |
| 114 | Lung | African Swine Fever | Pulmonary edema | |
| 115 | Heart | African Swine Fever | Hydropericardium | |
| 116 | Whole body | African Swine Fever | Bloody diarrhea | A terminal event caused by DIC and thrombocytopenia **(Gomez-Villamandos et al., J Comp Path 118:1-13 & 119:111-119, 1998, Sanchez-Cordon et al., J Comp Path 127: 239-248, 2002)** |
| 117 | Colon | African Swine Fever | Bloody diarrhea | |
| 118 | Stomach | African Swine Fever | Venous infarction | DDX: S. choleraesuis, typhimurium or typhisuis |
| 119 | Whole body | African Swine Fever | Poor growth | |
| 120 | Lymph node | African Swine Fever | Chronic lymphadenitis | |
| 121 | Heart | African Swine Fever | Pericardial fibrosis | |
| 122 | Lung | African Swine Fever | Pleural fibrosis | |
| 123 | Fetuses | African Swine Fever | Abortion | |
| 124 | **Eperythrozoonosis:** Haemoplasma, *Mycoplasma suis, that t*argets red blood cells. These are cell wall-less bacteria that are consistently pathogenic, but chronic or latent infections are common. The disease is distributed worldwide. Clinical signs include anemia and jaundice in piglets. Grossly, the spleen is enlarged, and infarcts may occur. | | | |
| 125 | Spleen | Eperythrozoonosis | Splenomegaly | |
| 126 | **GASTROINTESTINAL SYSTEM** | | | |
| 127 | Oral cavity | Laceration | Multifocal necroulcerative gingivitis and chelitis | Caused by careless trimming of “milk” or “needle” teeth |
| 128 | Oral cavity | T-2 toxin | Necroulcerative gingivitis and stomatitis | DDX: chemical burns |
| 129 | Tongue | Staphylococcus hyicus | Focally extensive ulcerative glossitis | Seen in about 1/3 of animals affected with exudative epidermitis |
| 130 | Tongue | FMD (Picornavirus, Aphtovirus) | Multifocal vesicular glossitis | DDX: swine vesicular disease, vesicular stomatitis, vesicular exanthema |
| 131 | Esophagus | Candida albicans | Diffuse pseudomembranous esophagitis (fibrinonecrotic) | |
| 132 | Stomach | Candida albicans | Diffuse pseudomembranous gastritis (fibrinonecrotic) | |
| 133 | Stomach | Candida albicans | Diffuse pseudomembranous gastritis (fibrinonecrotic) | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 134 | **Ulceration of the pars-esophagea:** Costly problem in the swine industry. Risk factors include: gender (barrows), genotype, season (summer), particle size of feed, anorexia (concurrent disease), Spiral organisms and/or Gastrospirillum sp. (now Helicobacter heilmannii) have been associated with ulcers **(Queiroz et al., 1996, Gastroenterology 111:19-27; Barbosa et al., 1995 Vet Path 32:134-139)**. However, inoculation studies did not demonstrate an effect of H. heilmannii, but did reproduce ulcers by feeding fermentative commensal bacteria with a high carbohydrate diet **(Krakowka et al., 1998, Vet Pathol 35: 274-282).** Recently, it was suggested that an imbalance between cell proliferation and programmed cell death is an underlying feature of gastric ulcers **(Preziosis et al. 2000, Res Vet Science 68: 189-196)**. Helicobacter pylori causes lymphoplasmacytic gastritis in experimentally inoculated pigs similar to human disease **(Poutahidis et al., Vet Path, 38: 667-678, 2001).** Experimental infection of the gastric mucosa of mice with tightly coiled spiral bacteria (candidatus H. suis) was closely associated with chronic gastritis and dysplastic lesions **(Park et al., J Comp Pathol, 2003, 129: 154-160)**. Heat shock proteins may play a role in the defense mechanisms of the gastric mucosa **(Marruchella et al., 2004, J Comp pathol 131: 10-17).** | | | |
| 135 | Oral mucous membrane | Ulceration of the pars esophagea | Anemia | "bleach outs" |
| 136 | Stomach, Colon | Ulceration of the pars esophagea | Ulceration of the pars esophagea with associated hemorrhage | Gastric hemorrhage, gastric ulcer-esophageal portion, digested blood in colon |
| 137 | Stomach | Ulceration of the pars esophagea | Gastric hemorrhage, gastric ulcer-esophageal portion | |
| 138 | Stomach | Ulceration of the pars esophagea | Gastric hemorrhage, gastric ulcer-esophageal portion | “Coffee ground” material (digested blood) in stomach |
| 139 | Stomach c/s | Ulceration of the pars esophagea | Gastric ulcer-esophageal portion | |
| 140 | Esophagus | Esophageal perforating ulcer | Esophageal perforation, necrotizing esophagitis | Gastric ulcer w/ reflux |
| 141 | Esophagus | Esophageal stenosis | Esophageal perforation secondary to gasstric ulcer | Gastric ulcer w/ reflux |
| 142 | Stomach | Salmonella typhimurium | Gastric ulcer, fundic portion | Secondary to vascular thrombosis |
| 143 | Stomach | Aspergillus fumigatus | Gastric ulcer, fundic portion | |
| 144 | Stomach | Hyostrongylus rubidus | Gastric nematodiasis, Hyostrongylus rubidus | Usually not pathogenic, but can induce hyperplasia and ulceration |
| 145 | Stomach | Anisakis | Gastric anisakiasis, focal ulcerative gastritis | Multiple fish species are intermediate host, pigs can be infected through fish meal, similar lesions in humans |
| 146 | Stomach | Hemagglutinating encephalomyelitis virus | Gastric Dilatation (Degeneration of intramural ganglia) | Syn.: Vomiting and Wasting Disease (Coronavirus), causes nonsuppurative encephalomyelitis **(Hirano et al., J Comp Path, 125: 8-14, 2001)** |
| 147 | Pig | Hemagglutinating encephalomyelitis virus | Vomiting pig | |

**File 3**

**DDX: Diarrhea in swine:**

**Without blood:** **Colibacillosis**
**Clostridium perfringens type A**
**Coccidiosis**
**Viral enteritis**
**Proliferative enteritis (except PHE)**
**Whipworms**
**Intestinal spirochetosis**

**With blood:** **Clostridium perfringens type C**
**Salmonellosis (dark digested blood)**
**Proliferative enteritis - PHE form**
**Swine dysentery**
**Whipworms**

| | | | |
|---|---|---|---|
| 1 | Small intestine | Normal | Chyle in the lymphatics. Pigs suckle ≈ every hour. The stomach should always contain milk. Lymphatics in the proximal ½ of the small intestine in a normal suckling pig should have chyle. Villus/crypt ratio normally decreases as gut is colonized by microflora. 2 day old pig: V:C = 10:1, 3-4 week old pig: V:C = 4-6:1 |
| 2 | E coli infections | | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 3 | Word slide ETEC | Hemolytic or non-hemolytic, small intestine only, colonize via fimbria: suckling pigs - K88, K99, 987P, F41; weaned pigs - K88, F18ac (2134P), F18ac and F18ab fimbriae can be involved in causing either postweaning diarrhea or edema disease **(DebRoy et al, JVDI, 2009, 21: 359-364)** accumulates due to enterotoxins (secretory diarrhea): LT (A and B units) activates adenylate cyclase > ↑ cAMP > increased secretion of Cl, Na, $HCO_3$, water into lumen; STa & Stb activates guanylate cyclase > ↑ GMP > inhibit Na/Cl cotransport system, Hpth.: Uniform colonization of the brush border **(Madec et al., 2000, Vet Microbiol 72: 295-310, Francis J 2002, Swine Health Prod 10: 171-175, Pritchard et al., JVDI 16: 108-115, 2004; Berberov et al., Infect Immun 2004; 72: 3914-3924).** A potentially new pathogenic ETEC fimbrial type has been described in piggeries in Vietnam **(Do et al., 2006, Vet Pathol 43:150-160).** | | |
| 4 | Word slide EPEC | AAEC - attaching and effacing E. coli, uncommon in 1-6 week old pigs, "classic AE lesion", small and large intestine, attach by *eae* gene product - 94 Kd protein "intimin", verotoxin negative, Hpth.: Colonization and degeneration of villous enterocytes; "cobblestone" appearance of brush border | | |
| 5 | Small intestine | Colibacillosis; E. coli | Catarrhal enteritis, congestion | Fluid distention, congestion |
| 6 | Small intestine | Colibacillosis; E. coli | Catarrhal enteritis, congestion | Homogeneous fluid contents |
| 7 | Small intestine | Colibacillosis; E. coli | Enteritis, hemorrhagic, mild | Homogeneous bloody fluid contents Detection of adhesion factor genes F4, F18 and intimin suggested when diagnosing post-weaning diarrhea **(Frydendahl, Vet Microbiol, 2002, 85: 169-182)** |
| 8 | Word slide Atrophic enteritis | Differentials for atrophic enteritis in pigs: TGE (Coronavirus), Rotavirus (A, C, B ), Coccidiosis (Isospora suis). Less likely differentials: chlamydia, adenovirus, enteric calicivirus, astrovirus, parvovirus. Protective immunity against enteric viruses depends on the magnitude, location, viral protein-specificity, and isotype of the antibody responses induced by vaccination. Therefore highly effective enteric viral vaccines should: (i) induce sufficient levels of intestinal IgA antibodies; (ii) include viral antigens that induce neutralizing antibodies; and (iii) require the use of effective mucosal adjuvants or antigen delivery systems for non-replicating oral or i.n. vaccines. **(Yuan et al., Vet Immunol Immunopath, 2002, 87: 147-160)** | | |
| 9 | TGE causes apoptosis infected and bystander cells in cell culture **(Sirinarumitr et al., 1998, Arch Virol 143: 2471-2485)** and has been shown to induce IFN-alpha producing cells in the small intestine **(Riffault et al., Vet Res, 32: 1, 71-79, 2001).** Chlamydophila suis replicated in mature enterocytes on the distal half of villi in gnotobiotic pigs and caused atrophic enteritis **(Rogers and Anderson, 1996, J Vet Diagn Invest 8: 433-440; 2000, J Vet Diagn Invest 12: 233-239)**. The same organisms were demonstrated as incidental findings almost exclusively in mature colonic enterocytes in finishing age pigs at an abattoir **(Szeredi et al., 1996, Vet Path 33: 369-374)**. | | |
| 10 | Pig | Transmissible gastroenteritis (Coronavirus) | Diarrhea, kachexia and dehydration | Hpth.: severe villous atrophy; V:C =2:1 Different strains vary in their virulence and affect different segments of small intestine **(Kim et al., J Comp Pathol, 2002, 126: 30-37)** |
| 11 | Small intestine | TGE virus | Atrophic enteritis | Lack of chyle absorption |
| 12 | Small intestine | TGE virus | Atrophic enteritis | Thin wall; maldigestion, PED in Europe, similar to TGE **(Pospischil et al., 2002, J Swine Health Prod. 10: 81-85, Kim et al., J Comp Pathol, 2003, 129: 55-60)** |
| 13 | Small intestine | TGE virus | Normal villi | |
| 14 | Small intestine | TGE virus | Loss of villi | |
| 15 | Word slide Isospora suis | Infections occur most commonly in pigs from 5 days to 4 weeks-of-age, rarely in older pigs (Eimeria debliecki). I. suis replicates through 2 sequential asexual cycles (schizogeny) and 1 sexual cycle (gametogeny) in the cytoplasm of the epithelial cells in the small intestine causing moderate to severe atrophic enteritis. Grossly, there is a bright yellow fibrinous mucosal pseudomembrane that can be removed with gentle scraping to reveal a glistening mucosa beneath **(Niestrath et al., J Vet Med Series B, 2002, 49: 176-180)**. | | |
| 16 | Small | Coccidiosis; Isospora | Diffuse catarrhal enteritis | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| | intestine | suis | | |
| 17 | Small intestine | Coccidiosis; Isospora suis | Diffuse fibrinonecrotic enteritis | Eimeria debliecki or spinosa cause similar disease in adult swine **(Yaeger et al., JVDI, 15: 387-389, 2003)** |
| 18 | Small intestine | Coccidiosis; Isospora suis | Diffuse fibrinonecrotic enteritis | Pseudomembrane; yellow, easily removed from mucosa. DDX: Subacute C. perfringens type C |
| 19 | Word slide Clostridium perfringens type C | Occurs in piglets less than 1 week of age. Some pigs may survive initially, but tend to grow poorly and die by 2 – 3 weeks-of-age. C. perfringens type C is present in small numbers in sow feces. Some C. perfringens strains out-compete "normal flora" in the gut of piglets causing segmental transmural necrohemorrhagic enteritis with subserosal and intramural emphysema **Review: Keel and Songer 2006, Vet Pathol 43: 225-240).** | | |
| 20 | Perineum | C. perfringens type C | Bloody diarrhea (necrohemorrhagic enteritis) | Usually 1-4 days of age |
| 21 | Small intestine | C. perfringens type C | Acute segmental necrohemorrhagic enteritis | Segmental, usually small intestine |
| 22 | Small intestine | C. perfringens type C | Acute necrohemorrhagic enteritis with subserosal emphysema | |
| 23 | Small intestine | C. perfringens type C | Acute necrohemorrhagic enteritis with subserosal emphysema | |
| 24 | Intestines | C. perfringens type C | Acute segmental necrohemorrhaic colitis with subserosal emphysema | Unusual location |
| 25 | Small intestine | C. perfringens type C | Diffuse subacute transmural necrohemorrhagic enteritis | Subacute disease in 1-2 week-old pigs; partial protection from lactogenic immunity |
| 26 | Word slide | C. perfringens type A | Putative cause of diarrhea in pigs 1-4 days of age; high morbidity, low mortality; rare gross (mesocolonic edema) or microscopic lesions (mild neutrophilic enteritis); overgrowth in intestinal contents. **(Collins, 1994, Swine Health and Production 2: 24-25). (Fernandez et al., JVDI, 2007, 19:184-186).** DDX: C. difficile, B. fragilis, E. coli. **Review: Songer JVDI, 17:528-536, 2005, Keel and Songer 2006, Vet Pathol 43: 225-240** | |
| 27 | Word slide | C. difficile | Diarrhea in 1-14 day old pigs; also with ascites, subcutaneous edema. Clostridium difficile toxins A and B can be detected in feces of pigs for the diagnosis of neonatal swine enteritis **(Post et al., JVDI 2002, 14: 258-259, Yaeger et al., JVDI, 2002, 14: 281-287, experimental study: Yaeger et al., 2007, JVDI 19: 52-59)** Reproduction of lesions with toxin A due to receptor mediated endocytosis **(Keel and Songer 2011, Vet Pathol 43: 369-380)** DDX: C. perfringens type A, B. fragilis, E. coli. | |
| 28 | Thorax and abdomen | C. difficile | Severe diffuse mesocolonic edema | **Waters et al., 1998, J Vet Diagn Invest 10: 104-108** Hpth.: Multifocal erosive colitis with marked fibrinosuppurative exudation |
| 29 | perineum | C. difficile | Severe diarrhea | |
| 30 | Colon | C. difficile | Severe diffuse mesocolonic edema | |
| 31 | Colon | C. difficile | Severe diffuse mesocolonic edema<br>Severe diffuse fibrinonecrotic colitis | |
| 32 | Word slide | Porcine proliferative enteropathy/enteritis | Lawsonia intracellularis, obligate intracellular curved rod-shaped bacteria, Koch's postulates fullfilled, recent reviews **(Lawson & Gebhardt, 2000, J Comp Path 122: 2-3, 77-100, Knittel, 1999, Comp Cont Edu 21: S53-S59)** There was recent evidence of the presence of an immunosuppressive mechanism operating in this disease **(MacIntyre et al., Vet Pathol, 40: 421-432, 2003).** L. intracellularis has been found in tonsils of pigs with PPE associated with necrosis **(Jensen et al., 2000, Res Vet Science 68: 23-26)** | |
| 33 | Ileum | Porcine proliferative enteropathy | Diffuse proliferative enteritis | Syn.: Porcine intestinal adenomatosis |
| 34 | Ileum | Porcine proliferative enteropathy | Diffuse proliferative enteritis | Syn.: Porcine intestinal adenomatosis |
| 35 | Ileum | Porcine proliferative | Diffuse proliferative enteritis | **Jensen et al., J Comp Path 2006,** |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| | | enteropathy | | **135: 176-182** |
| 36 | Ileum | Porcine proliferative enteropathy | Fibrinonecrotic enteritis | Syn.: Necrotic enteritis |
| 37 | Ileum | Porcine proliferative enteropathy | Fibrinonecrotic enteritis with intraluminal cast | |
| 38 | Colon | Porcine proliferative enteropathy | Fibrinonecrotic colitis | Often involves proximal 1/3 of the spiral colon |
| 39 | Perineum | Porcine proliferative enteropathy | Bloody diarrhea | **Bane et al., J Swine Health Prod., 9: 155-158, 2001** |
| 40 | Ileum | Porcine proliferative enteropathy | Hemorrhagic proliferative enteritis | Syn: Proliferative Hemorrhagic Enteropathy (PHE) |
| 41 | Ileum | Porcine proliferative enteropathy | Fibrinohemorrhagic proliferative enteritis | Syn: Proliferative Hemorrhagic Enteropathy (PHE) |
| 42 | Cecum | Porcine proliferative enteropathy | Hemorrhagic proliferative typhlitis | Often involves proximal 1/3 of the spiral colon |
| 43 | Cecum | Porcine proliferative enteropathy | Hemorrhagic proliferative typhlitis | |
| 44 | Word slide Brachyspira sp. (Serpulina sp.) in swine | 6 species of Brachyspira in swine **(Hulto et al., 1999, Vet Path 36: 412-422)**: Strongly β hemolytic - B. hyodysenteriae and hampsonii (Swine Dysentery); Weakly β hemolytic – B. pilosicoli (Intestinal Spirochetosis), B. innocens, S. intermedia* and S. murdochii* (all 3 are nonpathogenic or *occasionally mildly pathogenic). **(Lee et al., 1993, Vet Microbiol 34:273-285, Stanton et al., 1997, Int J System Bact 47:1007-1012)**. Brachyspira species cannot be differentiated based on morphology at a LM level (vissible with Victorian Blue or a Silver stain). Even when present with microscopic lesions typical of disease, positive identification by culture or PCR is needed to confirm a diagnosis. Restriction fragment length polymorphism analysis, or more recently, sequencing of the Brachyspira nox gene is used for differentiation of Brachyspira spec. **(Rohde et al., J Clin Microbiol, 2002, 40: 2598-2600; Townsend et al., JVDI 17: 103-109, 2005; Chander et al., JVDI 24:903-10; Burrough et al., JVDI 24:1025-34; Rubin et al., PloS One 8:e57146)** | | |
| 45 | Colon | Swine dysentery | Diffuse catarrhal and hemorrhagic colitis | Brachyspira hyodysenteriae |
| 46 | Colon | Swine dysentery | Severe diffuse catarrhal and hemorrhagic colitis | **Jensen et al., 2000, Vet Path 37: 22-32, Novotna et al. 2002, Vet Med 47: 104-109** |
| 47 | Colon | Swine dysentery | Severe diffuse fibrinonecrotic colitis | |
| 48 | Word slide | Intestinal spirochetosis Brachyspira pilosicoli | Weaned to adult animals, a mild catarrhal or fibrinous colitis with a loose "wet-cement-like" stool. B. pilosicoli transiently colonizes the surface of colonic mucosa creating a "false brush border" . Lesions are commonly of a mild superficial erosive colitis with goblet cell hyperplasia and mats of serpentine spirochetes in crypts **(Trott et al., 1996, Int J Sys Bact 46 (1): 206-215; Trott et al., 1996, Infec Immun 64:4648-4654; Thomson et al., 1996, Infec Immun 65:3693-3700, Worarach et al., J Thai Vet Med Assoc, 2002, 53: 25-33).** Recently, the weakly beta-hemolytic porcine spirochete *Brachyspira murdochii*, considered a normal intestinal commensal, was associated with a catarrhal colitis in a pig characterized by extensive spirochetal colonization of the surface epithelium **(Jensen et al., Vet Pathol, 2010, 47: 334-338)** | |
| 49 | Colon | Intestinal spirochetosis | Mild diffuse erosive colitis | |
| 50 | Colon | Intestinal spirochetosis | Mild diffuse fibrinocatarrhal colitis | |
| 51 | Colon | Multiple causes | Colitis cystica | Abscessed lymphoglandular complexes. A nonspecific lesion. |
| 52 | Ileum | Salmonella typhimurium | Severe diffuse fibrinonecrotic enteritis | **Asai et al., J Vet Med Sci. 2002, 64: 2, 159-160** |
| 53 | Colon | Salmonella typhimurium | Multifocal to coelescing fibrinonecrotic colitis | Early cellular invasion of S. typhimurium is rapid and non-specific (M cells, goblet cells and enterocytes). **Meyerholz et al., Vet Pathol. 2002, 39: 712-720** |
| 54 | Colon | Salmonella typhimurium | Severe diffuse fibrinonecrotic colitis | |
| 55 | Whole body | Salmonella typhimurium | Abdominal distention | |
| 56 | Colon | Salmonella | Megacolon | DDX: Lupin meal intoxication (— 10% |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
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| | | typhimurium | | of ration) |
| 57 | Rectum | Salmonella typhimurium | Rectal stricture | DDX: Sequel to rectal prolapse |
| 58 | Rectum | Salmonella typhimurium | Rectal stricture | DDX: Sequel to rectal prolapse |
| 59 | Rectum | Rectal prolapse | Rectal prolapse | Risk factors: genetic (Yorkshire), piling in cold weather, coughing, estrogenic mycotoxins (gilts) |
| 60 | Colon | Salmonella typhisuis | Severe chronic multifocal fibrinonecrotic ulcerative colitis | DDX: "button ulcers" in colon: Salmonella choleraesuis, typhimurium or typhisuis, Classical swine fever (Hog cholera). |
| 61 | Cecum | Trichuris suis; whipworms | Diffuse catarrhal typhlitis with many Trichuris suis | May cause colitis for ~ 1 week before emergence of adults; may be catarrhal, necrotic or necrohemorrhagic **(Chiou et al., Taiwan Vet J 2002, 28: 142-147)** |
| 62 | Cecum | Trichuris suis; whipworms | Diffuse hemorrhagic typhlitis with many Trichuris suis | |
| 63 | Cecum | Trichuris suis; whipworms | Diffuse typhlitis with many Trichuris suis | |
| 64 | Colon | Warfarin intoxication | Mesocolonic hemorrhage | Anticoagulant rodenticide |
| 65 | Whole pig | Warfarin intoxication | Ocular hemorrhage | Interference with vitamine K utilization |
| 66 | Abdomen | Inguinal hernia | Inguinal hernia with intestinal incarceration and infarction | Male>female, left>right (unilateral), Weakness of tunica vaginalis |
| 67 | Jejunum | Intestinal volvulus | Small intestinal volvulus and infarction | DDX: hemorrhagic bowel syndrome |
| 68 | Jejunum | Intestinal volvulus | Small intestinal volvulus and infarction | DDX: hemorrhagic bowel syndrome |
| 69 | Small intestine | Hemorrhagic Bowel Syndrome | Hemorrhagic Bowel Syndrome | Thin walled, blood filled intestines, no diarrhea, 3-6 months old pigs **(Waters et al., Vet Clin North-Am, Food Anim Pract 17: 517-534, 2001)**, unknown cause, but most likely non-infectious, involving intestinal volvulus **(Straw et al., J Swine Health Prod, 2002, 10: 75-79)** |
| 70 | Colon | Colocecal volvulus | Colocecal volvulus | Thick, edematous wall secondary to vascular accident |
| 71 | Colon | Colocecal volvulus | Colocecal volvulus | Infarction |
| 72 | Colon | Colocecal volvulus | Colocecal volvulus | |
| 73 | Colon | Gastric ulcer | Gastrointestinal hemorrhage | |
| 74 | Small intestine | Hypervitaminosis D | Serosal mineralization, small intestine | |
| 75 | Thoracic l.n. | Tuberculosis, Mycobacterium avium | Multifocal granulomatous lymphadenitis | DDX: Rhodococcus equi ? **Madarame et al., 1998, J Comp Path 119: 397-405** |
| 76 | Mesenteric l.n. c/s | Tuberculosis, Mycobacterium avium | Multifocal granulomatous lymphadenitis with mineralization | |
| 77 | Stomach | Tuberculosis, Mycobacterium avium | Multifocal granulomatous gastritis | DDX: candidiasis (poor choice) |
| 78 | Spleen | Tuberculosis, Mycobacterium avium | Multifocal granulomatous splenitis or multifocal splenic granulomas | DDX: lymphoma (poor choice) |
| 79 | Spleen | Tuberculosis, Mycobacterium avium | Splenic granuloma | DDX: abscess (poor choice) |
| 80 | Kidney | Tuberculosis, Mycobacterium avium | Extensive granulomatous nephritis | DDX: lymphoma (maybe a better choice!) |
| 81 | Meliodosis | | | |
| 82 | Spleen | Burkholderia pseudomallei | Multifocal splenic abscesses or multifocal splenic granulomas | Not currently in North America. **Najdenski et al., 2004, J Vet Med B 51: 225-230; Choy et al., 2000, Acta** |
| 83 | Liver | Burkholderia | Multifocal hepatic abscesses | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| | | pseudomallei | | **Trop. 74:153-158** (Review) |
| 84 | Lung | Burkholderia pseudomallei | Multifocal pulmonary abscesses | |
| 85 | Lymph node | Burkholderia pseudomallei | Multifocal abscesses | |
| 86 | Stomach | Ascaris suum, roundworms | Gastric ascariasis | Usualy in small intestine (bile duct) |
| 87 | Small intestine | Ascaris suum, roundworms | Intestinal ascariasis | **Kon et al., 2002, J Jap Vet Med Ass 55: 240-243** |
| 88 | Small intestine | Macrocanthyrhynchus sp. | Intestinal acanthocephalidiasis with a fibrous mucosal nodule | |
| 89 | **Liver** | | | |
| 90 | Liver | Pseudorabies (Porcine Herpesvirus) | Multifocal hepatic necrosis | Suckling pigs: DDX: Multifocal septic hepatitis caused by S. equisimilus or Listeria monocytogenes |
| 91 | Liver | Salmonella choleraesuis | Multifocal hepatic necrosis | DDX: Pseudorabies |
| 92 | Liver | Listeria monocytogenes | Multifocal suppurative hepatitis | Rare cause of abortion, Wild boars have been suggested as a reservoir of Y. pseudotuberculosis and L. monocytogenes in Japan **(Hayashidani et al., J Wildl Dis, 2002, 38: 202-205).** |
| 93 | Liver | Ascaris suum larval migrans | Chronic multifocal interstitial hepatitis | Syn.: milk spots **(Boes et al., J Parasitol, 2002, 88: 180-183)** |
| 94 | Liver | Ascaris suum larval migrans | Chronic multifocal interstitial hepatitis | Syn.: milk spots |
| 95 | Liver | S. dentatus larval migrans | Severe chronic interstitial hepatitis | Granulomatous hepatitis can be caused by infection with Schistosomas japonicum **(Iburg et al., J Comp Pathol, 2007, 136: 250-255).** |
| 96 | Word slide | Toxic hepatopathy | DDX: Xanthium sp. (Cocklebur), Gossypol, Hepatosis dietetica (Vit. E, Se def., Sulfur), Coal Tar, Aflatoxin (> 1200ppm), Fumonosin (>80ppm) | |
| 97 | Liver | Vitamin E/Selenium deficiency | Diffuse hepatic necrosis | Hepatosis dietetica |
| 98 | Liver c/s | Vitamin E/Selenium deficiency | Diffuse hepatic necrosis and hemorrhage | Hepatosis dietetica |
| 99 | Liver | Xanthium sp. toxicosis (Cocklebur) | Diffuse hepatic necrosis and hemorrhage | Dicotyledon stage is toxic |
| 100 | Liver c/s | Xanthium sp. toxicosis (Cocklebur) | Diffuse hepatic necrosis and hemorrhage | |
| 101 | Liver | Aflatoxicosis | Hepatic lipidosis, cholestasis | Atrophy? Low doses of aflatoxin depress growth and alter many aspects of humoral and cellular immunity in pigs **(Marin et al., J Anim Sci, 2002, 80: 1250-1257).** |
| 102 | Liver | Aflatoxicosis | Hepatic lipidosis, cholestasis | |
| 103 | Liver | Aflatoxicosis | Hepatic lipidosis, cholestasis | |
| 104 | Liver | PCV2 | Diffuse hepatic necrosis/atrophy, fibrosis and hemorrhage (cirrhosis) | Secondary to myocardial necrosis, chronic passive congestion |
| 105 | Liver c/s | PCV2 | Diffuse hepatic necrosis/atrophy, fibrosis and hemorrhage (cirrhosis) | Secondary to myocardial necrosis, chronic passive congestion |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| **Hepatitis E virus**: Hepatitis E virus causes mild to moderate hepatitis in experimentally inoculated pigs (Halbur et al., J Clin Microbiol, 39: 918-923, 2001), a recent review: (**Smith et al., J Food Protect, 64: 4, 572-586, 2001**). Evidence that hepatitis E virus is zoonotic is accumulating: severe hepatitis in humans. (**Wang et al., J Med Virol, 2002, 67: 516-521**). The putative capsid protein of the newly identified avian hepatitis E virus shares antigenic epitopes with that of swine and human hepatitis E viruses and chicken big liver and spleen disease virus (**Haqshenas et al., J Gen Virol, 2002, 83: 2201-2209**). | | | | |
| **File 4** | | | | |
| **Primary pulmonary pathogens:**<br>• **Bacterial: M. hyopneumoniae, A. pleuropneumoniae, B. bronchiseptica, S. choleraesuis, A. suis**<br>• **Viral: PRV, SIV, PRCV, PRRSV, PCV2**<br>**Viral respiratory pathogens were reviewed and the role of proinflammatory cytokines as mediators of viral respiratory disease analysed (Reeth & Nauwynck, 2000, Vet Res 31: 187-213)**<br>**Secondary pulmonary pathogens:**<br>• **Bacterial: P. multocida, S. suis, H. parasuis, A. pyogenes, others ..** | | | | |
| 1 | **RESPIRATORY SYSTEM** | | | |
| 2 | **Atrophic Rhinitis:** cytotoxin produced by toxigenic strains of primarily capsular serotype D (rarely serotype A) of Pasteurella multocida is absorbed and causes bony hyperplasia in the nasal turinates and physes of long bones by inhibiting osteoblasts, inhibiting chondrocyte proliferation and (most likely indirectly) stimulating osteoclasts **(Ackermann et al., 1996, AJVR 57: 848-851; Gwaltney et al., 1997, Vet Pathol 34:430-430).** | | | |
| 3 | Head | Atrophic rhinitis | Atrophic rhinitis | P. multocida, toxigenic strains; poor growth **(Ackermann et al., 1996, AJVR 57: 848-851)** |
| 4 | Nasal cavity | Atrophic rhinitis | Nasal turbinate atrophy, nasal septal deviation | P. multocida toxin inhibits osteoblasts and reduces chondrocyte replication, but increases numbers of osteoclasts and activates macrophages **(Gwaltney et al., 1997, Vet Pathol 34: 421-430)** |
| 5 | Nasal cavity | Atrophic rhinitis | Nasal turbinate atrophy, nasal septal deviation | |
| **Inclusion body rhinitis (Porcine Cytomegalovirus):** Cytomegalic viral inclusions in tubular glands of the nasal mucosa, usually clinically silent, but produces mild rhinitis in in pigs < 3 weeks of age, experimental inoculation produced systemic disease characterized by edema (interstitial pneumonia) and disseminated petechia. | | | | |
| **Cilia-associated respiratory bacillis:** silver staining to recognize in trachea, no gross lesions, experimental inoculation produced no lesions (**Nietfeld et al., JVDI 7: 338-342**) | | | | |
| 6 | Lung | Normal | Pliable, pink and collapsed | |
| 7 | **DDX: Interstitial pneumonia in swine:**<br><br>**Viral**: Pseudorabies (PRV)<br>Swine Influenza (SIV)<br>Porcine Respiratory Coronavirus (PRCV)<br>PRRS virus (PRRSV)<br>Porcine circovirus type 2 (PCV2)<br><br>**Septicemic**: S. choleraesuis<br>H. parasuis<br>S. suis<br>other<br><br>**Allergic:** Ascarid larval migration<br><br>Recent isolates of paramyxovirus from pigs with interstitial pneumonia, necrotizing bronchiolitis and encephalitis **(Janke et al., J Vet Diagn Invest, 13: 428-433, 2001)**. | | | |
| 8 | Lung | Salmonella cholerasuis | Hemorrhagic interstitial pneumonia | |
| 9 | Lung | Hemophilus parasuis | Hemorrhagic interstitial pneumonia | May be very mild without hemorrhage |
| 10 | **Swine influenza:** Swine influenza virus isolates have been shown to share genetic similarities to human influenza viruses (**Lim et al., Korean J Vet Publ Health, 2002, 26: 135-142**). The swine influenzavirus isolate H1 serotype has similar genetic characteristics to human influenzavirus based on the HA gene and antibodies against this serotype have been found more commonly in swine farmers. Swine farmers may represent an important sentinel population to evaluate the emergence of new pandemic influenza viruses (**Olsen et al., Emerg Infect Dis,. 2002, 8: 814-819**). There is seroepidemiological evidence that avian H4, H5, and H9 influenza A virus have been transmitted to pigs in southeastern China (**Ninomiya et al., Vet Microbiol,. 2002, 88: 107-114**). SIV has been detected mainly in the bronchial and bronchiolar epithelial cells by in-situ and IHC and a less intense signal was detected in the interstitial and alveolar macrophages (**Jung et al., Vet Pathol, 2002, 39: 10-16**). | | | | |
| 11 | Trachea | Swine influenza (Orthomyxovirus) | Submucosal hematoma, trachea | 2 subtypes H1N1 (more common in the US) and H3N2 **Choi et al., 2002, Arch Virol 147: 1209-1220 (**H1N2 in Korea: **Jung, JVDI 17: 176-178, 2005)** |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 12 | Lung | Swine influenza (Orthomyxovirus) | Diffuse intersitial pneumonia with lobular atalectasis | Necrotizing bronchiolitis "checkerboard pattern" **(Jung et al., Vet Path, 39: 10-16, 2002; Gramer , 2005, J Swine Health Prod 13: 157-160, Jung & Chae, 2006, Vet Pathol 43:161-167)** |
| 13 | Lung | Swine influenza and P. multocida | Diffuse interstitial pneumonia, broncho-pneumonia | Whereas SIV can be potentiated by concurrent infection with PRRSV and possibly PCV2, Mycoplasma hyopneumonia does not potentiate disease **(Thacker J Clin Microbiol, 39: 7, 2525-2530, 2001**) |
| 14 | Lung | Swine influenza and P. multocida | Diffuse interstitial pneumonia with multifocal hemorrhage, broncho-pneumonia | |
| 15 | **Swine influenza in humans:** Pigs are important role in inter-species transmission, because they have receptors to both avian and human influenza virus strains: "mixing vessel". Outbreaks and sporadic human infection with swine influenza have been occasionally reported. Influenza outbreak caused by swine H1N1 virus in Fort Dix, New Jersey in 1974 and an outbreak in Wisconsin in 1988 resulted in multiple human to human infections. People in contact with swine have higher antibody levels. Swine influenza viruses have been isolated from turkeys, indicating transmission between pigs and avian species. Pigs can be infected with the highly pathogenic avian influenza (HPAI) H5N1 virus. | | | |
| 16 | Lung | PRRS (Arterivirus) | Severe diffuse interstitial pneumonia with lobular atalectasis | |
| 17 | Lung | Pseudorabies | Severe diffuse interstitial pneumonia with hemorrhages | DDX: Salmonella cholerasuis |
| 18 | Lung | Ascaris suum larval migrans | Diffuse interstitial pneumonia with multifocal hemorrhage | |
| 19 | Lung | Ascaris suum larval migrans | Diffuse interstitial pneumonia with multifocal hemorrhage | |
| 20 | Lung | Actinobacillus suis | Multifocal embolic fibrinonecrotic pneumonia | |
| **Proliferative and necrotizing pneumonia** (**PNP; Can Vet J 35(8): 513-515, 1994**) has been associated with PRRSV, SIV, and PCV-2. Recent studies demonstrated that PRRSV was the only virus consistently and predominantly associated with PNP and should be considered the key etiologic agent for the condition (**Drolet et al., Vet Pathol, 40: 143-148, 2003, Morandi et al., J Comp Pathol, 2010, 142: 74-78**). | | | | |
| 21 | Lung | Tuberculosis, Mycobacterium avium | Multifocal granulomatous pneumonia | |
| 22 | **Mycoplasma hyopneumoniae:** The growth rates of pigs infected with M. hyopneumoniae were significantly reduced **(Czaja et al., Vet Rec, 2002, 150: 9-11).** Pathogenic M. hyopneumoniae activates receptors that are coupled to Gi or Go, which in turn activates a phospholipase C pathway, thereby releasing Ca2+ from the endoplasmic reticulum. Thus, an increase in Ca2+ may serve as a signal for the pathogenesis of M. hyopneumoniae **(Park Seung Chun et al., Infect Immun, 2002, 70: 2502-2506)**. The immune response around airways was recently characterized **(Sarradell et al., Vet Pathol, 40: 395-404, 2003; Choi et al., 2006 J Comp Pathol. 134: 44-50).** | | | |
| 23 | Lung | Mycoplasma hyopneumoniae | Catarrhal bronchointerstitial pneumonia | Mucus predominates in exudates within airways in mycoplasmosis. **Fano et al., 2005, Can J Vet Res 69: 223-228.** |
| 24 | Lung; c.s. | Mycoplasma hyopneumoniae | Mucopurulent bronchopneumonia "Enzootic pneumonia" | **Desrosiers et al.,** J Swine Health Prod, 9: 233-237, 2001 |
| 25 | Word slide | **Secondary Inhaled Pathogens:** P. multocida, M. hyorhinis, S. suis, A. pyogenes, H. parasuis, B. bronchiseptica | | |
| 26 | Word slide | **Pasteurella multocida:** Common in nearly all swine herds, non-toxigenic and toxigenic strains. The most common bacterial isolate from pneumonic lungs in slaughter swine. Most are capsular type A, most are serotypes 3 or 5 (of 16 total), toxin as a virulence factor?? Some strains cause pleuritis, abscessation. | | |
| 27 | Lung | Mycoplasma hyopneumoniae "Enzootic pneumonia" | Bronchopneumonia with localized fibrinous pleuritis | Second bacteria is "pleuritic" P. multocida suis, H. parasuis, A. pyogenes |
| 28 | Lung | "Primary" + "secondary" | Purulent bronchopneumonia | Mycoplasmosis with secondary bacteria: P. multocida, S. suis |
| 29 | Lung | Arcanobacterium pyogenes | Purulent bronchopneumonia with abscessation | DDX: Actinomyces hyovaginalis has been associated with disseminated necrotic lung lesions in slaughter pigs |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
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| | | | | **(Aalbæk et al., J Comp Pathol, 2003, 129: 70-77).** |
| 30 | Word slide | **Bordetella bronchiseptica:** Can be a primary or secondary inhaled pathogen. Colonizes and destroys cilia in upper respiratory tract, may colonize lung causing broncho-pneumonia (cranial and middle lobes). Acts as a primary pathogen during the first few weeks of life causing lobular necrohemorrhagic bronchopneumonia. Clinical signs include coughing, sneezing +/- epistaxis and mucopurulent nasal discharge, mild (reversible) turbinate atrophy (regressive atrophic rhinitis), chronic progressive bronchopneumonia and death | | |
| 31 | Lung | Bordetella bronchiseptica | Necrohemorrhagic bronchopneumonia | Bordetella bronchiseptica produces several purported virulence factors, including the dermonecrotic toxin, which is necessary to produce the lesions of turbinate atrophy and bronchopneumonia **(Brockmeier et al., Infect Immun, 2002, 70: 481-490)** |
| 32 | Lung | Bordetella bronchiseptica | Chronic necrotizing bronchopneumonia | Sometimes have localized fibrinous pleuritis |
| 33 | Word slide | **Actinobacillus pleuropneumoniae:** APP adheres to alveolar epithelial cells through proteins and carbohydrates. Multiple adhesins are involved **(Overbeke et al., Vet Microbiol, 2002, 88: 59-74).** Transmission between pigs is highly variable and isolation of APP from tonsils and the number of colonies isolated from the nasal swabs were correlated to the level of infectivity **(Velthuis et al., Epi Inf, 2002, 129: 203-214).** $NOS_2$ and TNF-alpha expression may play a role in the pathophysiology of pleuropneumonia **(Cho et al., Vet Pathol, 2002, 39: 27-32, Cho and Chae, Vet Pathol, 2003, 40: 276-282).** Expression of COX-2 has been shown in neutrophils in acute lesions and alveolar macrophages in chronic lesions **(Cho et Chae, Vet Pathol, 2003, 40: 25-31).** Exotoxins APX1 and APX2 are produced by highly pathogenic serovars **(Maldonaldo et al., JVDI, 2009, 21: 854-857).** Rare granulomatous hepatitis has been described in slaughter house pigs **(Ohba et al., J Comp Pathol, 2008, 139: 61-66),** DDX: Actinobacillus porcitonsillarum **(Ohba et al., J Comp Pathol, 2007, 137: 82-86),** | | |
| 34 | Head | Actinobacillus pleuropneumoniae | Epistaxis | |
| 35 | Thorax | Actinobacillus pleuropneumoniae | 1. Fibrinohemorrhagic pleuropneumonia<br>2. Fibrinous pleuritis | DDX for fibrinous pleuritis: H. parasuis, S. suis, M. hyorhinis |
| 36 | Lung | Actinobacillus pleuropneumoniae | Diffuse fibrinohemorrhagic pleuropneumonia | DDX for fibrinonecrotic pleuro-pneumonia: A. pleuropneumonia, A. suis, S. suis and S. choleraesuis, recnt case report: DDX: Morganella morganii **(Ono et al., Vet Path, 38: 336-339, 2001)** |
| 37 | Lung c/s | Actinobacillus pleuropneumoniae | Severe fibrinohemorrhagic bronchopneumonia | |
| 38 | Lung | Actinobacillus pleuropneumoniae | Severe fibrinohemorrhagic pleuropneumonia | Distribution tends to be dorsal (hilar) "red hepatization" |
| 39 | Lung, Heart | Actinobacillus pleuropneumoniae | 1. Fibrinohemorrhagic pleuropneumonia<br>2. Fibrinous peri- and epicarditis | Endotoxemia can cause sterile epicarditis, |
| 40 | Lung | Actinobacillus pleuropneumoniae | Unilateral fibrinohemorrhagic pleuropneumonia | Distribution may be unilateral; right lung is more commonly affected |
| 41 | Lung | Actinobacillus pleuropneumoniae | Multifocal pulmonary sequestra | May cavitate or form abscesses |
| 42 | Lung | Actinobacillus pleuropneumoniae | Chronic pulmonary sequestra | Cavitation |
| 43 | Lung | Streptococcus suis | Severe fibrinonecrotic bronchopneumonia with hemorrhage | **Reams et al., J Vet Diagn Inves, 7:406-408, 1995** |
| **Fumonosin intoxication:** Fumonosin inhibits sphingosine- and sphinganine-N-acyltransferase causing elevated levels of sphingosine and sphinganine in serum and tissues. Although there are ultrastructural changes in endothelial cells. **(Gumprecht et al., 1998, Env Tox Path 26:777-778)** Formalin-fixed lung and liver can be used to determine fumonisin B1–induced sphinganine and sphingosine alterations in swine **(Hsiao et al., JVDI, 2007, 19: 425-430)** | | | | |
| 44 | Thorax | Fumonosin toxicity Fusarium moniliforme | Hydrothorax, pulmonary edema | |
| 45 | Lung | Fumonosin toxicity Fusarium moniliforme | Severe pulmonary edema | DDX: Vit. E/Se deficiency; Vegetative valvular endocarditis $\Rightarrow$ heart failure |
| 46 | Lung | E. coli septicemia | Interstitial pneumonia with pulmonary edema | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 47 | Lung | Bacterial septicemia | Pulmonary arterial thrombosis with pulmonary edema | |
| 48 | Lung | Metastrongylus elongatus | Lobular emphysema, margins of diaphragmatic lobes | Lungworm |
| 49 | Lung | Metastrongylus elongatus | Catarrhal bronchitis, lobular atalectasis | Lungworm |
| 50 | Abdomen, Thorax | Congenital, acquired | Diaphragmatic hernia | Acquired: suspected cause: excess Vitamin E |
| 51 | Lung, Liver | Congenital | Pulmonary and hepatic melanosis | DDX: Metastatic melanoma; lung and liver, **Muller et al., J Anim Breed Gen, 8: 275-283, 2001;** Acorn ingestion can lead to aquired melanosis **(Lanteri et al., Vet Pathol, 2009, 46: 329-333)** |
| 53 | **CARDIOVASCULAR SYSTEM** | | | |
| 54 | Heart | Mulberry heart disease, Vit.E/Se deficiency | Multifocal myocardial hemorrhage and necrosis | Pigs range in age from 3 to 7 weeks, concentrations of Vit. E below 2 ppm are deficient, often selenium conc. are Within limits: **Pallares et al., JVDI, 2002, 14: 412-414** DDX: EMCV; fetuses neonates (PPV, PRRSV, PCV2 **Opriessnig et al., 2006 J Comp Pathol. 134: 105-110**) |
| 55 | Heart c/s | Mulberry heart, Vit.E/Se deficiency | Multifocal transmural myocardial hemorrhage | DDX: septicemia, Selenium intoxication: **Kim et al., J Anim Scien, 79: 942-948, 2001** |
| **Encephalomyocarditis Virus (EMCV):** viral myocarditis (multifocal necrosis and mineralization, not hemorrhage), EMCV also causes necrotizing pancreatitis and tonsilitis **(Papaioannou et al., J Comp Pathol, 2003, 129: 161-168).** | | | | |
| 56 | Heart | Gossypol toxicity | Hydropericardium and fibrinous epicarditis | DDX: Vit.E/Se deficiency, Bacterial epicarditis |
| 57 | Heart | S. suis | Fibrinous epicarditis and pericarditis; | DDX: Hemophilus parasuis, Myco-plasma hyorhinis, E. coli, (A. suis) |
| 58 | Heart | Erysipelothrix rhusiopathiae | Vegetative mural endocarditis, right ventricle | DDX: S. suis, A. suis, E. coli, A. pyogenes Sequelae: localized thrombosis, septic emboli and abscessation or infarction to lungs or myocardium, kidneys, etc. |
| 59 | Heart | Actinobacillus suis | Vegetative mural endocarditis, right ventricle | DDX: S. suis, E. rhusiopathiae, E. coli, A. pyogenes Sequelae: localized thrombosis, septic emboli and abscessation or infarction to lungs or myocardium, kidneys, etc. |
| 60 | Heart | S. suis | Fibrinous endocarditis | DDX: A. pyogenes |
| 61 | Heart | A. pyogenes | Thrombosis, right ventricle | Secondary to vegetative valvular endocarditis |
| 62 | Heart | A. pyogenes | Myocardial abscesses | |
| 63 | Heart | Vitamin D intoxication | Severe diffuse epicardial mineralization | Lameness, paralysis, death |
| **Taxus sp. intoxication:** Model and inefficiency of hypertonic sodium bicarbonate treatment **(Ruha et al., Acad Emerg Med, 2002, 9: 179-185)** | | | | |
| 64 | **INTEGUMENTARY SYSTEM** | | | |
| 65 | Snout, foot | Foot and Mouth disease (Aphtovirus) | Vesiculo-ulcerative nasal dermatitis, necrosis of the coronary band | DDX: Vesicular diseases: SVD (Entero) **Rodriguez 2002, Vir Res 85: 211-219**, VE (Calici), VS (Rhabdo) **Brown et al., J Swine Health Prod, 9: 239-242, 2001** |
| 66 | Snout | Foot and Mouth disease (Aphtovirus) | Chronic vesiculo-ulcerative nasal dermatitis | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 67 | Foot | Foot and Mouth disease (Aphtovirus) | Necro-ulcerative pododermatitis or necrosis and separation, coronary band | DDX: Can be a sequel to severe septicemia; Epithelial basal cells are early replication site **(Durand et al., J Comp Pathol, 2008, 139: 86-96)** |
| 68 | Foot | Foot and Mouth disease (Aphtovirus) | Necro-ulcerative pododermatitis or necrosis and separation, coronary band | Strains are not species specific **(Aggarwal et al., Vaccine. 2002, 20: 2508-2515)** A strong relation exists between dose of FMDV and length of incubation period **(Alexandersen et al., J Comp Pathol, 2003, 129: 268-282, Quan et al., 2004 Comp Pathol. 131: 294-307)** |
| 69 | Foot | Foot and Mouth disease (Aphtovirus) | Necro-ulcerative pododermatitis or necrosis and separation, coronary band | |
| 70 | Teat | Foot and Mouth disease (Aphtovirus) | Necro-ulcerative thelitis | |
| 71 | Snout | Vesicular stomatitis | Vesiculo-ulcerative nasal dermatitis | DDX: Vesicular diseases: FMD (Picorna), VE (Calici), SVD (Entero) |
| 72 | Foot | Vesicular stomatitis | Vesiculo-ulcerative pododermatitis, coronary band | DDX: Can be a sequel to severe septicemia |
| 73 | Nose | Fusobacterium necrophorum | Necrotizing cellulitis, nose | Syn.: Bull nose |
| 74 | Head | Exudative epidermitis, Staphylococcus hyicus | Focal exudative facial dermatitis | Syn.: Greasy Pig Disease<br>“milk or needle” teeth not removed |
| 75 | Body | Exudative epidermitis, Staphylococcus hyicus | Diffuse exudative dermatitis | |
| 76 | Snout | Exudative epidermitis, Staphylococcus hyicus | Exudative nasal dermatitis | |
| 77 | Foot | Exudative epidermitis, Staphylococcus hyicus | Exudative pododermatitis, coronary band | Secondary to wounds at the coronary band from poor flooring material |
| 78 | Skin | Erysipelas, Eryselothrix rhusiopathiae | Multifocal cutaneous infarction | DDX: A. suis |
| 79 | Skin | PDNS | Multifocal hemorrhagic cutaneous infarcts | May present as poorly defined macules, clearly defined macules or infarcts. |
| 80 | Skin | PDNS | Multifocal hemorrhagic cutaneous infarcts | |
| 81 | Pinna | Ear biting, fighting or vice | Multifocal cutaneous lacerations, pinna | |
| 82 | Pinna | Ear biting or vasculitis | Cutaneous ischemic necrosis, infarcts, dry gangrene; pinna | Ear necrosis, bacterial septicemia: S. cholerasuis, E. rhusiopathiae |
| 83 | Pinna | Frostbite | Acute cutaneous infaction, bilateral, pinna, abrupt demarcation from normal skin | Ear notches on the tip of the right ear are for identification and were present prior to the lesion |
| 84 | Pinna | Frostbite | Acute necrosis, skin and subcutis. | |
| 85 | Tail | Tail biting, vice | Necrosis, tail | |
| 86 | Navel | Bacterial wound contamination | Necrotizing omphalitis | Leads to septicemia or polyarthritis: A. pyogenes, Strep. sp. |
| 87 | Umbilicus | Arcanobacterium pyogenes | Umbilical abscess or abscessing omphalophlebitis | |
| 88 | Skin | Borellia suis | Spirochetal granuloma; Chronic cutaneous ulcer | |
| 89 | Skin | Dermatophytosis (Ringworm) | Hyperkeratotic dermatitis and focal alopecia | M. parvum or T. verrucosum most common, zoonotic **(Pittman et al., 2005, J Swine Health Prod 13: 86-90)** |
| 90 | Skin | Arthropod bites | Multifocal cutaneous hemorrhagic macules or multifocal necrohemorrhagic dermatitis | Infestation of underfloor manure storage pits with mosquitoes or other flying arthropods |
| 91 | Ear | Mange (Scabies) | Hyperkeratotic dermatitis, pinna | Sarcoptes scabei var. suis, **(Firkins et al., Vet Parasit 99: 323-330, 2001; Handa et al., J Jap Vet Med Assoc, 2002, 55: 498-500)** |
| 92 | Skin | Hematopinus suis (lice) | Pediculosis | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 93 | Skin | Hematopinus suis, Swine pox | Pediculosis (nits) and multifocal proliferative dermatitis | |
| **Swine pox:** Due to changes in swine production systems and availability of parenteral ectoparasiticides, lice and thus swine pox are/is rare. However, infrequently congenital swine pox occur sporadically in some lice-free herds affecting few pigs in few litters with a high case fatality rate by 10-days-of age **(Thibault et al., 1998, Swine Health and Production, 6: 276-278)**. This suggests that swine pox virus may be endemic in some swine herds. | | | | |
| 94 | Whole body | Swine pox | Multifocal proliferative dermatitis (pox) | Distribution follows lice habitat |
| 95 | Head | Swine pox | Multifocal proliferative dermatitis with central necrosis (pox) | |
| 96 | Skin | Swine pox | Multifocal proliferative and pustular dermatitis (dermal nodules, pustules) | Nodules are darker on pigmented skin |
| 97 | Pinna | Swine pox | Multifocal proliferative and ulcerative dermatitis (pox) | May appear mostly ulceratve |
| 98 | Whole body | Zinc deficiency; Parakeratosis | Severe diffuse hyperkeratotic dermatitis | DDX: Chronic solar dermatitis (sunburn), zinc toxicity causes pancreatic necrosis **(Gabrielson et al., 1996, Vet path 33: 692-696)** |
| **File 5** | | | | |
| 1 | **NERVOUS AND MUSCULOSKELETAL SYSTEMS** | | | |
| **DDX: Frequent causes of CNS disease in swine:**<br><br>**Suckling Pigs:**<br>Hypoglycemia<br>Streptococcal meningitis<br>Pseudorabies<br><br>**Weanling pigs:**<br>Streptococcal meningitis<br>Edema disease<br>Pseudorabies<br>Water deprivation<br>**(Brito et al., Vet Hum Tox, 43: 2, 88-90, 2001)**<br><br>**Grower/Finisher & Adult**<br>Streptococcal meningitis<br>H. parasuis meningitis<br>S. choleraesuis meningitis<br>Pseudorabies | | | | |
| 2 | Word slide | Viral encephalitis | **DDX:** Pseudorabies **(Narita et al., J Comp Pathol, 2004, 130: 277-284)**, Teschovirus (**Yamada et al., J Comp Pathol, 2009, 141: 223-229**), HEV (coronavirus, **Hirano et al., J Comp Pathol, 2004, 130: 58-65**), EEE virus, Rabies, Para-myxovirus (Blue eye[FAD] **(Hernández-Jáuregui et al., J Comp Pathol, 2004, 130: 1-6)**, recent US isolates of paramyxoviruses: **Janke et al., J Vet Diagn Invest, 13: 428-433, 2001**).Hog Cholera [FAD], PRRS virus, EMC virus, Cytomegalovirus, Nipah virus [FAD] **(Kurup, Infect Dis Clin Pract, 2002, 11: 52-57)**, Japanese B encephlitis [FAD] **(Yamada et al., Vet Pathol 41: 62-67, 2004)** | |
| 3 | Brain | Bacterial meningitis | Purulent meningitis | DDX: S. suis, H. parasuis, E. coli, S. choleraesuis, A. suis, M. hyorhinis |
| 4 | Cerebellum | Bacterial meningitis | Purulent meningitis | |
| 5 | Pons | Bacterial meningitis | Purulent meningitis, abscessed left trapezoid body (origin of VII, VIII) | Exudate gravitates to ventral brain stem; infection MAY extend via C.N. VIII from otitis media/interna. |
| 6 | Brain | Bacterial meningitis | 1. Multifocal cerebral abscesses<br>2. Cerebral cortical atrophy | |
| 7 | Middle ear | Otitis media | Purulent otitis media | DDX: A. pyogenes, P. multocida, M. hyorhinis **(Morita et al., AJVR 59:869-873, 1998)** M. hyorhinis eustachitis **(Morita et al., 1999, Vet Path 36: 174-178),** Otitis interna as a sequela to meningitis caused by S. suis **(Madsen et al., Vet Path, 38: 190-195, 200)** |
| 8 | Occipital cortex | Arcanobacterium pyogenes | Abcess, left occipital cortex | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 9 | Word slide | Edema disease | ETEEC - enterotoxemic E. coli: hemolytic colony types, somatic serotypes O139, O141 and O157, colonizes the S.I. via F18ab (F107) or K88 fimbria, secretes shiga-like toxin II variant (SLTIIvar) that induces characteristic systemic angiopathy. In the brain, lesions are most often in the brain stem. Hpth.: Vasculopathy; mural degeneration, mural and perivascular edema. **MacLeod et al., Vet Pathol, 28:66-73, 1991; Kausche et al., AJVR, 53:281-287, 1992.** One study identified production of verotoxin SLTIIvar and expression of F18 and K88 colonization factors only in isolates of the serogroups O139, O141, and O157, respectively **(Alexa et al., Vet Medicina, 2002, 47: 132-136).** The presence of the adhesin involved in diffuse adherence (AIDA) gene has been suggested as an important virulence factor **(Ha et al., JVDI, 15: 378-381, 2003).** | |
| 10 | Eyelid | Edema disease | Palpebral edema | SLTIIvar causes necrosis in endothelial cells and myocytes in experimentally infected pigs **(Matise et al., 2000, Vet Path 37: 318-327; MacLeod et al., 1991, Vet Path 28: 66-73, Kausche et al., 1992, AJVR 53:281-287)** |
| 11 | Stomach | Edema disease | Edema of gastric mucosa and mesentery | |
| 12 | Colon | Edema disease | Edema of mesocolon | |
| 13 | Brain | Harding's cerebrospinal angiopathy | Nodular arteritis with leptomeningitis | Middle cerebral artery |
| 14 | Brain | Harding's cerebrospinal angiopathy | Focal hemorrhage and malacia (infarct), basal ganglia | Lymphoproliferative vasculitis in multiple organs has recently been described with MCF (OHV-2) **(Alcaraz et al., JVDI, 2009, 21: 250-253)** |
| **Water deprivation / salt intoxication**, Hpth.: Cerebrum; edema, laminar necrosis, eosinophilic leptomeningitis **(Gelberg, Vet Pathol 2010, 47: 576-578)** | | | | |
| 15 | Word slide | **DDX: Posterior Paralysis or Paresis:** **Spinal cord**: enteroviral poliomyelitis (Hpth.: lymphoplasmacytic poliomyelitis), selenium intoxication (Hpth.: Bilateral poliomyelomalacia, ventral horns) – poliomyelomalacia (also: anorexia, alopecia, separation of hoof and skin at coronary band, degenerative changes in liver and kidney), fibrocartilagenous emboli and infarction secondary to disk rupture, contusion secondary to spinal fracture, lymphosarcoma (**(Haynes et al., 2000, J Vet Diagn Invest 11: 533-536, York et al., 2000, J Vet Diagn Invest 11: 352-357)** **Spinal column**: vertebral osteomyelitis or osteomalacia with secondary spinal fracture, fracture of lumbar spinal cord secondary to lightning **(Van Alstine and Widmer, JVDI, 15: 289-291, 2003)** **Bones and Muscles**: Ischial epiphysiolysis, rupture of the "hamstring", fractures +/- osteiomyelitis (Staph. aureus, **Jensen et al., J Comp Pathol, 2009, 141: 269**) or osteomalacia, arthritis **Peripheral nerves**: organic arsenical intoxication, sciatic damage from injections A vestibulocerebellar disorder has been described in pigs after consumption of broken rice contaminated with Aeschynomene indica seeds **(Riet-Correa et al., Vet Pathol, 40: 311-316, 2003).** | | |
| 16 | Thoracic spine | Arcanobacterium pyogenes | Paravertebral/vertebral abscess, thoracic spine | TNF-a and IL-10 are involved in the late reperatory phase of experimental disk lesions **(Holm et al. Vet Pathol 2009, 46:1293-1300)** |
| 17 | Spine | Arcanobacterium pyogenes | Purulent discospondylitis, ankylosing spondylosis | |
| 18 | Spine | Arcanobacterium pyogenes | Discospondylitis, spondylosis, pathologic fracture, hemorrhage | |
| 19 | Intervert. Disc | Degenerative disc disease | Necrosis of annulus fibrosus Rupture of nucleus pulposis | Can lead to fibrocart. emboli - ischemic necrosis of spinal cord |
| 20 | Sitting pig | Sciatic nerve damage | Posterior hemiparesis, necrotic pododermatitis | |
| 21 | Sciatic nerve | Sciatic nerve damage | Abscess and necrosis surrounding the sciatic nerve | Injection site trauma/infection |
| 22 | Skeleton | Osteochondrosis | Apophysiolysis tuberis ischidici | Dyschondroplasia of the ischial physis |
| 23 | Ischium | Osteochondrosis | Epiphysiolysis capitis femoris, muscular hemorrhage | Between 6-18 months of age, discrepancy between weight gain and sceletal maturation |
| 24 | Muscle | Trauma | Muscular necrosis and hemorrhage | |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| **Taenia solium:** The metacestodes of Taenia solium are the cause of cysticercosis in the skeletal muscle of pigs. They induce an inflammatory reaction in naturally infected pigs that may vary from a minimal inflammatory infiltrate consisting of eosinophils and a few mononuclear cells to severe granulomatous reaction associated with the destruction of the parasite. The organization of an active inflammatory response against the T. solium in pigs includes the sequential participation of CD4+, CD8+ and IgM+ lymphocytes. **(Perez-Torres et al., Parasitol Res, 2002, 88: 150-152).** | | | | |
| 25 | Femur | Lactogenic osteoporosis | Overriding midshaft femoral fracture | "Downer" sow syndrome, mobilization of calcium for milk production. Phosphorus-deficient diets induce bone loss **(Liesegang et al., J Anim Physiol Anim Nutr, 2002, 86: 1-16)** |
| 26 | Coxofem. joint | Arcanobacterium pyogenes | Abscessed coxofemoral joint | |
| 27 | **Lameness in Swine:**<br>**Bacterial arthritis**: S. suis, H. parasuis, , M. hyorhinis, S. equisimilis, E. rhusiopathiae, M. hyosynoviae, A. pyogenes, P. multocida capsular serotype A alone can cause not only pneumonia in pigs but also septicaemia or arthritis **(Ono et al., J Comp Pathol, 2003, 129: 251-258)**.<br>**Degenerative arthritis**: Osteochondritis dissecans (OCD), DDX.: chronic zinc intoxication **(Gabrielson et al., 1996, Vet Path 33: 692-696)**<br>**Injuries/Other**: Fractures, Ruptured cruciate ligaments, Overgrown hoofs, heel abscesses | | | |
| 28 | Foot, suckling pig | Abrasion, opportunistic bacteria | Pododermal abrasions, polyarthritis of the digits | DDX: A. pyogenes, E. coli, S. suis |
| 29 | Digit | Abrasion, opportunistic bacteria | phalangeal arthritis, abscess | DDX: A. pyogenes, E. coli, S. suis |
| 30 | Frontleg | Abrasion, opportunistic bacteria | Decubital ulcer | DDX: A. pyogenes, E. coli, S. suis |
| 31 | Coxofem. Joint | Mycoplasma hyosynoviae | Serofibrinous arthritis, coxofemoral joint | |
| 32 | Stifle joint | Erysipelas rhusiopathiae | Proliferative synovitis | |
| 33 | Carpal joint suckling pig | Streptococcus equisimilis | Chronic arthritis and periarthritis, carpo-phlangeal joint | DDX: A. pyogenes, E. coli, S. suis |
| 34 | Carpal joint weaned pig | Hemophilus parasuis | Fibrinous arthritis, carpal joint | DDX: necrotizing osteomyelitis and fibrinopurulent arthritis caused by APP **(Jensen et al., 1999, Vet Path 36: 258-261)** |
| 35 | Tibiotarsal joint | Arcanobacterium pyogenes | Physeal abscess | |
| 36 | Humeral head | Osteochondrosis | Epiphiseal subarticular osteonecrosis | Ischemic necrosis of the articular epiphyseal cartilage complex (AECC) |
| 37 | Humerus | Osteochondrosis | Osteochondritis dissecans (OCD), degenerative joint disease | Onset of lameness typically $\geq$ 4 months-of-age |
| 38 | Stifle joint | Trauma | Rupture of anterior cruciate ligaments, articular hemorrhage | |
| 39 | Hock joint | Trauma, recumbancy | Hygroma, plantar surface, hock | |
| 40 | Cervical region | Anthrax, B. anthracis | Cervical edema, ventral neck | 3 forms: pharyngeal, intestinal, septecemia, DDX: Clostridial infections or S. porcinus retropharyngeal lymphadentitis |
| 41 | Left rear leg | Clostridium septicum | Severe acute cellulitis, left rear leg | |
| 42 | Cervical region | Clostridium septicum | Gangrenous dermatitis, ventral neck | |
| 43 | Neck | Clostridium septicum | Gangrenous dermatitis and myositis | |
| 44 | Cervical mm. | Clostridium septicum | Severe diffuse necrotizing and emphysematous myositis | |
| 45 | Whole body | Porcine stress syndrome | Tetany | DDX: Tetanus |
| 46 | Epaxial | Porcine stress | Acute muscular necrosis, epaxial | "PSE" pork, |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| | mm.'s | syndrome | muscles | |
| **PSE:** The halothane gene has an effect on the quality characteristics of pork. Pigs can be of normal halothane genotype (HalNN), heterozygous genotype (HalNn) or homozygous recessive genotype (Halnn). The frequency of PSE carcasses is greater in the Halnn and in the HalNn pigs than in the HalNN. The halothane genotype does not affect muscle temperature and final pH. Related to pH and color, the quality of pork was inferior in the Halnn and the HalNn pigs than in the HalNN. **(Culau et al., Rev Brasil Zootec, 2002, 31: 954-961).** The role of carnitine in body composition and obesity has recently been reviewed. **(Carroll et al., Compend, 23: 45-52, 2001).** Also stress has an impact on sarcoplasmatic reticulum $Ca^{2+}$ transport in pig muscle **(Kuchenmeister et al., Meat Sci, 2002, 61: 375-380)** | | | | |
| 47 | Sceletal Muscle | Monensin intoxication | Acute muscular necrosis, lumbar muscles | Potentiated by Tiamulin **(Miskimins et al., 1996, J Vet Diagn Invest 8:396-397; Carpenter et al., 2005, J Swine Health Prod 13: 333-336.),** DDX: Vit. E/selenium deficiency |
| **Parasites of skeletal muscle:** Sarcocystis miescheriana: **Reiner et al., 2002, Vet Parasit 106: 99-113**, Distribution and density of cysticerci of Taenia solium: heart, tongue, internal and external masseters, triceps brachii, lungs, liver, kidneys, psoas, diaphragm and brain as well as the muscles from the forelimb, hind limb, abdomen, head and thorax, **Boa et al., 2002, Vet Parasit 106: 155-164;** Trichinella spiralis **(Kapel, Dansk Veterinaertidsskrift, 2002, 85: 11-15)** | | | | |
| 48 | **GENITAL SYSTEM** | | | |
| 49 | Mammary glands | Mammary edema | Severe diffuse mammary edema | DDX for bacterial mastitis: Klebsiella sp., E. coli, Citrobacter sp. (Mammary gland development in gilts. **Sorensen et al., Livest Prod Sci, 2002, 75: 143-148**). |
| 50 | Mammary glands | E. coli | Multifocal necrotizing mastitis | DDX for bacterial mastitis: Klebsiella sp., E. coli, Citrobacter sp. |
| 51 | Ovary | Active ovary | Regressing C L, multiple Graafian follicles | |
| 52 | Ovaries | Follicular cysts | Multiple follicular cysts | Small cyst: estrogen producing – nymphomania, large cysts: progesteron producing – inhibit estrous cyclicity |
| 53 | Word slide | Zeralenone | | |
| 54 | Vulva | Normal | | |
| 55 | Vulva | Estrogenic mycotoxins: Zeralenone (Fusarium roseum) | Vulvar swelling (edema) | Vulvovaginitis → vulvar and rectal prolapse; luteotropic → anestrus, pseudopregnancy; **Reischauer et al., 2005, Mycotoxin-Res 21: 143-146;** DDX: estrus |
| **Reproductive failure – failure of conception, early embryonic death, abortion:** Reproductive failure is a major cause of economic loss in the swine industry. Unfortunately, in cases of failure during breeding and early gestaion, embryos or very small fetuses are very rarely available for examination and have few lesions. Mid-gestational failure often results in mummification; again resulting in case submissions with few lesions. Fetuses aborted in the last third of gestation rarely have lesions, and lesions that are present are rather non-specific. The most recent edition of Diseases of Swine is probably the most recent and concise source of information. Each chapter focuses on a specific infectious agent or disease and includes sections on pathogenesis and diagnosis. There is much new information on the reproductive pathogenesis of PRRSV. Recent papers discussing uncommon or newly recognized diseases include: (**Thoma et al., 1997, Vet Pathol 34: 467-469:** Chlamydial abortion; **Stoffregen et al., JVDI, 2007, 19: 227-237:** Brucella abortus, **Ramirez-Mendoza et al., 1997, J Comp Path 117: 237-252:** Rubulaviral lesions in male reproductive tract; **Dubey et al., 1990, Vet Pathol 27: 411-418, Gelmetti et al., 1999, J Vet Diagn Invest 11: 87-90, Lunden et al., 2002, Scan J Infect Dis, 34: 362-365:** Toxoplasmal abortion; **West et al., 1999, J Vet Diagn Invest 11: 530-532:** PCV2 transplacental infection of fetuses and associated lesions; **Madson et al., Vet Pathol, 2009, 46: 707-716:** Intrauterine infection of naiv sows with PCV2 cause reproductive failure; **Opriessnig et al., 2006, J Swine Health Prod 14: 42-45:** PCV 2 in the testes and accessory sex glands. **Kazami et al., J Vet Med Sci, 2002, 64: 735-737:** Leptospira spp. causing premature births or stillbirths; **Hernández-Jáuregui et al., J Comp Pathol, 2004, 130: 1-6:** Rubulavirus causing Infertility in sows characterized by an increased number of returns to oestrus, stillbirths and mummified fetuses). Meconium staining of the skin has been associated with fetal hypoxia, stillbirths, weak-born piglets, neonatal mortality and aspiration leads to meconium aspiration syndrome **(Castro-Nájera et al., JVDI 2006, 18: 622-627).** | | | | |
| 56 | Uterus | Staphlococcus aureus | Fibrinohemorrhagic necrotizing metritis | **Can you tell this from intestine?** |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 57 | Uterus | Staphlococcus aureus | Fibrinohemorrhagic necrotizing metritis | |
| 58 | Uterus | Arcanobacterium pyogenes | Pyometra | Bladder helps to tell this is uterus |
| 59 | Uterus | E. coli | Fibrinohemorrhagic necrotizing metritis | |
| 60 | Placenta | Incidental finding | Placental mineralization | |
| 61 | Embryo Placenta | normal | Porcine embryo | Individual placentas |
| 62 | Uterus | Porcine parvovirus | Multiple intrauterine mummified fetuses | **Wilhelm et al., 2005, J Vet Med B 52: 323-326.** |
| 63 | Uterus | Porcine parvovirus | Multiple mummified fetuses | |
| 64 | Fetuses | PRRS virus | Fresh and partially mummified near-term fetuses | Note the characteristic but inconsistent umbilical lesions in the center fetus: necrotizing vasculitis causing umbilical edema and hemorrhage |
| 65 | Placenta | Bacterial placentitis | Acute ulcerative placentitis | DDX: multifocal necrotizing placentitis; Toxoplasma gondii **(Jauregui et al., J Clin Microbiol, 39: 2065-2071, 2001, Li et al., JVDI, 2010, 22: 442-444)** |
| 66 | Fetus, placenta | Mycobacterium bovis | Granulomatous placentitis | |
| 67 | Fetal lung | Nocardia asteroides | Diffuse bronchopneumonia | |
| 68 | Fetal skin | Mucor. sp. Dermal mucoromycosis | Multifocal necrotizing dermatitis | DDX: Streptomyces sp., Aspergillus |
| 69 | Fetus | Carbon monoxide intoxication | Carboxyhemoglobinemia | |
| 70 | Penis | Paraphimosis | Paraphimosis | |
| 71 | Penis | Fibrinonecrotic posthitis | Fibrinonecrotic posthitis | |
| 72 | Line drawing | b=preputial diverticulum | | |
| 73 | Whole body | Vice &/or mixed bacterial infections | Preputial diverticulitis | |
| 74 | Preput. divertic. | Mixed bacterial infections | Chronic necro-ulcerative preputial diverticulitis | |
| 75 | Bulbouret. gland | normal | | |
| 76 | Testicle | Trauma | Severe diffuse necrotizing orchitis | Brucella suis is rare, causes multifocal testicular abscesses or granulomas |
| **Clenbuterol:** Testicular damage can be caused by anabolic treatments with the beta2-adrenergic agonist clenbuterol (Blanco et al., Vet J, 2002, 163: 292-298). Clenbuterol treatment causes an increased volume fraction of the testicular interstitium especially in the Leydig cell population (Blanco et al., Vet Res, 2002, 33: 47-53). | | | | |
| 77 | **URINARY SYSTEM** | | | |
| 78 | Kidney | Salmonella cholerasuis | Renal cortical petechiae | DDX: Classical swine fever (Hog cholera), Erysipelas, PDNS, ASF |
| 79 | Kidney | Erysipelas | Renal cortical petechiae | DDX: S. choleraesuis, Classical swine fever (Hog cholera), ASF, PDNS |
| 80 | Kidney | Classical Swine Fever | Renal cortical petechiae | DDX: S. choleraesuis, African swine fever, Erysipelas, S. suis, PDNS |
| 81 | Kidney, lymph node | African Swine Fever | Renal cortical petechiae and lymph node necrosis | DDX: S. choleraesuis, Classical swine fever (Hog cholera), Erysipelas, PDNS |
| 82 | Kidney c/s | African Swine Fever | Renal cortical petechiae | DDX: S. choleraesuis, Classical swine fever (Hog cholera), Erysipelas, PDNS |
| 83 | Kidney, lymph node | PDNS | Renal cortical petechiae and lymphadenopathy | DDX: S. choleraesuis, African swine fever, Erysipelas, PDNS |
| 84 | Kidney | A. suis | Renal cortical petechiae | DDX: S. choleraesuis, Classical swine fever (Hog cholera), Erysipelas, S. suis, H. parasuis |

| No. | Tissue | Etiology/Disease | Gross Diagnosis | Notes |
|---|---|---|---|---|
| 85 | Kidney | A. suis | Multifocal embolic nephritis | DDX: H. parasuis |
| 86 | Kidney | Streptococcus suis | Multifocal renal abscesses (necrosis) | DDX: A. pyogenes, A. suis, E. coli |
| 87 | Kidney | Arcanobacterium pyogenes | Multifocal renal abscesses | Embolic nephritis |
| 88 | Kidney | E.coli | Multiple renal infarcts | |
| 89 | Kidney | E. coli | Multiple renal infarcts | |
| 90 | Whole body | Leptospira pomona | Icterus | Icterus and hemogloninuria in naturally occuring cases in pigs < 3 months of age, DDX: Salinomycin intoxication, Copper **(Plumlee et al., 1995, J Vet Diagn Invest 7: 419-420)** |
| 91 | Kidney | Leptospira pomona | Multifocal renal cortical petechia | In chronic cases lesions are confined to kidneys: interstitial nephritis. Review of leptospirosis in swine: **(Friis et al., Dansk Veterinaertidsskrift. 2002, 85: 6-11).** It was suggested that MHCII contributes to the intensity of inflammation in the kidneys **(Radaelli et al., Vet Pathol 2009, 46: 800-809)** review of pathogenesis of chronic kidney lesions **(Monahan et al. Vet Pathol 2009, 46: 792-799).** PPV and PCV1 and 2 were detected in kidneys with interstitial nephritis at an abattoir surveillance **(Drolet et al., Vet Rec, 2002, 150: 139-143).** |
| 92 | Urinary tract | Eubacterium suis | Bilateral pyoureter and pyelonephritis | DDX: E. coli, Klebsiellae sp. Streptococcal sp. |
| 93 | Kidney | Arcanobacterium pyogenes | Severe chronic pyelonephritis | |
| 94 | Kidney | Stephanurus dentatus, larval migrans | Diffuse interstitial nephritis | |
| 95 | Kidney | Vitamin D intoxication | Diffuse renal mineralization | |
| 96 | Kidney | Ochratoxin, Citrinin | Severe diffuse renal fibrosis | Ochratoxin – Aspergillus ochraceus **(Ballarini, 2002, Ob Doc Vet 23: 23-27, Stoev et al., Exp Tox Path, 52: 287-296, 2000),** Citrinin – Penicillium citrinin; DDX: Melamin intoxication **(Gonzalez et al. JVDI, 2009, 21: 558-563)** |
| 97 | Kidney | Ochratoxin, Citrinin | Severe diffuse renal fibrosis | |
| 98 | Kidney | Amaranthus sp. (pigweed) | Severe perirenal edema | |
| 99 | Kidney | Amaranthus sp. (pigweed) | Severe renal necrosis and hemorrhage | |

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