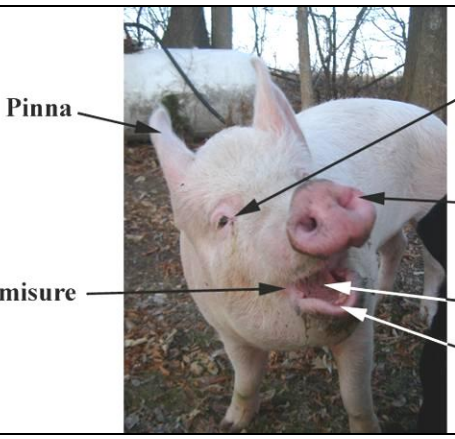
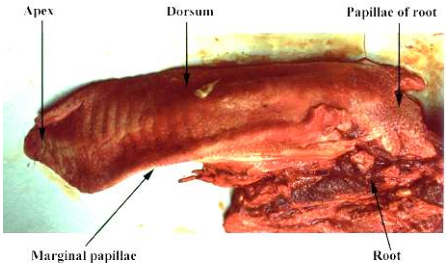
Disorders of the intestinal tract

Disorder present in	Australia	Europe/Asia	North America
Anatomy of the intestinal tract			
Abdominal catastrophe	Yes	Yes	Yes
Ascaris suum	Yes	Yes	Yes
Brachyspira colitis	Yes	Yes	Yes
Clostridium difficile	Yes	Yes	Yes
Clostridium perfringens	Yes	Yes	Yes
Coccidiosis of piglets	Yes	Yes	Yes
Non-specific colitis	Yes	Yes	Yes
Escherichia coli ï general	Yes	Yes	Yes
Bowel Oedema	Yes	Yes	Yes
Pre and post-weaning diarrhoea	Yes	Yes	Yes
Pre-weaning diarrhoea treatment			
Gastric ulceration	Yes	Yes	Yes
Porcine epidemic diarrhoea	No	Yes	No
Porcine proliferative enteropathy (Ileitis)	Yes	Yes	Yes
Pig parasites	Yes	Yes	Yes
Post-weaning ill-thrift syndrome	Yes	Yes	Yes
Rectal stricture	Yes	Yes	Yes
Rotovirus	Yes	Yes	Yes
Salmonellosis	Yes	Yes	Yes
Swine dysentery	Yes	Yes	Yes
TGE	No	Yes	Yes

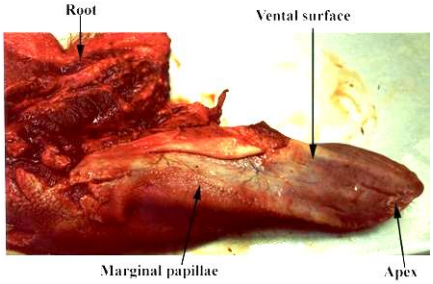
CLINICAL GROSS ANATOMY OF THE INTESTINAL TRACT



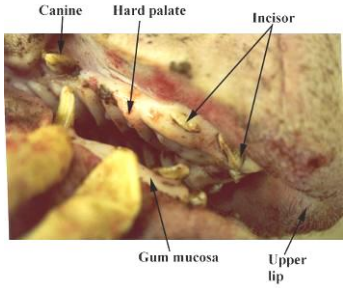
General view of the head of the pig



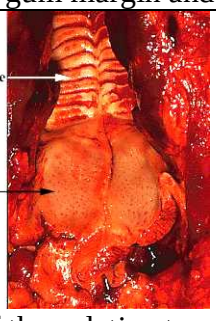
The tongue dorsal view



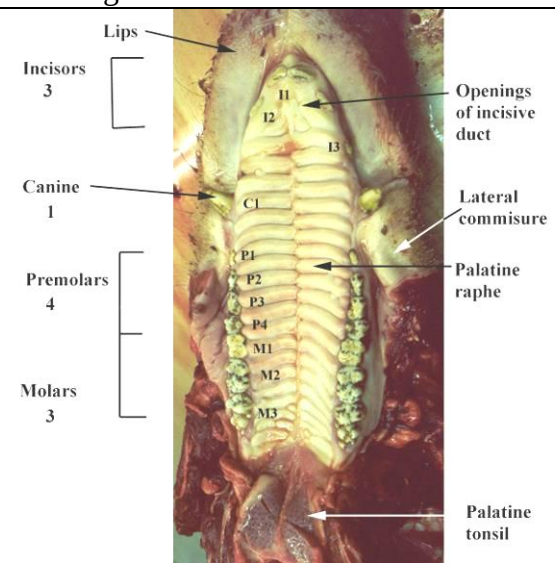
The tongue ventral view



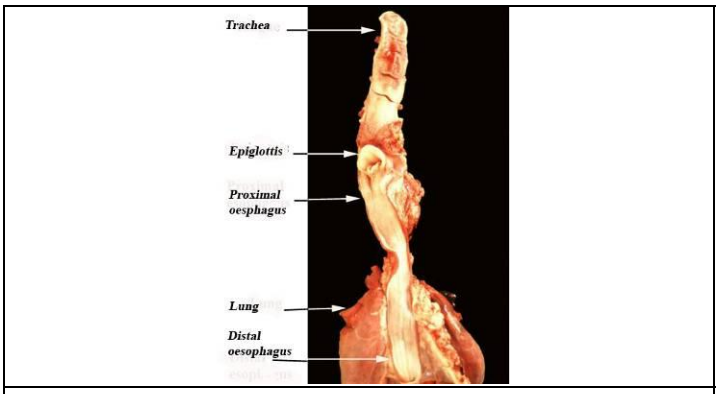
Detail of the gum margin and teeth



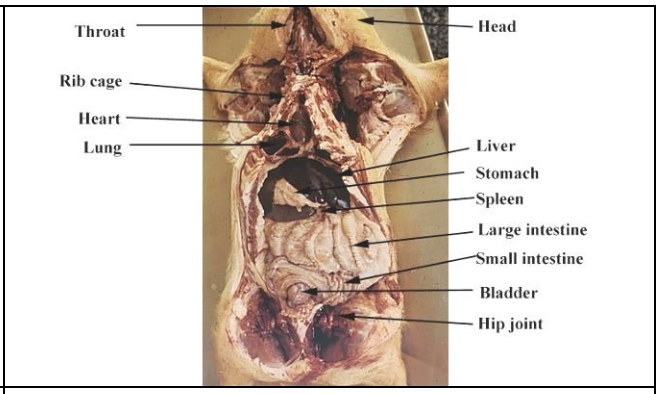
Detail of the palatine tonsil



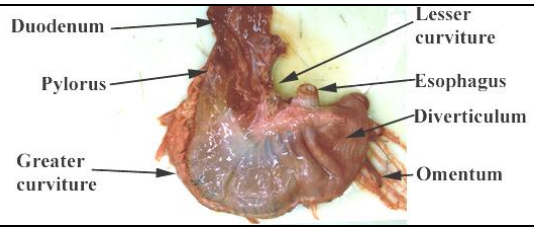
The hard and soft palate ï ventral view



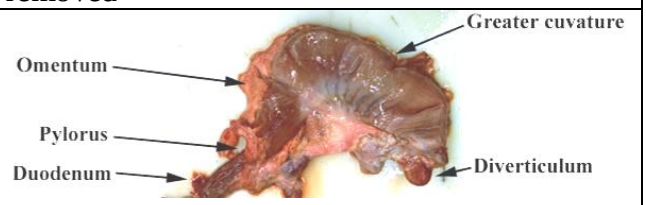
The oesophagus opened



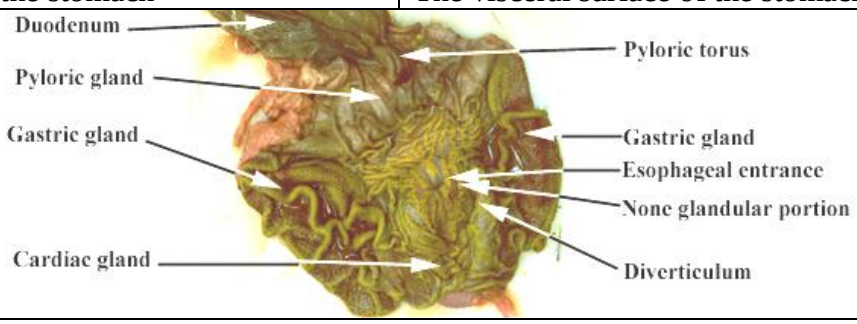
General view of the pig, ventral body wall removed



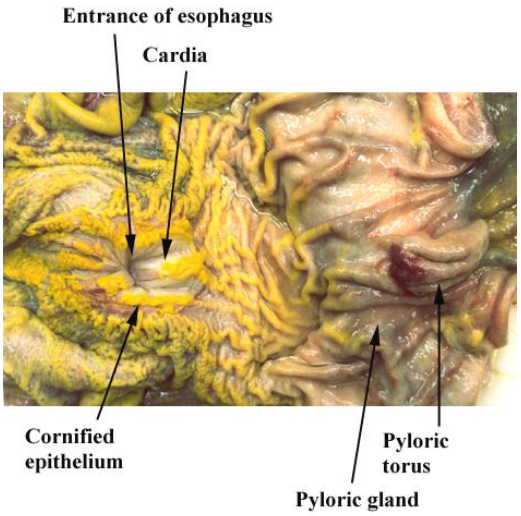
The parietal surface of the stomach



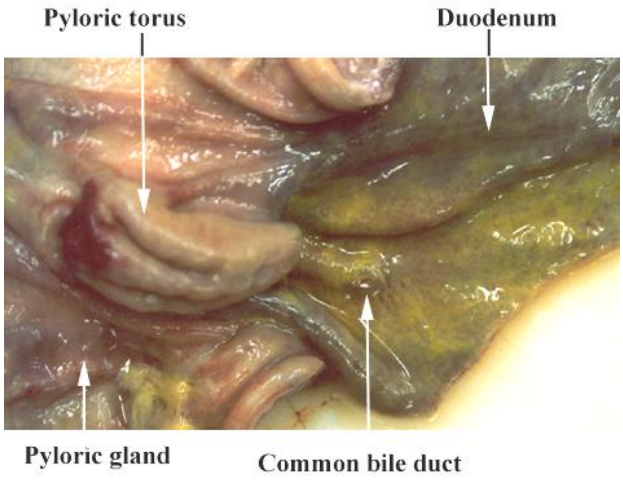
The visceral surface of the stomach



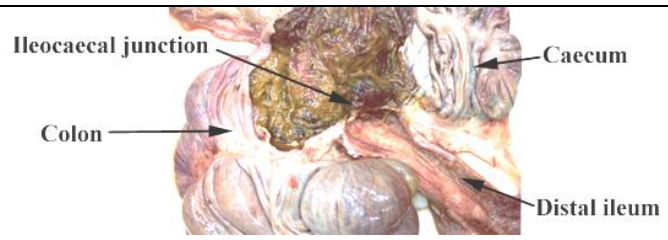
The mucosal surface of the stomach, the organ opened along the greater curvature



Detail of the oesophageal opening

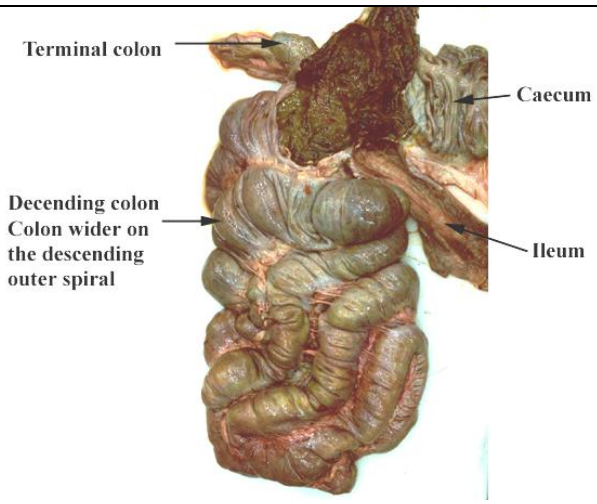


Detail of the pyloric sphincter opened

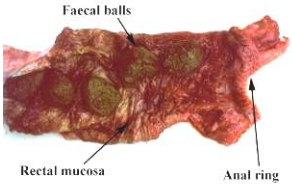


The ileocaecal junction

The ileocaecal ligament, useful in locating the ileocaecal junction

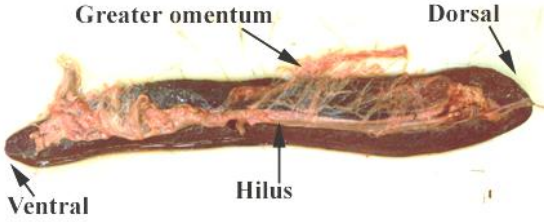


The spiral colon of a pig

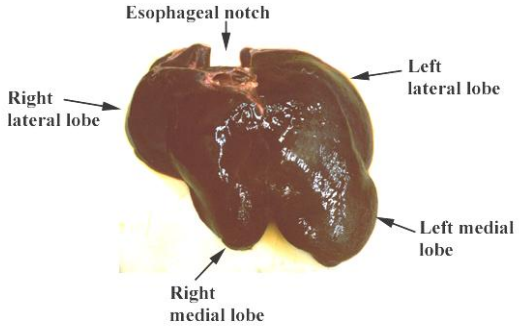


The rectum opened

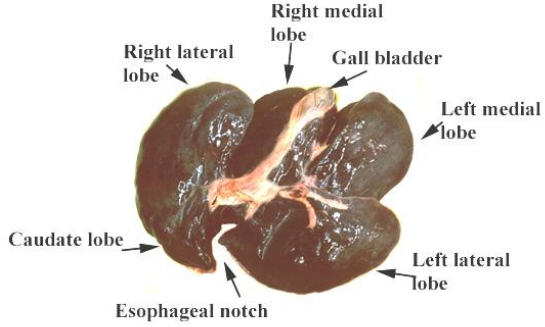
The spleen parietal surface



The spleen visceral surface



The liver parietal surface



The liver visceral surface

<table>
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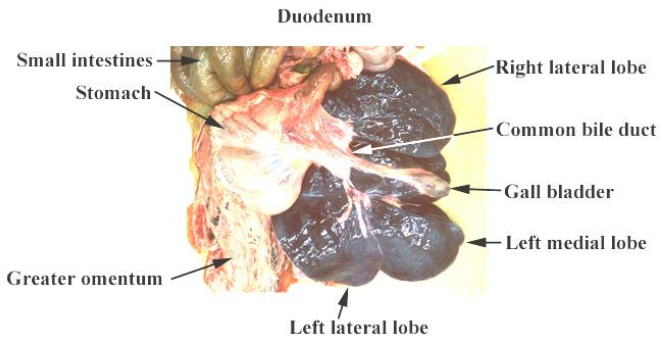
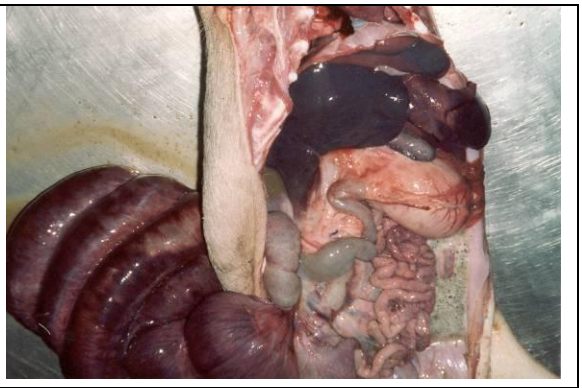
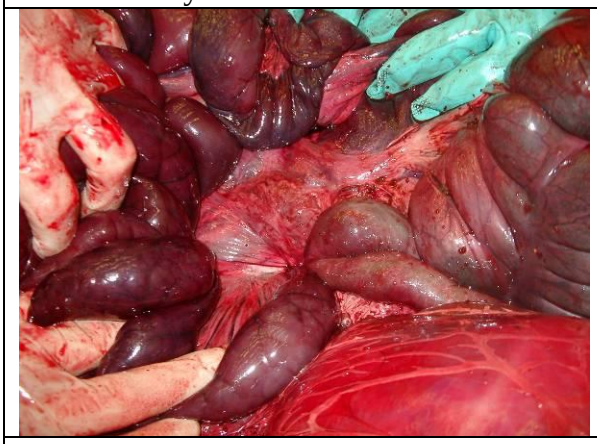
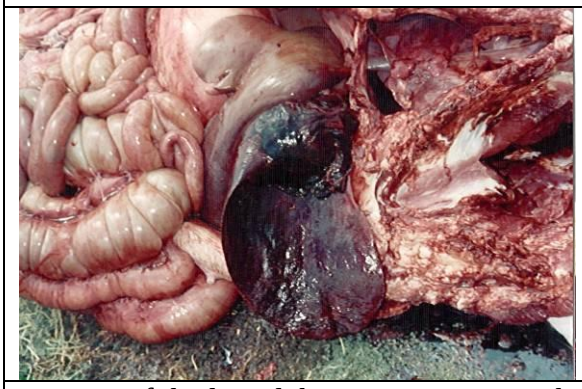
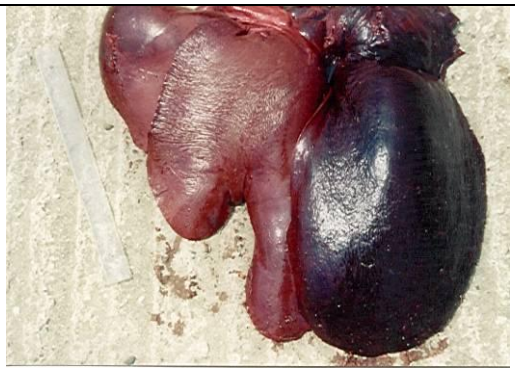
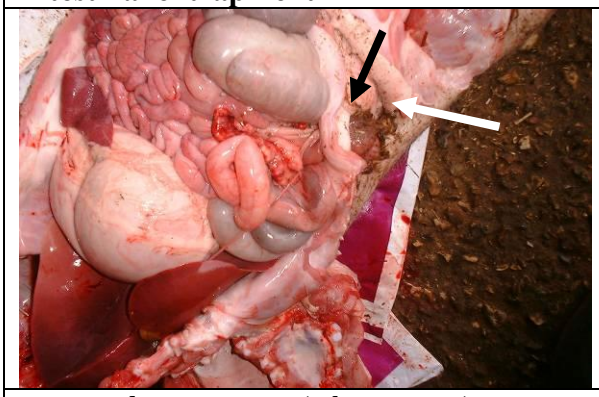
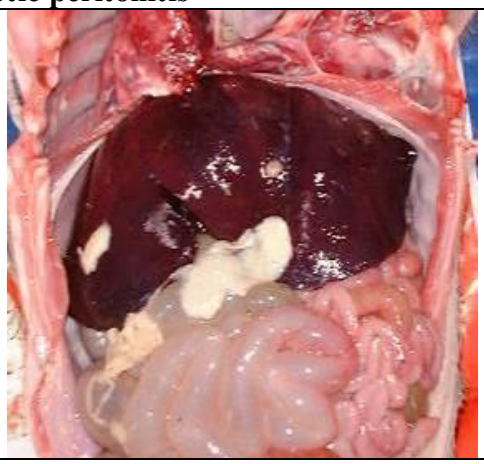
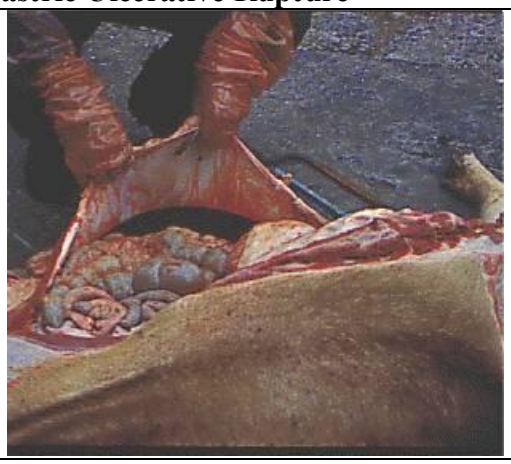
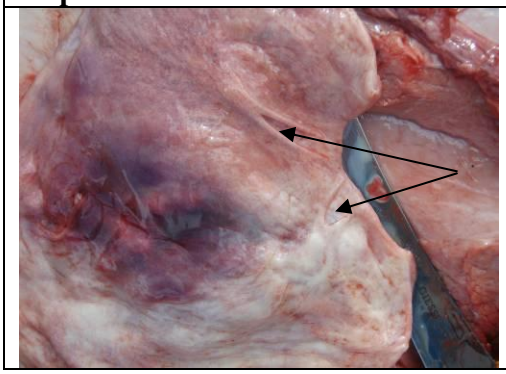
<td> 
</td>
<td>The liver, visceral surface showing the common bile duct.</td>
</tr>
<tr>
<td colspan="2">Faeces:
It is always important to note the consistency and colour of faecal pellets:</td>
</tr>
<tr>
<td>  </td>
<td>  </td>
<td>  </td>
</tr>
<tr>
<td>Constipated</td>
<td>Normal</td>
<td>Diarrhoea</td>
</tr>
</table>

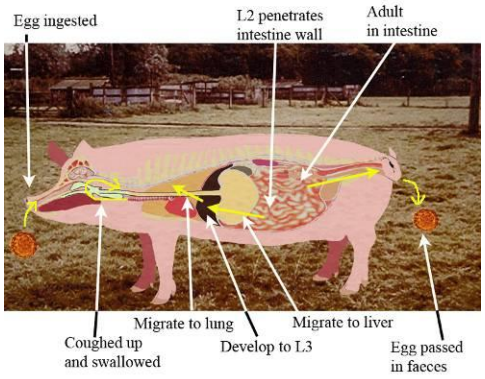
ABDOMINAL CATASTROPHE

A common cause of sudden death in adults is an abdominal catastrophe, characterized by a torsion/twist of the abdominal contents. The twist is commonly associated with the intestinal tract at the mesentery root or associated with a mesenteric tear. Occasionally, the spleen or a liver lobe can become associated with a twist.

<table>
<tr><td>  </td><td>  </td></tr>
<tr><td>General view of an abdominal twist. The abdomen may be more bloated</td><td>Gross post-mortem view of the pig on the left demonstrating the intestinal torsion</td></tr>
<tr><td>  </td><td>  </td></tr>
<tr><td>A twist at the mesenteric root</td><td>Twist/torsion of the spleen at the greater omentum</td></tr>
<tr><td>  </td><td>  </td></tr>
<tr><td colspan="2">Torsion of the liver lobe ï appearance in the abdomen and the liver laid out ï note the dark congested left lateral liver lobe</td></tr>
</table>

Intestinal entrapment	**Intestinal perforation**
	
Intestinal entrapment (white arrow) in a scrotal hernia (black arrow)	This is uncommon, but can result in sudden death associated with an acute peritonitis and release of abdominal contents into the peritoneum. Plastic hard brush bristles have been found in a few cases
Septic peritonitis	**Gastric Ulcerative Rupture**
	
Acute peritonitis associated with abscessation in the peritoneal cavity	Sudden death which revealed a large ruptured gastric ulcer releasing blood into the peritoneal cavity
Rupture of the urethra/ureter	
	This may occur during mating; the boar enters the urethra rather than the anterior vagina. Tearing of the urethra and ureterovesical junction occurs, with the sow dying due to haemorrhage and/or gangrene Torn ureterovesical junction arrowed

ASCARIS SUUM

Causal agent	*Ascaris suum* ï the large round worm of pigs The adult worm is large the female is 20-40 cm long and the male slightly shorter at 15-20 cm. The adult female may lay 2 million eggs per day. An adult can live 6 months		
Life cycle		Day Zero 2-3 3-7 8-10 10-15 21-30 10 13-18	Egg+L_2 stage ingested and swallowed L_3 hatch from egg in intestines L_3 penetrate intestine wall and migrate to liver L_3 develop in liver L_3 migrate from liver to lung L_3 leave lung, coughed up and swallowed L_3 develop to L4 in intestines Young adult develop Eggs are passed L_1 develop in 10 days L_2 develop in 13-18 days **Prepatent period 40-52 days**
Age group	Affects all age groups of pigs		
Clinical signs			
Normally	No clinical signs. May be some reduction in growth rates due to competition between pig and worm for food.		
Acute pneumonia	Ascaris, during the lung phase, may result in an asthmatic cough and the pig may have problems breathing. Ascaris will exacerbate other pneumonic conditions, especially swine influenza.		
Intestine	In young pigs severe infestation may results in intestinal blockage, rupture and death		
Infectivity			
	The eggs are very infective. They are extremely resistant and can survive for more than 7 years in the environment. Generally disinfectants have little effect on the eggs. However, steam cleaning and direct sunlight will kill the eggs.		
Transmission			
	The eggs are extremely sticky and will be easily transported onto the farm by pigs, insects, birds and equipment		
	Note workers boots are a significant source		

Post-mortem Lesions

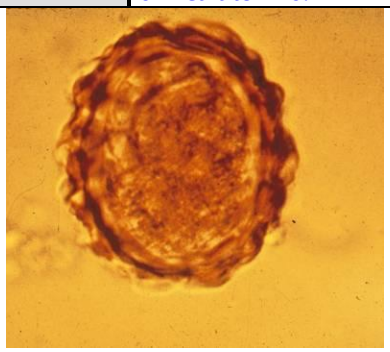
Liver	ñWhite spotò on the liver develop within days of infestation. However, they heal within 25 days
Lung	Small lesions may be seen in the lung, easier on histology
Intestine	The presence of the adult worms may be seen in the intestine lumen

ñWhite spotò in the liver ï severe case

Heavy worm infestation in the intestine ï seen at slaughter

Diagnosis

	White spot on liver in the slaughterhouse or at post-mortem
	Presence of adult worms post anthelmintic treatment
	Worm eggs seen during faecal examination using flotation technique. Note Ascaris eggs require $ZnSO_4$ flotation; normal salt solution will not work. In addition, they may still be difficult to find.



Egg in flotation

Adult worms seen after worming the pig

Treatment

Anthelmintics	Many anthelmintics work against Ascaris larvae and adults
	The problem with treatment is the rapid re-infestation and lesions heal within 25 days. Withdrawal times may preclude use prior to slaughter
	The adult worm in the gut does reduce the development of future larval migration

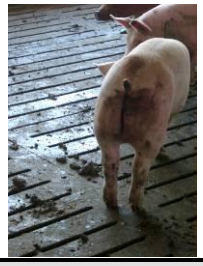
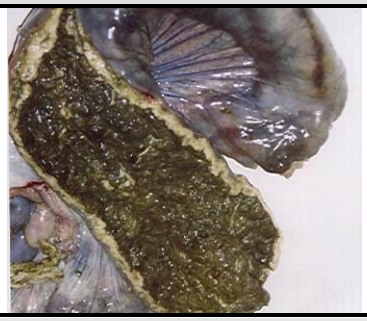
Common differentials

	Stephanurus dentatus (kidney worm) in the early stages may result in a ñmilk spotò liver. Later stages liver damage much more severe

Zoonotic

	No zoonotic implications. The human *Ascaris lumbricoides* worm is a separate species

BRACHYSPIRA COLITIS

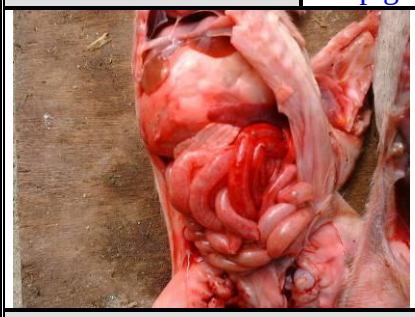
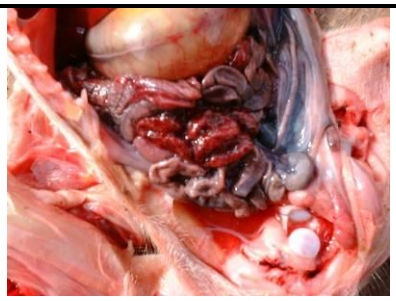
Other names	Porcine Colonic spirochaetosis, PCS. *Serpulina pilosicoli.*	
Causal agent	*Brachyspira pilosicoli* a bacteria	
Age group	Mainly affects 10-20 week old growers/finishers (30-90 kg)	
Clinical signs		
Naive herds 	A non-fatal wasting diarrhoea disease of growing pigs	
	Results in increased days to finish	
	Results in a reduction in feed efficiency	
	Produces watery/grey brown diarrhoea or loose stools	
	The clinical signs are more common 10-14 days after mixing and change of feed i.e. to the grower ration	
	50% of pigs may show transient to persistent watery to mucoid green to brownish diarrhoea without blood -resembles a cow pat.	
Infectivity		
	Pigs are infected by faecal-oral transmission	
	There are numerous other associated hosts:- dogs, mice, birds, guinea pigs, primates and probably also humans	
Incubation period		
	6 to 14 days	
Stress factors		
	Reduce stressors - transport, overcrowding, commingling, resorting, abrupt dietary changes, improper ventilation, wide fluctuation in temperature and inadequate feeders and water allocation	
Post-mortem Lesions		
		The colon and small intestine may demonstrate areas of inflammation, both acute and chronic. The spiral colon contains abundant watery green or yellow mucoid and frothy contents. Erosions in the colonic mucosa may be evident
Pathogenesis		
	The organism results in intestinal mucosal damage and inflammation resulting in enteritis/colitis reducing the surface area of the large intestine available, which reduces the absorptive capacity of the intestine reducing efficiency of feed utilisation. The large intestine is critical for absorption of fluids and nutrients therefore resulting in diarrhoea. Damage to the intestinal wall may also aid the disease and their toxins to gain access to the rest of the body resulting in systemic effects.	

Diagnosis	
	Bacteriological culture needed, however, samples need to be transported in a media such as Amies transport media
	PCR (DNA analysis) can identify the organism
	Histological analysis by silver stains
	Review the health records
Treatment	
	Aim to identify subclinical infected carriers
	Reduce environmental contamination
	Increase sanitation
	Antimicrobial therapy in both water and feed may be useful
	Reduce access to wildlife, birds and rodents for example in feed stores
	Reduce concurrent causes of enteritis/colitis
	Eliminate all draughts and chilling
	Move towards all-in/all-out
	Reduce scrape through passageways
Common differentials	
	Swine dysentery, Salmonellosis, TGE, PE (ileitis), Intestinal parasites - *Trichuris suis* or *Isospora suis* (whip worms or Coccidiosis)
Zoonotic	
	It is possible that the disease may be similar to human colonic inflammation and may therefore have a health significance

CLOSTRIDIUM DIFFICILE

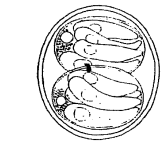
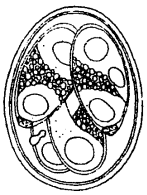
Causal agent	*Clostridium difficile*
Age group	Pre-weaning predominately before 10 days of age
Clinical signs	
Acute	Yellow pasty diarrhoea, unresponsive to many antibiotics
	May be mild abdominal distension, in the un-castrated male there might be scrotal oedema
Normal pig	No clinical signs
Infectivity	
	Normal contaminant of the environment
Post-mortem Lesions	
	Mesoclonic oedema and fluid intestinal contents
	Microscopic examination demonstrates acute multifocal, diffuse erosive colitis with large gram positive rods on the mucosa
	 4 day old piglets with a pasty diarrhoea  Post-mortem with liquid intestinal contents and moderate oedema of the colonic loops.
Diagnosis	
	Demonstration of organism
	Demonstration of the toxins
	Lack of other obvious causes
Treatment	
Treatment	Review antimicrobial treatment programmes. In many cases previous overuse of antimicrobials likely
	Supportive therapy, *Clostridium difficile* resistant to penicillin
Control	Initiate a feed-back programme of piglet faeces and intestines of affected piglets to sows 6 weeks pre-farrowing and gilts in acclimatisation
Common differentials	
	Causes of pre-weaning diarrhoea in piglets less than 10 days of age. Check ventilation system for chilling and draughts etc.
Zoonotic	
	Clostridium difficile may cause serious problems in children

CLOSTRIDIAL ENTERITIS

Causal agent	Bacterial. *Clostridial perfringens* Type C occasionally A or other types
Age group	Piglets less than 1 week (often within 3 days of birth) sudden death
	Piglets 2-3 weeks of age a chronic enteritis
Clinical signs	
Neonatal piglets	Sudden death. Anus often bright red. Other piglets very weak and pale
Older piglets	Diarrhoea, which may be intermittent. Piglets emaciated but can be active and alert. Eventually piglets die. Often seen in outside farming.
Post-mortem findings	
Neonatal piglets	Intestines full of blood
Older piglets	Chronic thickened enteritis, which make absorption of food very difficult for the piglet
 Acute haemorrhagic enteritis in a 3 day old piglet	 Chronic enteritis with a thickened bowel. Note the intestines have gas bubbles visible on their surface
Infectivity	
	Clostridial organisms are very common in the normal environment The clostridial spores are very resistant
Diagnosis	
	Post-mortem examination of affected piglets
	Identification of clostridial organisms in the intestinal tract
	Identification of clostridial toxins, which cause many of the clinical signs
Treatment	
Affected piglets	Oral or Injectable antibiotics with demonstrated efficacy against the clostridium to affected piglets and litter mates
Prevention	Vaccinate sows and gilts against clostridial organisms. Note commercial vaccines do not contain *Cl. perfingens* A, however, autogenous vaccines can be made
	All-in/all-out hygiene
	Effective farrowing house cleaning programmes
	Oral antibiotics to sows pre and post farrowing to reduce spread from the sow. Bacitracin may prove useful
Common differentials	
	Coccidiosis
	Salmonellosis
	Thrombocytopaenia and other neonatal blood disorders
	Trauma from the sow
Zoonotic	
	None

COCCIDIOSIS OF PIGLETS

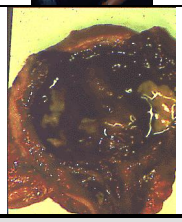
Causal agent	*Isospora suis*. This is a coccidia a parasite
Age group	Occurs between 8 and 15 days of age. Chronic cases can be seen post-weaning
Clinical signs	
Acute 	Diarrhoea tends to occur in individual pigs from approximately 6 days of age, but most of the litters have diarrhoea at 8-10 days. At the start of the diarrhoea, vomiting may be seen
	Diarrhoea ranges from white to pasty cream faeces through to a yellowy watery scour
	Piglets tend to be in poor condition, hairy and growing more slowly than other piglets within the litter
	Mortality rates reach 20% in acute cases
	The diarrhoea is not responsive to antibiotic therapy -excluding sulph medicines
	Piglets which survive often become unthrifty finishers when exposed to high pathogen concentrations, as they have little passive immunity and an immature gut
	Failure to recognise the disease leads to increased farrowing house mortality, poorer weaning weights and increased post-weaning problems
Sub-clinical	Most farms will have Coccidiosis at a low level affecting piglet performance
Infectivity	
	The coccidial egg, or oocyst infects young piglets by mouth, and relatively heavy infections are needed to cause disease. After ingestion, the organisms move down to the small intestines, where they invade the gut wall. In successive stages of a complex life cycle, they emerge from the wall at 5-9 days and again at 11-14 days after infection, and it is this emergence which causes diarrhoea.
Transmission	
	For clinically significant disease to occur, piglets need to be infected with relatively high numbers of oocysts
	Oocysts survive well in farrowing house environments. They are resistant to drying and most disinfectants
	Infection from the sow plays only a small part in disease development. Sows excrete a different coccidia called Eimeria
	Most piglets are infected by oocysts carried over from previous litters
	Once the piglet starts with diarrhoea, the intestinal wall is damaged, and treatment will not work effectively
	Failure to recognise the disease leads to increased farrowing house mortality, poorer weaning weights and increased post-weaning problems
Incubation period	
	The incubation period is 5 days

Post-mortem Lesions	
	There may be very few postmortem findings ï steatorrhea (increase of fat in faeces). Sometimes there is generalised enteritis. Scrapes from the intestinal wall may reveal the coccidial parasite. Histological examination are also used, however, note coccidia are also very common (normal?)
Diagnosis	
	Can be very difficult. Coccidial oocysts are only excreted in the faeces long after the clinical disease has passed
	Response to treatment with Toltrazuril or ponazuril (US not licensed for pigs). Dose 7mg/kg.
	  *Isospora suis* oocyst only has two merozoites within the egg  Eimeria (from the sow) has four merozoites within the egg, allowing easy differentiation
Treatment	
	Give electrolytes
	Give extra bedding. Stop creep feeding. Increase heat to piglets
	Treat with toltrazuril an oral preparation given at 4 and 10 days of age. Note this can make the pigs vomit. Sulpha-antibiotic medicines may also be used as treatment
Control	Prevent carryover of oocysts from previous litters. Clean the farrowing house with an occide disinfectant such as Oocide (Antec International)
	Control of rapid multiplication in infected piglets can be achieved by early and sometimes repeated treatment
	Reduce/cease cross-fostering after 2-3 days of age
	Use as a preventive measure toltrazuril oral doser. Pigs should be dosed at 4 and 10 days of age. Note some piglets vomit after being treated
	Sows should be washed/disinfected before entering the farrowing house
	Do not enter pens
	Use separate brushes, forks, shovels in each farrowing room
	Control flies
	Reduce draughts and other environmental stress factors
Common differentials	
	Other causes of preweaning diarrhoea. Stress induced diarrhoea
Zoonotic	
	None

NON SPECIFIC COLITIS

Causal agent	None identified. Diarrhoea of weaned pigs of any age from weaning to slaughter. Diarrhoea can occur within hours of consuming a new batch of pelleted feed and they can dramatically cease within hours of the removal of the suspect feed. Considered important factors are nutrition, infectious agents, and draughts
Age group	Weaning to slaughter pigs, most common between 25-30 kg pigs
Clinical signs	
	Softening of faeces with/without mucus and/or blood Diarrhoea can develop Decreased growth rate. Feed conversion increases Most common between 8-10 weeks of age More commonly seen in fast growing pigs on high density diets
Infectivity	
	Agent not recognised, but can appear to spread around and between farms The syndrome classically occurs with a particular feed and when this is changed the ódiseaseô disappears. Nutritional factors known to affect digestion are: Presence of trypsin inhibitors in peas, beans and soya Poor quality fat
Post-mortem Lesions	
	The colon and small intestine may demonstrate areas of inflammation both acute and chronic. The spiral colon contains abundant watery green or yellow mucoid and frothy contents. In some cases there may be no gross lesions. The photograph shows raised rugae in the inside of the large bowl but few other lesions
Diagnosis	
	Based on the clinical signs and absence of other specific organisms
Treatment	
	Improve the environment, remove draughts and ensure that the stocking rate is correct ï photo shows a growing shed being examined for draughts Do not place pigs into buildings which are damp and cold Check and clean the water supply Ensure the correct pig is placed in the building Change the feed to a meal Establish an all-in/all-out programme
Common differentials	
	Other causes of post-weaning diarrhoea

E. COLI GENERAL NOTES

Name	*Escherichia coli*	
Agent	Bacteria belonging to the Enterobacteriacae. Other members include salmonella and klebsiella	
Diseases	0-3 days of age associated with toxins	Sudden death, often with few clinical signs. Those that survive can be very stunted with a gaunt hair staring coat. Reddening of the skin of the perianal area and under-side of tail may be present.
	Pre-weaning diarrhoea 3 to 10 days of age	Loose to watery brown, white or cream coloured diarrhoea. Reduced gain and loss of weight, depression and loss of appetite, rough hair coat, sunken eyes, unthrifty appearance with ribs and backbone highly visible. One or two days of diarrhoea pre-weaning can add 5 days to finishing.
	Post-weaning diarrhoea 1 - 10 days post-weaning	Loose to watery brown, white or cream coloured diarrhoea. Reduced gain and loss of weight, depression and loss of appetite, rough hair coat, sunken eyes, red streaking or soreness in the anal region, unthrifty appearance with ribs and backbone highly visible. One or two days of diarrhoea post-weaning can add 5 days to finishing.
	Bowel oedema	Toxins from *E. coli* in the stomach and intestines in the post-weaning period result in oedema throughout the animal which presents with swollen eyelids and death and ill thrift. Some neurological signs can be seen. Presenting signs seen most commonly second week after weaning.
	Mastitis	Immediately after farrowing, reddening hard and hot mammary gland. Most *E. coli* mastitis is actually from toxins from the intestines rather than mammary gland. Toxin works against the hormone: Prolactin
	Cystitis and some involvement in pyelonephritis	Many sows have cystitis after farrowing or service associated with poor urination or hygiene. If the infection is complicated with other bacteria, such as *Actinobaculum suis* from the boar's prepuce, the kidneys can become infected as well (pyelonephritis).
The organism	The bacterium is the large oval in the centre. Surrounding the bacteria are 'hairs' called fimbriae which contain the adhesion factors which the bacteria use to stick to the host cells. Flagellae are long and used to move the bacteria. The example shown has no flagella.	

Classification	By cell wall	O antigens O147 for instance
	Fimbriae	F antigens (use to be K antigens) F1,F4 (K88), F5(K99), F18 (F107), F6 (987P), F41 and FP
	Flagellae	H antigens (not used to classify *E. coli)*
	Therefore the *E. coli* can be given a code which is useful to indicate its likely role in the disease. For instance: diarrhoea is often associated with O147, F4, F5 this is *E. coli* Abbotstown Bowel Oedema is associated with F18 fimbriae.	

	Recognising the code is important in the selection of the correct vaccine	
	Toxins	*E. coli* can produce a number of toxins
	Exotoxins	LT and ST toxins are actively produced by some strains. These are important in causing scour. Shigela-like toxin type II variant (SLT-IIe), Stx2e, verotoxin oedema disease principle act on the wall of the small arteries resulting in oedema. Enteroaggregative (EAST1) toxin.
	Endotoxins	From breakdown products from the cell wall which plays a role in mastitis as it acts against Prolactin (the hormone which releases milk) and in urinary tract diseases. Major cell wall toxin called Lipid A.
Diagnosis		
	Laboratory cultures. The collection of the sample is critical and requires a rectal swab from a recently infected (ideally untreated piglet) or if the problem is severe the submission of live recently ill pigs provides the best material.	
	Checking for and ruling out viral or parasitic causes	
	Examine intestinal pH, when at 8 or above suggestive of *E. coli*	
	Check herd history, past treatments, feed medication, incidence of scour	
	Determine age of affected pigs, disinfectants used, pig flow and environmental effects	
Treatment and control		
Hygiene	Adequate pressure washing. Utilise All-in/All-out. Sick animals are not put back through the system.	
Pre-weaning scour	Check for draughts and colostrum intake	
Post-weaning scour	Check mixing routines, draughts and temperature variations/cooling curves.	
Antibiotics	Ideally after the organism has been grown in the lab. The *E. coli* is grown and then tested against a range of antibiotics to determine which will provide the best cure	
Vaccines	To the sow and via colostrum to the piglet. Note vaccine storage and administration have got to follow prescribed protocols. Water vaccines are available for bowel oedema	
Farming Practices	To reduce stressors on the pig, particularly in neonate. Many cases of 'scour' associated with *E. coli* will respond poorly to antibiotics as the real cause is draughts. Examine each area in detail and remove as many of the stress factors as possible. Even a barely perceptible draught can result in a wind chill 3ÅC below thermometer readings. Draughts can be considered chilling air with a speed in excess of 0.2m/s	
Colostrum	The vital ingredient to survival pre-weaning. Feed back of weaner faeces is given to sows and gilts to ensure that her colostrum is adequate	
Feeding routines	Ensure good quality feed is being used as creep. Check feed quality actually being fed not just in the bag. Check feed for allergic factors, i.e. plant proteins being fed too early. In pre- and post-weaners provide small amounts of fresh feed several times a day, this helps to minimise digestive stress	
Dry sow feeding	Effective and disciplined feeding regimes are required to reduce udder oedema, mastitis and constipation	
Adequate Water	The lack of water is a major stress factor on the pig at all ages. However, insufficient marginal water supplies play a major role in the cause of cystitis and kidney disease in the pig	
Genetics	With bowel oedema in particular, only pigs which have the correct genes to allow the F18 fimbriae to attach will result in the disease. Changing the genetics may result in removal of the disease from the farm. There are F4 negative pigs, but these have not been exploited commercially. F18 negative pigs are commercially available. Do not select from sows/gilts who have any history of diarrhoea in the farrowing house. Over time this will select away from susceptibility towards *E. coli* types especially F4.	
Zoonotic implications		
	E. coli is a potential pathogen in humans. However, the more significant strain *E. coli* VTEC 0157 is only very rarely found ï less than 0.3% of carcases in a UK study	

BOWEL OEDEMA

Causal agent	*Escherichia coli* with the STx2e toxin. Normally associated with the F18 (rarely F4) fimbriae attachments. The *E. coli* is ß haemolytic.
Age group	2 weeks post-weaning
Clinical signs	
	Sudden death 2 weeks post-weaning.
	Pigs present with diarrhoea and neurological signs
	Leg paralysis, splaying, staggering, circling and severe ataxia
	Swollen eyelids
 	Dying weaner with swollen eyelids.
Transmission	
	From pig to pig ï faecal oral route
Post-mortem lesions	
	Oedema of the mesocolon, stomach, eyelids and forehead
	The Stx2e toxin is a vasotoxin causing microangiopathy ï leakage of the capillary vessels. This leads to oedema. The blood pressure increases to 20 mm Hg resulting in oedema of the brain and neurological signs.
	**Oedema in the loops of the spiral colon**
Diagnosis	
	Isolation of the F18 antigen and the STx2e toxin.
Treatment	
Affected litters	Acidify the water supply with 22g citric acid per litre drinking water for the first 21 days post-weaning
	Zinc Oxide at 2300g/tonne. Ensure dosage is accurate.
	Antibiotics where possible.
	All-in/all-out
	Thoroughly clean between batches
	Minimise utensil spread
Vaccine	Sow vaccination will not prevent bowel oedema
	Vaccinate with the non-toxigenic F18 at weaning, one week post-weaning and 4 weeks post-weaning through the water supply.
Genetics	Possible to use resistant pigs ï recessive gene
	Change boar line and possibly sow line.
Common differentials	
	Meningitis associated with Streptococci or *Haemophilus parasuis*.

ESCHERICHIA COLI PRE AND POST-WEANING DIARRHOEA

Causal agent	*Escherichia coli* ï *E. coli*. A gram negative rod bacteria
Age group	From birth to around 10 days of age. First few weeks post-weaning
Clinical signs	
0-3 days	Sudden death, with very few clinical signs. Possibly some diarrhoea
3-10 days	Acute and chronic diarrhoea, pasty yellow colour. Piglets may be found dead, but most have clinical signs which lead to dehydration and ultimately death.
Post-weaning	Acute and chronic diarrhoea. Weaners may demonstrate ill-thrift. The diarrhoea progressively leads to dehydration and death.

Neonatal death

Diarrhoea in farrowing

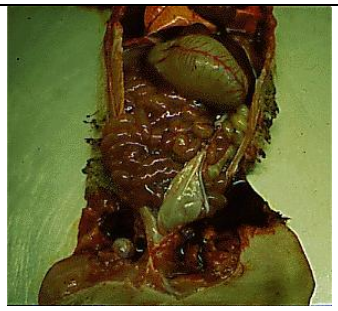
Piglet with *E. coli* diarrhoea

Diarrhoea on the walls

Diarrhoea post-weaning

E. coli diarrhoea in weaners

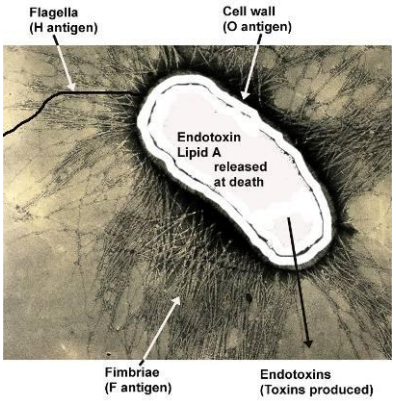
Transmission	
	E. coli are ubiquitous (everywhere). Some types are more pathogenic depending on fimbriae. The organism may be easily transmitted around the farm.
Post-mortem Lesions	
	There may be very few gross pathological findings. Dilated swollen small intestines fluid filled. Sometimes congestion of the stomach or small intestines may be seen. The intestinal contents will be alkaline.



Gross-postmortem findings dilated small intestines and stomach

Loops of bowel with very watery alkaline diarrhoea

Diagnosis



Drawing of the classification of *Escherichia coli*

Pathogenic swine *E. coli* are normally classified by:

Have one of the following four fimbriae (attachments) F4 (K88), F5 (K99), F6 (987P) or F41

Haemolytic. Produce toxins (exotoxins) ï LT, Sta or STb.

Most *E. coli* problems are associated with management and environmental factors, in particular draughts. Examination of the environment is an essential component to any diagnosis.

- As soon as diarrhoea starts, submit rectal swabs
- If piglets die, submit piglet for post-mortem examination
- If diarrhoea continues, submit live piglets before treatment

Treatment

Individual litters	Treat whole litter as soon as one piglet starts to show symptoms
	Place a trough drinker filled with water, electrolytes and glucose. This must be replaced at least 4 times daily. Keep clean
	If creep is provided, remove the creep
	All piglets to be given colostrum
	Administer antimicrobial oral dose measured doses once a day by mouth, see the practice. Ensure you do not use this medicator for healthy piglets
	Inject with a suitable antimicrobial.
	Clean up diarrhoea as soon as possible
	The stockpeople must wash their hands after treating the piglets and dip their boots in disinfectant
	Syringes or oral dosers must not be used on a sick piglet and then used on a healthy piglet
	If diarrhoea occurs after day 10 consider coccidiosis
	Ensure iron injections are appropriate especially with post-weaning scours
Cross-fostering	Pigletôs movements must be carefully monitored. A lot of scouring is associated with fostering. Review cross-fostering protocols
Utensil movement	All rooms should have their own brush and shovel
	Each brush and shovel should be placed in disinfectant when not in use. Ensure disinfectant is still working
	Teeth clipping, tattooing, notching and tail docking equipment must be thoroughly cleaned between rooms
	Ensure that the rooms are thoroughly cleaned between batches
	Consider lime-washing to enhance hygiene and disinfection
Control	Pressure washing principles must be exemplary
	Practice lime washing if possible
	Practice all-in/all-out ï pig flow
	If diarrhoea is a persistent problem have the environment examined in detail i.e. for draughts
	Ensure preventative protocols are adhered to, i.e. vaccination and feed-back
	F4 genetically resistant pigs are available but not used commercially.

Common differentials

Viral diarrhoea ï normally acid intestinal contents. Coccidiosis

Zoonosis

E. coli has the potential of being zoonotic

PRE-WEANING SCOUR PROTOCOLS

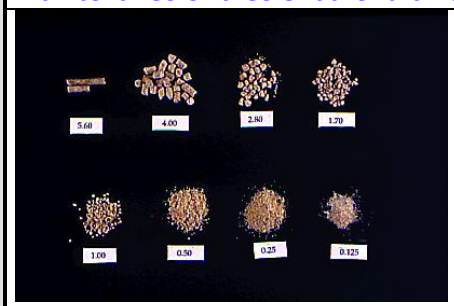
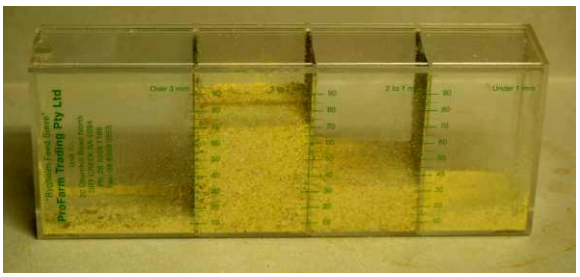
Piglet treatments	
1	Treat whole litter as soon as one piglet starts to show symptoms
2	Place a cube drinker filled with water, electrolytes and glucose. This must be replaced at least 4 times daily. Keep clean
3	If creep is provided, remove the creep
4	All piglets to be given colostrum
5	Administer antimicrobial oral dose measured doses once a day by mouth, see the practice. Ensure you do not use this medicator for healthy piglets
6	Inject with a suitable antimicrobial.
7	Clean up diarrhoea as soon as possible
8	The stockpeople must wash their hands after treating the piglets and dip their boots in disinfectant
9	Syringes or oral dosers must not be used on a sick piglet and then used on a healthy piglet
10	If diarrhoea occurs after day 10 consider coccidiosis
Cross-fostering	
1	Pigletôs movements must be carefully monitored. A lot of diarrhoea is associated with fostering. Review cross-fostering protocols
Movements of utensils and people from one room to the next	
2	All rooms should have their own brush and shovel
3	Each brush and shovel should be placed in disinfectant when not in use. Ensure disinfectant is still working
4	Teeth clipping, tattooing, notching and tail docking equipment must be thoroughly cleaned between rooms
5	Ensure that the rooms are thoroughly cleaned between batches
6	Consider lime-washing to enhance hygiene and disinfection
Investigation	
1	As soon as diarrhoea starts, submit rectal swabs
2	If piglets die, submit piglet for post-mortem examination
3	If diarrhoea continues, submit live piglets before treatment
Long term	
1	Pressure washing principles must be exemplary
2	Practice lime washing if possible
3	Practice all-in/all-out ï pig flow
4	If diarrhoea is a persistent problem have the environment examined in detail i.e. for draughts
5	Ensure preventative protocols are adhered to, i.e. vaccination and feed-back

Classic “causes” of pre-weaning diarrhoea

1	A range of disease agents ï *E. coli*, TGE, Coccidiosis, Clostridia, Rotovirus etc.
2	Almost any air movement is undesirable >0.2 m/sec (> 50 feet/min) is a draught
3	Chilling of the piglets, check lying patterns and creep temperatures (ideally 30ÅC)
4	Variable temperatures in the creep
5	Damp floors particularly in the creep area
6	Poor colostrum intake
7	No milk in the sows, check udder line ï mycotoxins and management
8	Degree of cross-fostering
9	Piglet treatments not been clean enough, Check cross-contamination between healthy and sick piglets
10	Infection transfer - is there a separate brush and scrape for each room, foot baths, personal hygiene
11	Poor room cleaning between batches
12	Number of sows farrowing each week, application of all-in all out and pig flow
13	Presence of udder oedema
14	Amount of navel bleeding
15	Type of iron injection utilised, more post-weaning scour
16	Vaccine storage protocols

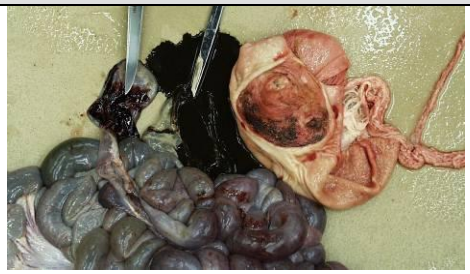
As part of the investigation, if post-mortem examinations are required, select an acutely sick piglet, not a chronic piglet which is likely to have secondary infections which may mask the actual causal agent(s).

GASTRIC (STOMACH) ULCERATION

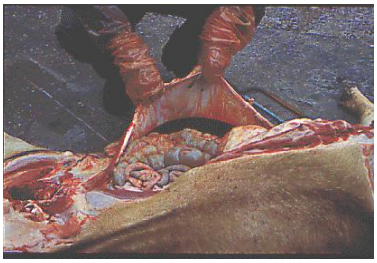
Age group	Any age group can present with a stomach ulcer. However they are more significant in lactating sows and grower pigs older than 8 weeks. The condition can occur in 100% of groups of pigs with levels of 50% of sows and 60% of growers being commonly seen.
Clinical signs	
Peracute	Death or collapse of apparently healthy animals. The animal may be pale
Acute	Animals weak and wobbly on their legs. The animals are anaemic with increased respiration. They may grind their teeth and wag their tail in pain. Animals lie down and fidget trying to find a comfortable position. The animal passes bloody tarry faeces (melaena). Vomiting may be noted. The animal is generally anorexic. The rectal temperature is normal; however, if subnormal it generally indicates a poor prognosis. The animal may be pale.
Chronic	Either presents as an extended duration of acute symptoms with weaker animals. This may be misdiagnosed as pneumonia in growers.
	In some chronic cases the oesophageal entrance becomes narrow and a stricture occurs. The pigs vomit shortly after feed and run off rapidly.
	Or no symptoms and the lesion is found as an incidental finding at post-mortem
Pathogenesis	
General	While bacteria and fungi are often found in association with ulcers no specific infectious cause has been confirmed in pigs. Note in man *Heliobacter pylori* are associated with ulcers. Other associated conditions may be copper and zinc toxicity, stress/psychological reasons in particular starvation (for only 12 hours), transportation, crowding and mixing with unfamiliar pigs.
Feed related	It is important to minimise the number of small particles in the feed below 500 μm (0.5 mm). Whey may be associated with gastric ulcers. Feed with high concentrations of unsaturated fatty acids especially together with a vitamin E deficiency are particularly prone to gastric ulcerations. Mycotoxins may also play a role in gastric ulcers. Other factors that have been associated are a low protein diet, a high energy diet and diets containing more than 55% wheat. Note the wheat type may also have a role as high yielding wheat can have sharp spicules.
No feed	Pigs which do not eat are very likely to develop stomach ulcers, especially if the period exceeds 24 hours. The effect of fine ground feed may be more significant in the maintenance of ulcers rather than their cause.
	  Feed examination of particle size

Post-mortem findings

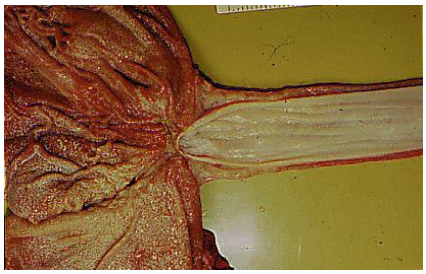
Normal oesophageal entrance

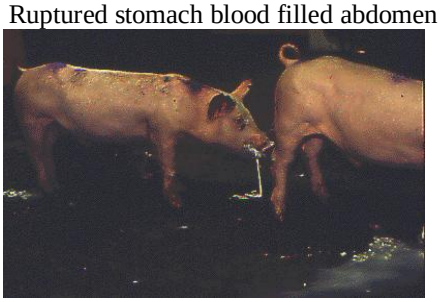


Gastric ulcer in a weaner



Ruptured stomach blood filled abdomen





Chronic ulcer with scarring of oesophageal entrance, this can lead to a stricture of the oesophagus and the pigs vomit shortly after feeding

In peracute/acute cases the stomach may be filled with dark/black blood sometime with a large blood clot. In the more chronic cases there may be black streaks in the stomach contents. The large intestine is full of black tarry faeces. The ulcer varies from mild erosion to a large ulcer with a thickened scarred wall boundary. Note chronic ulcers may bleed more than the acute looking ulcer due to capillary blood vessels oozing blood over a long period of time. Acute death is associated when the ulcer invades an underlying large blood vessel

Diagnosis

	Clinical signs. Necropsy finding. Blood in faeces -melaena.

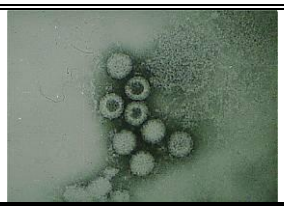
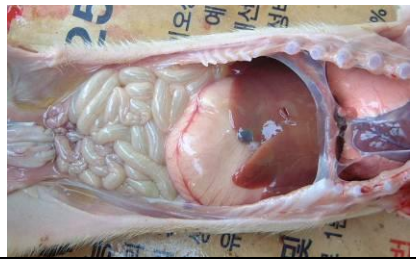
Treatment

Individuals	Treatment with aluminium hydroxide or magnesium silicate can help to line the stomach and protect the ulcer from the stomach acids and thus healing can take place. Feeding straw/hay may help to increase the fibre content and may help to heal the ulcer. The use of injectable painkillers may relieve some of the pain. In expensive individual pigs H_2 blockers or alternatives may be useful but are unrealistic in commercial situations. Get the pig to eat ï use milk/rice/beer mix to encourage eating
Herd	Vit E may be helpful at 100 g/tonne. Check feed sieve size not less than 3.5 mm. Ensure feed is clean and stored adequately. Reduce stress factors. Increase straw in diet. Increase body condition of sows. Increasing the feed size to a mean of 750 µm for 2 weeks may help the ulcer to heal. This may be useful to consider for new gilts as part of their introduction/isolation protocol.

Common differentials

	Swine dysentery, *Salmonella choleraesuis*, PIA, torsion of intestine, warfarin poisoning, copper poisoning, other cause of sudden death.

PORCINE EPIDEMIC DIARRHOEA

Causal agent	Porcine Epidemic Diarrhoea Virus a Coronavirus - RNA virus		The virus particles are called ñcoronaò because of their appearance of a sun with the surrounding corona
Other names	PEDV		
Age group	Type 1 affects all age groups but not suckling piglets Type 2 affects all age groups, clinical signs particularly severe in naive piglets but is not TGE virus ï another coronavirus. The disease is reported in Europe and Asia but not in the Americaôs or Australia		
Clinical signs			
Acute Naive herd	Explosive outbreak similar to TGE		
	Mortality of piglets may reach 80%		
	Acute watery diarrhoea in piglets		
	Diarrhoea and vomiting may be seen in all age groups		
	Problems persist for 4-6 weeks		
	The condition can occur at any time but is more severe in the winter		
Endemic herd	Little or no problem virus progressively dies out		
	Disease may be more persistent than TGE		
			
Piglets with PEDV		Sow diarrhoea with PED	Dilated small intestine
Incubation period			
	Pathological findings can be seen in 12 hours. Infection can take up to 5 days		
Transmission			
	Transmission is via the faecal oral route		
	Role of formites is very important		
	Mechanism of persistence is not known		
Post-mortem Lesions			
	Lesions are mainly in the jejunum and ileum. The duodenum is less affected. The lesions are villus atrophy. The pH of the intestine changes to acidic. In *Escherichia coli* infections the pH becomes more alkaline		

Diagnosis	
	The virus is distinct from TGE and PRC
	Immunohistochemistry of the infected intestines
	Antibody tests useful, but virus and antibodies may be present without clinical signs
Treatment and control	
Treatment	No specific treatment
	Provide supportive electrolytes for affected piglets. Piglets may require supportive therapy for 2 weeks, until intestinal tract heals
	Ensure all piglets get colostrum
	As the virus does not spread rapidly around the farm, attempt to isolate the next 3 weeks worth of farrowing sows
In an outbreak	Feedback farrowing house diarrhoea and gut materials from infected piglets to sows from breeding to 3 weeks pre-farrowing. Ensure gilts in isolation receive this material as well
	Stop introduction of new animals into the herd for 6 weeks
Control	Ensure gilts receive feed back
	Good biosecurity and practice all-in/all-out
	Purchase gilts and boars from known negative herds
Common differentials	
	Transmissible Gastroenteritis. Salmonellosis

PORCINE PROLIFERATIVE ENTEROPATHY

Other names	Ileitis, PIA, Porcine Intestinal Adenomatosis, Red Gut. Porcine Enteropathy.	
Causal agent	*Lawsonia intracellularis* an intracellular curved bacterium.	
Age group	Acute form occur in young breeding stock or finishing pigs	
	Young growing pigs exhibit chronic forms of the disease	
	The disease, albeit not apparent, can affect 15 to 50% of the growing herd	
Clinical signs		
Acute Form Proliferative haemorrhagic enteropathy (PHE)		Death (often the first signs seen). Pale and anaemic animals. Depression, reduced appetite and reluctance to move. Retarded growth. Watery, dark or bright red diarrhoea. The hindquarters may be stained with bloody faeces. Abortion may occur in recovering animals often within 6 days of the onset of clinical signs. More often seen in young adults greater than 70 kg.
Chronic forms		More often seen in growing animals 20 to 70 kg. The clinical signs depend on the extent of the intestinal lesions.
PIA Necrotic ileitis Regional Ileitis		With Porcine intestinal adenomatosis (PIA) clinical signs can be very slight with irregular periods of diarrhoea and anorexia. Animals with Necrotic ileitis have more severe clinical signs in individuals with severe loss of condition and often persistent scour. Death is not uncommon. Major effect is the increased FCR and thus feed costs necessary to finish the animal. In chronically affected herds, days to slaughter may be extended by up to 14 days Animals with regional ileitis also present with severe loss of condition and sometimes a terminal peritonitis
Infectivity		
	The pigs are infected by oral contact with faeces from infected pigs and the disease can be shed for at least 10 weeks. Nearly all farms have the organism present on the unit	
Incubation period		
	The incubation period is 13 days	
Diagnosis		
	Examination of faecal samples for *L. intracellularis* by specific antibody stains	
	PCR (DNA) analysis of faeces for the bacteria. The bacterium does not grow in media. There are blood tests available	
	Histology of the intestine may be useful for presumptive diagnosis	

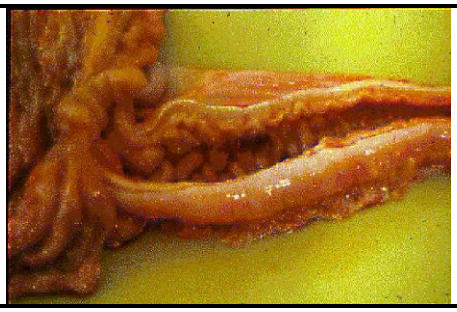
Post-mortem Lesions

Post-mortem findings	Post mortem findings generally restricted to the terminal 50 cm of the small intestine and possibly just into the spiral colon and caecum.

PHE The small intestine and large intestines are dilated and filled with a formed blood clot. The colon contains black tarry faeces. The intestinal contents are rarely liquid. The intestines bulge out of the abdomen once opened.

PIA: The intestinal wall thickens often with oedema to varying degrees. The mucosa is thrown into folds and may result in sharply defined plaques or marked multiple polyp formation.

Necrotic ileitis: There is necrosis of the underlying PIA lesion resulting in yellow/grey cheesy masses that adhere tightly to the wall.



Regional ileitis: The lower intestine becomes thickened and ridged. Often referred to as hosepipe gut. Ulceration can be seen in the mucosa.

Treatment

	Oral vaccines can be very effective.
	Antimicrobial therapy, Tylosin (Valnemulin) and Tiamutin very effective.
	Wash and disinfect pens
	Minimise mixing of pigs
	Use all-in/all-out
	Maintain proper pig density, water and feeder space
	Maintain proper building temperature and ventilation
	Match health history of incoming pigs to those of the farm
	Reduce scrape through passageways

Common differentials

	Intestinal twist, Haemhorragic bowel syndrome, gastric ulceration, Swine Dysentery, Salmonellosis, Whip worms and chronic TGE Salmonella - colon and focal ulcers and lymph node enlargement Swine dysentery - large intestine only, without swelling of lymph nodes Whip worms - pin point lesions in colon and adult worms in large intestine

Zoonotic

	Unsure. *Lawsonia intracellularis* has been recognized in other animals ï horses and hares.

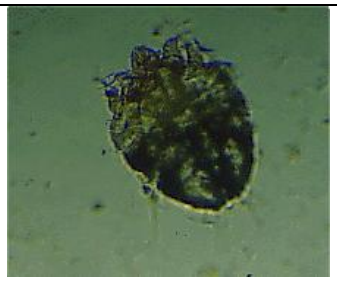
PIG PARASITES

External Parasites

Mange

Mange is caused by infection with the microscopic burrowing mite *Sarcoptes scabiei* var *suis*. All parts of the its life cycle, the egg, larvae, nymph and adult develop below the surface of the skin and only require 15 days to complete. Experimentally the mite can live for up to 3 weeks off the pig, however, at temperatures higher than 25C the mite does not survive more than 3 days.

The consistent clinical sign is rubbing and scratching. All ages can be affected from weaner to adult and the worse cases can be in the growing pig with PRRS infections. Classical signs are excess wax in the ear form which the mites can be identified; however, it can take 25 scrapes, even from infected herds, to find the mite. Monitoring of mange is carried out in the slaughterhouse. Treatment and control is by the establishment of mange free herds or in chronically infected herds by the use of in-feed or injection of ivermectins or pour on pesticides. It is estimated that about 70% of the UK national finishing herd is infested with mange. Up to 10% loss in FCR and growth rates have been reported. The mite is not infective to man.



The mange mite

Dirty ears in an adult boar

Lice

The pig biting louse is *Haematopinus suis*. These are the biggest louse known to man and are readily observed. The life cycle occurs on the body and takes 30 days to complete from egg to adult, however, the louse cannot live for more than 3 days away from the pig making control technically easier than with mange although in practice this has proven more difficult. It is possible that swine pox may be carried by lice. Lice are very sensitive to standard mange treatments.

Internal Parasites

Ascaris –milk spot

Ascaris suum is a large round worm which lives in the intestinal tract of pigs, with a prevalence of between 50 and 75% of herds. The female worm produces around 2 million eggs per day; however, production is very variable. The eggs are difficult to find in the faeces and special techniques are employed to attempt to find the egg. The eggs are very sticky and are resistant to most disinfectants, but heat (steam) are direct sunlight are effective in destroying the eggôs viability. The eggs are able to survive for more than 7 years in pasture or housing.

Once ingested the egg hatches and the larvae pass through the intestinal wall and migrate via the blood stream to the liver. The worm is only in the liver for 5 days. The liver damage heals by scarring, producing the white marks on the liver surface or ómilk spot liverô. These lesions heal within 25 days. The larvae leave the liver and migrate to the lungs where they contribute to respiratory diseases such as Enzootic pneumonia or Swine Influenza. Ascaris may cause a cough in piglets in the farrowing house. The larvae are then coughed up and swallowed and once back in the intestinal tract they mature to adults. The time interval from ingestion to producing eggs can be as short as 40 days. Ascaris is important to the pig industry as the disease reduces growth rates and feed conversion and may aggravate other diseases. It also has a direct economic loss to the slaughtering industry though liver condemnations.

Egg

Worm from anus

White spots on the liver surface

Strongyle worms

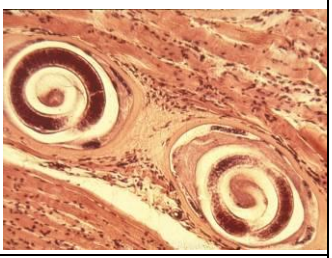
There are two important strongyle worms in the pig. *Hyostrongylus rubidus*, the red stomach worm and *Oesophagostomum dentatum* which lives in the large intestine. Neither of these worms migrates around the body, but lives in the wall and lumen of the intestines causing local damage which results in poor food conversion and growth. They both contribute to the óthin sow syndromeô and while controlled in housed sows may become an increasing problem again with loose housing. The level of infestation is calculated through the worm egg count.

Whip worms

Trichuris suis is the pig whip worm and lives in the large bowel and causes local damage to the intestinal wall. These worms do not migrate around the body. This may play a marginal role in the óthin sow syndromeô. They are readily recognized through a worm egg examination through their bipolar egg shape.

Trichinella spiralis

This is an important parasite of the pig, but is rare in the undeveloped countries. The worm is important as man may become infected resulting is severe muscular pains and swelling of the face. The life cycle is different from the worms described so far. The adult worm lives in the intestine of pigs, but no eggs are laid. The larvae develop within the female worm. The larvae are released from the female and migrate through the intestinal wall moving through the body eventually localizing in muscle tissues. Here they wait (for up to 24 years) until the muscle in eaten by another pig, a rat or man, when the live cycle starts again. Diagnosis of trichinella is through examination of muscle tissues, especially the diaphragm.

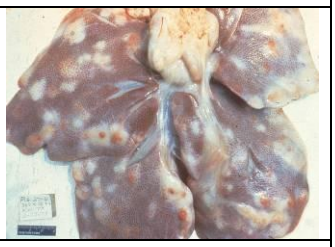


Lung Worm

The pig lung worm is called *Metastrongylus apri.* This adult worm lives in the bronchi and bronchioles of the pig where it can cause local damage and coughing. The eggs containing fully formed larvae are laid by the female, coughed up, swallowed and passed out of the pig via the faeces. The larvae are then eaten by an earthworm where it remains in the blood vessels. The earthworm is eaten by the pig and the larvae migrate through the intestinal wall to the lungs where the cycle starts again. Earthworms are able to live up to 7 years and so once pasture is infected it will take a long time to eliminate the parasite.

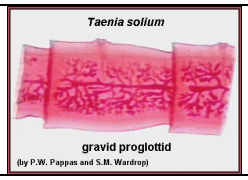
Other round worm parasites

There are a number of other parasites especially in the warmer climates, two worthy of note are *Stephanurus dentatus* (the kidney worm), very common in the southern states of the USA where the migrating larvae can cause severe liver damage and *Strongyloides ransomi* which is a worm which can kill piglets due to diarrhoea resulting from damage to their intestinal tract.
Photo shows liver damage with *Stephanurus dentatus*



Tape worms

With increased vigilance in the slaughterhouse, tape worms of pigs are now rare. Of particular important is *Taenia solium*, where the pig is the intermediate host. The larvae develop into a cyst and when infected pork is eaten by man the tape worm develops in the intestinal tract.



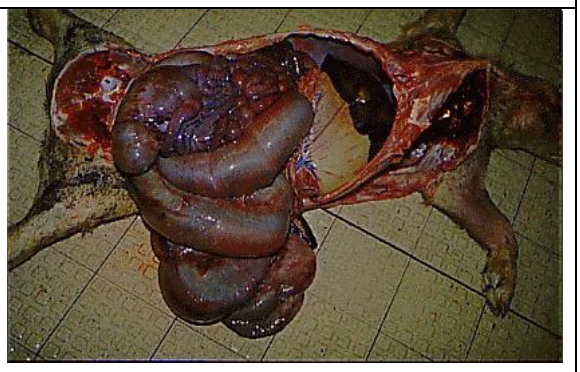
POST-WEANING ILLTHRIFT SYNDROME

Causal agent	**None – starvation. The piglets fail to learn to eat and drink post-weaning**
Age group	**Immediately post-weaning – not dependent on weaning age but more common when piglets weaned before 17 days of age**
Clinical signs	
	Severe emaciated weaners 10-15 days post-weaning
	Weaners gaunt, dehydrated often incoordinated and lethargic
	Affected weaners often exhibit signs of vice – penile sucking, sham nursing
	Other weaners look normal
	 
Infectivity	**None**
Transmission	None
Post-mortem Lesions	
	In the classical case the stomach and small intestines will be empty of food. The stomach may be filled with fluid and possibly just straw (if housed on bedding)
	However, the weaner may have just figured out how to eat.
	Absence of body fat and the superficial inguinal lymph nodes may be more prominent
	Liver may be pale
	Histological changes in small intestine include villus atrophy and fusion
	 
	Stomach and intestines empty / Liver paler than normal
Diagnosis	
	Small intestinal histology indicative of not eating
Treatment	
	Improve management of the post-weaning period
	Gruel feeding
	Ensure that gruel feeding does not continue beyond day 5 post-weaning or a double weaning effect will occur
	Examine pig flow and weaning age. Increase weaning age if possible
	Feeding creep feed pre-weaning appears to have little impact on the progression of the condition **Ensure**
Common differentials	**PMWS/PCVAD – but normally occurs after 15 kg – pigs with PMWS have learned to eat post-weaning**
Zoonotic	**None**

RECTAL STRICTURE IN THE PIG

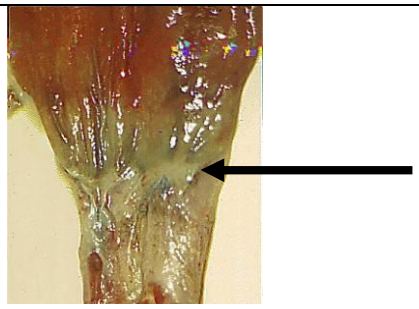
Appearance of a rectal stricture in the grow finish house

Gross post-mortem appearance of a rectal stricture.



Gross view of the abdomen showing grossly dilated large bowel.

X ray of a rectal stricture, indicated by the white arrow.



Post-mortem internal view of the rectum, the scarred rectum indicated by the black arrow

Treatment: There is no effective treatment for rectal strictures. Once recognised euthanasia is the only option.

Causes: Possible causes include: trauma of the rectum during defecation (bitten by another pig), end-stage of rectal prolapse, Salmonellosis, mycotoxins.

Control: Review causes of rectal prolapse. Review salmonella and feed management. Note certain cereal grains may break into sharp spicules increasing large bowl irritation. Change from grinding to rolling as part of the investigation process.

Rotovirus

Causal agent	Virus ï Rotovirus Double stranded RNA non enveloped Type A most common, but many strains. Also 5 types A-E are known in pigs.
Age group	Clinical signs normally between 7 and 14 days of age. Clinical signs unusual over 28 days of age
Clinical signs	
No colostral immunity	Severe clinical signs in young pigs below 14 days of age. Severe profuse diarrhoea. Diarrhoea watery, yellow, white with flecks of tissue.
	Diarrhoea continues 3-5 days and mortality may reach 100%
Colostral immunity	No clinical signs or mild diarrhoea. May contribute to other causes of diarrhoea ï *E. coli* for example

Watery and yellow diarrhoea

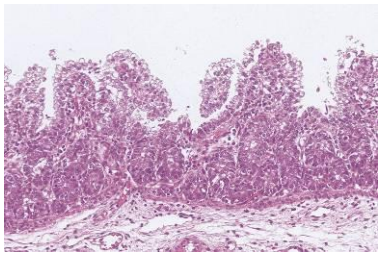
Diarrhoea may also contain vomit

Infectivity	
	The virus is extremely resistant to temperature, chemicals and disinfectant, pH changes. The virus will survive 3 months or more in the environment.
Transmission	
	The virus is ubiquitous (everywhere).
	Transmission is by the oral-faecal route.
	Sows may excrete the virus at the time of farrowing

Post-mortem Lesions

	Very watery diarrhoea and dilated small intestines
	The small intestinal villi will be shorter ï up to $^{1}/_{10}$ length of normal
	pH of the intestinal contents acidic (*E. coli* normally alkaline)
	Note recovery of intestine may occur within 72 hours so postmortem findings more severe in acute case.

Normal small intestine villi



Shortened villi with Rotovirus

Diagnosis

	Difficult as the organism is common and antibodies are normal
	Histological examination of the intestine in acute cases

Treatment

Sick pigs	Supportive treatment with electrolytes
Preventative	
Feedback	Essential to ensure that the gilt is provided with experience of the farmôs Rotovirus population so she can pass this immunity on to her piglets
	Feedback to sows 6-4 weeks pre-farrowing during outbreak
Colostrum	Review colostrum availability- especially fostering protocols
Hygiene	All-in/all-out and good hygiene between groups will help reduce clinical signs
Vaccine	Available in some countries but many not be effective due to the number of different strains

Common differentials

	TGE and EPD. Play a common role in other piglet diarrhoeaôs ï *E. coli* and Coccidiosis

Zoonotic

	Rotovirus is a common virus of Man, but direct transmission not demonstrated.

SALMONELLOSIS

Causal agent	Bacteria belonging to the genus Salmonella. There are numerous types of Salmonella that can cause a variety of 'disorders' in pigs. The classic salmonella of pigs is *Salmonella choleraesuis* var *kunzendorf* which is rare in EU but common in the USA and *Salmonella typhimurium* which is much more common worldwide.
	Salmonella infections should be distinguished into two different problems 1. Salmonella infections as a disease of pigs which is covered in this note 2. Salmonella contamination of pork and retail products
Age group	All ages can be affected
Clinical signs	
	The clinical signs differ depending on the type of salmonella infecting the individual pig. Many salmonella show no clinical signs in the pig
Septicaemia 	Septicaemic salmonellosis is often associated with *S. choleraesuis*
	This is generally seen in pigs 3 weeks to 5 months of age
	It is rare in suckling pig, probably due to intestinal lactobacilli predominance
	The disease presence as a piglet reluctant to move, anorexic, with a high temperature 40.5-41.6ÅC
	The piglet may have a shallow cough
	The piglets are generally huddled
	A few piglets may be found dead with purple (cyanotic) extremities
	After a couple of days a yellow soft faeces/diarrhoea may be seen.
	Diarrhoea faeces are often golden coloured
	S. choleraesuis is a pathogen that can cause pneumonia and diarrhoea in the same pig
	Mortality of infected pigs may be high
Enterocolitis	Enterocolitis is generally associated with *S. typhimurium*
	The piglets present with a watery, yellow diarrhoea initially without blood or mucus
	The diarrhoea may reoccur over the period of a couple weeks
	Mortality is low, mainly associated with dehydration and potassium loss
	A few pigs may remain unthrifty and some may develop rectal strictures

Clinical signs of enterocolitis may only be mild wasting and diarrhoea. Initial treatment may be disappointing

Pigs may present with cyanosis of the ears, nose and extremities

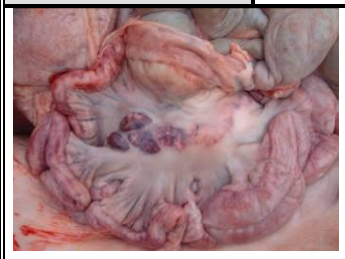
Infectivity	
	Salmonella are hardy and ubiquitous (everywhere)
	Salmonella can persist for weeks or even years in the right environment
	However, they are readily destroyed by heat, desiccation and many common disinfectants
	There is a carrier status for *S. typhimurium* which may last for 5 months
	Salmonella is spread through contact with infected pigs
	Salmonella is spread through contact with infected pigs faeces
	Salmonella is spread through contact with contaminated water supplies
	Salmonella cholerae suis is only rarely found in feed
	A disease outbreak is more likely to occur in an animal which is stressed or has other diseases
	Salmonella found in pork may be contracted during the short time spent in the slaughterhouse lairage and have nothing to do with the farm conditions. Salmonella may be found in intestinal lymph nodes within 30 minutes of oral ingestion of the salmonella.

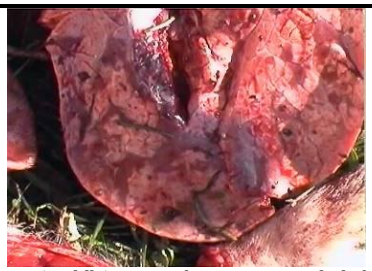
Incubation period

	24 to 48 hours to produce clinical signs in the pig. Note lairage contamination within minutes

Post-mortem Lesions

Septicaemia	With septicaemia there is cyanosis of the ears, feet, tail and abdomen Together with an enlarged spleen. The lungs are congested possibly with interlobular oedema. Jaundice is not uncommon. There may be millary white foci of necrosis in the liver. If the pigs survive the initial stages it may also present with a necrotic enterocolitis
Enterocolitis	With enterocolitis there is focal or diffuse necrotic colitis and typhlitis (infected colon and caecum). This may also extend into the small intestine. Necrotic lesions may also be seen as adherent grey, yellow debris on the red roughened mucosal surface of an oedematous spiral colon and caecum. These may be well demarcated into button ulcers. The mesenteric lymph nodes are often greatly enlarged.





In enterocolitis intestinal changes may be mild with swollen lymph nodes (left) to a necrotic enteritis (middle). Lung changes may include blotchy consolidation (right)

Diagnosis

	Isolation of the organism

Treatment

In many countries all infected cases and isolates have to be reported to local authorities

Individuals	Salmonella live inside cells and are thus cannot be reached by many antimicrobial agents. Treatment can therefore be difficult and unrewarding. Provide water and electrolytes as the main component of your treatment regime. Consider using probiotics to restore gut microflora.
Control	Minimum bacterial spread
	Note that one diarrhoetic pig will massively infect the environment
	Remove all sick pigs and materials and isolate the pig
	Scrupulously adhere to cleaning regimes
	Pay particular attention to water supplies. Reduce pH to less than 4
	Restrict staff and utensil movements
	Reduce stress factors where possible
	Apply strict all-in/all-out
	It is possible to vaccinate, but many vaccines are overwhelmed in the face of a serious challenge. This may be used in a *S. cholerasuis* outbreak

Common differentials

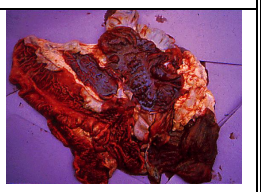
Septicaemia	Aujeszky's disease (liver changes), Actinobacillus pleuropneumonia, Erysipelas, Classical Swine Fever
Enterocolitis	Swine Fever, Swine Dysentery, PE (Ileitis), Coccidiosis, Clostridial enteritis and other causes of diarrhoea

Zoonotic implications

	Salmonella can infect human beings and may result in a fatal infection

SWINE DYSENTERY

Other names	Blood dysentery, bloody scours
Causal agent	Bacterial Brachyspira *hyodysenteriae* (formally known as *Serpulina hyodysenteriae*). 12 serotypes are known
Age group	Typically affects pigs from 15 to 70 kg
	However, in acute outbreaks in naive herds it can affect all age groups from suckling piglets to adult sows
Clinical signs	
	Diarrhoea - with or without blood, and severity is very variable. A careful search of the pen normally reveals the presence of blood and mucus in some places
	Diarrhoea with large amounts of mucus in faeces and afterwards with flecks of blood
	Death of one or two pigs before other pigs show any signs
	Rapid loss of condition in some pigs and pigs look hairy
	Clinically affected pigs in a group can reach 50%
	Reduction in FCR of 0.6 while disease present with extension of finishing by 20 days
On established herds	Dehydration, pigs with a painful abdomen and some pigs weak and incoordinated
	The disease appears to be cyclic and reappears at 3-4 week intervals
Infectivity	
	Pigs may transmit the bacteria for 90 days
Brachyspira hyodysenteriae survives in:	
	Faeces for 61 days at 5ÅC
	Soil for 18 days at 4ÅC
	Flies for 4 hours
	Mice can shed for over 180 days
	Cats and dogs can carry for 13 days
Incubation period	
	Incubation period 10-14 days
Common differentials	
	Colitis
	Salmonellosis
	PIA - Haemorrhagic Porcine Intestinal Adenomatosis - Ileitis
	Other Brachyspira spp can cause very similar signs, including the mortality.

Post-mortem Lesions	
	Confined to the large bowel - caecum, colon and rectum
	Reddening to haemorrhage of the large bowel
Post-mortem findings	Typical changes in the acute phase include hyperaemia and oedema of the walls of the large intestine. Colonic submucosal glands may be more prominent and appear white. The mucosa is usually covered by mucus and fibrin with flecks of blood and the colonic contents are soft to watery and contain exudate. The photograph represents a severe case with extensive haemhorrage into the colon. 
Diagnosis	Isolation of *Brachyspira hyodysenteriae* in the faeces. Note there are a number of other spirochetes that are normal in the pigs' large bowel and other may be associated with colitis syndromes. PRC is available and may be used on faecal samples. Immunohistochemistry can be useful on tissues samples.
Treatments	
Acute outbreak	Treatment via the water supply is essential for acute cases of swine dysentery as the affected pigs will not eat and to all animals in drainage contact
	Follow by in-feed medication once pigs start to eat, to all affected pigs and all animals in drainage contact
	Very sick and weak pigs respond better using injection antibiotics
	Water supply should be supplemented using electrolytes
Herd eradication	
Typical procedures without total depopulation	Requires detailed preparation and attention to detail
	Attempt in the late spring/summer
	Reduce herd size to as small as possible ï consider a partial depopulation
	Have an effective rodent control programme
	Drain all slurry pits
	All buildings not containing pigs should be cleaned, disinfected and fumigated
	Medicate all remaining pigs as prescribed
	After 1 week of medication all equipment used for handling pigs, feed and manure should be cleaned and disinfected
	Clean and disinfect floor as often as possible
	Treat all farm cats and dogs as prescribed
Spread of swine dysentery	
	Pigs Purchased only from Swine Dysentery free herds
	Faeces
	Boots, clothing, stockpeople
	Truck wheels
	Rats and Mice
	Cats
Zoonotic	
	None

TRANSMISSIBLE GASTROENTERITIS

Definition	TGE
Agent	TGE is caused by a virus belonging to the virus family coronavirus
	Related viruses 1 PRCV Porcine Respiratory Coronavirus which is a mutant of TGE 2 Porcine Epidemic Diarrhoea virus I and II 3 Haemagglutinating Encephalomyelitis virus The emergence of PRCV in 1986 effectively vaccinated the European herd against TGE. In America PRCV only appears to reduce the clinical signs
Clinical signs	
Initially	 Watery diarrhoea (foul smelling yellowish-green often containing flecks of undigested milk particles in the piglet); vomiting and loss of appetite in pigs of all ages. The disease spreads rapidly around the farm. Piglets less than 21 days of age are all affected and generally die. Weaners become unthrifty. Growers, finishers and adults are generally mildly affected and will survive if their water supplies are adequate. Outbreaks on smaller herds generally only last 3 weeks.
Chronic	On large herds the disease can persist for some time contributing to post-weaning diarrhoea.
Spread	Spread occurs directly or indirectly through contact with infected faeces. Starlings in particular are implicated in the spread of the virus. The virus is relatively fragile and susceptible to disinfectants and drying. However, can survive a few days in the cold, hence the disease is more severe in the winter
Incubation	18 hours to 3 days
Treatment and Control measures	
Treatment	There is no specific treatment. Vaccines are generally disappointing. However, nursing and enhanced management of the piglet may reduce loss
	⁞ Provide warmth, extra bedding and fluids (electrolytes)
	⁞ If sows go off their milk provide milk replacer/creep
	⁞ Cross-suckle affected piglets onto recovered sows
	⁞ Early wean into warm dry flat decks or similar accommodation
	⁞ Use antibiotics as directed by your vet to control secondary infections
	Ensure that all non-pregnant and pregnant (up to 3 weeks pre-farrowing) are exposed to the TGE virus (intestines and feaces of affected piglets).
	Sows about to farrow must not be exposed or they will infect their offspring and have inadequate colostrum to provide adequate cover
Control	Critically assess your general hygiene and disease control measures. Including the avoidance of unwanted visitors
	Provide specific loading/unloading areas for pigs and keep them clean
	Utilise adequately isolation facilities for introduced animals
	Bird proof pig units where practical
	Avoid spillage of feed around hoppers, and where food is split clean it up
	On yarding systems cover all feed hoppers
Zoonotic implications	
	None