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PORCINE NEONATAL COCCIDIOSIS IN CUBA

In 1934, Biester and Murray (Biester H. E., Murray C., 1934: J. Am. Vet. Med. Assoc. 85: 207–219) recognized pig diarrhea and enteritis with coccidia and identified new species of porcine coccidia as *Isospora suis*. After its original description, *I. suis* has been reported in many countries. In the late 1970s, veterinary diagnosticians recognized a distinct clinical and morphologic diarrheal disease in 1- to 2-week-old pigs, renewing interest in coccidiosis and rediscovering *I. suis* as an important disease of swine. The disease was reported in the USA (Sangster L. T., Seibold H. R., Mitchell F. E., 1976: Proc. Am. Ass. Vet. Lab. Diag. 19: 51–55; Stuart B. P., Lindsay D. S., Ernst J. V., Gosser H. S., 1980: Vet. Pathol. 17: 84–93; Eustis S. L., Nelson D. T., 1981: Vet. Pathol. 18: 21–28), Canada (Morin M., Robinson Y., Turgeon D., 1980: Can. Vet. J. 21: 65; Sanford S. E., Josephson G. K. A., 1981: Can. Vet. J. 22: 282–285), Scotland (Roberts L., Walker E. J., 1981: Vet. Rec. 108: 62), Belgium (Coussement W., Ducatelle R., Geeraerts G., Berghen P., 1981: Vet. Q. 3: 57–60) and Czechoslovakia (Koudela B., Vitovec J., 1984: J. Protozool. 31: abstr. No. 186). Porcine neonatal coccidiosis in tropical and subtropical zones has been identified only in Brasil (Lima J. D., Oliveria A. R. S., Martins N. E., Boretti L. P., 1983: Arg. Bras. Med. Vet. Zoot. 35: 33–40). Our present communication is based on search for *Isospora suis* infection in piglets in Cuba.

This study was carried out in Cuban large-scale piggerie (Complejo integral de cerdos Caonao) in October 1986. First, herd history was obtained and whole litters of piglets and their sows, including diarrheic animals, were sampled and the animals were individually marked so that the same animals could be examined over a number of days. This study was performed on 85 piglets (10 litters) 4–18 days of age, which were individually coprologically examined by floation using Sheather's sugar solution. Oocysts of *Isospora suis* were present in 38 samples of piglets (44,7 %) and oocysts of *I. suis* were not found in any of the samples from sows. Outbreaks of porcine neonatal coccidiosis were most often marked by the onset of profuse watery diarrhea in piglets. Only a portion of the litter was usually affected at one time and morbidity within litters varied from one pig to all piglets in the litter. The diarrhea generally lasted 2–4 days.

Two piglets containing oocysts of *I. suis* in faeces were killed and routine necropsies were performed. Samples for histopathological examination were collected immediately after killing. At first we took a specimen of ileum from a spot distant not more than 5 cm from ostium ileocecale. More specimens were collected to 15 cm, 50 cm and then at each point distant successively 50 cm from ostium ileocecale so that the last one was taken from duodenum. In the large intestine we took one specimen from apex or corpus of the caecum, two from the colon and one from the rectum. Specimens for the histology were also collected from liver, kidneys, spleen and mesenterial lymph nodes. As a part of post-mortem examination we took scrapings of mucosa from different portions of the gut, smeared them and stained by Giemsa to evaluate the incidence of endogenous stages of coccidia as recomended by Stevenson and Andrews (Stevenson C. W., Andrews J. J., 1982: Vet. Med. /SAC 77: 111–115). Samples from the gut and other organs were fixed in 10 % neutral formol and paraffin tissue section were cut using conventional methods. The sections were stained by hematoxylin-eosin and azur-eosin.

The colon contents from the piglets were placed in 2.5 % potassium dichromate aqueous solution, poured into petri dishes and incubated at 25 °C, 30 °C and 37 °C. Oocysts cultures incubated at 30 ° and 37 °C were examined every 4 h and those incubated at 25 °C every 6 h. Sporulated oocysts of *I. suis* were cleaned of fecal debris and enumerated for inoculation. Eight 3-day old piglets were inoculated via stomach tube with 200,000 sporulated *I. suis* oocysts. Two control piglets and infected piglets were observed daily for clinical signs of illness and for the shedding of oocysts. Experimental piglets were killed at various times after inoculation with oocysts and examined in the same way as spontaneously infected piglets.

The macroscopical lesions caused by spontaneous isosporosis manifest themselves as catharral enteritis. Microscopically, they consisted of more or less extensive atrophy of villi whose apical areas were covered by metaplastic pavement epithelium. Predilected was a portion of caudal sector of middle jejunum and the cranial sector of the caudal jejunum. Endogenous stages of *Isospora suis* found within the described morphological alteriation swere situated in parasitophorous vacuoules.

Endogenous stages of *I. suis* in smears prepared from mucosal scrapings matched with those detected in histological preparations from the respective sector of gut. Compared with histology, the method of mucosal scraings is simple and renders diagnostic results within a very short period of time after necropsy. Similar results were reported by Stevenson and Andrews (Stevenson C. W., Andrews

J. J., 1982: Vet. Med. SAC 77: 111–115).

The sporulation was completed within 8 h at 37 °C, 12 h at 30 °C and 36 h at 25 °C. In the present study, the sporulation times of *I. suis* oocysts were shorther, as reported by Lindsay (Lindsay D. S., 1982: J. Parasitol. 68: 861–865). The differences in sporulation times reported by different investigators may be due to the use of different isolates of *I. suis* and use of different laboratory techniques. The rapid sporulation of *I. suis* oocysts is probably an important factor in the spread of *I. suis* infections between litters of neonatal pigs in the farrowing houses. The temperatures between 32° and 36 °C in farrowing houses in Cuba create suitable enviroment for a rapid sporulation of *I. suis* oocysts.

No coccidia were seen in histological sections of intestine of piglets killed 6 h after inoculation. The piglet killed 48 h after inoculation had no large lesions and few coccidia were found in villous epithelium. Severe diarrhea developed in all piglets 60 h after inoculation and continued for 4 days. Villous atrophy and fusions were marked in middle jejunum, and focal erosions to necrosis of villi and adhered necrotic debris predominated in the caudal jejunum and ileum in the piglets killed 3 and 4 days after inoculation. Many endogenous stages of *I. suis* were seen in the epithelim of jejunum and ileum.

Oocysts of *I. suis* were at first in faeces of piglets 5 days after inoculation, prepatent period of *I. suis* was 120 h. Microscopically the piglets killed 9 days after inoculation had marked atrophy and increased lymphocytic and neurophylic infiltrate in the lamina propria of jejunum and ileum. Few endogenous stages of *I. suis* were observed. The lesions induced in this study are similar to the morphological observations in natural infection and are in agreement with the results of other authors (Stuart B. P., Lindsay D. S., Ernst J. V., Gosser H. S., 1980: Vet. Pathol. 17: 84–93; Robinson Y., Morin M., Girard C., Higgins R., 1983: Can. J. Comp. Med. 47: 401–407; Harleman J. H., Meyer R. C., 1985: Vet. Rec. 116: 561–565). The endogenous stages of *I. suis* in our experiment were similar to the stages in the study by Lindsay et al. (Lindsay D. S. Stuart B. P., Wheat B. E., Ernst J. V., 1980: J. Parasitol. 66: 771–779).

The results of this work confirm that *I. suis* may become a cause of neonatal diarrhea in piglets in Cuba and represents a new enteropathogen for piglets in this region.

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