Coccidioidomycosis (Coccidioides immitis) in the Collared Peccary (Tayassu tajacu: Tayassuidae) in Texas

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have low levels of total protein and gammaglobulins (Okoshi et al., 1968, Jap. J. Vet. Sci. 29: 337–345). In the cat, most immunoglobulins are transferred via colostrum, rather than the placenta (Okoshi et al., 1968, op. cit.; Schultz et al., 1974, Infect. Immun. 9: 391–393). Probably this cub did not receive adequate colostrum at birth; but whether this inhibited its ability to combat infection is unknown.

Aspergillosis in cats is primarily a bronchopulmonary disease (Bright, 1981, *In* Pathophysiology in Small Animal Surgery, M. J. Bojrob (ed.), Lea and Febiger, Philadelphia, Pennsylvania, pp. 335–349) and seldom involves the brain.

The portal of entry in this case for the central nervous system was probably via the nasal cavity, with direct extension into the brain. The bloody nasal discharge occurred before other respiratory signs, and the finding of a blood clot extending to the base of the brain from the nasal cavity reinforces this hypothesis. Pulmonary infection was probably simultaneous with the nasal infection, and therefore hematogenous spread from the lungs to the central nervous system cannot be dismissed. Rapid hematogenic dissemination does occur, at least in turkey poults exposed to aerosols of *A. fumigatus* spores (Richard and Thurston, 1983, Avian Dis. 27: 1025–1033). In humans with direct spread from paranasal sinuses to the CNS, most had signs of sinusitis before meningeal involvement was noted (Bhalla et al., 1980, Acta Neurochir. 55: 135–139; Mohandas et al., 1978, J. Neurol. Sci. 38: 229–233). The severe meningitis seen in this case points to a "break" in the normal CNS barrier (Rippon et al., 1974, Sabouraudia 12: 157–161), rather than just seeding by septic emboli, although both processes may have been involved.

The source of the infection in this case was unknown. The cub's littermate was unaffected. Whether this unusual infection was due to an overwhelming insult to an immature immune system, or to an immune deficiency, is also unknown. The documentation of hypogammaglobulinemia in snow leopards suggests that some may have an impaired immune function. However, even a normal cub might be susceptible to opportunistic mycotic infection after being temperature stressed, colostrum deprived, and treated with broad spectrum antibiotics. Nevertheless, the possibility of multiple immune deficiencies in this endangered species should not be overlooked, and warrants investigation.

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Coccidioidomycosis (*Coccidioides immitis*) in the Collared Peccary (*Tayassu tajacu*: Tayassuidae) in Texas

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Coccidioidomycosis is primarily a respiratory disease caused by the fungus *Coccidioides immitis*. The fungus thrives in soil (especially rodent burrows) and produces arthrospores which usually are inhaled by mammals, causing a primary lung infection. The disease has been reported in a variety of free-ranging and captive wild mammals and domestic

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mammals living in the Lower Sonoran Life Zone of the United States (Cornell et al., 1979, J. Wildl. Dis. 15: 373–378; Jungerman and Schwartzmann, 1972, Veterinary Medical Mycology, Lea and Febiger, Philadelphia, Pennsylvania, pp. 89–105; Maddy, 1954, J. Am. Vet. Med. Assoc. 124: 456–464). Only a limited number of individuals exposed to *C. immitis* in the endemic area actually develop clinical signs (Jungerman and Schwartzmann, 1972, op. cit.). Disseminated coccidioidomycosis, in which liver, spleen, kidney, and osseous tissues commonly are infected, occurs most frequently in horses and dogs while other species rarely show infection outside the thoracic cavity (Jungerman and Schwartzmann, 1972, op. cit.).

This report describes a single case of disseminated coccidioidomycosis and three cases of primary respiratory coccidioidomycosis in a herd of captive collared peccaries located in Brazos County, Texas. Coccidioidomycosis has not been reported previously in tayassuids. In the United States, collared peccaries inhabit the Lower Sonoran Life Zone in areas of Texas, New Mexico, and Arizona.

An outdoor enclosure housing 25 adult collared peccaries was constructed in 1981 at Texas A&M University. All animals were collected originally as wild adults from southern Texas and released into the captive herd at various times since its establishment. In late January 1984, female peccary R20 was observed exhibiting signs of a neurological-like disorder, prompting close monitoring of her behavior. During her illness R20 appeared disoriented and was largely unresponsive to external stimuli. Her head usually was lowered with snout just above the pen floor; she often salivated profusely. Signs developed rapidly during the first 2 wk following detection of the disorder. No respiratory difficulties were apparent, although an occasional dry cough was observed. R20 was seen running in circles on numerous occasions. Muscular tremors (anterior and posterior) sometimes were apparent, and convulsive seizures lasting 15–20 sec followed by a 5–10 min period of complete immobilization were noted twice. Locomotion was characterized by incoordination in hindlimbs and goose-step placement of forelimbs. R20 had been housed in a 2 m by 3 m pen with another female (R8) for 2 mo before developing the above signs.

A general antibiotic (Combiotic) was administered to R20 when clinical signs were first noted. Appetite was maintained despite the disorder; there was no loss of body weight and gross body condition appeared normal. Four wk after the first signs were noted, the animal was euthanized. Blood was drawn at this time, and a metabolic profile showed elevated enzyme levels, hypercholesterolemia, and slightly elevated phosphorus and total bilirubin levels (Table 1). R20 also had a mild leukopenia (7,400 cells/mm^3; 10,000–13,000 cells/mm^3 normal) which often occurs in infected humans (Swatek, 1975, *In* The Epidemiology of Human Mycotic Diseases, Al-Doory (ed.), Charles C Thomas Publ., Springfield, Illinois, pp. 74–102).

Female R8 was penned with female R20 for 3 mo but failed to show any of the clinical signs exhibited by R20. R8 and 11 other adult female peccaries from the captive herd (none exhibiting obvious clinical signs of the disease) were immobilized, blood drawn, and euthanized during late February 1984. Metabolic profiles of these 12 adults appeared normal (Table 1). All females were necropsied.

Gross examination of the lung surface in R20 revealed multiple granulomas containing purulent centers. Granulomas were light in color, slightly protuberant and 5–10 mm in diameter. Throacic lymph nodes were enlarged, but contained no grossly visible granulomas. Two discrete white granulomas 2 mm in diameter were visible on the surface of the left kidney; none were found on the right kidney. The brain

TABLE 1. Concentrations of serum metabolites in four captive female collared peccaries infected with *Coccidioides immitis*, and normals.

Serum constituent	Infected peccary no.				Normals* ($\bar{x} \pm$ SE)
	R20	R8	R31	R33	
Alkaline phosphatase (I.U./liter)	343	9	17	12	13 ± 2
Lactate dehydrogenase (I.U./liter)	4,360	846	811	785	1,261 ± 233
Aspartate aminotransferase (I.U./liter)	280	21	26	28	31 ± 3
Alanine aminotransferase (I.U./liter)	51	25	18	16	26 ± 2
Cholesterol (mg/dl)	207	115	77	123	108 ± 4
Phosphorus (mg/dl)	7.4	4.8	3.5	4.3	4.0 ± 0.2
Total bilirubin (mg/dl)	0.9	0.1	0.2	0.2	0.2 ± 0.01

* Lochmiller, 1984, Unpubl. Ph.D. Dissertation, Texas A&M University, College Station, Texas, 216 pp.

appeared grossly normal, with no evidence of a granulomatous meningitis which has been reported in some cases of disseminated coccidioidomycosis (Emons, 1963, Medical Mycology, Lea and Febiger, Philadelphia, Pennsylvania, 380 pp.). The liver was fatty and was severely infected with *Ascaris suum*. Specimens of *A. suum* were found throughout the small intestine. Ascarid infections were not severe in any other animal. Multiple granulomas also were present on the lung surface in three (R8 who was penned with R20, R31, and R33) of the other 12 females necropsied. Other organs appeared normal grossly.

Histologic examination of samples of kidney, lung, and spleen from R20 revealed spherical vegetative forms of a fungus identified as *Coccidioides immitis*. With hematoxylin and eosin stains, numerous basophilic staining, round to oval, double-walled spherules, some having undergone endosporulation, were discernible (Fig. 1). Most spherules measured from 10 to 60 μm in diameter and many contained granular material or numerous endospores (round or oval) 2–15 μm in diameter. The spherule's double wall measured 1.5 to 2.8 μm thick. Spherules in all stages of maturation were present, but small immature spherules which had not undergone endosporulation were most abundant. Neutrophilic infiltration was intense in infected tissues. Similar spherules were found only in lung tissue taken from R8, R31, and R33. Histologic examination of brain tissue from each female revealed no signs of infection.

The presence of *C. immitis* spores in four of 13 adult female peccaries is interesting since the Brazos County area of eastern Texas is thought to be outside the endemic range of the fungus (traditionally southwestern to western Texas). It is speculated that six wild-caught peccaries (four males, four females) obtained from the Chaparral Wildlife Management Area in southwestern Texas during September 1983 and subsequently released into the outdoor enclosure in Brazos County may have introduced the fungus to the captive herd. However, these introduced peccaries showed no clinical signs of the disease and two males possessed no visible lesions of *C. immitis* at necropsy. The four females showing infections of *C. immitis* were collected from southwestern Texas (within the endemic range) 2 yr previously (1981–1982), indicating that the disease might have been acquired after their release into the captive herd. However there is a possibility that these females had an infection of *C. immitis* when captured in the wild. In general, infected mammals are not considered to be direct infection threats to other mammals. However, feces, urine, and saliva of infected animals may

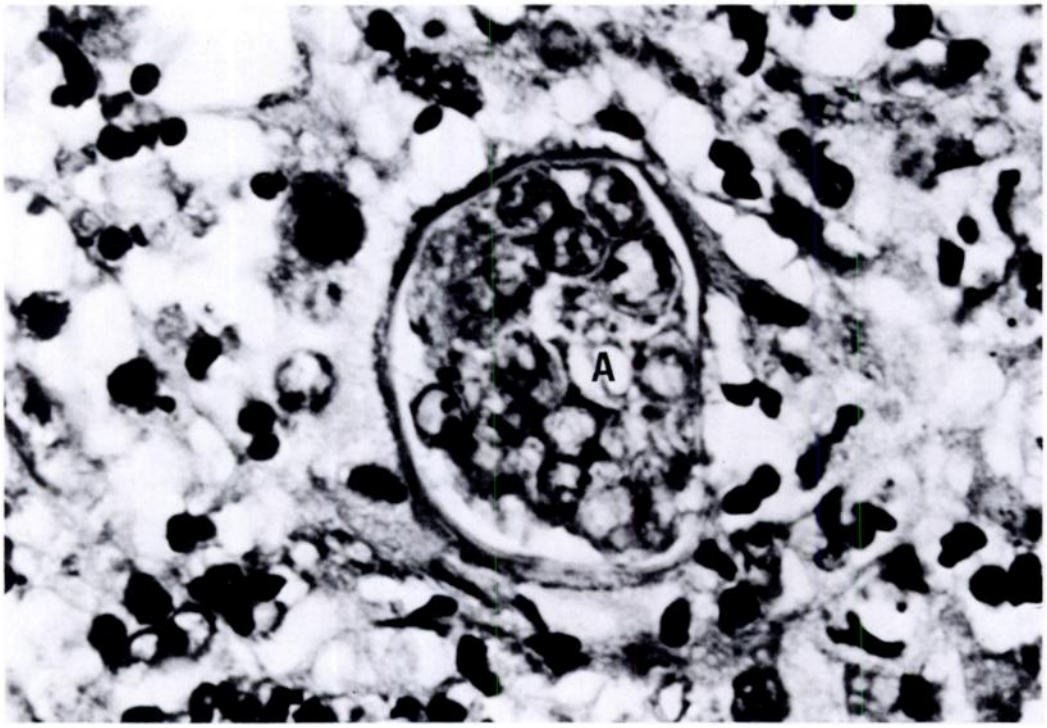

FIGURE 1. Sporulating spherule (A) of *Coccidioides immitis* in lung of a collared peccary. Hematoxylin and eosin stain, ×700.

contain sporangia which might produce mycelia and infective arthrospores when a suitable environment exists outside the body (Maddy, 1959, Vet. Med. 54: 233–242).

Signs of disease differed in many respects between this study and other reported cases of disseminated coccidioidomycosis. Loss of appetite and intermittent diarrhea leading gradually to cachexia have been described in dogs with disseminated coccidioidomycosis (Jungerman and Schwartzmann, 1972, op. cit.) but were not observed in peccaries. The muscle tremors and convulsive seizures have not been reported previously for other animals with dissseminated coccidioidomycosis, although the central nervous system is sometimes infected (Emmons et al., 1963, op. cit.).

Blastomycosis and histoplasmosis produce clinical signs of disease similar to coccidioidomycosis and the nonbudding form of these organisms (5–20 μm in diameter) resemble the small immature spherules in coccidioidomycosis. However, no budding spores were found in infected tissues of peccaries and the abundant endosporulation distinguished the disease organisms as *C. immitis*.

Wild collared peccaries live in close association with soil throughout the southwestern United States where *C. immitis* is endemic which suggests that contact with infected dust particles is quite prevalent. Digging or rooting with the snout occurs frequently and could greatly increase chances for inhalation of viable arthrospores in the soil. However it appears that only a few of the total number of peccaries exposed to *C. immitis* spores in the wild actually develop clinical signs of the

disease. This is supported in part by the absence of previous reports of coccidioidomycosis in peccaries, and the observation that only one of four infected females showed clinical signs in this study.

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Experimental Coccidiosis (*Isospora suis*) in a Litter of Feral Piglets

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Neonatal coccidiosis caused by *Isospora suis* is a serious problem in the swine production industry. Clinical signs of the disease consist of nonhemorrhagic diarrhea, weight loss, and dehydration in 5- to 14-day-old nursing piglets. Mortality is usually low to moderate. Microscopic lesions of villous atrophy, villous erosion and necrotizing enteritis have been reported from naturally and experimentally infected piglets (Stuart et al., 1980, Vet. Pathol. 17: 84–93; Eustis and Nelson, 1981, Vet. Pathol. 18: 21–28; Robinson et al., 1983, Can. J. Comp. Med. 47: 401–407). The present study was conducted to determine the responses of neonatal feral piglets to experimental infections of *I. suis*.

The feral sow and nursing piglets used in this study were the progeny of animals that were caught live on Ossabaw Island (Chatham County, Georgia). The population of swine on this island has perpetuated in a feral state for several hundred years (Brisbin et al., 1977, *In* Research and Management of Wild Hog Populations, G. W. Wood (ed.), B. W. Baruch Forest Science Institute of Clemson University, Georgetown, South Carolina, pp. 71–90).

Prior to farrowing and during lactation the sow was confined to a farrowing crate on an elevated plastisol-coated floor and fed water and commercial sow ration ad libitum. The sow farrowed six piglets which were used for the experimental studies. On the day of birth, each piglet received an intramuscular injection of iron-dextran solution (Nonemic®, Burns-Biotec, Laboratories Division, Chromalloy Pharmaceutical, Inc., Omaha, Nebraska 68103, USA) and was ear-notched for identification. When 3 days old, each piglet was inoculated via a stomach tube with sporulated oocysts of *I. suis*. Two piglets received 15,000 oocysts (Nos. I and II), two piglets received 50,000 oocysts (Nos. III and IV), and two piglets received 100,000 oocysts (Nos. V and VI). Piglets were observed daily for clinical signs of disease.

Six days postinoculation (PI), one piglet from each dosage group was killed by methoxyflurane anesthesia (Metofane®, Pitman-Moore, Inc., Washington Crossing, New Jersey 08560, USA). Sections of the upper and mid-jejunum, and lower ileum were removed, fixed in 10% neutral buffered formalin solution, and processed for histological examination. Mucosal smears were made from the mid-jejunum and examined for the presence of endogenous stages of *I. suis* (Lindsay et al., 1980, J. Parasitol. 66: 771–779) using Nomarski interference contrast microscopy.

Three days PI the piglets appeared somewhat inactive, but were otherwise

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