Locomotor Disease
What you will learn in this session:

- What conditions affect the locomotor system of pigs in Australia.
- How they impact on the health, welfare & performance of the pigs & the herd.
- How to diagnose them.
- How to treat them.
- How to minimise their impact on-farm.
Diagnostic methods

- History (records)
- Evaluation of pigs on farm (by group/location or by age)
- Walk through observing all pigs (buildings, rooms, pens)
- Evaluation of individual pigs (observe, hands-on)
- Consider environment (especially floors)
- Consider nutrition (quality control)
- Laboratory diagnosis (post-mortem)
  - Adequate numbers of pigs should be provided
  - Untreated, clinically affected pigs, (i.e. soon after onset of signs)
  - History and preliminary findings should also be provided
What conditions are we talking about?

- Splayleg in piglets
- Neonatal polyarthritis
- Superficial injury/trauma
- PSS
- Mycoplasma hyorhinus
- Vitamin E/Selenium deficiency
- Mycoplasma hyosynoviae arthritis
- Degenerative joint disease
- Rickets & osteomalacia
Splayleg

Q1. Is it a common problem?
Q2. What pre-disposes to this?
Q3. How do you treat affected pigs?
Neonatal polyarthritis

Q1. What are the major pathogens?
Q2. How do they infect the joints?
Q3. What can you do to prevent it?
Superficial injury/trauma

- May result in cellulitis and/or osteomyelitis in deeper tissues.
- Clinical signs: Lameness involving one or more limbs; unwilling to stand.
- “Bushfoot” a big problem among sows.
Deep cellulitis
“Bushfoot”
Superficial injury/trauma

- **Diagnosis**: Observe wounds on wall, sole or heel of hoof, swollen coronary band.

- **Prevention**: Examine, repair (sharp edges on galvanized woven wire, sharp slat edges, wide grooves in expanded metal, rough concrete, damp floors) and improve (epoxy paint, paper, carpet, rubber mats, straw) the floors.

- **Treatment**: Relies on topical and/or parenteral antibiotic - must be used early in pathogenesis of lesions. Also treat the animal with an anti-inflammatory.
Porcine stress syndrome (PSS)

• A “side-effect” of the “Halothane gene” - used in the past to increase lean yield.

• PSS is less of a problem in the Aust. Pig industry with pressure to select against the “Halothane” gene

• Lesions: Rapid development of rigor mortis. Pale, soft, "watery" musculature - due to denaturation of muscle protein.
Porcine Stress Syndrome (PSS)

Pathogenesis:
Stress $\rightarrow$ XS adrenaline $\rightarrow$ rapid muscle glycogenolysis, ATP breakdown and excess formation of muscle lactate $\rightarrow$ high metabolic rate $\rightarrow$ increased muscle temperature $\rightarrow$ death

Clinical signs:
- Muscle and tail tremor at outset
- Acute lameness and collapse
- Muscle rigidity
- Reddened skin
- Hyperthermia Temp. 107-113 degrees F.
- Shock $\rightarrow$ death
PSS

- **Diagnosis:** clinical signs + blood test for stress gene.
- **Prevention & Treatment:**
  - Rest; Reduce temperature rapidly; use hose with cold water; Tranquilizer.
  - Avoid mixing stress-susceptible animals
  - Market animals on dry, cool days and withdraw feed for 12-24 hours beforehand
  - **Genetic selection:** Tests incoming animals for stress gene
Mycoplasma hyorhinis

- Uncommon cause of polyarthritis in Australia.
- Transmitted from the oro-nasopharynx of the sow to piglet, and from carrier pigs to naïve younger pigs.
- Disease occurs after local damage to respiratory epithelium -> septicaemia -> polyserositis/polyarthritis.
- Clinical signs: fever (39C+), lameness and abdominal discomfort with polyserositis.
- Diagnosis: Necropsy - polyserositis, fibrinous exudate; Culture organism
- Treatment: Tylosin/Lincomycin/Tiamulin + anti-inflammatory
Vitamin E / Selenium deficiency

- Selenium and Vitamin E reduce levels of peroxide in tissues to prevent excessive lipid peroxidation of cell membranes, etc.
- Deficiencies: (1) absolute Se/Vit E deficiency, (2) Vitamin oxidation in rancid food and/or (3) rapidly growing pigs exceed average requirements in the diet and become deficient.
- Disease most common in weaner pigs.
- Mulberry heart disease (most common) and Hepatosis dietetica.
- Pigs with Mulberry heart disease die suddenly (usually “good pigs found dead”). This can occur quite commonly in pig herds in Australia.
MULBERRY HEART DISEASE
Vitamin E / Selenium deficiency

Diagnosis:
- History, clinical signs, and characteristic lesions
- Unexpected deaths in best pigs
- Heparinized blood for glutathione peroxidase
- Histology - coagulative necrosis of muscle fibers

Prevention:
- Se/Vitamin E in unthrifty animals - IM sodium selenite, 0.55 mg = 0.25 mg Se; Vit E, 50 mg = 68 I.U.
- Selenium supplement in diet - 0.3ppm Se (sodium selenite)
Mycoplasma hyosynoviae arthritis

- Found in the snouts from 60-70% of pigs at slaughter
- Disease most commonly seen in weaners & growers, but may affect gilts -> low to moderate morbidity and very low mortality.
- Transmission vertical (“Carrier” sows tonsil) or horizontal (at weaning).
- Clinical signs:
  - Painful, swollen joints, but usually afebrile
  - Acute or subacute lameness lasting up to 10 days in 10-20 week old animals
  - Excess synovial fluid may distend joint
**Mycoplasma hyosynoviae arthritis**

**Diagnosis:**
- Age of onset
- Afebrile and lacking polyserositis
- Lesions restricted to synovium at necropsy
- Culture organism (only likely for up to 3-4 days after onset)
- Response to treatment with antibiotics

**Treatment:**
- Tylosin / Lincomycin / Tiamulin
- + Corticosteroid / Flunixin IM
Degenerative Joint Disease

- Generalized condition with predilection sites, e.g. AECC of medial humeral and femoral condyles, and growth plates of costochondral junctions, distal parts of ulnae, and ischial tuberosities.
- Clinical signs occur from 4 months / > 100kg.
- Results in high cull rates (34-100%) in breeding pigs.
- Aetiology unknown-multifactorial:
  - genetics (muscular, heavy pigs)
  - nutrition- quantity vs. quality
  - environment / management.
Degenerative Joint Disease - pathogenesis

- OCD-dyschondroplasia-results in disturbed endochondral ossification in the physeal component of growth cartilages. Hypertrophied chondrocytes persist in the metaphysis.

- Osteochondrosis-a generalized condition-many sites may be affected and a single growth cartilage may have one or more focal lesions. Cartilage flaps, or areas with loss of cartilage result in exposure of subchondral bone to the joint cavity results in pain & lameness.
Epiphysiolyis-gilt hip
DJD

- **Clinical signs:**
  - Moderate/severe lameness in 1 or more legs - "kneeling" walk or on tip toes
  - Age of onset can be as early as 4 months. Onset may be insidious or sudden following trauma
  - Chronic, progressive lameness - probably when osteoarthrosis develops
  - Involvement of synovial joints of vertebrae -> kyphosis
DJD

- **Lesions:**
  - Radiolucency and sclerosis, Joint cartilage has folds and flaps, craters expose bone, Excess synovial fluid, Villous proliferation, Joint “mice” ossified, Osteophytes

- **Diagnosis:** Clinical signs; Post-mortem lesions

- **Prevention:** None since cause unknown; Good floor surfaces - bedding or dirt lots

- **Treatment:** Rest & anti-inflammatory
Rickets and Osteomalacia

- Rare today on commercial farms due to well-formulated diets. May be due to absolute Ca, P deficiencies, imbalanced ratio of Ca:P, feed mixing issues and/or cost cutting.

- Clinical signs:
  - Rickets: Anorexia and unthriftiness, stunted growth by 10 weeks, pigs unwilling or unable to stand-will sit, enlarged joints, skull may seem disproportionately large, bowed, truncated limbs, possibly fractured long bones, ribs or vertebrae (paresis or paralysis likely)
  - Osteomalacia: lameness or inability to stand late in pregnancy, during lactation or at weaning - seen when sows are in crates or at weaning
Rickets and Osteomalacia

- Lesions:
  - Rachitic lesions: Enlarged costochondral junctions & thickened, hemorrhagic growth cartilages at ends of long bones (seen on slabs of bone), pliable ribs - some cut with a knife, bones poorly mineralized, fractures and/or calluses
  - Osteomalacic lesions: Classically, poorly mineralized bone (Osteoporosis is a decreased amount of bone), fractures frequently seen - femoral neck & shaft, humerus or vertebrae, secondary osteomyelitis at fracture site
Rickets and Osteomalacia

- **Prevention:**
  - Provide adequate diet - Ca, P and Vit D in adequate amounts and suitable proportions during the grower/finisher phase for adequate skeletal development and during lactation.
  - Monitor home-mixed rations - difficult if batches are mixed frequently, but the problem may develop only if there is repeated error in mixing.
  - ? Adequate exercise

- **Treatment:** Provide diet as above; Parenteral Vit. D
CASE STUDY

- Commercial breeder site-producing weaners for an off-site weaner facility
- Received gilts at 24 weeks of age from a multiplier unit
- Gilts were housed in a straw-bedded shelter from 22-26 weeks, then moved inside to a concrete-based shed.
CASE STUDY

- 15 lame gilts in the past 6 weeks. 11 of the 15 failed to recover after isolation on solid flooring & were destroyed.
- Gilts do not appear to be lame on arrival. Lameness occurs after days-to-weeks post-arrival in ecoshelters.
QUESTIONS

- Q1. What are your DDX?
- Q2. What will you do to confirm the diagnosis?
- Q3. What will you do to treat affected pigs?
- Q3. What will you do to prevent further occurrences?
ANSWERS

Q1. DDx

- Trauma-deep, soggy bedding
- Injury and cellulitis
- DJD
- Infectious causes
  - Erysipelas
  - Mycoplasma hyosynoviae
  - Mycoplasma hyorhinis
Q2. How would you confirm the diagnosis?

- Do a farm visit & examine affected animals
- 5-10% of current gilt population affected with mainly hind-leg lameness, seeming to originate from the hip. 2 pigs appear to have problems originating in the back, with stiffness and spine curvature.
- Necropsy
- Focus on joints – take one back to lab for examination, culture, PCR (Mycoplasma)
Fibrous tissue-elbow
Elbow joint opened
Culture results

- 2 gilts euthanased
  - 1 hip-bilat femoral separation of head from shaft + floccular, serosanginous material in the joint
  - 1 elbow joint filled with pus extending out to form fibrous tissue & draining abscess around area

- Results: 3 of 5 swabs grew Actinomyces pyogenes
Q3. What will you do to treat affected pigs?

- Identify & isolate affected gilts. Treat all with anti-inflammatories (Dexapent or Flunixin) for 3 days.
- Treat with parenteral Pencillin
Q4. What will you do to prevent further occurrences?

- Change the bedding between batches of gilts
- Ensure there are no sharp edges etc they can damage themselves on
- Treat group with soluble Amoxycillin on arrival for 5 days @ 10mg/kg
What you should have learnt in this session:

- What locomotor conditions exist in pigs in Australia.
- How they impact on the health, welfare & performance of the pigs & the herd.
- How to diagnose them.
- How to treat them.
- How to minimise their impact on-farm.