

Munson L. 1993. Diseases of captive cheetahs (*Acinonyx jubatus*): results of the Cheetah Council Pathology Survey, 1989-1992. *Zoo Biology* 12(1):105-24.

Keywords: 3NAm/*Acinonyx jubatus*/captive breeding/captivity/cheetah/chronic gastritis/disease/feline infectious peritonitis/*Gastrospirillum*/glomerulosclerosis/*Heliobacter*/parovirus cysts/veno-occlusive disease/veterinary/VOD/zoo

Abstract: Knowledge of the diseases of cheetahs is essential to prevent and treat conditions that can modulate fertility and longevity. Toward this aim, a comprehensive pathology survey was conducted under a directive from the Cheetah Species Survival Plan. To date, 31 adult cheetahs and nine cubs from 16 zoological parks have been evaluated. Also, liver biopsies from 67 female cheetahs from 22 zoological parks were examined. Veno-occlusive disease (VOD) affected 82% of deceased cheetahs and 51% of live female cheetahs, and was the cause of death in nine cheetahs. Glomerulosclerosis and nephrosclerosis affected 84% and 39% of the population, respectively, and caused renal failure in eight cheetahs. The severity of VOD and glomerulosclerosis increased with age, and was not associated with infertility. Chronic gastritis was noted in 91% of the study population, and 95% of these cases also had spiral bacteria. Feline infectious peritonitis caused the death of two cheetahs. Male cheetahs had testicular degeneration, atrophy, and/or spermatogenic arrest, but these cheetahs also had severe systemic illness. Most females did not have reproductive tract lesions that would cause infertility, including those with parovarian cysts. Ovarian histology suggested that infertile cheetahs were not ovulating. Most cubs died from pneumonia or other systemic infections. The results of this study indicate that serious diseases are prevalent in the North American cheetahs, but these diseases do not appear to be the cause of infertility in the population. However, these diseases do limit the life span and well-being of cheetahs in captivity.

Diseases of Captive Cheetahs (*Acinonyx jubatus*): Results of the Cheetah Research Council Pathology Survey, 1989-1992

Linda Munson

Department of Pathobiology, College of Veterinary Medicine, University of Tennessee, Knoxville

Knowledge of the diseases of cheetahs is essential to prevent and treat conditions that can modulate fertility and longevity. Toward this aim, a comprehensive pathology survey was conducted under a directive from the Cheetah Species Survival Plan. To date, 31 adult cheetahs and nine cubs from 16 zoological parks have been evaluated. Also, liver biopsies from 67 female cheetahs from 22 zoological parks were examined. Veno-occlusive disease (VOD) affected 82% of deceased cheetahs and 51% of live female cheetahs, and was the cause of death in nine cheetahs. Glomerulosclerosis and nephrosclerosis affected 84% and 39% of the population, respectively, and caused renal failure in eight cheetahs. The severity of VOD and glomerulosclerosis increased with age, and was not associated with infertility. Chronic gastritis was noted in 91% of the study population, and 95% of these cases also had spiral bacteria. Feline infectious peritonitis caused the death of two cheetahs. Male cheetahs had testicular degeneration, atrophy, and/or spermatogenic arrest, but these cheetahs also had severe systemic illness. Most females did not have reproductive tract lesions that would cause infertility, including those with parovarian cysts. Ovarian histology suggested that infertile cheetahs were not ovulating. Most cubs died from pneumonia or other systemic infections. The results of this study indicate that serious diseases are prevalent in the North American cheetahs, but these diseases do not appear to be the cause of infertility in the population. However, these diseases do limit the life span and well-being of cheetahs in captivity. Further research is needed to elucidate the causes of these diseases. © 1993 Wiley-Liss, Inc.

Key words: veno-occlusive disease, glomerulosclerosis, gastritis, parovarian cysts, *Gastrospirillum*, *Helicobacter*

Received for publication August 5, 1992; revision accepted November 2, 1992.

Address reprint requests to Dr. Linda Munson, Department of Pathobiology, College of Veterinary Medicine, University of Tennessee, P.O. Box 1071, Knoxville, TN 37901-1071.

INTRODUCTION

The North American captive cheetah population has had poor reproductive performance and shortened life spans in comparison to other captive large felids or free-ranging cheetahs [Marker and O'Brien, 1989]. Because fertility and longevity are essential for a viable population, the future of cheetahs in zoological parks depends on reversing these trends. However, the cause of mortality and infertility must be known before strategies can be developed to improve captive breeding and management of cheetahs. Toward this aim, the American Association of Zoological Parks and Aquariums (AAZPA) Cheetah Species Survival Plan (SSP) Advisory Council selected a Cheetah Research Council in 1988 to investigate the biological basis of these problems in the captive cheetah population.

Because disease can modulate fertility and longevity, one mandate of the Cheetah SSP was to determine the diseases affecting captive cheetahs. At that time, information on most cheetah diseases was sparse and limited principally to causes of death. The genetic homogeneity of cheetahs was predicted to result in higher susceptibility to infectious diseases [O'Brien, et al, 1985], and, in fact, feline infectious peritonitis (FIP) and rhinotracheitis (feline herpes virus) had higher morbidity in captive cheetahs than domestic felids [Heeney, et al, 1990; Junge, et al., 1991]. However, no comprehensive survey of non-infectious health problems in captive cheetahs had been conducted. In order to define the types and prevalences of diseases affecting the captive cheetah population, a complete pathology survey of all cheetahs that died was proposed. Evaluating the prevalence of veno-occlusive disease in live cheetahs also was recommended, because veno-occlusive disease previously had been reported to be prevalent in deceased cheetahs [Munson and Worley, 1987; Gosselyn, et al 1989]. This report summarizes the results of this Cheetah Research Council Pathology Survey since its inception.

MATERIALS AND METHODS

Population

Deceased cheetahs. All United States zoological parks participating in the Cheetah SSP were requested to conduct complete necropsies on deceased cheetahs, and to provide a copy of the necropsy report and formalin-fixed tissues from all organs. Tissues of 31 cheetahs (13 males and 18 females) greater than 1 year old were received from 16 zoological parks. Reproductive tracts, but no other tissues, were submitted from 2 male and 3 female cheetahs. Samples of all organs were not available from all 31 subadult and adult cheetahs that died. The number of organ samples from individual cheetahs for this study were: 30 livers, 31 kidneys, 23 stomachs, 24 pancreases, 29 lungs, 27 spleens, 18 uteri, 13 ovaries, 10 testis, and 1 prostate. Tissues of nine cheetah cubs (1 to 16 days old) from 3 zoos were also received.

Breeding information was obtained on all cheetahs. Cheetahs then were designated as "fertile" if they had produced cubs, "infertile" if they had been housed with the opposite sex and had breeding opportunities, but had not produced cubs, or "undetermined" if they did not have the opportunity to breed. The study population contained 4 male and 3 female fertile, 8 male and 11 female infertile, and 1 male and 4 female undetermined.

All fixed tissues were embedded in paraffin, sectioned at 7 μm , and stained with hematoxylin and eosin (H&E). Sections of liver and kidney were stained with Masson's trichrome to delineate fibrosis, and sections of stomach were stained with modified Steiner's stain for spiral bacteria.

Live cheetahs. Liver biopsies were obtained from 67 female cheetahs at 22 zoological parks participating in the Cheetah Research Plan Reproductive Survey. Thirteen females were fertile, 29 infertile, and 25 undetermined. The cheetahs were anesthetized and three liver biopsies were obtained from the hepatic margins via laparoscopy as described [Wildt et al, 1993]. Two biopsy samples were fixed in Trump's solution, and one was frozen at -70°C . Fixed biopsies were embedded in paraffin, sectioned at 7 μm , and stained with H&E and Masson's trichrome.

Histopathological Analysis

All tissues were evaluated histologically for pathological changes. Specific diseases were defined and graded for severity as follows:

Veno-occlusive disease (VOD). Fibrosis surrounding or occluding hepatic sinusoids and central and/or sublobular veins of the liver. Grade 1: Mild fibrosis surrounding sinusoids or veins without occlusion. Grade 2: Moderate fibrosis surrounding sinusoids or veins with or without partial occlusion. Grade 3: Severe fibrosis surrounding sinusoids or veins with total occlusion of some or all veins.

Glomerulosclerosis (GS). Partial to total fibrosis or hyalinosis of renal glomeruli. Grade 1: Sclerosis of 1-3 glomeruli/three 100X fields. Grade 2: Sclerosis of 4-8 glomeruli/ three 100X fields. Grade 3: Sclerosis of >8 glomeruli/ three 100X fields.

Nephrosclerosis. Diffuse or locally extensive fibrosis or hyalinosis of the renal parenchyma. Locally extensive lesions were considered healed renal infarcts.

Glomerulonephritis. Changes involving 80% or more of the glomeruli, characterized by thickening of the basement membrane, increased cellularity of the mesangial matrix, and/or adhesions between glomerular tufts and Bowman's capsule.

Chronic interstitial nephritis (CIN). Inflammatory disease of the kidney, characterized by lymphocytes and plasma cells in the interstitium, and increased interstitial fibrosis.

Renal papillary necrosis. Coagulative necrosis of the renal papilla.

Pyelonephritis. Acute inflammatory reaction with neutrophils, fibrin, and necrosis, ascending from the renal pelvis to the cortex.

Gastritis. Acute or chronic inflammation of the gastric mucosa, characterized by necrosis of epithelial cells, infiltrations of lymphocytes, plasma cells, or neutrophils in the lamina propria and ulceration of the mucosa. Severity of the lesion was graded 0 to 3. Grade 1 gastritis consisted of mild inflammatory cell infiltrates and rare necrotic epithelial cells. In Grade 2 gastritis, inflammatory cell infiltrates filled the lamina propria and some dilated glands with necrotic cells were present. In Grade 3 gastritis, the lamina propria was expanded with inflammatory infiltrates, glands were dilated, many epithelial cells were necrotic, and erosions or ulcers were present.

Pancreatitis. Inflammation and necrosis of the pancreas, either acute or chronic.

Pancreatic atrophy. Loss of acinar cells or decreased zymogen granule content within lobules of the exocrine pancreas.

Pancreatic ductal ectasia. Dilation of pancreatic ducts with or without concentric fibrosis.

Bronchiectasis. Dilation of bronchi or bronchioles with or without associated fibrosis and inflammatory response.

Lipoma/myelolipoma. A benign mass of adipocytes without or with myelopoietic cells.

Feline infectious peritonitis (FIP). A systemic disease characterized by fibrinopurulent (acute) or pyogranulomatous (chronic) inflammatory reactions associated with blood vessels or serosal surfaces.

Testicular degeneration. Degeneration of advanced stages of spermatogenesis resulting in lack of spermatids, formation of spermatogenic giant cells, and sloughing of degenerate cells into the lumen. Affects some or all seminiferous tubules.

Testicular atrophy. Loss of spermatogonia and other components of the germinal epithelium, resulting in seminiferous tubules lined by Sertoli cells only.

Spermatogenic arrest. Arrest in developmental stages of the germinal epithelium, usually at the primary spermatocyte stage.

Determination of Cause of Death

Causes of death were determined by the severity of lesions in an individual cheetah, correlated with clinical pathology data (urea nitrogen and creatinine levels in serum) and clinical histories when available.

Statistical Analyses

Associations between lesions or between age and lesion severity were analyzed by Pearson correlation coefficients. Relationships between variables were assessed by least squares linear regression analyses. Chi-square contingency tables were used to determine the association between infertility and disease, and were analyzed by Fisher exact tests. For all analyses, the statistical significance was set at $P < 0.05$.

RESULTS

Cheetahs submitted for the survey represented 38% (31 of 82 cheetahs) of the subadult and adult cheetahs, and 35% (9 of 26 cubs) of the cubs less than one month old that died from January 1989 through May 1992. No tissues were received from the other cheetahs that died during that period. The majority of deceased cheetahs were less than 10 yrs old, and only 7 cheetahs were geriatric (15 yrs or older). The age distribution of this population is in Figure 1. Cub ages ranged from < 1 day to 16 days. The causes of death of subadult and adult cheetahs is in Table 1, and of cubs is in Table 2.

Diseases of Cubs

Pneumonia was the most prevalent disease in cubs, affecting cubs from 1 to 16 days old. Four of five cubs with pneumonia were being hand-raised, and in three of these four, the histological appearance of pneumonia was compatible with aspiration. One cub with pneumonia also had placentitis, and its littermate had septicemia.

Diseases of Subadults and Adults

Liver diseases. Veno-occlusive disease (VOD) was present in 82% of deceased cheetahs, and in 51% of live female cheetahs. The severity of VOD in these populations is depicted in Figure 2. In deceased cheetahs, the prevalence was comparable

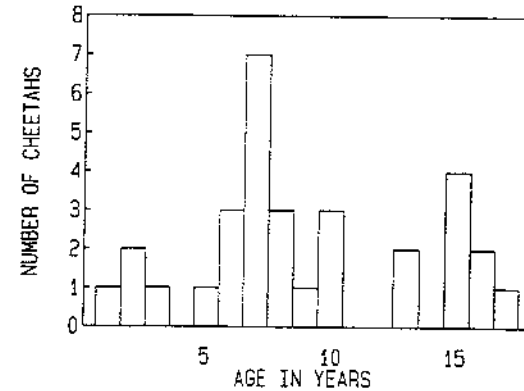


Fig. 1. Age distribution of subadult and adult cheetahs from North American zoos in the Cheetah Research Council Pathology Survey.

TABLE 1. Causes of death of subadult and adult cheetahs from North American zoos. (N = 31)

Cause of death	Number
Veno-occlusive disease	9
Glomerulosclerosis/Nephrosclerosis	8
Cagemate aggression	5
Anesthetic complications	3
Feline infectious peritonitis	2
Euthanasia/gastritis/papillary necrosis	1
Pyothorax	1
Enteritis	1
Rattlesnake bite	1

TABLE 2. Causes of death of cheetah cubs from North American zoos (N = 9)

Cause	No. of Cubs
Pneumonia	5
Septicemia	1
Enteritis	1
Undetermined	2

in both sexes, and the severity of VOD increased with age (Table 3). VOD was the sole cause of death in 9 cheetahs (30%), and contributed to the death of two other cheetahs with kidney disease. In live cheetahs, severe (Grade 3) VOD was less prevalent than deceased cheetahs, but the severity of VOD also increased with age (Table 4). The prevalence of VOD varied noticeably between zoos (Table 5).

Telangiectasia was noted in the livers of 6 deceased cheetahs (20%), and some cases had associated vascular thrombosis. There was no association between telangiectasia and VOD. Mild amyloidosis was noted in livers of 2 deceased and 3 live cheetahs. Lipomas/myelolipomas were noted in seven (23%) livers of cheetahs that died.

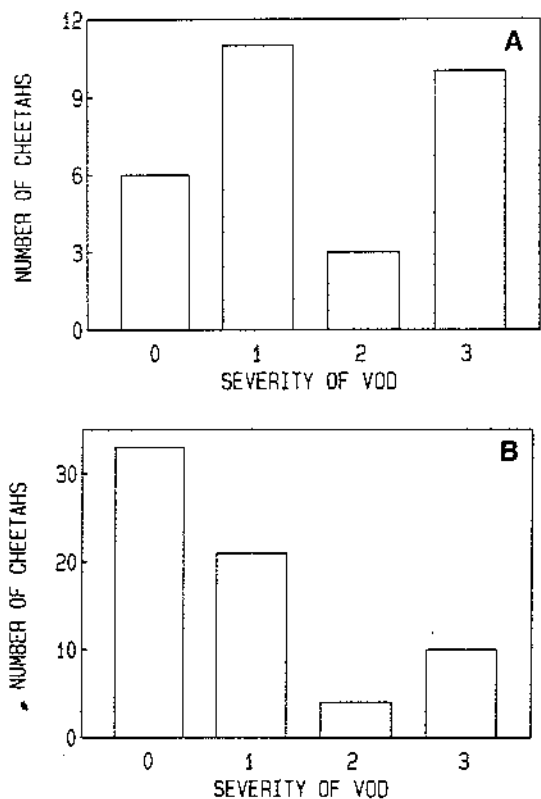


Fig. 2. Severity of veno-occlusive disease (VOD) in deceased (A) or live (B) cheetahs from North American zoos.

TABLE 3. Correlation* between severity of veno-occlusive disease (VOD) and age in deceased cheetahs from North American zoos (N = 31)

Age (in years)	VOD Grade 0 ^a	VOD Grade 1	VOD Grade 2	VOD Grade 3
1-4	2	1	0	0
5-8	3	5	1	4
9-12	1	2	1	0
13-17	0	3	1	6

* $r = 0.49$, $P < 0.01$.

^aSee text for description of grades.

Kidney diseases. Kidney failure due to acute or chronic kidney disease was the cause of death of 8 cheetahs (26%), and contributed to the death of 3 other cheetahs with VOD. The types and prevalences of renal disease in the study population are

TABLE 4. Correlation* between severity of veno-occlusive disease (VOD) and age in live female cheetahs from North American zoos (N = 67)

Age (in years)	VOD Grade 0 ^a	VOD Grade 1	VOD Grade 2	VOD Grade 3
1-4	24	6	0	0
5-8	8	13	1	4
9-12	1	2	2	2
13-17	0	0	1	3

* $r = 0.58$, $P < 0.0001$.

^aSee text for description of grades.

TABLE 5. Prevalences of veno-occlusive disease (VOD) in representative North American zoos

Zoo	No. of Cheetah	% With VOD	VOD Grade 0 ^a	VOD Grade 1	VOD Grade 2	VOD Grade 3
1	13	31	9	4	0	0
2	13	38	8	2	2	1
3	15	47	8	6	0	1
4	6	66	2	1	0	3
5	5	80	1	0	0	4
6	10	90	1	2	3	4
7	5	100	0	2	0	3

^aSee text for description of grades.

in Table 6. Kidney lesions were present in 90% of the population, and were moderate to severe in 52% of the deceased cheetahs.

Glomerulosclerosis was the most prevalent renal disease (Fig. 3). There was a significant association between the severity of glomerulosclerosis and age of the cheetah (Table 7). All cheetahs with nephrosclerosis had some degree of glomerulosclerosis, and in most of these cases the severity of glomerulosclerosis exceeded that of nephrosclerosis, suggesting that the lesion originated in the glomeruli. That glomerulosclerosis precedes nephrosclerosis also could be inferred from four cases that had mild glomerulosclerosis without any nephrosclerosis. Nephrosclerosis was mild in 4 cheetahs, moderate in 4, and severe in 4 cheetahs.

Membranous glomerulonephritis was the most common form of glomerulonephritis in the population (18 of 23), and all cheetahs with membranous glomerulonephritis also had glomerulosclerosis, including all seven cheetahs with grade 3 glomerulosclerosis. The material causing thickening of the glomerular basement membrane had staining characteristics of collagen with Masson's trichrome stain. This suggests that the membranous changes in glomeruli may be due to collagen and not immune complex deposits, and these changes may represent the earliest lesion in the progression toward glomerulosclerosis and nephrosclerosis.

Twenty-two cheetahs (92% of the cheetahs with VOD) had both VOD and glomerulosclerosis. Many cheetahs had severe degrees of both diseases. However, for the population as a whole, no correlation was apparent between the severity of VOD and glomerulosclerosis ($r = 0.23$, $P > 0.05$), and one cheetah with severe VOD had no glomerulosclerosis.

TABLE 6. Types and prevalences of kidney disease in North American cheetahs (N = 31)

Disease	Prevalence (% of population)
Glomerulosclerosis	84
Nephrosclerosis	39
Glomerulonephritis	74
Chronic interstitial nephritis	29
Healed infarcts	19
Interstitial amyloid	10
Pyelonephritis	6
Papillary necrosis	3

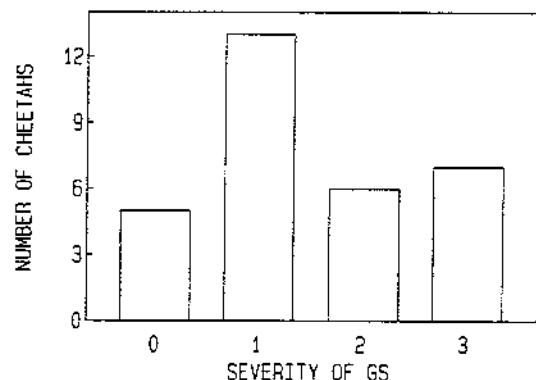


Fig. 3. Severity of glomerulosclerosis in cheetahs from North American zoos.

TABLE 7. Association* between severity of glomerulosclerosis (GS) and age in cheetahs from North American zoos

Age (in years)	GS Grade 0 ^a	GS Grade 1	GS Grade 2	GS Grade 3
1-4	2	2	0	0
5-8	2	7	1	3
9-12	1	1	1	1
13-17	0	3	4	3

* $r = .49, P < 0.01$.^aSee text for description of grades.

Four cheetahs had mild mesangioproliferative glomerulonephritis with adhesions, and only two of these four had mild glomerulosclerosis, suggesting that these lesions were not associated. One cheetah with mesangioproliferative glomerulonephritis had FIP.

Some degree of chronic interstitial nephritis was prevalent in the population. However, the pattern of inflammation suggested that the inflammation might be secondary to damage from glomerulosclerosis and not due to a primary kidney infection. For example, all cheetahs with chronic interstitial nephritis also had glom-

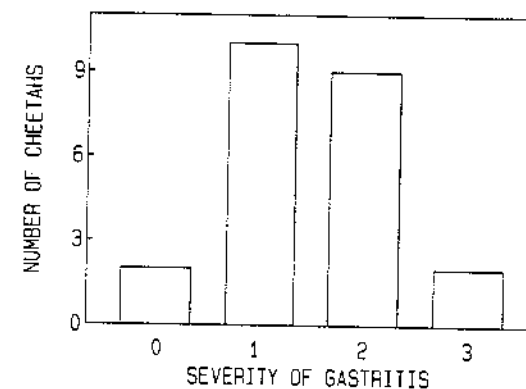


Fig. 4. Severity of gastritis in deceased North American cheetahs.

erulosclerosis, and the severity of glomerulosclerosis was greater than the degree of inflammation. Also, the inflammatory response in the kidneys was minimal and vascular changes were not noted except for cases with pyelonephritis and FIP. The possibility that nephrosclerosis was secondary to chronic interstitial nephritis was not likely, because 58% of the kidneys with glomerulosclerosis had no inflammation. Both cheetahs with pyelonephritis were females.

Bacterial gastritis. Gastric was present in 91% of the study population from 16 zoological parks, although in 10 of 21 cheetahs, the inflammatory reaction was mild, and not associated with ulceration (Fig. 4). Seven zoos had more than 1 cheetah with gastritis. Spiral bacteria (presumptive *Gastrospirillum* sp. or *Helicobacter pylori*) were noted in 95% of the cheetahs with gastritis, and in one cheetah with no gastritis. The one cheetah with gastritis but no bacteria, had been on a treatment protocol to eliminate gastric spiral bacteria. There was no association between the density of bacteria (histologically) and severity of gastritis ($r = 0.02, P > 0.1$).

Pancreatic diseases. One 16-year-old female cheetah had pancreatitis. Pancreatic atrophy was noted in 46% of the study population, but atrophic changes were mild in 9 of 11 cases. The 2 moderate cases of atrophy were geriatric animals (15 yrs). Pancreatic ductal actasia was present in 33% of the study population, and was associated with pancreatic atrophy in 6 of 8 cases. Fatty infiltration of the interstitium was a common incidental finding.

Diseases of the lungs. Mild chronic bronchitis was present in 7 cheetahs (24% of the lungs submitted to the study), and 4 cheetahs had bronchiectasis. Thirteen cheetahs (45%) had small multifocal aggregations of foamy macrophages in subpleural alveoli ("foam cell foci"). Grossly, these lesions appear as small (1-5mm) yellow or tan foci on the surface of the lung.

Lesions of the spleen. Splenic lipomas/myelolipomas were present in 17 cheetahs (63% of the spleens submitted to the study). Many spleens had multifocal irregular aggregations of adipocytes in the parenchyma. Seventy-eight percent of the cheetahs had lymphoid depletion at the time of death.

Feline infectious peritonitis. Two cheetahs that died had typical fibrinopurulent pleuritis, peritonitis, and vasculitis of multiple organs. Two additional cheetahs

had small multifocal granulomatous or pyogranulomatous reactions on pleural and peritoneal surfaces that are typical of chronic FIP, but these lesions were not the cause of death. One of these latter cheetahs had blunting and fusion of small intestinal villi, a lesion that is characteristic of chronic corona virus infection.

Lesions in the Male Reproductive Tract

All 10 testes had abnormal or absent spermatogenesis. Three cheetahs had atrophy of the germinal epithelium, two cheetahs had spermatogenic arrest, and four cheetahs had testicular degeneration. All three cheetahs with active spermatogenesis also had abundant degenerating germinal cells in some tubules. Notably, one 15 year old cheetah still had active spermatogenesis, although some degeneration also was present. Six cheetahs had considerable interstitial fibrosis, and this fibrosis was not associated with testicular atrophy in all cases. Infiltration of the testis interstitium with adipocytes was a common incidental finding. One cheetah had mild cystic hyperplasia of the prostate. The possibility that testicular lesions were due to significant diseases in other organs was examined. All cheetahs with fibrosis and from whom other tissue also were available also had considerable fibrosis of the liver and kidneys (severe glomerulosclerosis and/or VOD). All cheetahs with spermatogenic arrest, testicular degeneration or testicular atrophy also had moderate to severe forms of these diseases. However, one cheetah with normal spermatogenesis also had severe VOD, and one had moderate glomerulosclerosis.

Because spermatogenic arrest can be normal in seasonal breeders, the month of death was compared between cheetahs with normal and arrested spermatogenesis. All three cheetahs with active spermatogenesis died in January, but spermatogenic arrest also was noted in cheetahs dying during this period (1 in December, 2 in January, and 2 in April). Cheetahs with normal or arrested spermatogenesis were located geographically throughout the United States zoos at death.

Lesions and Histological Findings in the Female Reproductive Tract

Parovarian cysts were submitted from 6 females, and reported in the necropsy report of one other female. One cheetah with parovarian cysts was only 2 years old. Grossly the cysts were discrete fluid-filled structures, ranging from 1–15 mm diameter, and located in the ligament between the oviduct and the ovary. Histologically, cysts were lined by a cuboidal to flattened epithelium, were surrounded by a smooth muscle wall, and were filled with proteinaceous material. In no case did the cysts occlude the adjacent oviductal lumen, although the duct was compressed in some areas.

Histological findings in ovaries are summarized in Table 8. It is notable that both cheetahs with corpora lutea had produced cubs and that 8 cheetahs without corpora lutea or corpora albicans had been with males and had not produced cubs. Although some follicles were present in ovaries of the five cheetahs 12 years or older, no oogonia were noted in the sections examined, suggesting depleted germ cell reserves. One cheetah had cysts of the rete ovarii, and one had a small dysgerminoma.

The uterine horns of most cheetahs were narrow (0.5–0.8 cm diameter) and flattened, similar in appearance to uteri that have not had steroidal stimulation. In fact, the uteri of four cheetahs ranging from 6 to 14 yrs, were histologically similar to those of one- and two-year old cheetahs. However, the endometrial epithelia of

TABLE 8. Histological findings in ovaries of North American cheetahs (N = 13)

Cheetah	Age (in years)	FOL ^a	CL ^b	AF ^c
1	1	++	-	+
2	1	++	-	+
3	2	++	-	+
4	3	++	-	++
5	6	++	+	++
6	8	++	+	+
7	10	++	-	+++
8	11	++	-	++
9	12	++	-	+++
10	14	+	-	+++
11	15	-	-	++
12	15	+	-	++
13	17	+	-	+++

^aTertiary follicles.

^bCorpora lutea.

^cAtretic follicles.

most cheetahs with active folliculogenesis had a histological appearance characteristic of estrogen stimulation, indicating that steroidogenesis had occurred, and the endometria were responsive. Seven cheetahs had mild cystic endometrial hyperplasia consisting of a few hyperplastic dilated glands (N = 7), and two cheetahs had cystic endometrial hyperplasia that caused marked thickening of the endometrium (> 2 times normal). One of these cases of severe cystic endometrial hyperplasia had only a region of the uterus affected. Cheetahs with cystic endometrial hyperplasia ranged from 6 to 17 yrs, and all had been with males without producing cubs. Two cheetahs had leiomyomas of the myometrium. Another infertile cheetah had chronic endometritis.

Association of Diseases With Reproductive Success

Some zoos producing litters during 1989–1992 had lower prevalences of severe VOD than the population as a whole, or than zoos not producing litters, although one zoo with several cases of severe VOD also produced litters (e.g., Table 5, Zoo # 1, 2, 3, and 6 produced litters). There was no statistical difference between prevalences and severities of VOD in fertile and infertile cheetahs (Fig. 5) (all age groups had equivalent numbers of fertile and infertile cheetahs, so age was not biasing this statistic).

The presence or severity of glomerulosclerosis was not different between proven fertile and infertile cheetahs (Fig. 6). However, proven infertile cheetahs had more severe gastritis than fertile cheetahs (Fig. 7) and this difference was significant.

Only two males from which testis were available were fertile and only one of these had normal spermatogenesis. The other fertile male had spermatogenic arrest and testicular degeneration at death, but also had severe VOD and glomerulosclerosis.

Two of four fertile females had parovarian cysts. None of the fertile females had cystic endometrial hyperplasia or other lesions of the uterus.

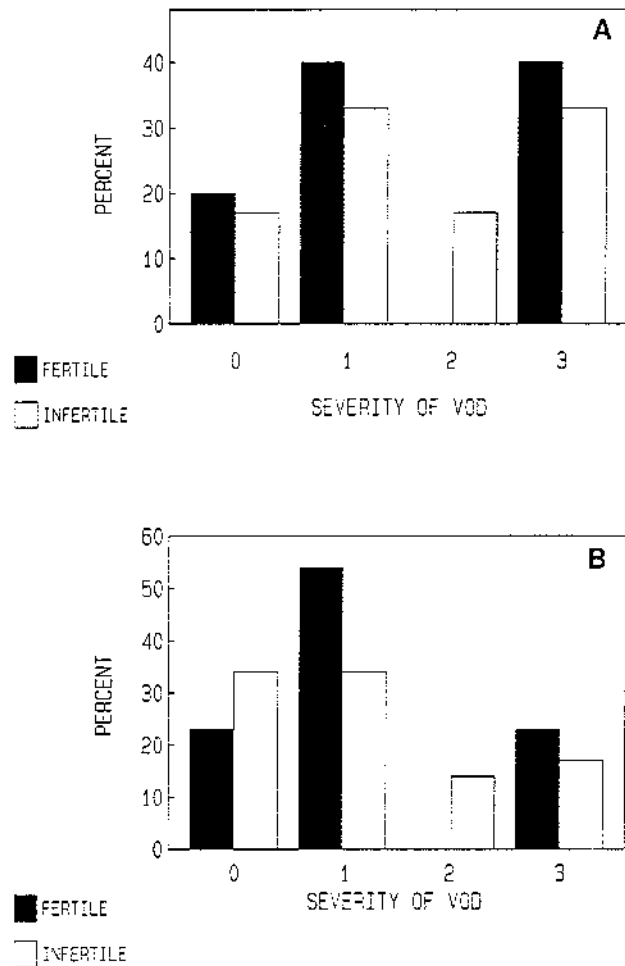


Fig. 5. The prevalence and severity of veno-occlusive disease in deceased (A) or live (B) North American cheetahs that were fertile or infertile.

DISCUSSION

Disease surveys in SSP-managed species provide valuable information for customizing the management and medical care of those species to maximize their survival. Knowledge of disease susceptibility is applied to quarantine procedures, pre-shipment serological screening, and nutritional management of that species. Disease surveys also focus veterinary medical research on critical unsolved questions in zoological medicine, and provide data for epidemiological studies.

This pathology survey has documented that three uncommon diseases, bacterial

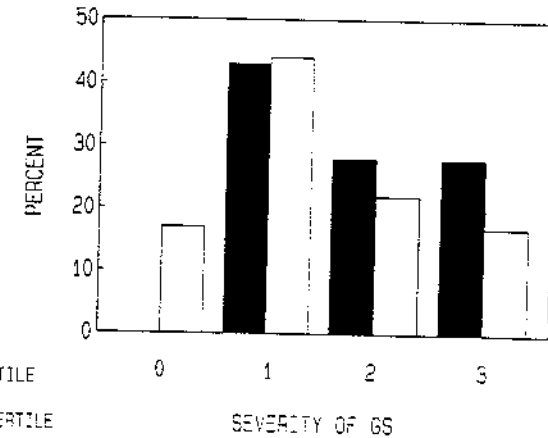


Fig. 6. The prevalence and severity of glomerulosclerosis in fertile and infertile North American cheetahs.

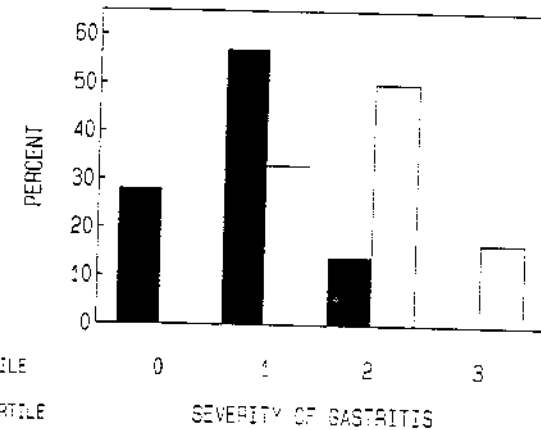


Fig. 7. The prevalence and severity of gastritis in fertile and infertile North American cheetahs.

gastritis, glomerulosclerosis/nephrosclerosis, and veno-occlusive disease, are highly prevalent in the North American cheetah population, and that these diseases are diminishing population viability by causing premature death of potential breeders. This data has revealed that bacterial gastritis is considerably more prevalent than previously assumed, and has confirmed that veno-occlusive disease and glomerulosclerosis/nephrosclerosis are the principal causes of death in captive cheetahs. The survey results indicate that most of these diseases do not directly impede fertility, but that they progress with age, thereby decreasing individual cheetah longevity.

The high prevalence of VOD in live female cheetahs of all ages in this study

provides further evidence that VOD is widespread in the North American cheetah population. The notable increase in severity of VOD with age in both live and deceased cheetahs suggests that VOD is a progressive disease. Earlier studies reported that VOD was prevalent in cheetahs that died [Dinnes and Henrickson, 1970; van den Ingh, 1981; Munson and Worley, 1987; Gosselyn, et al., 1988], but the prevalence in apparently healthy, reproductively active cheetahs was not known, and the progression of the disease could only be deduced. With liver biopsies now available from 68 cheetahs, the progression of this disease can be followed in the future.

Renal diseases have been known to be major causes of death in cheetahs [Marker and O'Brien, 1989], but these diseases have been poorly characterized pathologically. In this study, the most prevalent pathological process affecting the kidneys of captive cheetahs was glomerulosclerosis. The initial lesion appeared to be deposition of collagen along the basement membranes of glomerular capillary loops and in the mesangial matrix (membranous glomerulonephritis). In more advanced lesions, randomly distributed glomeruli had undergone hyalinosis, and were surrounded by fibrosis. Because nephrosclerosis was always noted in cheetahs with glomerulosclerosis, nephrosclerosis likely represents a progression of glomerulosclerosis in cheetahs, and will be considered the same disease in this survey. This is because the majority of the renal blood supply traverses the glomeruli, and sclerosis of the entire kidney would result from ischemia, if the glomerular vasculature was damaged or obstructed.

Glomerulosclerosis to the extent noted in this study is an unusual lesion in any species. Glomerulosclerosis occurs in some systemic diseases of humans, including vascular, metabolic, or immunologic diseases, and is a sequelae to chronic glomerulonephritis [Klahr, 1988]. It is interesting to note that VOD and glomerulosclerosis often were concurrent (84% of cheetahs with VOD had glomerulosclerosis, and most cases with severe glomerulosclerosis also had severe VOD), suggesting a common pathogenesis of these diseases. Both diseases appear to be caused by the progressive fibrosis of vascular spaces, which could be due to hypoxia from vascular damage and vasoconstriction. Nephrosclerosis is a common sequelae to chronic hypertension in humans [Robbins, 1989], but no vascular lesions typical of hypertension were noted histologically in these cheetahs. Both hepatic and glomerular vasculature could be susceptible to circulating toxins or vasoconstrictors (endogenous or exogenous), because they are exposed to the majority of the blood supply. Conversely, immune-mediated vascular injury could be the basis for these lesions. Unfortunately, FIP titers were not available from these cases to determine if these titers correlated with disease. In humans, glomerulosclerosis is common in infantile kidneys (up to 6 months old), but is considered abnormal if present to this degree after 6 months, because the affected nephrons atrophy ([Beckwith, 1992] in Sternberg, p. 674). In cheetahs, the lesion did not appear to be either a developmental anomaly or primarily an aging change, because no neonates were affected, and many affected cheetahs were 7 to 12 years old.

Because end-stage kidney disease can result from chronic interstitial nephritis or pyelonephritis, the inflammatory lesions of the kidney also were evaluated. Inflammatory lesions of the kidney were minimal except for two cheetahs with pyelonephritis, and one cheetah with FIP. This lack of notable inflammatory lesions in the population suggest that nephrosclerosis was not secondary to inflammation. In fact,

the inflammatory reaction noted in the study population may have been secondary to ischemic damage from glomerulosclerosis.

Chronic gastritis was noted at 16 zoological parks, indicating that this disease is very widespread in the North American captive cheetah population. Although spiral bacteria have not been proven to be the cause of gastritis unequivocally, the identification of bacteria by modified Steiner's stain in all but one affected case strongly suggest a causative relationship. One cheetah, however, had abundant gastric spiral bacteria without any gastritis, suggesting that the organisms are not pathogenic in all cheetahs. In an indepth study at one zoological park, *Helicobacter pylori* and *Gastrospirillum* sp. were cultured from cheetah stomachs with gastritis, and were observed in degenerating gastric epithelial cells by electron microscopy [Eaton, et al., 1992]. Because the pathogenicity of spiral bacteria may relate to the length of exposure, prospective studies documenting the progression of lesions in infected domestic cats are in progress (K. Eaton, personal communication). It should be noted that gastritis in this study was interpreted liberally, and included cases with mild lymphoplasmacytic infiltrates that might otherwise be considered normal. These cases were considered Grade 1 gastritis, because they represented the mildest form of a spectrum of inflammatory lesions noted in this study. Furthermore, modified Steiner's stains were necessary to identify organisms that were not evident in hematoxylin and eosin-stained sections.

Some cases of gastritis noted in this study were severe enough to have caused serious debility. Clinically, cheetahs with gastritis have chronic vomiting [Eaton, et al., 1992] and, in fact, one cheetah with moderate gastritis died from aspiration pneumonia due to regurgitation during anesthesia, and one cheetah with gastritis was euthanized because of severe debility from chronic vomiting. The results of the survey have serious implications for the captive cheetah population. Because the mode of transmission of spiral bacteria, susceptibility of the organism to heat, cold, light, or disinfectants, and species susceptibility are unknown, the potential health risks of moving known positive cheetahs to known negative facilities should be considered. However, at this time, no negative facilities have been identified. Treatment to reduce bacterial loads at one zoological facility have been successful, but are prolonged and expensive (Kramer L. and Wack R., personal communication).

Feline infectious peritonitis continues to cause morbidity and mortality in the cheetah population [Evermann, et al., 1991], but the prevalence of clinical FIP has dramatically decreased since the epizootic in 1982-83 [Evermann, et al., 1983]. In 1991-92, one zoo had several cheetahs seroconvert, and two cheetahs died of FIP after the introduction of cheetahs from another facility (M. Stetter, personal communication). This small epizootic reaffirms the need to screen cheetahs for FIP before movement. However, one of the cheetahs with clinical FIP had a consistently low positive titer, indicating that a rising titer does not always coincide with clinical disease. The health of the two other cheetahs in the survey with minor lesions, compatible with chronic FIP, was not likely compromised by the FIP infection at the time of death.

Pancreatic atrophy and ductal ectasia were prevalent in the study population, but likely did not affect the health of the cheetahs. In contrast, pancreatitis is life-threatening and was noted in one cheetah in this study and in other cheetahs not included in the study population (L.M., unpublished). Pancreatic ductal ectasia can result from chronic ductal inflammation, secondary to bacterial infections ascending

from the intestines. Pancreatitis can also result from ascending intestinal bacteria, and cheetahs with pancreatic ductal ectasia would be more susceptible, because the duct would not serve as a barrier.

Lipomas/myelolipomas of the liver and spleen were considered incidental findings, because the adjacent parenchyma was undamaged. These "tumors" appear to arise from aggregations of adipocytes, rather than from clonal expansion of a neoplastic adipocyte. Therefore, they likely represent a hyperplastic, rather than a neoplastic, lesion. They are significant only in that they are radiolucent and hypoechoic, and must be distinguished from clinically significant diseases such as tumors or abscesses. Adipocyte infiltrations are also common findings in the pancreas and testis of cheetahs, and likely are a normal species anatomical variation.

Hepatic amyloidosis and telangiectasia in the liver were not likely clinically significant, because the lesions were mild, and involved only small portions of the parenchyma. Foam cell foci in the lungs also were minor lesions that are common incidental findings in most species of cat. Most cases of bronchitis were mild, except for one cheetah that had a history of chronic mycoplasma bronchitis. Bronchiectasis could be a sequelae to chronic bronchitis, although only one cheetah had both lesions at death.

Associating disease conditions at death with reproductive failure during life has serious limitations, because the disease may not have been present during the period of normal productivity. This could be true for the association between gastritis and infertility noted in this study, although it is possible that chronic gastritis modulated reproductive functions through decreased intake of essential nutrients. It is more notable that many of the cheetahs with proven fertility also had severe VOD and glomerulosclerosis, because these diseases could affect the metabolism and excretion of hormones and toxins that modulate reproductive functions.

Correlating infertility with lesions in the testes of cheetahs with chronic diseases is inappropriate, because normal spermatogenesis is disrupted by most systemic diseases. Testicular degeneration occurs initially, but is succeeded by spermatogenic arrest and atrophy. All the testicular lesions noted in this study could be due to the other diseases in these cheetahs. It is improbable that these testicular lesions are the basis for high numbers of abnormal spermatozoa in cheetahs [Wildt, et al, 1983]. Spermatogenic arrest does not appear to be a normal physiological change in cheetahs, because both active and arrested spermatogenesis occurred during the same season in North American cheetahs from a wide geographic range. Most cheetahs with testicular atrophy and interstitial fibrosis were less than 10 years old, suggesting that these lesions were not aging changes, as is true in other species [McEntee, 1990, Robbins, et al. 1989]. It is interesting to note that two cheetahs with notable interstitial fibrosis also had fibrosis in the liver and kidney interstitium. Severe interstitial fibrosis could affect Leydig cell production of testosterone, which, in turn, could disrupt normal spermatogenesis. Conversely, low levels of testosterone can result in spermatogenic arrest, testicular atrophy, and eventually interstitial fibrosis.

The histological findings, that parovarian cysts did not obstruct the oviducts of female cheetahs, should allay concerns that these cysts interfere with fertility. These cysts are common incidental findings in all felid species, and are not associated with infertility. The cysts develop in remnants of the Wolffian ducts (mesonephric duct) that did not completely regress during embryogenesis [McEntee, 1990]. Because these ductal remnants are blind-ended, and are lined by a secretory epithelium, they

enlarge gradually until the smooth muscle wall restricts further expansion, and the epithelium atrophies from compression. Cysts are also more obvious in older females, because of the gradual fluid accumulation. Multiple cysts are common.

Uteri of most cheetahs in the study population were small in comparison to other felid species of similar ages, and cheetahs had less prevalent and less marked cystic endometrial hyperplasia than most other felid species [Munson, 1991]. Because uterine development and cystic hyperplasia are processes under steroid hormone control, this data provide indirect evidence that ovarian steroidogenesis in cheetahs is minimal. All but two cheetahs with cystic endometrial hyperplasia had mild lesions that should not have interfered with fertility. Also, the absence of cystic hyperplasia in females that had produced cubs supports data from other species, indicating that endometrial remodeling during pregnancy prevents these lesions [McEntee, 1990]. Except for those cheetahs with severe endometrial hyperplasia, and the cheetah with chronic endometritis, there were no findings to support that infertility in cheetahs is due to uterine diseases. In fact, most of the lesions noted (juvenile uteri, abundant atretic follicles, and cystic endometrial hyperplasia) would be the consequences, not the causes, of infertility.

Folliculogenesis was evident histologically in most ovaries, but the presence of tertiary follicles may not necessarily indicate that steroid synthesis was adequate for normal reproductive function. In fact, vaginal cornification (which is an indicator of estrogen activity) was not present in one cheetah with numerous secondary and tertiary follicles. The absence of corpora lutea or luteal scars in the ovaries of most cheetahs in this study indicates that ovulation had not occurred. Also, remnants of atretic follicles were abundant in most ovaries, and these are the morphological "footprints" of follicles that did not ovulate. If cheetahs are induced ovulators, these findings may reflect lack of breeding activity. Collectively, these data indicate that there is no anatomical basis for infertility in female cheetahs, and that folliculogenesis is occurring in many cheetahs that have failed to show estrus or breed.

Infections caused death in most cubs, and the character of the inflammatory response suggested that bacteria were the cause. Pneumonia appeared to have resulted from aspiration of milk in some cases. Two cubs had placentitis, indicating that they had been infected in utero.

Research Priorities

Several areas requiring further research were identified by this survey.

1. The cause of veno-occlusive disease needs to be identified. Research on the pathogenesis of this disease may assist in identifying the causative agent or condition.
2. The pathogenesis of glomerulosclerosis and nephrosclerosis needs elucidation. Studies further characterizing these lesions by electron microscopy are in progress, and may direct future research efforts to identify the cause.
3. The source and mode of transmission of gastric spiral bacteria should be determined and measures taken to reduce bacterial loads and transmission. Studies to fulfill Koch's postulates are also needed, and are in progress.
4. Serological monitoring the population for FIP should continue. All epizootics should be investigated for source and biotype of virus.
5. Additional ovaries from cheetahs should be evaluated to further our under-

standing of their failure to cycle. Morphological studies should be correlated with endocrinological studies when possible.

6. Centralization of all cheetah pathology data should continue to monitor changes in disease prevalences and emergence of new diseases.

7. Diseases of free-ranging cheetahs and of captive cheetahs in successful breeding facilities should be compared to the results of this survey to determine if these diseases are unique to cheetahs or are diseases of captivity.

CONCLUSIONS

This survey identified major and minor health problems in the North American cheetah population.

1. Glomerulosclerosis/nephrosclerosis are the main causes of renal failure in cheetahs. The earliest lesion is hyalinization and deposition of collagen in glomeruli, then progressive fibrosis of glomeruli and the renal interstitium.

2. Venocclusive disease is prevalent in live and deceased cheetahs, and appears to progress with age. The prevalence and severity of VOD differs between zoological parks, a factor that may aid in identifying the cause. Most cheetahs with VOD also had glomerulosclerosis.

3. Gastritis associated with spiral bacteria was identified in 16 zoos, and was severe enough in some cases to cause debility.

4. Pyelonephritis, papillary necrosis, membranoproliferative glomerulonephritis, and chronic interstitial nephritis also cause kidney disease in the population.

5. Feline infectious peritonitis was confirmed to cause the death of two cheetahs, and reported to cause death in one other during the 3.5-year survey.

6. Folliculogenesis was evident in most female cheetahs, but the reproductive tracts lacked morphological evidence of estrogen stimulation. Corpora lutea and luteal scars were noted in only two fertile females, indicating that the majority of the population had not ovulated.

7. Parovarian cysts commonly caused compression, but not occlusion, of the adjacent oviduct, and should not interfere with fertility. Cysts were present in fertile females.

8. Testicular degeneration, atrophy, and/or spermatogenic arrest were noted in deceased cheetahs with severe liver and kidney disease. These lesions likely were caused by these other diseases, and probably are not the basis for abnormal spermatozoa in cheetahs.

9. Uterine diseases were mild and not prevalent in the population. These findings reject the hypothesis that infertility in cheetahs is due to uterine diseases.

Continued surveillance of diseases in captive and free-ranging cheetahs will be necessary to acquire the information needed to provide optimal health care to captive cheetahs, and to direct initiatives in biomedical research that will improve the longevity and well-being of cheetahs in captivity

ACKNOWLEDGMENTS

The author is indebted to the following zoos for providing the tissues and information for this study: Audubon Park and Zoological Gardens, Binder Park Zoo,

Caldwell Zoo, Cleveland Metroparks Zoological Park, Cincinnati Zoo and Botanical Gardens, Columbus Zoological Gardens, Fossil Rim Wildlife Center, Fort Wayne Children's Zoo, Henry Doorly Zoo, Houston Zoological Gardens, Jackson Zoological Park, Knoxville Zoo, King's Island Wild Animal Habitat, Louisville Zoological Garden, Metropolitan Toronto Zoo, New York Zoological Park (Bronx Zoo), Oklahoma City Zoological Park, The Phoenix Zoo, Rio Grande Zoological Park, San Diego Wild Animal Park, St. Louis Zoological Park, San Antonio Zoological Gardens, Toledo Zoological Gardens, Wildlife Safari, and White Oak Plantation. I especially thank Dr. David Wildt and Dr. Mitch Bush for providing the liver biopsies. I am grateful for the generosity of the Department of Pathology at the National Zoological Park, and the Department of Pathobiology at the University of Tennessee College of Veterinary Medicine for providing the expert histotechnical assistance of Vera Bonshock, Brenda Collins, and Yolanda Bradley. I also acknowledge the contributions of Dr. Erby Wilkinson as a consultant on renal lesions, and Melissa Mason and Jan Grady in manuscript preparation.

REFERENCES

- Beckwith, J.B. Pediatric Kidney. Pp 669-675 in HISTOLOGY FOR PATHOLOGISTS. S.S. Sternberg, ed. New York. Raven Press, 1992.
- Dinnes, M.R.; Henrickson, R. Liver disease in cheetahs: a preliminary report. Pp 110-115 in PROCEEDINGS OF THE AMERICAN ASSOCIATION OF ZOOLOGICAL VETERINARIANS, 1970.
- Eaton, K.A.; Radin M.J.; Kramer, L.; Wack, R.; Sherding, R.; Krakowka, J.G.; Fox, J.G.; Morgan, D.R. Epizootic gastritis in cheetahs associated with gastric spiral bacteria. VETERINARY PATHOLOGY, 30:55-63, 1993.
- Evermann, J.F.; McKeirnan, A.J. Infectious disease surveillance in the cheetah with primary emphasis upon feline coronavirus and feline herpesvirus infections. Pp 191-197 in PROCEEDINGS OF THE AMERICAN ASSOCIATION OF ZOOLOGICAL VETERINARIANS, 1991.
- Evermann, J.F.; Burns, G.; Roelke, M.E.; MacKeirnan, A.J.; Greenlee, A.; Ward, A.C.; Pfeifer, M.L. Diagnostic features of an epizootic of feline infectious peritonitis in captive cheetahs. Pp 365-382 in TWENTY-SIXTH ANNUAL PROCEEDINGS OF THE AMERICAN ASSOCIATION OF VETERINARY LABORATORY DIAGNOSTICIANS, 1983.
- Gosselin, S.J.; Loudy, D.L.; Tarr, M.J.; Balistreri, W.F.; Setchell, K.D.R.; Johnston, J.O.; Kramer, L.W.; Dresser, B.L. Venocclusive disease of the liver of captive cheetah. VETERINARY PATHOLOGY 25:48-57, 1988.
- Henney, J.L.; Evermann, J.F.; McDeirnan, A.J.; Marker-Kraus, L.; Roelke, M.E.; Bush, M.; Wildt, D.E.; Meltzer, D.G.; Colly, L.; Lukas, J.; Manton, V.J.; Caro, T.; O'Brien, S.J. Prevalence and implications of feline coronavirus infections of captive and free-ranging cheetahs (*Acinonyx jubatus*). JOURNAL OF VIROLOGY 64:1964-1972, 1990.
- Junge, R.E.; Miller, R.E.; Boever, W.J.; Scherba, G.; Sundberg, J. Persistent cutaneous ulcers associated with feline herpesvirus type 1 infection in a cheetah. JOURNAL OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION 198:1057-1058, 1991.
- Klahr, S.; Schreiner, G.; Ichikawa, I. The progression of renal disease. NEW ENGLAND JOURNAL OF MEDICINE 318:1657-1666, 1988.
- Marker, L.; O'Brien, S.J. Captive breeding of the cheetah (*Acinonyx jubatus*) in North American Zoo (1871-1986). ZOO BIOLOGY 8:3-16, 1989.
- McEntee, K. REPRODUCTIVE PATHOLOGY OF DOMESTIC MAMMALS. New York. Academic Press, Inc., 1990.
- Munson, L.; Worley, M.B. The prevalence and morphology of liver disease in captive large cats: a comparison between snow leopards and cheetahs. P. 492 in ANNUAL PROCEEDINGS OF THE AMERICAN ASSOCIATION OF ZOOLOGICAL VETERINARIANS, 1987.
- Munson L.; Mason, R.J. Pathological findings in uteri of progesterone-implanted exotic felids. Pp. 311, 312 in PROCEEDINGS OF THE AMERICAN ASSOCIATION OF ZOOLOGICAL VETERINARIANS, 1991.
- O'Brien, S.J.; Roelke, M.E.; Marker, L.; Newman, A.; Winkler, C.A.; Meltzer, D.; Colly, L.; Evermann, J.F.; Bush, M.; Wildt, D.E. Genetic basis for species vulnerability in the cheetah. SCIENCE 227:1428-1434, 1985.
- Robbins, S.L.; Cotran, R.S.; Kumar, V. PATHOLOGICAL BASIS OF DISEASE, 4th ed. Philadelphia, W.B. Saunders Company, 1989.

van der Ingh, T.S.G.A.M.; Zwart, P. Heistab, A. Veno-occlusive disease (VOD) of the liver in cheetahs and snow leopards. *SCHWEIZ ARCHIVE TIERHEILKE* 123:323-327, 1981.

Wildt, D.E.; Bush, M.; Howard, J.G.; O'Brien, S.J.; Meltzer, D.; Van Dyk, A.; Ebedes, H.; Brand, D.J. Unique seminal quality in the South African cheetah and a comparative evaluation in

the domestic cat. *BIOLOGY OF REPRODUCTION* 29:1019-1025, 1983.

Wildt, D.E.; Brown, J.L.; Bush, M.; Barone, M.A.; Cooper, K.A.; Grisham, J.; Howard, J.G. Reproductive status of cheetahs (*Acinonyx jubatus*) in North American Zoos: The benefits of physiological surveys for strategic planning. *ZOO BIOLOGY*, 12:45-80, 1993.

Infectious Disease Surveillance in Captive and Free-Living Cheetahs: An Integral Part of the Species Survival Plan

James F. Evermann, M. Karen Laurenson, Alison J. McKeirnan, and T. M. Caro

Department of Veterinary Clinical Medicine and Surgery and Washington Animal Disease Diagnostic Laboratory, Washington State University, Pullman (J.F.E., A.J.M.); Department of Zoology, University of Cambridge, Cambridge, United Kingdom (M.K.L.); and Department of Wildlife and Fisheries Biology, University of California, Davis (T.M.C.)

During the formulative stages of developing the Species Survival Plan (SSP) for the cheetah, the impact of infectious disease upon its survival in captivity was of prime consideration, together with genetics, nutrition, physiology, and behavior. This paper summarizes the results of an infectious disease surveillance program, initially designed to monitor the infectious agents associated with clinically normal and clinically ill cheetahs in captivity, but subsequently supplemented with data from free-living cheetahs. The focus was on two viral infections, feline infectious peritonitis (FIP) and feline rhinotracheitis virus. Results indicated that between 1989 and 1991, there was an increase in the seroprevalence (number antibody-positive animals) of cheetahs to feline coronavirus from 41% to 64% in captivity. During this same time period, there were only two documented cases of FIP in cheetahs in the United States. The results suggest that feline coronavirus (feline enteric coronavirus-feline infectious peritonitis group) or a closely related coronavirus of cheetahs is becoming endemic in the captive cheetah population. Further serologic results from 39 free-living cheetahs demonstrated that there was a high seroprevalence (61%) to feline coronavirus, although serum antibody titers were considerably lower than those encountered in captive cheetahs. The observation of a high percentage of free-living cheetahs, which were seropositive to feline herpesvirus (44%), was unexpected, since it has been generally regarded that this infection is primarily associated with cheetahs in captivity.

© 1993 Wiley-Liss, Inc.

Key words: coronavirus, herpesvirus, management

Received for publication July 17, 1992; revision accepted November 10, 1992.

Address reprint requests to James F. Evermann, Department of Veterinary Clinical Medicine and Surgery, College of Veterinary Medicine, Washington State University, Pullman, WA 99164.