ENTERIC DISEASES
What you will learn in this session:

- What enteric diseases 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.
## Common causes of scour in suckers

<table>
<thead>
<tr>
<th>Agent</th>
<th>Age of occurrence</th>
<th>sample</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>NHEC</td>
<td>2 hr- 5 days</td>
<td>Rectal swab</td>
<td>Mild to severe Scour, dehydration death</td>
</tr>
<tr>
<td>Coccidia</td>
<td>5-15 days</td>
<td>2g Faeces</td>
<td>Scour, death uncommon</td>
</tr>
<tr>
<td>HEC</td>
<td>+10 days through to postweaning period.</td>
<td>swab</td>
<td>Sudden death-endotoxic shock or scour + dehydration</td>
</tr>
</tbody>
</table>
Non-hemolytic E. coli
## Common NHEC serotypes

Serotyping of isolate is used to determine if it is a recognized pathogen

<table>
<thead>
<tr>
<th>Fimbrial (synonym) Pilus types</th>
<th>Associated O serogroups</th>
<th>Toxin Types</th>
</tr>
</thead>
<tbody>
<tr>
<td>F4 = K88</td>
<td>O8, O9, O20, O64, O101</td>
<td>LT_STa</td>
</tr>
<tr>
<td>F5 = K99</td>
<td>O8, O9, O64, O101</td>
<td>STa</td>
</tr>
<tr>
<td>F6 = 987P</td>
<td>O8, O9</td>
<td>STa</td>
</tr>
<tr>
<td>F41</td>
<td>O101</td>
<td>STa</td>
</tr>
</tbody>
</table>
Scouring litter
Poor environments predispose
Coccidiosis - *Isospora suis*

- scour from 5 – 15 days of age
- more commonly seen in piglets at 7-10 days of age
- ingest oocysts soon after birth from infected faeces
- minimum of 5-6 days after ingestion to produce intestinal lesions
- These lesions resolve 10 to 14 days after infection.
- villous atrophy in the distal one-third of the small intestine.
- malabsorption diarrhoea (steatorrhoea - fatty faeces)
Fat droplets
Villous destruction
Diagnosis

• The excretion of oocytes (mean of 5.4 days in 19 days) and faecal fat (mean of 4.0 days in 19 days) is intermittent and has an important effect on reaching an accurate diagnosis.

• Examination of 3 faecal samples from 5 typically affected litters

• Pooling of faeces from each litter would be sufficient and more cost effective.

• Prevention: treatment of pigs at 4 days of age with Baycox®
Haemolytic E.coli (HEC)

• Esp. serotype K88: O149.

• Excretes Labile toxin and Stable toxin type 1 & 2.

• “Piglet attachment antigen” ie PAA

• K88: O149. causes sudden death or scouring and death in pigs from about 10 days of age.

• Corresponds to a decline in the IgG levels in the milk of the sow at this stage of lactation.
Hemolytic E.coli

- Diagnosis-isolation from a rectal swab (or ileal swab from a dead pig).

- Preventing the disease is not easy.
  - It is highly resistant, some strains resistant to registered antibiotics.
Quick quizz

Q1. Is vaccinating the sow IM at farrowing going to help prevent disease due to hemolytic E coli?

Q2. Explain your answer.

Q3. What would be a better way to go?
## Uncommon causes of scour in suckers

<table>
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<tr>
<th>Agent</th>
<th>Age of occurrence</th>
<th>Sample</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rotavirus</td>
<td>+ 7 days into weaners</td>
<td>2g Faeces</td>
<td>Scouring, acidic faeces</td>
</tr>
<tr>
<td>Strongyloides ransomi</td>
<td>10 days to weaning</td>
<td>faeces</td>
<td>severe diarrhoea and dehydration</td>
</tr>
<tr>
<td>Clostridial perfringens type A</td>
<td>1- 21 days</td>
<td>2g Faeces</td>
<td>Non-responsive scour, affects growth rate</td>
</tr>
<tr>
<td>Clostridial perfringens type C</td>
<td>2 + days</td>
<td>2g Faeces, whole animal, small intestine</td>
<td>Severe haemorrhagic enteritis</td>
</tr>
<tr>
<td>Salmonella</td>
<td>5 days of age to weaning</td>
<td>Rectal swab, whole animal, caecum terminal ileum, mesenteric lymph node</td>
<td>Scour, poor doing, emaciation, +/- pneumonia</td>
</tr>
<tr>
<td>Cryptosporidum</td>
<td>5 days of age to weaning</td>
<td>2g Faeces, whole animal, small intestine</td>
<td>Scour, poor doing</td>
</tr>
</tbody>
</table>
Common causes of scouring post-weaning

<table>
<thead>
<tr>
<th>Agent</th>
<th>Age of occurrence</th>
<th>sample</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemolytic E.coli</td>
<td>0-14 days post weaning</td>
<td>Faecal swab or ileum swab</td>
<td>Sudden death, scour dehydration</td>
</tr>
</tbody>
</table>
# Common HEC serotypes

<table>
<thead>
<tr>
<th>Fimbriae</th>
<th>Associated O serogroups</th>
<th>Toxin Types</th>
</tr>
</thead>
<tbody>
<tr>
<td>F4 = K88</td>
<td>O147, O149, O157</td>
<td>LT_STb</td>
</tr>
<tr>
<td>F4 = K88 ***</td>
<td>O8, O149</td>
<td>Sta_STbLt</td>
</tr>
<tr>
<td>Will affect suckers and weaners</td>
<td>O138, O141, O149, O157</td>
<td>Sta_STb</td>
</tr>
</tbody>
</table>
Control & Prevention

* Individual treatment of affected pigs with an injectable antibiotic is needed.
* Group in-water medication is warranted to control the problem.
* Vaccination of sucker pigs orally (Autovac<sup>R</sup>) 1 week before weaning is highly effective.

- The K88:O149 organism as an oral vaccine itself can cause disease - unless sows have been orally vaccinated during gestation. Use a PAA negative strain.
# Less common causes of scours in weaners

<table>
<thead>
<tr>
<th>Agent</th>
<th>Age of occurrence</th>
<th>Sample</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPE (Lawsonia intracellularis)</td>
<td>6- 10 weeks</td>
<td>Faeces for PCR, whole animal, terminal ileum and caecum</td>
<td>Scour, poor doing, emaciation,</td>
</tr>
<tr>
<td>salmonella</td>
<td>0- 6 weeks post weaning</td>
<td>Swab, whole animal, distal ileum, caecum</td>
<td>Scour, poor doing, emaciation, +/- pneumonia</td>
</tr>
<tr>
<td>Rotavirus</td>
<td>0- 3 weeks post weaning</td>
<td>2g Faeces</td>
<td>Scouring, acidic faeces</td>
</tr>
<tr>
<td>Clostridium perfringens Type A</td>
<td>5-7 weeks of age</td>
<td>2g faeces</td>
<td>Scour to soft faeces</td>
</tr>
<tr>
<td>Swine dysentery</td>
<td>6- 10 weeks</td>
<td>Swab, whole animal, spiral colon</td>
<td>Scour, poor doing, emaciation, +/- pneumonia</td>
</tr>
<tr>
<td>Trichuriasis</td>
<td>6- 9 weeks of age</td>
<td>Faeces, whole animal, large intestine</td>
<td>Scour, poor doing, emaciation,</td>
</tr>
</tbody>
</table>
Rotavirus

- *Rotavirus is ubiquitous - 100% of herds thought to have virus.
- *Virus invades & destroys enterocytes of small intestine resulting in malabsorption diarrhoea
- *Infection much more common than clinical disease.
- *Synergistic with other enteropathogens eg. E.coli, coccidia
- *Diarrhoea seen after 5 + days of age or 5 + days post weaning
Rotavirus diagnosis

* Detection of viral particles in faeces using an ELISA
* Samples need to be taken early in onset of disease as virus disappears once enterocytes destroyed
* Lacteals of distal small intestine contain no chyle and associated mesenteric lymph nodes appear small and tan
* Small intestine appears thin walled, flaccid, dilated with large volumes of watery yellow or grey fluid.
** Large intestine fluid filled and contents acidic (c/f E.coli alkaline)
* Villous atrophy of distal SI can be seen with hand lens.
Scouring in 8-24 week-old pigs

- Swine Dysentery (SD)
- Proliferative enteritis
- Intestinal spirochaetosis
- Salmonella
- Internal parasites
Brachyspira spirochaetes

- Delicate, anaerobic, difficult to culture
- Do not survive dessication or exposure to air for long periods
- Can survive in moist faeces
- Host-dependent
What’s in a name?

- *Treponema* then *Serpula* then *Serpulina* now *Brachyspira*
- *hyodysenteriae* (pig dysentery)
- *pilosicoli* (hairy colon)
<table>
<thead>
<tr>
<th>Common name</th>
<th>Swine dysentery</th>
<th>Intestinal spirochaetosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Causal agents</td>
<td><em>Brachyspira hyodysenteriae</em></td>
<td><em>Brachyspira pilosicoli</em></td>
</tr>
<tr>
<td>Species affected</td>
<td>Pigs, farmed rheas</td>
<td>Pigs, poultry, dogs, humans</td>
</tr>
<tr>
<td>Clinical signs</td>
<td>Severe colitis with mucus and blood</td>
<td>Mild colitis with mucus but never blood</td>
</tr>
<tr>
<td>Affect on production</td>
<td>High morbidity, some mortalities, decreased production, cost of medication ($&gt;100/sow)</td>
<td>Variable morbidity, no mortality, affect on production unknown</td>
</tr>
<tr>
<td>Ages affected</td>
<td>Any age but mainly growers and finishers</td>
<td>Post weaning to finishers, younger pigs tend to be more affected</td>
</tr>
<tr>
<td>Means of spread</td>
<td>Pig to pig, rats, mice, dogs, contaminated boots, effluent</td>
<td>Pig to pig, birds, dogs Other??</td>
</tr>
<tr>
<td>National herd prevalence</td>
<td>25%</td>
<td>Unknown</td>
</tr>
</tbody>
</table>
Faecal appearance
Swine Dysentery

- An infectious mucohemorrhagic colitis, characterized by wasting and diarrhoea.
- Faeces contains mucus, blood and necrotic material. Characteristic foul smell.
Clinical signs

- Mild fever, depressed, off feed
- Pigs fall back in condition
- Diarrhoea containing blood, mucus and necrotic material. Characteristic smell.
- Paper test- mucus sticks paper together
Brachyspira hyodysenteriae
42.4. *S. hyodysenteriae* in colonic crypt and epithelium...
Transmission

- Infection by oral route from sow to offspring.
- Then from clinically affected to pen mates
- Organism invades colonic crypts
  - Interferes with absorption of fluid and electrolytes
  - Death from dehydration and ion imbalance
Necropsy

- Confined to large intestine
- Reddened and edematous
- Colon most affected
- Lumen:
  - Brown fluid feces
  - Mucus
  - Necrotic material
  - Flecks of blood
Diagnosis

- Clinical signs (variable)
- Submit large intestine to Lab
- Spirochetes seen on dark field exam
  - Spirochaetes attached by one end to the mucosal surface (= pilosicoli)
- Histopathology
- Culture and biochemical tests to confirm species (can be really difficult unless lab is specialised)
- Polymerase Chain Reaction (PCR) on faeces
Differential diagnosis

- Ileitis or gastric ulcer
  - Not large intestine, tarry feces
- Whipworm.
  - Eggs, parasite,
- Salmonella.
  - No mucus or blood, culture, pneumonia?
Treatment

- Scouring pigs - injection for 3 days
- In contact pigs - water medicate
- Prevention - in feed medication
- Lincomycin, Tiamulin
Scours- treatment in general

- You must inject individual affected animals!
- Water medicate affected pens
- Don’t contaminate other pens
- Clean & disinfect where possible
Prevention

- Clean pens
- Don’t floor feed
- Minimise stress
- Strategic water medication
- In feed medication
Eradication

- Depopulation if respiratory disease
- Medication eradication
- High level for 7 days and then clean
- Medium level for 14 days during cleanup
- Low level for 56 days to allow bugs in environment to disappear
- Or DMZ for 6 months
Control rodents!
Proliferative enteropathy

- Collective name for different manifestation of same disease complex
- *Lawsonia intracellularis* invades crypt cells of the enterocytes
- Cells fail to mature and become hyperplastic
- Mucosa becomes thickened and invaded by other bugs
Etiology

- *Lawsonia intracellularis.*
  - Intracellular bacteria of intestinal epithelium (enterocytes)
  - Needs cell culture for grow at lab
Uncontrolled proliferation of enterocytes
Non-haemorrhagic PE
Porcine Intestinal Adenomatosis (PIA)
Necrotic enteritis
Hemorrhagic PE
Proliferative haemorrhagic enteroparphy (PHE)

- Sudden death
- Pale, anemic pigs or carcasses
- Black, tarry feces
Differential diagnosis

- Intestinal spirochaetosis
- Salmonella
- Swine Dysentery
- Whip worms
- Haemorrhagic gastric ulcer
Effect on production

- Diarrhoea usually mild
- Animals fall back - slab sided
- Poor weight gain
- PHE sudden death in older pigs or replacement breeders
- Can reduce ADG and FCE by 6-20%
- Causes large variation in batch of pigs which causes grid penalties.
Diagnosis of PE

- Clinical signs
- Necropsy & histo
- PCR on faeces
- IFA on sera
- Response to chlortetracycline
Treatment and prevention

- Antibiotics - Chlortetracycline, lincomycin, tylosin
- Inject + In-water
- Control strategy varies among farms & infection pressure - target exposure & immune development
- 1. Determine when shedding & infection is occurring.
- 2. Either allow non-medicated exposure for 10-18 days OR..
- 3. Medicate at low levels for 2-3 weeks
Salmonellosis

- Rare in Australia - usually due to *S. typhimurium* in rodents, birds, feed, by-products - infection by ingestion from feed, environment or dung.

- *S. cholerasuis* host adapted serotype not diagnosed in Australia for 30+ years

- Systemic invasion, localizes in lymph nodes, lung, intestine etc.

- Seen as outbreak of septicaemia, pneumonia, acute and chronic enteritis-scouring or death.
Clinical signs

- All ages can be affected, usually weaner/grower
- Sudden death with evidence of septicaemia
- Enteritis acute or chronic
  - Rarely blood in feces but necrotic material
- Chronic poor doers.
Necropsy

- Septicaemic form
  - Skin discoloration
  - Petechial and ecchymotic hemorrhages
  - Enlarged lymph nodes
- Acute enteritis.
  - Inflamed intestines fluid contents
- Chronic enteritis
  - Emaciated, thickened intestine, necrotic
  - Pneumonia 50% of cases
Diagnosis

- Always include it as DDx for scouring
- Non lactose fermentors on MacConkey
- Culture mesenteric lymph nodes *, liver spleen lung and intestines
- Culture on primary (take care with enrichment)
- Differential diagnosis
  - SD large intestine and often blood
  - Could be mistaken for necrotic enteritis
  - Pneumonia
Treatment and prevention

- Treat affected animals
- Water and feed medicate at risk group
- Vaccinate gilts?
- QA program - biological standards
  - Rodents
  - Cleaning
  - By products
  - Approved suppliers
Whipworm
Whipworm

- Uncommon - rule out Swine Dysentery or Ileitis
- Eggs require 3 weeks to infectivity
- Prepatent period 42 day
- Clinical signs
  - Anorexia
  - Mucoid to bloody scour
  - Dehydration maybe death
Necropsy and treatment

- Colon grossly enlarged
- Masses of worms evident
- In feed medication of sows and progeny
- Clean up environment- eggs live 6 years
- Pulse treat sows every 5 weeks
- Ivermectin not very effective
- FBZ, Hygromix?
Ascaris suum (pig roundworm)

- Very common, found in SI
- males 15-25 cm, females 25-40 cm
- eggs are thick shelled, sticky coated
- very resistant, but killed by heat (steam) and bright sunlight
Migration through liver (L3) to lung within 1wk after ingestion

Ingested eggs hatch in intestine to release stage 2 larvae

Stage 3 larvae are coughed up & swallowed, & are in SI 2wks after ingestion (L4)

Mature worms after 6-7 weeks post ingestion (min 40 days)

Eggs passed out in faeces

Stage 1-Stage 2 larvae develop & eggs become infective in 3-5 weeks
Pathogenesis

- **Caused by migrating larvae**
  - destruction of liver tissue, fibrosis
  - multiple focal pulmonary haemorrhages and inflammation

- **Caused by adults**
  - enteritis
  - migration causing blockages, perforations
Clinical signs

- Usually a condition of younger pigs
- Cough, dyspnoea, secondary pneumonia
- Afebrile diarrhoea
- Weight loss, illthrift, death
- Usually a herd problem
- Diagnosis by slaughter checks, PMs, faecal exam.
Worm Issues

- Long-term survival in environment
- Stickiness - readily transmitted by birds, insects, dirty boots etc----hard to eradicate
- Short “incubation” (1-2 weeks post-ingestion) before it migrates and potentially does damage
• The final two parasitic molts (L3 to L4 to L5 [the adult stage]) are completed in the small intestine (F) by 30 days after infection.
• The adult worm (L5) matures and begins to lay eggs approximately 6 to 8 weeks after infection.
• The minimum period between ingesting an egg containing an L2 stage and adult (L5) egg-laying worm being found in the pig is 42 days.
• This is called the Pre-Patent Period.
• If animals are treated with an effective wormer before 42 days after infection the life cycle of the worm is broken and no eggs are laid.
• Fenbendazole (FBZ) is 100% effective against all larval and adult stages of the worms of pigs. The recommended dose rate is 5mg/kg/week ie around 0.7 mg/kg/day
Balantidium coli

- Normally a commensal.
- Can invade 2’ to another pathogen.
- May cause anemia, enteritis and decreased growth rate.
- Fix primary cause.
Non- infectious GIT causes of death in pigs

- Oesophago-gastric ulcers
- Porcine intestinal distension syndrome
Progression of an ulcer in a stomach

- Normal
- Keratinised (worse)
- Scarred (non-patent) ulcer
- Haemorrhagic ulcer
- Ulcer (worse still)
Porcine intestinal distension syndrome (twisted bowels)
Note – caecum faces cranially
What you should have learnt in this session:

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- How to minimise their impact on-farm.