AN EPIZOOTIC OF BESNOITIOSIS IN CAPTIVE CARIBOU (RANGIFER TARANDUS CARIBOU), REINDEER (RANGIFER TARANDUS TARANDUS) AND MULE DEER (ODOCOILEUS HEMIONUS HEMIONUS)

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ABSTRACT: *Besnoitia* sp. was diagnosed in two caribou (*Rangifer tarandus caribou*) which died of pneumonia at the Assiniboine Park Zoo (Winnipeg, Manitoba, Canada) in 1983. During the following 3 yr besnoitiosis spread to an isolated herd of caribou, to mule deer (*Odocoileus hemionus hemionus*) and to reindeer (*Rangifer tarandus tarandus*). Reduction of exposure to biting insects appears to have reduced the transmission of besnoitiosis within the reindeer herd. The morbidity rate was approximately 82% in caribou and 67% in mule deer over the age of 2 mo. Most animals with clinical signs were euthanized; this precluded an estimation of the disease-related mortality rate. Twenty-eight caribou, 10 mule deer and three reindeer have been euthanized or died as a result of this epidemic. Attempts to artificially transmit the disease to potentially susceptible intermediate and definitive hosts were unsuccessful.

Key words: *Besnoitia* sp., besnoitiosis, caribou, reindeer, mule deer, *Rangifer tarandus caribou*, *Rangifer tarandus tarandus*, *Odocoileus hemionus hemionus*, epidemic, insect vector.

INTRODUCTION

Besnoitiosis is widespread among cattle (*Bos taurus*) in Africa, Asia, Europe and South America (Bigalke, 1967). Species of *Besnoitia* (Apicomplexa: Sarcocystidae) have obligatory heteroxenous life cycles, utilizing predator-prey relationships to ensure transmission (Frenkel, 1977). The intermediate host becomes infected by ingesting sporulated oocysts in feed or water contaminated with feces. The parasite then undergoes schizogony leading to cyst formation in connective tissue. Cysts, each within a single fibroblast, contain bradyzoites (infectious to definitive hosts) produced by endopolyogeny. The cysts are thick-walled and often become macroscopic. Following ingestion by the definitive host, bradyzoites undergo schizogony and gametogony in the intestine; sporogony is exogenous (Frenkel, 1977).

Several species of *Besnoitia* have been reported from various reptilian and mammalian intermediate hosts. Unfortunately, species designations within the genus *Bes-*noitia are unclear; most *Besnoitia* spp. have a broad range of intermediate hosts. Where life cycles have been completed (i.e., *B. besnoiti*, *B. darling* and *B. wallacei*), cats (*Felis* sp.) are suitable definitive hosts (Frenkel, 1977; Peteshev et al., 1974; Smith and Frenkel, 1977).

Cysts of *Besnoitia tarandi* occur in reindeer (*Rangifer tarandus tarandus*) and caribou (*R. tarandus caribou*) in Alaska, northern Canada, Scandinavia and the Soviet Union (Choquette et al., 1967). The definitive host of *B. tarandi* is unknown. The purpose of this study is to describe the occurrence of an epidemic of besnoitiosis (presumably caused by *B. tarandi*) in captive caribou, reindeer and mule deer (*Odocoileus hemionus hemionus*) at the Assiniboine Park Zoo (Winnipeg, Manitoba, Canada).

MATERIALS AND METHODS

Study sites

The Assiniboine Park Zoo kept two herds of caribou (herd A and B), a herd of reindeer and

a herd of mule deer at the time besnoitiosis was first diagnosed in August 1983 (Fig. 1). Caribou herd A consisted of nine animals and was separated from nine mule deer by a single page wire fence. Caribou herd B consisting of five animals was maintained in a holding corral approximately 100 m from herd A. The reindeer were maintained in a field approximately 20 m from caribou herd B. The number of animals in each herd varied during the time the observations were made due to new births. No animals were imported into these herds during this period; however, three caribou were moved from herd B to other zoos in the summer of 1985.

History of epidemic

Large numbers of encysted *Besnoitia* sp. were found in ulcerative lesions of the nasal and pharyngeal epithelium of two caribou which died of bacterial pneumonia in August and September 1983. One of these animals had extensive cutaneous lesions but skin lesions were not present in the other animal which died. Lesions characteristic of besnoitiosis were observed in one other caribou but these lesions did not appear to be progressive. In 1984 more than 50% of the caribou in herd A developed areas of alopecia and rugose hyperkeratosis typical of besnoitiosis; no mortalities occurred. Four of the six mule deer in the paddock beside caribou herd A developed extensive and progressive lesions of besnoitiosis throughout the summer of 1985. Fawns also were affected. Progression of lesions in young mule deer was more rapid than observed in mature animals. One mule deer fawn died of septicemia resulting from extensive cutaneous lesions in January 1986. The other nine members of the mule deer group were euthanized between November 1985 and February 1986 to prevent suffering and further disease transmission.

Skin lesions developed in caribou herd B in the fall of 1985 and throughout the summer of 1986. One animal was euthanized in January 1986 and six animals in March 1986. The six remaining animals of this group had clinical signs of besnoitiosis by September 1986. Three of these animals were euthanized and three animals were used for study of the disease. The remaining six caribou in herd A were euthanized in October and November 1986.

Two of 19 reindeer had early clinical signs of besnoitiosis when examined in October 1986. These animals were euthanized.

Three caribou had been moved to two other zoos from herd B in 1985. The single animal shipped to one of the zoos was euthanized in early 1986. Antemortem and postmortem lesions of this animal were consistent with those observed in caribou infected with *Besnoitia* sp.
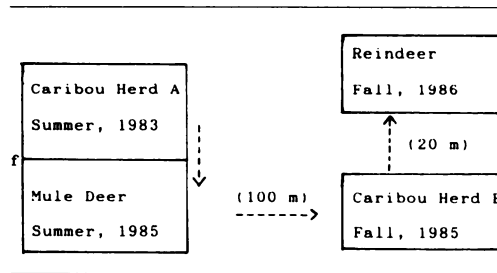

FIGURE 1. Sequence of spread of *Besnoitia tarandi* in the Assiniboine Park Zoo. The letter f denotes a single wire fence separating these groups.

The caribou sent to the second zoo were not euthanized despite having skin and ocular lesions of besnoitiosis. Subsequently, a mule deer death associated with *Besnoitia* sp. infection was reported at this facility.

Control procedures

Initially, we believed the transmission of *Besnoitia* sp. in caribou required ingestion of oocysts shed by a definitive host. The opportunity for transmission by this method is reduced within the zoo by (1) the zoo structure, which includes a complete chain link circumference fence in addition to individual enclosure fencing, (2) a continuous program of feral predator detection and removal using live traps and (3) husbandry techniques within the zoo such as daily cleaning of feed areas and monitoring for the presence of feral carnivores using live traps. Transmission by insect vectors was suspected when besnoitiosis was detected in caribou and reindeer maintained further than 100 m from the site of the initial disease occurrence. Supporting this hypothesis was the observation that most new clinical cases of besnoitiosis were diagnosed in the late summer and fall after the season when biting insects occur in this locality.

The first attempt to control this disease outbreak was based on clinical examination and euthanasia of affected animals. Clinical examination included a visual inspection of physically restrained animals for skin lesions and scleral cysts. In animals without clinical signs, the subcutaneous tissues of the lateral, midshaft metacarpal region was visually examined for characteristic cysts through surgical exposure under general anesthesia. When control was not achieved by the euthanasia of animals with besnoitiosis lesions detected by these procedures, all caribou and mule deer exposed to infected animals were euthanized.

Since 1987 the reindeer herd has been isolated in a fly-controlled barn during the fly season in an attempt to prevent further transmission of the disease. Reindeer calves of this group were

removed for hand-raising in a separate facility. The adult herd is being monitored for the development of new clinical cases.

Intermediate and definitive host studies

A study was designed to determine if several species of artiodactyle are susceptible to infection as intermediate hosts. Ten to 14 cysts containing bradyzoites of *Besnoitia* sp. were dissected free of surrounding caribou tissues under a stereomicroscope and suspended in heparinized blood from an infected caribou. This suspension was injected intravenously into one chinese water deer (*Hydropotes inermis*), one mule deer (*Odocoileus hemionus hemionus*), one white-tailed deer (*Odocoileus virginianus*), one Formosan sika deer (*Cervus nippon taiouanus*), one domestic sheep, and one domestic calf. These animals were euthanized 14 wk following exposure and tissues were examined for the presence of *Besnoitia* sp. cysts.

A balanced saline solution (0.9% Sodium Chloride Injection USP, Travenol Canada Inc., Mississauga, Ontario, Canada L4V 1J3) was used to suspend the cyst material for intraperitoneal injection into six laboratory mice, six laboratory rats and four domestic rabbits. A similar number of control animals of each species was housed with the experimental animals. An experiment also was performed to determine if the raccoon (*Procyon lotor*), domestic cat and arctic fox (*Alopex lagopus*) were potential definitive hosts of the species of *Besnoitia* involved in this epidemic. Approximately 150 g of fresh caribou tissues containing large numbers of *Besnoitia* sp. cysts were fed in one meal to two juvenile, zoo raised raccoons, two 8-mo-old house-raised cats and one juvenile zoo-raised arctic fox. Fecal samples collected from these animals at 48 to 72 hr intervals for 30 days post-exposure were examined by direct smears and flotation.

Feces were collected from the carnivores and rodents used in the experiments described above 7 days prior to exposure to *Besnoitia* sp. cysts. No protozoa were observed on direct smear and flotation (1.2 specific gravity sodium nitrate solution) preparations of feces samples.

Preparation of diagnostic specimens

Necropsy specimens were fixed in 10% neutral buffered formalin, sectioned at 6 μm and routinely stained with hematoxylin and eosin. Formalin-fixed reindeer nasal mucosa for electron microscopy was transferred to a 1% glutaraldehyde–4% formaldehyde solution (McDowell and Trump, 1976), washed in 0.1 M Sorensen's phosphate buffer pH 7.3, postfixed in 1% osmium tetroxide (in the above buffer), dehydrated in a graded series of alcohols and embedded in epon-araldite (JB EM Services Inc.,

Poite-Clare, Dorval, Quebec, Canada H9R 4S8). Thin sections were contrasted with uranyl acetate and lead stain; specimens were examined with a Hitachi 7000 scanning and transmission electron microscope (Nissei Sangyo Canada Inc., Rexdale, Ontario, Canada M9W 6A4).

RESULTS

Clinical signs

Lesions of besnioitiosis were similar in both mule deer and caribou but appeared to progress more rapidly in mule deer and in animals of both species less than 4 mo of age. Before cutaneous lesions became apparent, whitish 0.5 to 0.7 mm areas were observed on the ocular sclera (Fig. 2). These lesions were confirmed histologically to be cysts of *Besnoitia* sp. Although scleral cysts protruded above the surface of the sclera and often occurred in large numbers, they did not cause ocular irritation. Unless infected animals were examined closely these lesions were overlooked.

Gross lesions were usually first observed as areas of alopecia around the ocular orbit and on the dorsal aspect of the face (Fig. 3). The skin of the lower legs, particularly the areas overlying the distal metatarsal and metacarpal region, was affected early in the course of the infection (Fig. 4).

The areas of alopecia became rugose and appeared dry. Lesions progressed proximally on the legs, most severely affecting the areas overlying joints; crusting of affected areas followed. Eventually, the skin became indurated, inflamed and ulcerated; this resulted in epithelial loss, bleeding and the development of fissures. The skin of the muzzle also was affected (Fig. 5) resulting in partial obstruction of the nasal passages, respiratory stirdor and mouth breathing. Most animals were euthanized before lesions progressed beyond those described above. Ulcerative lesions developed on the legs and ventral aspects of the body of animals which were not euthanized.

Besnoitiosis was not observed clinically or on necrospy of animals <2-mo-old. Excluding animals <2-mo-old, the preva-
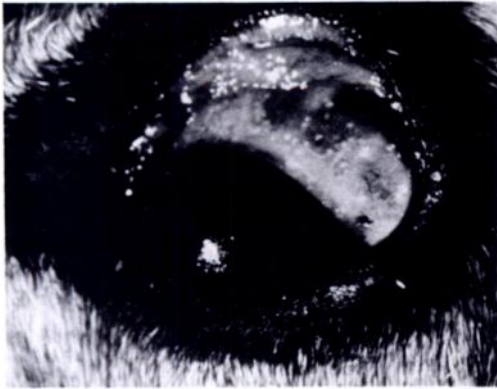
FIGURE 2.   Multiple *Besnoitia* sp. cysts on bulbar and palpebral conjunctiva of a caribou.

lence of clinical disease was 82% in caribou and 67% in mule deer (Table 1). Because most of the clinically affected animals were euthanized, an accurate disease related mortality rate cannot be calculated.

Gross pathology

Clinically affected animals had varying degrees of alopecia, primarily affecting the distal extremities, eyelids and periorbital skin and lips. The severity of the skin lesions varied from mild thickening with slight superficial scaling to marked thickening associated with hyperpigmentation, fissuring and epidermal ulceration with serous exudation and crust formation. Animals which had severe generalized exudative dermatitis were cachexic. Opalescent nodules, approximately 0.5 mm in diameter were present in the dermis and subcutaneous tissues including the panniculus, epimysium, peritendinous fascia and periosteum. Nodules in the tendon fascia and periosteum formed small pits or depressions in the underlying tendon and bone. Heavily affected areas had a coarse granular appearance typical of "corn meal" disease described in caribou (Fig. 6) as described previously by Choquette et al. (1967). Similar nodules were present on the sclera (Fig. 2), palpebral conjunctiva and the nasal turbinate mucosa (Fig. 7). Rarely, small numbers of cysts were present within the viseral serosa.

FIGURE 3.   Caribou with alopecia and rugose hyperkeratosis of periocular and nasal areas caused by besnoitiosis.

Within the group of animals necropsied, cysts were most consistently present in the subcutaneous tissues of the carpal and tarsal regions. Thorough ocular examination of the sclera of early cases of besnoitiosis was not performed precluding the calcu-

TABLE 1.   Incidence and severity of besnoitiosis in caribou (*Rangifer tarandus*) and mule deer (*Odocoileus hemionus hemionus*).

	Caribou (1983–1986)[a]	Mule Deer (1985–1986)[a]
Number of animals with		
lesions of Besnoitiosis	23	8
Mild[b] lesions	6	4
Moderate[b] lesions	15	0
Severe[b] lesions	2	4
Mortalities	1	3
Number of animals without		
lesions of Besnoitiosis	14	8
<2-mo-old	9	4
Total number of animals	37	16

[a] Time from the first diagnosis to the time of herd removal.
[b] Mild, scleral lesions only; Moderate, local alopecia; Severe, ulcerative skin lesions.
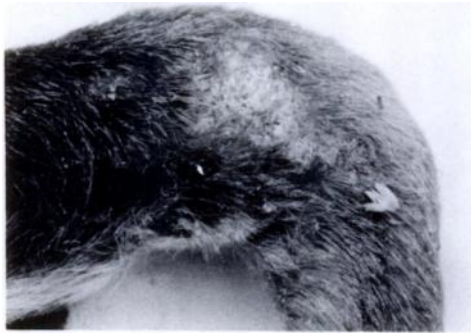
FIGURE 4. Mule deer showing early signs of besnoitiosis; hair loss on lower legs with hyperkeratosis and crusting.
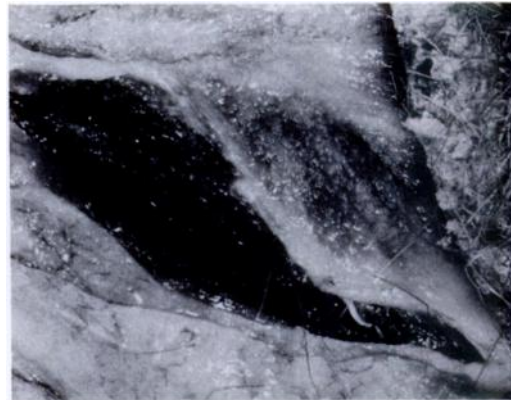

FIGURE 6. Large numbers of *Besnoitia* sp. cysts in the subcutaneous tissues and muscles of a caribou leg. This lesion resulted in besnoitiosis of caribou being referred to as "corn-meal disease."

lation of incidence of this lesion. Cyst numbers did not always correlate with the severity of skin lesions because massive numbers of subcutaneous cysts frequently extended to the pectoral region with minimal skin lesions.

Histopathology

The opalescent nodules observed grossly were thick walled spherical cystic parasitic profiles 240 to 373 μm in diameter. The cysts were composed of a four layered wall

typical of the genus *Sarcocystis* (Jubb et al., 1985; Terrel and Stookey, 1973) 14 to 56 μm thick enclosing hypertrophied and hyperplastic host cell nuclei and a central mass of closely crowded fusiform bradyzoites measuring approximately 1 × 3 μm. The outer wall segment was fibrillar condensed collagen, the intermediate segment pale staining and hyaline and the inner

FIGURE 5. Mule deer fawn with extensive facial lesions of besnoitiosis extending into the nares.

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FIGURE 7.   A granular appearance of the nasal turbinate mucosa of a caribou is created by massive numbers of *Besnoitia* sp. cysts.

basophilic layer was associated with the host cell nuclei. The primary cyst wall appeared as a thin inner membrane surrounding the central mass of bradyzoites. It was most easily visualized when separated from the cell cytoplasm by shrinking artifact. Both the absolute and relative thickness of the outer three layers (sec-

ondary cyst wall) varied with the cyst maturity. As cysts mature they increase in size, the cyst wall becomes relatively thinner and darker staining, host cell nuclei become more compressed and eventually are completely disrupted and replaced by a zone of streaming basophilic nuclear material as described by Basson et al. (1970).

The cysts were most numerous in the dermis and subcutaneous tissues. Rarely were cysts observed in parenchymatous organs and blood vessels. The inflammation associated with the cysts ranged from none to a chronic mixed inflammation of varying intensity. Plasma cells, lymphocytes and macrophages predominated with small numbers of eosinophils and globoid leukocytes. Giant cells were not observed in any of the sections examined.

Intermediate and definitive host studies

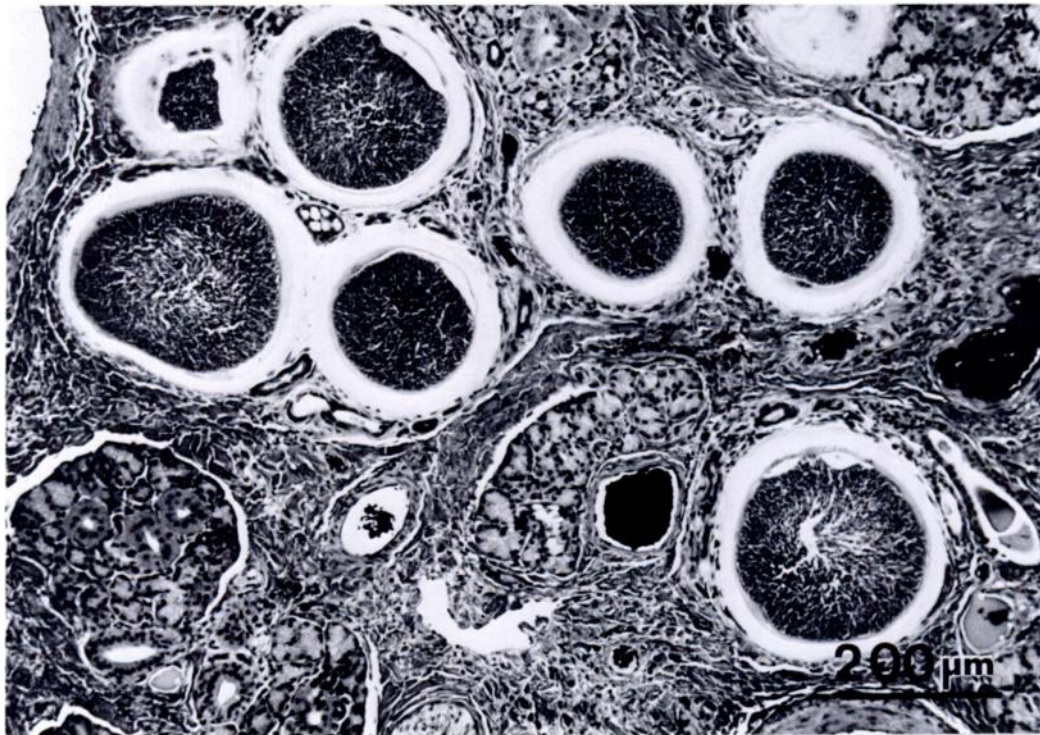No evidence of infection was found on necropsy of the test animals in the inter-

FIGURE 8.   Overview of caribou nasal turbinate mucosa containing numerous cysts of *Besnoitia* sp. H&E.

TABLE 2. Clinical examination record of the reindeer herd for the presence of scleral cystic structures.

| Zoo number | Date of examination[a] | | | |
	September 86	April 87	October 87	April 88
69	N[b]	N	S[c]	N
73	S	N	P[d]	P
79	P	N	P	P
81	S	N	S	S
83[e]	P			
85	N	N	S	N
86	P	P	S	P
87	N	N	N	N
90	P	P	P	P
95	N	N	S	N
101[e]	P			
107[e]	P			
108	N	N	N	N
115[f]	S			
118[f]	N			
119	N	N	N	N
122	N	N	N	P
124	N	N	N	N
126	N	N	N	N
127[f]	N			
(Offspring of 1987)				
128			N	N
129			N	N
130			N	N
132			N	N

[a] Consisted of visual examination of the ocular sclera.
[b] N, No cystic structures were observed.
[c] S, ≤3 cystic structures were observed on either sclera.
[d] P, >3 cystic structures were observed on each sclera.
[e] These animals had many ocular cysts on visual examination and were subsequently euthanized. Diagnosis of besnoitiosis was confirmed on necropsy.
[f] Caribou born in 1986 that died of causes unrelated to besnoitiosis.

mediate host experiment which were sacrificed 14 wk following exposure. Lesions of besnoitiosis also were not observed in the laboratory rodents examined at necropsy 12 wk after inoculation. Oocysts of *Besnoitia* sp. were not found in the feces of the raccoons, cats and the fox in the definitive host experiment that were fed caribou tissues containing *Besnoitia* sp. cysts.

Control procedures

Besnoitiosis has not been diagnosed in animals at the Assiniboine Park Zoo other than the caribou, mule deer and reindeer. Although none of the reindeer have developed progressive cutaneous lesions observed in the caribou and mule deer, six of the 17 animals currently in the herd do have scleral lesions of the disease (Table 2). Six offspring of the reindeer herd have been successfully hand-raised. None of these animals have developed any clinical signs of infection with *Besnoitia* sp.

DISCUSSION

The diagnosis of besnoitiosis was confirmed by the histological and ultrastructural features observed in mature cysts. These features include cyst formation in fibroblasts, formation of a primary cyst wall, hypertrophy and hyperplasia of the original fibroblast nucleus, bradyzoite formation (infectious to definitive hosts) and formation of a secondary cyst wall surrounding the parasitized fibroblast (Figs. 9 and 10) (Frenkel, 1977; Heydorn et al., 1984). Although the criteria differentiating the various *Besnoitia* sp. are not clear, the fusiform bradyzoites observed histologically were similar to those described for *B. tarandi* (Choquette et al., 1967) as opposed to the crescent-shaped bradyzoites described for *B. besnoiti* (Levine, 1973) and *B. jellisoni* (Frenkel, 1977). There is a need for more critical studies on transmission, immunology and ultrastructure in order to confirm the validity of previously described species of *Besnoitia* as has been done for the closely related genus *Sarcocystis* (Dubey et al., 1989).

Besnoitia tarandi has been reported in wild caribou in North America but has never been reported in an intermediate host species other than *R. tarandus*. Lesions of besnoitiosis in mule deer progressed more rapidly than lesions observed in caribou or reindeer. This could indicate that mule deer are aberrant hosts and less immunologically competent to respond to infection with the *Besnoitia* sp. involved.

The high prevalence of clinical disease in the caribou herd in this epidemic deviates from the epidemiological and clin-
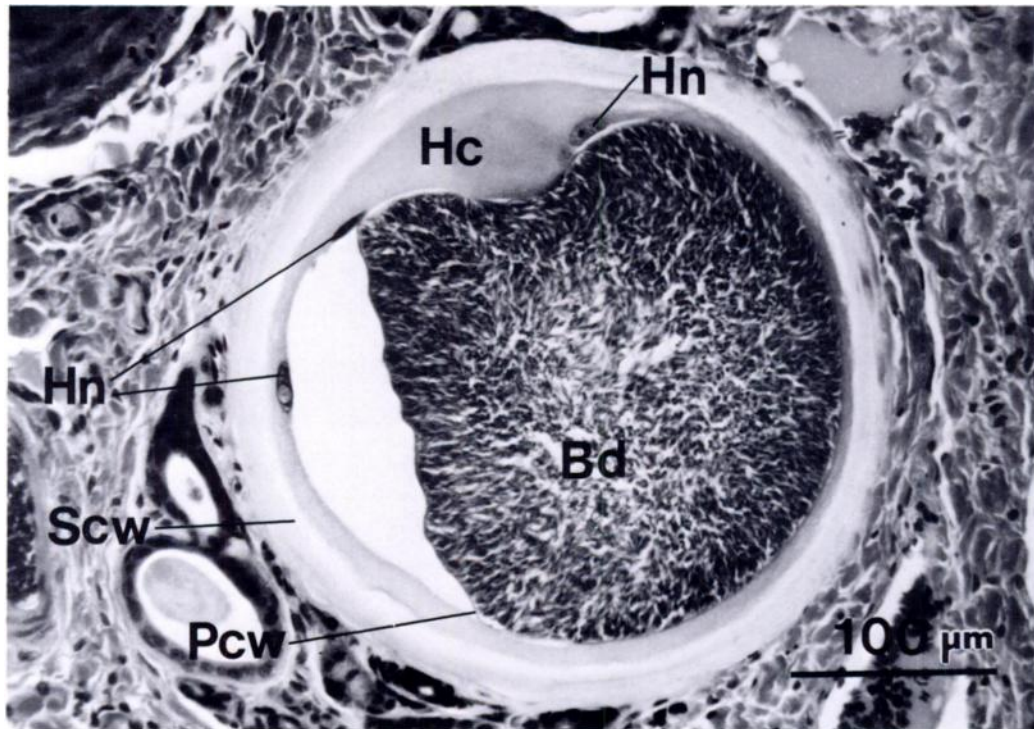
FIGURE 9.   Detail of a mature *Besnoitia* sp. cyst, in caribou nasal turbinate mucosa. Abbreviations: Bd, bradyzoites; Hc, host cell cytoplasm; Hn, host cell nucleus; Pcw, primary cyst wall; Scw, secondary cyst wall. H&E.

ical observations made previously in reports of *B. tarandi* in North America. Immunological naivete of the zoo animals may account for the severity of the disease. Alternatively, the species of *Besnoitia* causing this epizootic may be more virulent than the *Besnoitia* sp. endemic to the caribou herds of northern Canada.

Besnoitia tarandi of northern Canada, Alaska and northern Scandinavia is characterized by a chronic, relatively mild disease with a low prevalence of serious cutaneous lesions but large numbers of cysts in the subcutaneous fascia and periorbital areas (Choquette et al., 1967; Hadwen, 1922; Rehbinder et al., 1981). Besnoitiosis in reindeer in the Soviet Union (Nikolaevskii, 1961) causes serious cutaneous lesions and clinical disease.

Two strains of *B. besnoiti* occur in Africa. The viscerotropic *B. besnoiti*, occurring naturally in wild antelope in Africa,

is less virulent but immunologically indistinguishable from the dermatotrophic *B. besnoiti* which causes clinical disease in the domestic cattle from the same region (Bigalke, 1967). *Besnoitia besnoiti* cysts in antelope are infrequently found outside the cardiovascular system (McCully et al., 1966) whereas cysts in cattle are found most frequently in the skin of the legs and scrotum, in skeletal musculature and in mucous membranes of the nares, upper respiratory tract and larynx (Shultz, 1960). *Besnoitia besnoiti* infection of cattle in Africa rarely results in acute death and more commonly occurs as a chronic disease characterized by rugose hyperkeratosis, alopecia, scleroderma, nasal crusts and male infertility (Nobel et al., 1977).

Besnoitiosis may have been introduced into the Assiniboine Park Zoo by an inapparent carrier caribou. The increased incidence of animals with clinical signs of
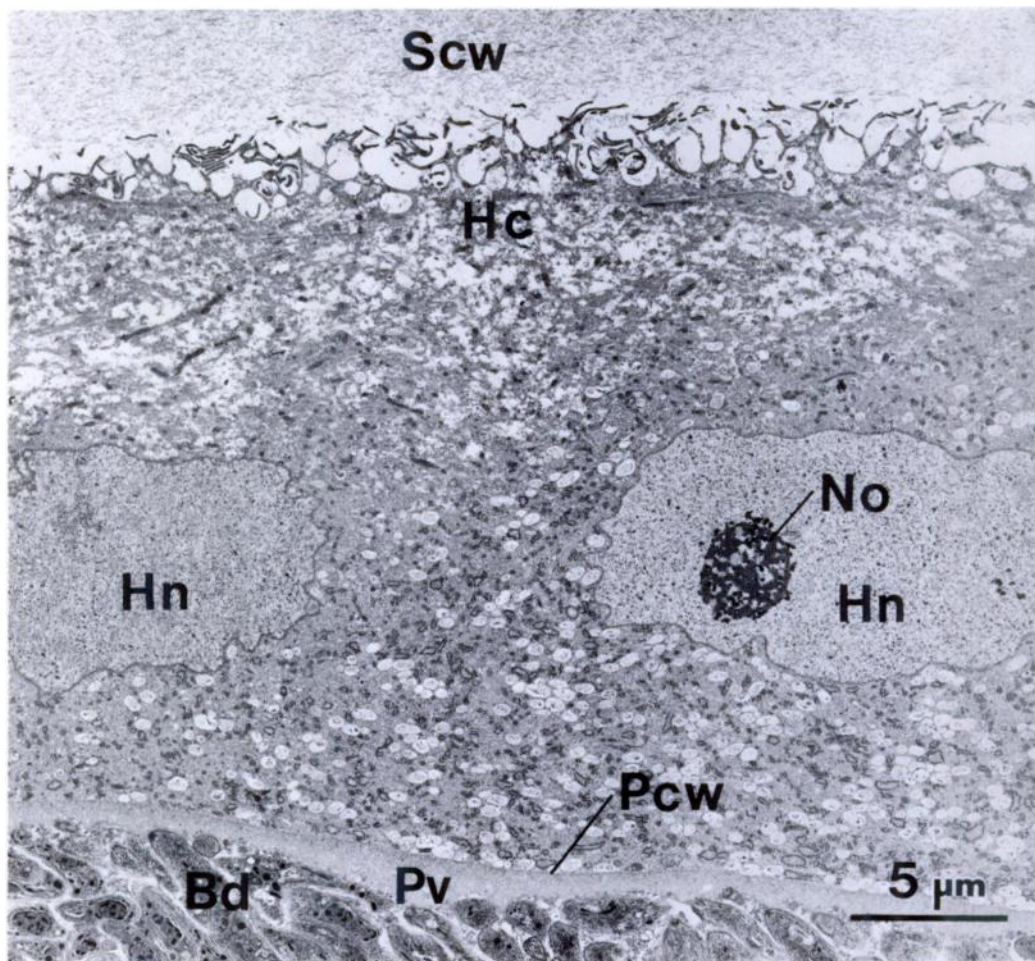
FIGURE 10.   Electron photomicrograph of a portion of a mature *Besnoitia* sp. cyst in caribou nasal turbinate mucosa. Abbreviations: Bd, bradyzoite; Hc, host cell cytoplasm; Hn, host cell nucleus, No, nucleolus; Pcw, primary cyst wall; Pv, parasitophorous vacuole; Scw, secondary cyst wall. Uranyl acetate and lead citrate.

besnoitiosis during the summer and the transfer of the disease to an isolated herd suggests that biting flies were the vectors of the etiological agent. Insect vector transmission of *B. besnoiti* has been observed (Bigalke, 1960).

Disregarding the possibility of other means of transmission between intermediate hosts, the rapid spread of besnoitiosis following the winter season when there are no potential flying arthropod vectors at the Assiniboine Park Zoo indicates that animals with besnoitiosis remain infectious for more than 7 mo. Two methods of arthropod transmission of besnoitiosis are sug-

gested. Bradyzoites of *Besnoitia* sp. may be transferred mechanically on the mouthparts of biting insects. Alternatively, the disease may be spread hematologically by blood-sucking insects. If hematological transmission occurs, carrier animals may be intermittently infectious when appropriate forms of the protozoan circulate in the blood vascular system such as (1) immediately after infection in the acute precystic period of the disease or (2) intermittently in chronically infected animals when infectious forms of *Besnoitia* sp. are released from mature cysts into the blood.

The failure to transmit besnoitiosis to

artiodactyles experimentally may have resulted from the transfer of insufficient infective material to establish an infection. Alternatively, the thick walled cysts transferred may have been isolated and inactivated by the recipients' immune system or perhaps the bradyzoite stage within the cysts was non-infective to the recipients. It remains unknown which species are susceptible to infection with this disease. This question should be considered before moving wild-caught animals to zoological parks and before relocating free-ranging caribou and reindeer to new geographical areas.

ACKNOWLEDGMENTS

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