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EXPERIMENTAL INFECTION OF COLLARED PECCARY *(Dicotyles tajacu angulatus)* WITH SWINE KIDNEY WORM *(Stephanurus dentatus)*

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Abstract: Two captive-born juvenile collared peccaries (*Dicotyles tajacu angulatus*) were given 3000 infective larvae of *Stephanurus dentatus* per os. One peccary harbored viable *S. dentatus* sub-adults in the liver 50 days post-infection. The other peccary had no larvae but did have diffuse fibrotic hepatic lesions and bile duct hyperplasia 213 days post-infection; however, the lesions may have been partially due to a concurrent *Ascaris suum* infection. A domestic pig (*Sus scrofa domesticus*) infected as a control was severely but non-patently parasitized 170 days post-infection.

INTRODUCTION

A project was initiated in February, 1975 to investigate the susceptibility of the collared peccary (*Dicotyles tajacu angulatus*) to infection with swine kidney worm (*Stephanurus dentatus*). The possibility of peccary susceptibility was postulated during collection of feral swine (*Sus scrofa*) on the Aransas National Wildlife Refuge, Aransas County, Texas. Feral swine on the refuge are infected with this parasite and the question of infection in another suiform (*D. t. angulatus*) arose because peccaries also inhabit the refuge.[2,4]

Though taxonomically separated into different families, the peccary and feral swine have some ecological, physiological and behavioral similarities which warrant the examination of disease agents that may affect both species. Other taxonomically distinct species, such as cattle,, become naturally-infected with *S. dentatus*.[3,5] Experimental, non-patent infections have been successful in cattle,[1,6] rabbits, and guinea pigs.[6]

MATERIALS AND METHODS

Adult swine kidney worms and egg-laden urine were collected from three adult feral swine on the Aransas National Wildlife Refuge. The viable eggs were cultured using a modified technique described by Batte.[1] Approximately 200,000 larvae were cultured from a combined sample of one fluid liter of urine.

Two captive-born juvenile collared peccaries and one juvenile male domestic pig (*Sus scrofa domesticus*) were used as experimental animals. All animals were tranquilized with Ketamine hydrochloride to facilitate passage of a stomach tube for per os inoculation of six ml. of culture media containing an average of 500 viable larvae/ml. A 2.5 month-old Lancaster male domestic pig was used as a control to determine the viability of the larvae. The animals were housed separately in roofed welded-wire pens with concrete floors.

Blood samples were drawn from each animal at monthly intervals for total and differential leukocyte determination. The

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peccaries also were tranquilized for blood collection using a dart or intramuscular injection. They were euthanatized and examined at necropsy at 50 (peccary A), 170 (pig), and 213 (peccary B) days post-infection.

RESULTS

Peccary A, infected at four months of age, accidentally fractured the right femur during capture for blood collection and was therefore euthanatized at 50 days post-infection. The animal was not moribund prior to the trauma and the euthanasia. Significant gross lesions were limited to the liver and consisted of numerous white nodules ranging in size from 1.5 cm to 4.0 cm in diameter on the hepatic seroosal surface. Many nodules contained suppurative exudate and a gray, liquified debris. Sectioning the entire liver revealed 15 live *S. dentatus* larvae, 1.5 to 2.0 cm in length. Nodular foci were not present deep in the parenchyma but live nematode larvae were present. No helminth parasites were found in the digestive tract. Microscopic examination of the liver revealed numerosus microabscesses, many containing dead and decomposing nematode larvae. Live larvae in the hepatic parenchyma were surrounded by hemorrhage and eosinophils. A marked periportal infiltration by eosinophils and macrophages also was observed. A peribronchial lymphocytic aggregation and a mild interstitial pneumonitis was seen in the lungs.

The experimentally infected domestic pig remained in excellent body condition but was moderately stunted. The animal was euthanatized and examined at necropsy 170 days post-infection. Approximately 300 ml. of clear serous fluid was present in the abdominal cavity. The lungs contained miliary palpable nodules detected as slight parenchymal thickenings. The liver was pale, contracted, diffusely fibrotic and contained live and dead larvae of *S. dentatus*. The portal vein contained an occluding thrombus, which extended into the liver, that was composed almost entirely of *S. dentatus* larvae. Larvae also were present in the wall of the caudal vena cava immediately cranial to the liver, and in the perirenal tissue and renal medulla. The larvae measured approximately 2 to 2.5 cm in length. Microscopically, the lung contained diffuse verminous emboli, many of which were necrotic.

Peccary B, infected at three months of age, developed a rough hair coat and a distended abdomen at two months post-infection. Peccary B was euthanatized and examined at 213 days post-infection. Approximately 50 ml. of serous fluid was present in the abdominal cavity. Consolidated areas approximately 25 mm. in diameter were seen in the diaphragmatic lobes of the lung. Numerous fibrous adhesions were present between the diaphragm and the liver. White streaks were observed on the serosal surface of the liver. The streaks were more prominent at the periphery of the lobes. Thin sections of the liver revealed diffuse white fibrous streaks throughout the parenchyma, giving it a mottled appearance. No other gross lesions were observed; however, two adult *Ascaris suum* were found in the jejunum. Microscopically, portal fibrosis and bile duct hyperplasia were the most prominent hepatic lesions. Several foci of eosinophils were observed in the liver, but nematode larvae were not found. The diaphragmatic lobes of the lungs had thickened alveolar septae and contained numerous eosinophils.

In all three animals the number of eosinophils in the peripheral blood progressively increased, while the total numbers of leucocytes fluctuated from 4,000-20,000/mm^3. Previous relative normals established were 1 to 6% eosinophils. Eosinophilia in the domestic pig were 45%, while in the peccaries the relative increase was only 12%.

DISCUSSION

The primary objective was to evaluate the susceptibility of the collared peccary to swine kidney worm. Successful experimental infection of peccary A with a single, relatively low dose of L_3 larvae indicates that the collared peccary is susceptible.

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The pathogenicity of *S. dentatus* for peccaries as indicated by the lesions in peccary A, is not as severe as in the domestic pig. The lesions in the control domestic pig were similar to those of a natural infection.[2,7] Although live larvae were present in peccary A at 50 days post-inoculation, as in other successful experimental infections of unnatural hosts,[1,6] patent infections are unlikely.

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