

DOI:

<https://doi.org/10.61900/SPJVS.2025.04.19>

THE MOST IMPORTANT HELMINTHS INFECTION OF PIGS IN EXTENSIVE BREEDING

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Abstract

Parasitic infections are constant companions of pig production, regardless of the method of breeding. They are equally found in farm and extensive rearing in all production and age categories of animals. Extensive pig breeding has a long tradition. Although this kind of behavior provides pigs with a much more comfortable life than farming (after all, organic production was developed on its principle), it also has many negative consequences for their health. Pigs are exposed to the risk of outbreaks of infectious and parasitic diseases that can cause large losses in production. Extensive and semi-extensive breeding means that pigs have direct contact with a multitude of transitional hosts of parasites, they are in contact with wild swine, and there is also frequent contamination of the area where they are discharged with the excrement of wild animals. Parasitic infections, along with other infectious diseases, are extremely common in this way of breeding. Infection is most often with biohelminths (*Metastrongylus* sp, causative agents of verminous gastritis, *Macracanthorhynchus hirudinaceus*) and zoonotic parasites to which pigs are transitional (*Trichinella spiralis*, *Taenia solium*, *Ascaris ssum*) or parent hosts (*Alaria alata*). There are also species that are equally present in extensive and farm farming, such as e.g. *Trichuris suis*, *Oesophagostomum* sp. or *Strongyloides ransomi*.

Key words: pigs, extensive breeding, parasitic disease, helminth, zoonoses,

Extensive pig breeding has a long tradition in world and in Serbia. This way of breed can take many forms. Herding means keeping pigs in herds with a larger number of animals from different owners on pastures where they spend most of the year. There they move freely, feed, and these animals often come into contact with wild boars, and they often mate. This upbringing was common until the mid-sixties of the 20th century in the northwestern parts of Serbia but today it is much rarer. Another form is keeping a smaller number of pigs in individual farms and they are in half drains with free movement. Today, such a way of breeding is conditioned by the fencing of the areas where the pigs are kept in order to prevent contact with wild pigs (which often fails).

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In farm breeding, there are large agglomerations of animals in a limited space, and the danger of the occurrence and spread of disease increases along with the density of the population of animals in the pens and boxes (Perez Brincones and Alvarez Fernandez, 1977, Loskot *et al.*, 1988, Pavlović *et al.*, 2011, 2013).

Extensive and semi-extensive breeding means that pigs have direct contact with a multitude of transitional hosts of parasites, they are in contact with wild swine, and there is also frequent contamination of the area where they are discharged with the excrement of wild animals (Bečkei *et al.*, 2011, Stokić-Nikolić *et al.*, 2011, stojanov *et al.*, 2018).

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Infection is most often with biohelminths (*Metastrongylus* sp, causative agents of verminous gastritis, *Macracanthorhynchus hirudinaceus*) and zoonotic parasites to which pigs are transitional (*Trichinella spiralis*, *Taenia solium*, *Ascaris suum*) or parent hosts (*Alaria alata*) (Pavlović and Ivanović, 2005, Gavrilović *et al*, 2019, Pavlović *et al*, 2016, 2025). There are also species that are equally present in extensive and farm farming, such as e.g. *Trichuris suis*, *Oesophagostomum* sp. or *Strongyloideus ransomi* (Pavlović *et al*, 2011).

BIOHELMINTH INFECTIONS

Metastrongylidiosis

Out of a total of 6 species of nematodes in this genus, *Metastrongylus elongatus* and *M. pudendotectus* were found in Serbia (Pavlović *et al*, 2005a.). Metastrongylides belong to biohelminths whose transitional hosts are lumbricids (earthworms). Larvae are released from the eggs and become infectious when they are eaten by earthworms from the genera *Eisenia*, *Dandreobena*, *Allopbophora*, *Lubricus* and *Octolasmus* (Kruse, 1978 Tričković, 1978, Pavlović *et al*, 2005b). The clinical picture depends on the degree of infection. In weak infections, a weak cough is most often present, and in strong infections, dyspnoea and frequent vesicular breathing. In the beginning, there is a weak and later a harsh cough, which is in the form of an attack when the animals are stressed (running, etc.) (Nakauchi *et al*, 1991, Pavlović *et al*, 2005a).

Gastritis verminosis

Verminous gastritis is a common name for a disease caused by a large number of nematodes belonging to the order Spiruridea and Trichostrongylidae. (Pathison *et al*, 1980, Pavlović *et al*, 2008).

Ascarops strongyllinae (syn. *Arduenna strongyllinae*) has larval development via Coleoptera from the genera *Aphodius*, *Onthophagus* and *Gymnopleurus*. If the infected coleopterans are eaten by the parent hosts - birds, small mammals, amphibians or reptiles. L2 larvae from their digestive tract migrate into the muscles where they cocoon again. Pigs become infected when they eat infected arthropods or the meat of infected parental hosts. In the stomach of pigs, the parasites mature, attach to the stomach lining, and there we find them in a large amount of mucus. (Corwin and Stewart, 1992, Pavlović *et al*, 2008).

Physocephalus sexalatus lives in the stomach, less often in the small intestine, of pigs and less often in donkeys, camels and cattle. Larval development takes place through Coleoptera from the genera: *Scarabeus*, *Aphodius*, *Gymenopleurus*, *Geotrupes* and *Onthophagus* (Pavlović *et al*, 2008). Pigs and other true hosts become infected when they eat infective arthropods or the flesh and organs of infected transitional hosts. In pigs, inflammatory processes can only be seen in severe infections. Weak infections pass almost imperceptibly through visible pathological changes, especially in older pigs.

Hyostrongylus rubidus does not have transitional hosts and is often found in pig farms where the necessary hygienic conditions do not prevail (Pavlović *et al*, 2008). At the time of laying, the eggs are furrowed into blastomeres. In the external environment, they continue to develop into larvae and become infectious. After oral infection, the larvae remain in the stomach mostly around the pylorus and fundus. The pathological effect is manifested by the larvae during their histotropic phase, and then by the adults with their mechanical and toxic effects, and due to feeding on the blood of the host, they can cause anemia in young animals.

Gnathostoma hispidum lives in the stomach wall of pigs and, more rarely, beef and humans. The eggs in the external environment have developed blastomeres, in them a larva forms which, after two moults, leaves the egg and penetrates into Cyclops shrimps. In the development of gnathostomes, there are also parental hosts - birds, amphibians and reptiles that are infected in the same way and in which the larvae migrate into organs and muscles. Once in the host's stomach, the larvae pierce the mucous membrane with their front end, most often in the area of the pylorus. In those places, damage appears as big as a millet grain, round with a sharp red edge (Pavlović *et al*, 2008).

Macracanthorhynchosis

Macracanthorhynchus hirudinaceus is a large acanthocephalus with clearly manifested sexual dimorphism. Females lay oval eggs with an embryo surrounded by four membranes armed with several small hooks (Pavlović *et al*, 2007). They are infected by transitional hosts, coleopterans from the Scarabidae family from the genera *Melolontha*, *Cetonia*, *Polyphylla*, *Anomala*, *Scarabeus* (*Ateuchus*), *Tropinota*, *Anisoplia*, *Amphimallon*, and others. (Olsen, 1986; Crompton and Nickol, 1995) When the scarab larvae eat the eggs of the

macracantharhynchus in the digestive tract, the larvae (acanthor) are released and remain in that stage throughout the entire metamorphosis of the coleoptera (Pavlović *et al*, 2010). Pig infection occurs when pigs eat infected pupae, larvae or adult coleopterans. Parasites are found in the small intestine where they pierce the wall of the intestine to the submucosa with their reeds, and in those places an ulcer the size of a millet grain to the head of a needle forms. The bottom of the ulcer is necrotic and the edge thickened due to chronic inflammation, which leads to the formation of visible nodules on the outside of the intestinal wall (Crompton, 1992, Pavlović *et al*, 2007).

ZOONOTIC HELMINTHS

In extensive keeping, pigs are exposed to contact with eggs or larvae of several significant zoonotic parasite species, where they are transitional or parental hosts.

Swine cysticercosis

Cysticercus suum is the encysted larval form of the tapeworm *Taenia solium*, which has a human as its first host. The eggs of the parasite are found in the excrement of infected people, and the infection of pigs and wild boars occurs through the ingestion of tapeworm eggs from human faeces due to the coprophagous habit of pigs, contaminated food or water (Wu *et al*, 2017, Trevisan *et al*, 2018). After infection of pigs, embryos are released in the intestines, which break through the intestinal wall and through the bloodstream, less often lymphoma, reach the muscle tissue where cystic formations known as pig berries (*Cysticercus cellulosae*) develop (Garcia and DelBrutto, 2000). There are most of them in the chewing muscles, heart, diaphragm, gluteal, neck and intercostal muscles, tongue, and they can also be found in the brain, lungs, liver and other organs. Human infection is caused by insufficiently thermally processed infected pork meat and leads to the complete development of the parasite (Gamble, 1997, Pavlović and Ivanović, 2005).

Trichinosis

Trichinosis is a parasitic zoonosis caused by larvae of *Trichinella spiralis*. The infection occurs through the consumption of thermally unprocessed meat and meat products or dead animals in which larvae of *T. spiralis* are cocooned (Fransen *et al*, 2017). When a host eats meat from

an infected animal, trichinella cysts break open in the intestine and larvae grow into adult roundworms. The female produces other larvae that move through the gut wall and into the bloodstream. The larvae invade muscle tissues, including the heart and diaphragm. They can also infect the lungs and brain. The resulting cysts can remain alive for years (Gottstein *et al*, 2009). The largest number of cocooned larvae is in the muscles of the diaphragm, tongue and chewing muscles, but they are also present in all other muscles. In pigs, trichinosis cannot be observed during life (Pavlović and Ivanović 2005).

Alariosis

Alaria alata is a trematode whose true hosts are foxes and other canid species (Pavlović, 1994). It lives in the small intestine of the host, from where the eggs of this parasite are eliminated into the external environment through feces. Alariae have complex indirect life cycles involving two transitional hosts, a water snail from the genus *Planorbis* and tadpoles in which mesocercariae develop. Mesocercariae cocoons are infective to the true hosts, the carnivores, but also to a number of parietic hosts, through which the frogs enter the food chain, including birds, wild pigs and finally humans. (Paulsen *et al*, 2013, Berger and Paulsen, 2014, Riehn *et al*, 2014, Gavrilović *et al*, 2019)

Ascaridiasis

Pig ascaridiasis is the most common, significant and widespread parasitic disease of domestic and wild pigs caused by the nematode *Ascaris suum*. (Pavlović, 1999, Pavlović *et al*, 2025) Recent serological studies of screening of ascarid-infected humans for specific antibodies to *A. suum* conducted in Denmark, the Netherlands and Sweden yielded extremely high seroprevalence values (Nejsun *et al*, 2001; Vlaminck *et al*, 2014; Schneider *et al*, 2015). Numerous case reports from Japan have reported pulmonary, hepatic, and cerebral symptoms caused by *A. suum* larvae following ingestion of infected raw meat (liver) or vegetables contaminated with *A. suum* eggs (Maruyama *et al*, 1996; Inatomi *et al*, 1999; Izumikawa *et al*, 2011).

OTHER HELMINTHS

Trichurosis

Trichurosis is a chronic disease that usually does not lead to significant health disorders. It is

caused by the helminth *Trichuris suis*. The eggs embryonate in the external environment. After oral infection, the larvae first crawl into the mucous membrane, the intestines into nodules, which leave in 11 days and settle in the large intestine. Clinical symptoms include watery-mucous diarrhea, sometimes with blood, anemia and reduced progress (Pavlović *et al*, 1996, Pavlović and Rogożarski, 2017).

Oesophagostomosis

The causative agents of the disease are helminths *Oesophagostomum dentatum* and *Oe. longicaudatum*. Infectious larvae emerge from the eggs in the external environment in 6-8 days. Pigs become infected when they eat the larvae. After infection, the larvae enter the large intestine, where they burrow into the mucous membrane and form nodules. In 5-10 days, the larvae leave the nodules, go into the intestinal lumen and become sexually mature. (Pavlović and Rogożarski, 2017).

Strongyloidosis

This is primarily a disease of young pigs in the first months of life, mostly in farm conditions. The causative agent is *Strongyloides ransomi*. The genus *Strongyloides* is characterized by a particular developmental cycle, which can be homogenous or heterogenous. Infection occurs with larvae through the skin, colostral, placental and peroral, and adult parasites are found in the intestines of pigs. In older animals, the larvae reach the subcutis, transverse striated musculature, where they remain as dormant larvae. Immediately after birth, the larvae are activated and migrate to the mother's gland, so they are excreted in colostrum and milk. Excretion of larvae with milk lasts about 3 weeks. In oral infections (including lactogenic infection), larvae usually develop to adulthood in the small intestine without extraintestinal (pulmonary) migration. The pre-patent period for pigs is 3 days. There is also the possibility of prenatal infection through transplacental penetration of larvae into the fetus. High-intensity infections most often occur through colostrum and are characterized by bloody diarrhea, cachexia, anemia, dehydration and sudden death (Eysker *et al*, 1994, Pavlović and Rogożarski, 2017).

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