

SHORT REPORT: SECONDARY TRANSMISSION IN PORCINE CYSTICERCOSIS: DESCRIPTION AND THEIR POTENTIAL IMPLICATIONS FOR CONTROL SUSTAINABILITY

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Abstract. *Taenia solium* taeniasis/cysticercosis is one of few potentially eradicable infectious diseases and is the target of control programs in several countries. The larval stage of this zoonotic cestode invades the human brain and is responsible for most cases of adult-onset epilepsy in the world. The pig is the natural intermediate host, harboring the larvae or cysticerci. Our current understanding of the life cycle implicates humans as the only definitive host and tapeworm carrier (developing taeniasis) and thus the sole source of infective eggs that are responsible for cysticercosis in both human and pigs through oral-fecal transmission. Here we show evidence of an alternative pig-to-pig route of transmission, previously not suspected to exist. In a series of four experiments, naive sentinel pigs were exposed to pigs that had been infected orally with tapeworm segments (containing infective eggs) and moved to a clean environment. Consistently in all four experiments, at least one of the sentinel pigs became seropositive or infected with parasite cysts with much lower cyst burdens than did primarily infected animals. Second-hand transmission of *Taenia solium* eggs could explain the overdispersed pattern of porcine cysticercosis, with few pigs harboring heavy parasite burdens and many more harboring small numbers of parasites. This route of transmission opens new avenues for consideration with respect to control strategies.

During experiments devoted to standardize an experimental infection model for porcine cysticercosis, an unexpected infection was noticed in a control pig. This observation prompted us to house two sentinel piglets together with four piglets that were being infected with one proglottid each. All pigs were naive, 1-month-old animals. To rule out pen contamination during infection, experimental pigs were moved to a new, clean pen before hosting them with the sentinel pigs. Exposure length was 3 days, starting from the day of infection. Pigs receiving oral proglottid infections became seropositive and at necropsy demonstrated 189, 353, 499, and 601 cysts, respectively, most of them viable as proven by *in vitro* evagination. Both naive piglets seroconverted to EITB (enzyme-linked immunoelectro transfer blot—to cystic glycoprotein antigens)¹ positive with antibodies directed against 3 and 2 antigen bands, respectively. On necropsy, the 3-band piglet showed 3 degenerated and 3 healthy cysts that evaginated. No cysts were found in the 2-band piglet. This experiment and those described below were reviewed and approved by the Committee on Animal Research of the School of Veterinary Medicine, Universidad de San Marcos.

Further evidence supporting secondary infection came from another experiment in which two naive 1-month-old piglets (sentinel) were housed in the same pen with six other piglets of the same age that were being orally infected with one tapeworm proglottid each. All of the primary infected pigs became EITB positive with 3 bands each¹ and had 25, 86, 86, 118, 1,179, and 2,275 cysts on necropsy, respectively. Both sentinel piglets became seropositive between 6 and 8 weeks after exposure and were shown to have 10 (50% evaginated)

and 16 (60% evaginated) cysts at necropsy, respectively. These cysts were infective in hamsters.

A third experiment investigated the sows' potential to infect their piglets. Four sows with their litters were used in this experiment. One was a naturally infected sow, tongue positive (with palpable cysts in the tongue),² another was EITB positive/tongue negative, and the other two were naive EITB-negative sows. Dams had 3, 11, 9, and 3 piglets, respectively. All sows received orally two proglottids each when the piglets were 1 week old. Piglets from both seropositive sows were also EITB positive at baseline, presumably due to maternally transferred antibodies. Both seropositive sows remained seropositive; both seronegative sows became seropositive. Sows were not necropsied, but one of the initially seronegative sows died of other causes and was found to be heavily infected. Six weeks after infection, the piglets from the tongue-positive sow became seronegative, and none of the piglets from seronegative sows seroconverted. However, the three piglets from the seropositive, tongue-negative sow remained seropositive, and all three had cysts on necropsy. One piglet had one healthy and four degenerated cysts and the other two had 2 degenerated cysts each. Interestingly, petechiae-like lesions were found in the piglets from the tongue-positive sow and all piglets from the naive sows, which remained negative for EITB.

A fourth experiment was performed to characterize the time periods during which secondary infections occur. We used fourteen 1-month-old seronegative piglets from a cysticercosis-free farm and two other pigs infected with one proglottid each. Piglets were divided into four homogenous groups that were exposed at different times to the primary-infected pig: i) from Days 1 to 6, ii) from Days 7 to 12, iii) from Days 13 to 18, and iv) from Days 19 to 24. Piglets were humanely killed 6 weeks after exposure. As shown in Table 1, pigs exposed in the first 18 days after infection developed

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TABLE 1

Times of exposure and results of necropsy (6 weeks after initial exposure) of piglets exposed to second-hand infection

Group	Exposure dates (days postinfection)	Cysts at necropsy (values for individual pigs)
1	1 to 6	91, 31, 20
2	7 to 12	60, 0, 0
3	13 to 18	87, 10, 0
4	19 to 24	0, 0, 0

cysts. Figure 1 shows the macroscopical and microscopical appearances of a degenerated cyst recovered from a pig exposed to the primary infected pig between 13 and 18 days after oral infection. Second-hand infections appear to not only dilute the infective dose but may also dilate the time of infection.

Identifying and characterizing the sources and mechanisms of infection are important to the goal of developing interventions aimed at controlling and/or eradicating *Taenia solium*. Our data strongly suggest that a single dose of infective *T. solium* proglottid administered to an index, or primary, pig is capable of passing the infection to a second pig (secondary infection). Both egg dispersal and herd immunity may be related to secondary parasite infection. Also important, one of the experiments demonstrated that piglets can get infected with cysticercosis as early as 1 week of age, thus limiting the efficacy of control measures to be applied in older pigs.

Whether secondary infection is only attributable to coprophagic habits has yet to be demonstrated. Regardless of the mechanism that would eventually explain the phenomenon, secondary infection yields significantly lower burdens of infection. Studies with *Taenia hydatigena* and *Taenia ovis* in sheep and dogs demonstrated that immunity in the intermediate host is acquired after the ingestion of as few as 10 eggs, is life-long in the presence of eggs, does not depend on the presence of larvae from a previous infection, and can be lost between 6 and 12 months in the absence of eggs.³ Perhaps a missing feature in the human/pig *T. solium* cycle is another means to disperse eggs in a manner that immunity would become a density-dependent constraint and produce the aggregation shown in porcine cysticercosis. After all, it has been

suggested that endemic stability arises if the force of infection is high enough that acquisition of functional immunity occurs in the population at a relatively young age.⁴

Endemic stability describes a dynamic epidemiologic state in which clinical disease is rare in spite of a high incidence of infection within a population.⁴ The concept of "endemic stability," where the rate of transmission of infection is low enough as to not result in clinically apparent disease yet sufficiently high to immunize susceptible animals, has been an accepted epidemiologic concept for decades.⁵ A key observation that strongly suggests the endemic stability of *T. solium* is the ability of the parasite to return to original levels of disease after eliminating part of the tapeworm population—an event documented in Peru on two different occasions.⁶ The endemic stability of an organism is influenced by the organism's basic reproductive ratio and density-dependent constraints. Secondary transmission may potentially be a major contributor to this dynamic and deserves a closer look.

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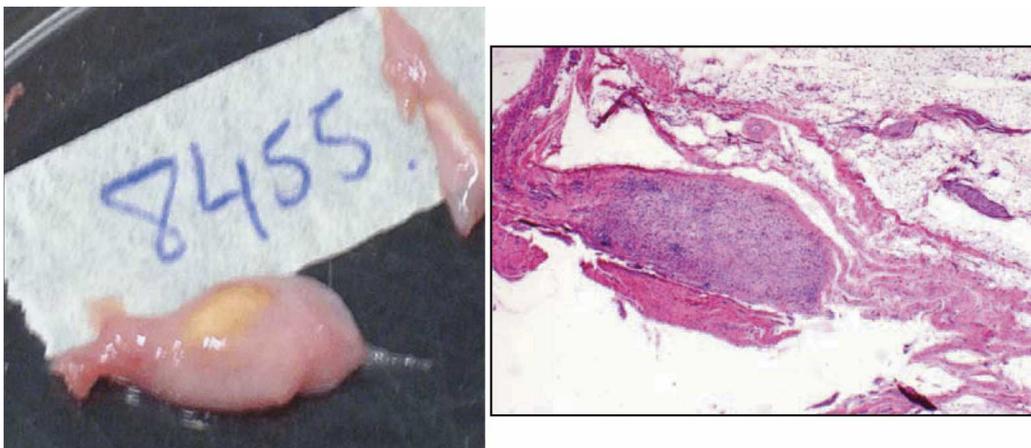


FIGURE 1. Degenerated cyst from a pig exposed to secondary infection on Days 13 to 18 after primary infection of an adult pig (right panel: H&E, ×100).

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