Internal parasites of pigs

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Animal Biosecurity & Welfare

**Introduction**

Pig performance is influenced by internal parasites, most dramatically in the young, growing pig. Internal parasites can kill, but loss of appetite, reduced daily rate of gain, poor feed conversion, and increased susceptibility to other pathogens are the more common results of parasitism. Performance effect varies greatly in relation to geographical region, type of housing, management, nutrition, pig breed, and species of parasite.

Appendix 1 lists the common internal parasites of pigs.

**Stomach**

*Hyostrongylus rubidus* (red stomach worm) is a blood-sucking trichostrongyloid nematode and it is essentially a parasite of pastured pigs. Adults are <10 mm in length and are bright red when first removed from the host. Infections are usually not pathogenic, but if enough worms are present hyperaemia, catarrhal gastritis, submucosal oedema, hyperplasia of the gastric gland area, erosion of the mucosa, and ulcer formation may result.

Affected pigs lose condition and pallor of the skin and mucous membranes may be apparent in adult pigs. The parasite is thought to be involved in ill-thrift and the ‘thin-sow syndrome’. Adult pigs can die following a sudden blood loss when ulcers perforate. It is uncommon in growing pigs.

Eggs are almost indistinguishable from those of *Oesophagostomum* species so larval culture is best for diagnosis.

**Small intestine**

*Ascaris suum* (large roundworm) is the most important gastrointestinal worm of pigs. It is more common in growing pigs than in adult pigs. Adult worms are large and pinkish-yellow: females are 20-40 cm in length and males 15-25 cm.

Heavily infested pigs may have up to twenty five worms blocking the small intestines and bile duct causing loss of appetite, vomiting, jaundice and death if the small intestine is ruptured. In less extreme cases there is reduced appetite, poor feed efficiency and slow growth. Growth rate and feed efficiency can be depressed by up to 10%.

Female worms are very prolific producing 0.5 to 2 million eggs per day and these can survive outside the pig for many years. They are resistant to drying and freezing, but sunlight kills them in a few weeks.
Internal parasites of pigs

Ascaris suum

Worms in the small intestine: photo courtesy of Merial Pty Ltd

Larvae migrate through the liver and cause haemorrhagic foci. On repeated exposure to larvae, there is an increase in connective tissue and infiltrating eosinophilis, and dilation of lymphatics, which grossly appear as whitish spots commonly referred to as ‘milk spots’.

‘Milk spot’ lesions on the liver

In the lungs, migrating larvae cause a verminous pneumonia, which may result in death if large numbers of larvae are involved. Clinical signs are those of pneumonia. Pigs have an asthmatic cough and may breathe with difficulty.

Strongyloides ransomi

Strongyloides ransomi (intestinal threadworm) is more common in the warmer climatic regions, where it is an important parasite of suckling pigs. Adults are practically microscopic, measuring about 4 mm in length.

Diarrhoea followed by progressive dehydration is the common clinical sign. In heavy infections, death generally occurs before piglets are 10-14 days old, but stunting and unthriftiness are the more usual sequelae of infection.

No specific lesions are associated with this parasite. Larvae can be distributed widely in most tissues of the body and lesions are dependent on the number of larvae and host response. Diagnosis is achieved by identifying eggs in fresh faeces or finding adults in the small intestine at necropsy.

Isospora suis

Neonatal coccidiosis caused by Isospora suis is the most important protozoal disease of pigs. Yellowish scours is the main clinical presentation in piglets between 7 and 11 days of age. Coccidiosis should be suspected if there is a diarrhoea problem in piglets from 7-21 days of age that does not respond well to antibiotics.

Piglets usually continue to suckle, develop a rough hair coat, become dehydrated, and have depressed weight gains. Not all piglets within a litter are equally affected. Concurrent bacterial, viral or other parasitic infections may lead to extreme mortalities and complicate diagnosis.

Necropsy examination may demonstrate gross lesions of neonatal coccidiosis characterised by a fibrinonecrotic membrane in the jejunum and ileum, but this is seen only in severely infected piglets. Microscopic lesions consist of villous atrophy, villous fusion, crypt hyperplasia, and necrotic enteritis.

Faecal samples for laboratory confirmation should be taken from semi-recovered pigs rather than scouring pigs.

Coccidiosis can be treated with trimethoprim-sulphonamide (available only from veterinarians), but for the treatment to be effective it must be given prior to invasion of the intestinal wall; once clinical signs have appeared the damage has been done.

Toltrazuril given as an oral drench on day 3-6 prevents diarrhoea, prevents oocyst shedding, and can maintain piglet growth.

Particular attention must be given to hygiene in control as the high levels of oocysts passed by piglets and their ability to survive for months makes environmental contamination important. Farrowing crates must have all organic matter removed prior to steam cleaning.

Large intestine

Trichuris suis

Pigs are considered the natural hosts of Trichuris suis (whipworm) although primates and humans may be infected. Trichuris infection is a fairly
common problem in pigs. Adult females are 6-8 cm in length and males 3-4 cm.

Trichuris infections cause enterocyte destruction, ulceration of the mucosal lining, loss of capillary blood +/- secondary bacterial infection.

Clinical signs include anorexia, mucoid to bloody diarrhoea, dehydration, and death. Trichuris infection must be considered in the differential diagnosis of swine dysentery that does not respond to antibiotic therapy.

The spectrum of gross lesions may be oedema with formation of nodules containing exudates surrounding portions of worms to the formation of a fibrinonecrotic membrane. Erosion of capillary beds and vasodilation result in haemorrhage, anaemia, and hypoalbuminaemia.

Eggs can remain viable for many years outside the pig and are easily identified in pig faeces.

**Oesophagostomum species**

Oesophagostomum species are strongyloid nematodes. The larval stage produces nodules in the lower small intestine and the large intestine. Adults are stout, white, and slightly curved. Females are 1-2 cm in length and males slightly shorter.

Formation of nodules from the caecum to the rectum is the major change. Walls of the caecum and colon become oedematous from extensive thrombosis of the lymphocytes. Secondary infection may occur and enhance clinical signs of depression, anorexia and scouring. The damage leaves the intestines unsuitable for use as sausage casings.

Eggs are typically strongyloid and may be confused with hyostrongylus. Larval culture aids in differentiation, but necropsy is the most reliable means of diagnosis.

**Respiratory tract**

Metastrongylus species occur in the trachea, bronchi and bronchioles, especially in the diaphragmatic lobes, and exclusively in pigs. Adults are slender and white: females are 50 mm in length and males 25 mm.

Dissecting the bronchioles reveals mucoid plugs in the diaphragmatic lobes of the lungs, which are filled with adults and eggs. Parasites, mucus and cellular exudate cause occlusion and induce atelectasis (collapse of lung tissue) observed as coughing or ‘thumps’.

It is difficult to find metastrongylus eggs in a faecal examination, but it is important to look for ‘fuzzy’ areas in which eggs are held in mucus. At necropsy, worms can be extruded by clipping the posteroverentral margins of the diaphragmatic lobes.

**Urinary tract**

*Stephanurus dentatus* (kidney worm) is a strongyloid nematode of pigs. Infestations occur mainly in pigs kept in paddocks or earth-floored
Internal parasites of pigs

Patent kidney worm infection is more common in breeding stock 2 years and older. Adults are thick-bodied with a black and white mottling and 2-3 cm in length. Kidney worms are found in cysts in peri-renal fat with fistulous openings into the ureters, in the kidney and in ectopic sites such as the pancreas, lumbar muscles, spinal cord and lungs. As well as the adult worms the cysts contain greenish pus. The immature or larval form is often found in the liver.

Posterior paralysis has been associated with larval migration around the spinal cord. Female worms can shed up to 1.5 million eggs per urination. Blood is often passed in the urine. Gross pathological changes may be found where there is migration: mesenteric lymph nodes are oedematous and swollen. Liver changes include inflammation, eosinophilia, abscessation and extensive fibrosis making this infection easily differentiated from ascarid migration. Similar lesions can be seen in other organs. Worms, abscessation, and liver scarring can be seen at necropsy. Eggs in urine can be confirmed ante-mortem.

Public health

Echinococcus granulosus

The hydatid cyst is the larval stage of Echinococcus granulosus, a tapeworm of dogs. Intermediate hosts are sheep, goats, cattle, horses, pigs, wildlife, and humans. The primary host becomes infected by eating offal of intermediate hosts.

Infection of intermediate hosts is by ingestion of eggs from the primary host. Hydatid cysts in humans can cause life-threatening disease.

Cysticercus cellulosae

Porcine cysticercosis due to Cysticercus cellulosae (pork measles) has never occurred in Australia. This disease is notifiable in Australia. Cysticercus cellulosae is the larval stage of the human tapeworm Taenia solium. The pig is the intermediate host and becomes infected by eating food contaminated with human faeces or sewage. Humans are infected by eating uncooked pork.

Trichinella spiralis

Trichinella spiralis infection is notifiable in Australia. The main importance of Trichinella spiralis is as a parasitic zoonosis associated with eating raw or improperly cooked infected meat, especially pork.

Spirometra erinacei

The zipper tapeworm Spirometra erinacei is significant because the second larval stage causes sparganosis in humans. Humans risk contracting the disease if they eat pork infected with the immature form of the parasite. Feral pigs can be a source of infection. Adult tapeworms are found in dogs, cats, foxes and dingoes. Eggs may be deposited in areas of water where they hatch and are ingested by pigs drinking the water.

Parasite control

Good sanitation and adequate nutrition are very important in controlling infections and reducing the adverse effects of parasites. The major mode of transmission of worm parasites is through contamination of food, soil or bedding with faeces or urine. Worm eggs need moisture and warmth to develop and survive.
The usual disinfectants used on the farm do not kill eggs and larvae of worms such as ascarids. Control options include:

- Thorough cleaning of buildings, pens and equipment with detergent and steam
- Fire and burning of surfaces with a flame gun (where the structure of the building will permit). Such burning must be thorough, but only carried out by trained operatives in a safe manner
- Hydrated lime mixed with water to form a thick emulsion results in pH 13-14. Due to its caustic nature, lime washing is a hazardous procedure for which operatives must be fully trained and provided with 100% protective clothing coverage including eye protection

In outdoor situations the management approach must be to avoid a build-up of worm eggs and larvae where pigs are kept. This can be achieved by regular strategic worming combined with movement of pigs to clean areas. However, it must be remembered that *Ascaris suum* and *Trichuris suis* eggs are capable of surviving many years so a return to previously used areas too soon will risk re-infestation.

For small piggeries, especially in backyard situations, pigs may be set-stocked for many years and as such there is always a risk of parasite build-up and the development of ‘pig-sick’ land. If this occurs, it may be necessary to permanently remove pigs from such land. To avoid such build-ups in the first place, regular de-worming of backyard pigs is essential.

For pet pigs, often kept within the owner’s living area, routine collection and disposal of faeces in exactly the same way as would be done for dogs will prevent re-infestation and worm build-up and it is often not necessary to treat adult pet pigs for worms. They do however often require regular sarcoptic mange treatment and as such are usually de-wormed at the same time.

Therapeutic treatment of pigs with anthelmintics may present only a temporary solution unless the conditions under which the parasites were acquired are altered. No drug is effective against all parasites and damage done by developing worms or migrating larvae cannot be cured.

Appendix 2 lists some of the registered products for the treatment and control of common internal parasites of pigs. Further information is available at https://portal.apvma.gov.au/pubcris.
# Appendix 1: Common internal parasites of pigs

<table>
<thead>
<tr>
<th>Location</th>
<th>Common name</th>
<th>Scientific name</th>
<th>Intermediate host</th>
<th>Mode of infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stomach</td>
<td>Red stomach worm</td>
<td><em>Hyostrongylus rubidus</em></td>
<td>None</td>
<td>Ingestion of infective larvae</td>
</tr>
<tr>
<td></td>
<td>Large roundworm</td>
<td><em>Ascaris suum</em></td>
<td>None</td>
<td>Ingestion of infective eggs</td>
</tr>
<tr>
<td>Small intestine</td>
<td>Intestinal threadworm</td>
<td><em>Strongyloides ransomi</em></td>
<td>None</td>
<td>Larvae by percutaneous, oral, transcolostral, prenatal</td>
</tr>
<tr>
<td></td>
<td>Coccidia</td>
<td><em>Isospora suis</em></td>
<td>None</td>
<td>Ingestion of infective oocysts</td>
</tr>
<tr>
<td>Large intestine</td>
<td>Whipworm</td>
<td><em>Trichuris suis</em></td>
<td>None</td>
<td>Ingestion of infective eggs</td>
</tr>
<tr>
<td></td>
<td>Nodular worm</td>
<td><em>Oesophagostomum dentatum</em></td>
<td>None</td>
<td>Ingestion of infective larvae</td>
</tr>
<tr>
<td>Lungs</td>
<td>Lungworm</td>
<td><em>Metastrongylus elongatus</em></td>
<td>Earthworm</td>
<td>Ingestion of infected earthworms</td>
</tr>
<tr>
<td>Kidneys</td>
<td>Kidney worm</td>
<td><em>Stephanurus dentatus</em></td>
<td>Earthworm</td>
<td>Larvae by percutaneous, oral, possibly prenatal, and ingestion of infected earthworms</td>
</tr>
</tbody>
</table>
## Appendix 2: Registered worm treatment chemicals for pigs*

<table>
<thead>
<tr>
<th>Active</th>
<th>Trade name</th>
<th>Manufacturer</th>
<th>Withholding periods</th>
<th>Dosage</th>
<th>Label claim/recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toltrazuril</td>
<td>Baycox</td>
<td>Bayer</td>
<td>Meat days</td>
<td>ESI days</td>
<td>1 mL/piglet orally at 3-6 days old</td>
</tr>
<tr>
<td></td>
<td>(50 g/L)</td>
<td></td>
<td>70</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>Abamectin</td>
<td>Virbamec antiparasitic injection for pigs</td>
<td>Virbac</td>
<td>21</td>
<td>-</td>
<td>1 mL/33 kg</td>
</tr>
<tr>
<td></td>
<td>(10 mg/mL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doramectin</td>
<td>Dectomax injectable</td>
<td>Pfizer</td>
<td>35</td>
<td>-</td>
<td>1 mL/33 kg</td>
</tr>
<tr>
<td></td>
<td>(10 mg/mL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ivermectin</td>
<td>Bomectin injectable</td>
<td>Bomac</td>
<td>28</td>
<td>-</td>
<td>1 mL/33 kg</td>
</tr>
<tr>
<td></td>
<td>(10 mg/mL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ivermectin</td>
<td>Noromectin premix for pigs</td>
<td>Norbrook</td>
<td>7</td>
<td>7</td>
<td>100 ug/kg ivermectin daily for 7 consecutive days</td>
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<tr>
<td></td>
<td>(6/kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ivermectin</td>
<td>Imax soluble wormer</td>
<td>Bomac</td>
<td>7</td>
<td>7</td>
<td>1 mL/100 kg daily 7 consecutive days</td>
</tr>
<tr>
<td></td>
<td>(10 mg/mL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Levamisole</td>
<td>Nilverm pig and poultry wormer</td>
<td>Coopers</td>
<td>3</td>
<td>14</td>
<td>5 mL/10 kg oral drench, in-feed or added to milk</td>
</tr>
<tr>
<td></td>
<td>(14 g/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Piperazine</td>
<td>Piperazine solution</td>
<td>Inca</td>
<td>0</td>
<td>21</td>
<td>4 mL/5 kg oral drench, in-feed or added to water</td>
</tr>
<tr>
<td>anhydrous</td>
<td>(172.5 g/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>