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SEROSURVEY OF VIRAL INFECTIONS IN FREE-RANGING NAMIBIAN CHEETAHS (*ACINONYX JUBATUS*)

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ABSTRACT: Cheetahs (*Acinonyx jubatus*) in captivity have unusually high morbidity and mortality from infectious diseases, a trait that could be an outcome of population homogeneity or the immunomodulating effects of chronic stress. Free-ranging Namibian cheetahs share ancestry with captive cheetahs, but their susceptibility to infectious diseases has not been investigated. The largest remaining population of free-ranging cheetahs resides on Namibian farmlands, where they share habitat with domestic dogs and cats known to carry viruses that affect cheetah health. To assess the extent to which free-ranging cheetahs are exposed to feline and canine viruses, sera from 81 free-ranging cheetahs sampled between 1992 and 1998 were evaluated for antibodies against canine distemper virus (CDV), feline coronavirus (feline infectious peritonitis virus; FCoV/FIPV), feline herpesvirus 1 (FHV1), feline panleukopenia virus (FPV), feline immunodeficiency virus (FIV), and feline calicivirus (FCV) and for feline leukemia virus (FeLV) antigens. Antibodies against CDV, FCoV/FIPV, FHV1, FPV, and FCV were detected in 24, 29, 12, 48, and 65% of the free-ranging population, respectively, although no evidence of viral disease was present in any animal at the time of sample collection. Neither FIV antibodies nor FeLV antigens were present in any free-ranging cheetah tested. Temporal variation in FCoV/FIPV seroprevalence during the study period suggested that this virus is not endemic in the free-ranging population. Antibodies against CDV were detected in cheetahs of all ages sampled between 1995 and 1998, suggesting the occurrence of an epidemic in Namibia during the time when CDV swept through other parts of sub-Saharan Africa. This evidence in free-ranging Namibian cheetahs of exposure to viruses that cause severe disease in captive cheetahs should direct future guidelines for translocations, including quarantine of seropositive cheetahs and preventing contact between cheetahs and domestic pets.

Key words: *Acinonyx jubatus*, canine distemper virus, cheetah, feline coronavirus, Namibia, serosurvey.

INTRODUCTION

The cheetah (*Acinonyx jubatus*) is globally endangered with the remaining free-ranging populations located principally in southern and eastern Africa. The largest free-ranging population of cheetahs resides in the farmlands of north-central Namibia, where contact with domestic pets and feral animals is likely (Marker-Kraus et al., 1996). Common viruses, such as feline coronavirus (FCoV; or feline infectious peritonitis virus [FIPV]), feline herpesvirus 1 (FHV1), and feline panleukopenia virus (FPV) or canine parvovirus (CPV) cause unusually severe or persistent clinical diseases in captive cheetahs (Evermann et al., 1988; Junge et al., 1991; Munson, 1993; Steinel et al., 2000). Whether free-ranging cheetahs are similarly affected by these viruses has not been investigated. Free-ranging cheetah populations share ancestry with captive cheetahs, and both populations lack the genetic variability typical of most species, including the major histocompatibility complex genes that determine, in part, the host response to viral infections (O'Brien and Wildt, 1983; O'Brien et al., 1985). Therefore, viral diseases of domestic pets may pose a threat to free-ranging populations of cheetahs.

In captive cheetahs, some viruses appear to be highly pathogenic and cause persistent viral infections that affect the management of captive populations. Feline infectious peritonitis occurs more commonly in FCoV-infected cheetahs than in FCoV-infected domestic cats, and FIP epidemics with high morbidity and mortality have occurred in captive cheetahs worldwide (August, 1984; Van Rensburg and Silkstone, 1984; Evermann et al., 1988; Heeney et al., 1990). Furthermore, FCoV-infected cheetahs persistently shed virus in their feces despite the presence of circulating antibodies (Kennedy et al., 2001). Feline herpesvirus infections also tend to be persistent and unusually pathogenic in cheetahs. In contrast to the mild upper respiratory disease caused by FHV1 infection in domestic cats, some FHV1-infected cheetahs develop a severe debilitating ulcerative and eosinophilic dermatitis concurrent with upper respiratory signs (Junge et al., 1991; Munson et al., 2003). Canine parvovirus and FPV infections also are unusually persistent in cheetahs despite vaccination and result in chronic debility from enteritis (Steinel et al., 2000). One suspected risk factor for developing severe disease and failing to clear infections is persistent stress, because captive cheetahs have elevated corticoid levels in comparison to free-ranging cheetahs (Terio, 2000). Therefore, it is possible that free-ranging cheetahs held under stressful conditions during rehabilitation or translocation also may develop these usually severe and persistent infectious diseases.

Other viruses of concern in free-ranging cheetah populations include canine distemper virus (CDV), feline leukemia virus (FeLV), and feline immunodeficiency virus (FIV). During the 1994 Serengeti CDV epidemic, several cheetahs were observed with myoclonus, a sign of permanent neurological damage from CDV infection (Appel, 1987; Roelke-Parker et al., 1996). Feline leukemia virus infection has been linked to fatal lymphoma in one free-ranging caught cheetah, and the source of infection was suspected to be domestic cats (Marker et al., 2003b). Feline immunodeficiency virus is prevalent in some African carnivore populations (Brown et al., 1994) and has the potential to affect immune function in nonadapted hosts (Brown et al., 1993), further compromising population health. These actual and potential disease threats should be cause for concern in Namibia, where conservation strategies include holding and translocating cheetahs.

Home ranges of Namibian cheetahs are extensive (averaging 1,776 km^2), encompassing several continuous farms and often bordering towns and cities (Marker, 2000), most of which have both domesticated and feral cats and dogs. Many domestic dogs and cats in Namibia are unvaccinated, and cases of CDV, FCoV, FPV, CPV, and FHV1 infection have been reported (Schneider, 1991). This close proximity of free-ranging cheetahs to infected carnivores provides ample opportunity for viral exposure. Additionally, free-ranging cheetahs are often trapped by Namibian farmers to protect their livestock, and these cheetahs are held in pens near domestic pets or other free-ranging carnivores, such as leopards (*Panthera pardis*), before being translocated to new regions (Marker-Kraus et al., 1996). These capture cages or holding pens would further facilitate viral concentration and transmission. The act of translocating cheetahs may, in turn, carry pathogens to previously uninfected regions, thereby increasing disease risks to indigenous animals. Transmission of viruses among free-ranging cheetahs would then be facilitated through the territory-marking behavior of depositing feces at play trees throughout the farmlands (Marker-Kraus and Kraus, 1995).

Because trapping and translocating cheetahs is part of the regional conservation plan, knowledge of the prevalence and distribution of viral infections in the Namibian farmland regions is needed to assess the health risk of these actions. The aim of this study was to determine the

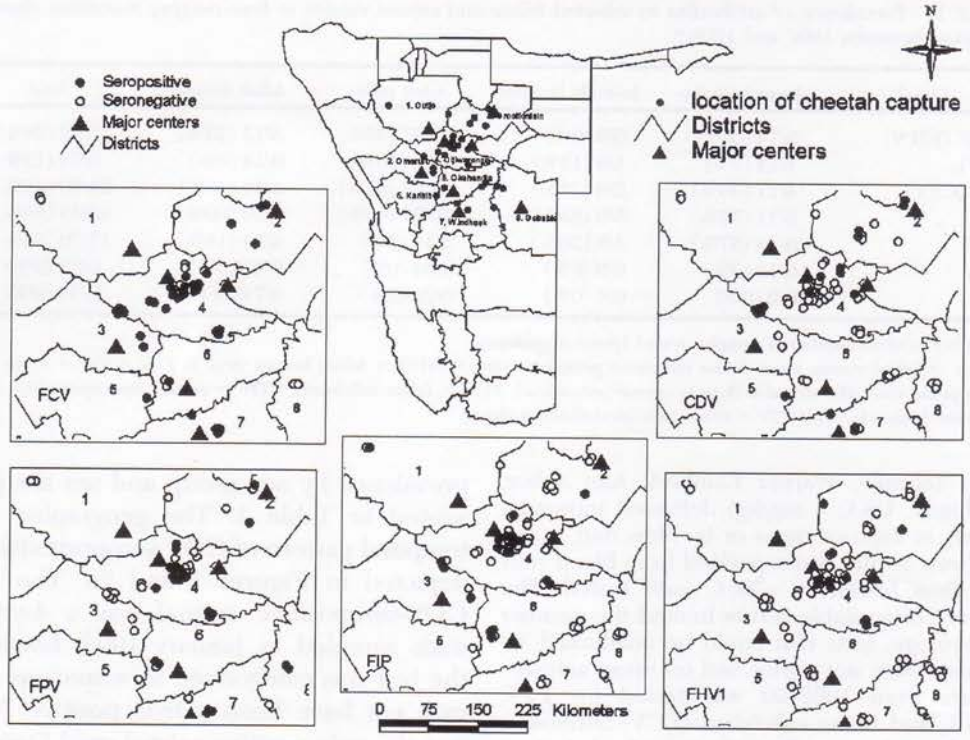

FIGURE 1. Spatial distribution of farms in Namibia where seropositive and seronegative cheetahs were trapped. Map of Namibia shows magisterial districts and population centers. Distribution of seropositive and seronegative animals are in relationship to population centers.

prevalence and spatial distribution of free-ranging Namibian cheetahs with antibodies to selected feline and canine viruses and determine temporal patterns of seropositivity.

MATERIALS AND METHODS

Serum was obtained opportunistically from 81 free-ranging cheetahs that were trapped between 1992 and 1998 by landowners in the north-central farmland regions of Namibia because of perceived threats to livestock. The number of animals sampled per year were six, 17, 13, 17, six, 10, and 12 for 1992–98, respectively. Animals were trapped in a region extending from 19°30′S to 23°30′S and 16°E to 19°E, including the magisterial districts of Gobabis, Windhoek, Okahadja, Omaruru, Otjiwarango, and Grootfontein (Fig. 1). Only cheetahs held in capture cages for less than 6 days were included in the study to assure that antibodies measured reflected exposure to infectious agents in the wild.

Age classification was based on descriptions from previous studies or personal experience and took into account body weight, body size, tooth wear, gum recession, wear on pads, pelage and scarring, social groupings of animals caught together, and reproductive condition (Burney, 1980; Caro, 1994; Marker and Dickman, 2002). To give confidence to the above procedures, lower premolars were categorized by cementum aging, and these results correlated with other age estimates (Marker, 2002). The age distribution of the study population was six cubs from two litters (2 mo and 3.5 mo), 22 juveniles and subadults (6–23 mo old; median age = 8 mo), and 53 adults (median age = 48 mo). Cubs were included in the study despite the possibility that their antibodies were acquired passively, because these antibodies would reflect maternal exposure in that trapping region (Spencer, 1992). However, cubs were excluded from prevalence statistics to avoid overrepresentation of maternal antibody status. The population included 57 males and 24 females. More males were included in the study group because traps are placed primarily at play trees, which are visited more frequently by males because of territorial behavior (Marker-Kraus and Kraus, 1995).

Serum samples were obtained under general anesthesia (tiletamine-HCl and zolazepam-

TABLE 1. Prevalence of antibodies to selected feline and canine viruses in free-ranging Namibian cheetahs collected between 1992 and 1998.[a]

Virus[b]	Juvenile males	Juvenile females	Adult males	Adult females	Total
FCoV (FIPV)	5/14 (36%)	0/8 (0%)	13/37 (35%)	3/13 (23%)	21/72 (29%)
FHV1	1/14 (7%)	1/8 (13%)	7/38 (18%)	0/14 (0%)	9/74 (12%)
FPV (CPV)	4/11 (36%)	2/6 (33%)	14/23 (61%)	4/10 (40%)	24/50 (48%)
FCV	8/11 (72%)	5/6 (83%)	10/22 (45%)	9/10 (90%)	32/49 (65%)
CDV	8/14 (57%)	4/8 (50%)	3/34 (9%)	2/14 (14%)	17/70 (24%)
FeLV	0/14 (0%)	0/8 (0%)	0/34 (0%)	0/13 (0%)	0/69 (0%)
FIV	0/6 (0%)	0/4 (0%)	0/22 (0%)	0/7 (0%)	0/39 (0%)

[a] Number positive/number of samples tested (percent positive).
[b] FCoV = feline corona virus (feline infectious peritonitis virus); FHV1 = feline herpes virus 1; FPV (CPV) = feline panleukopenia virus (the test also detects canine parvovirus); FCV = feline calicivirus; CDV = canine distemper virus; FeLV = feline leukemia virus; FIV = feline immunodeficiency virus.

HCl; Telazol®, Warner Lambert, Ann Arbor, Michigan, USA; 4 mg/kg) delivered intramuscularly in capture cages or by blow dart in enclosures. Serum was separated from blood cells and then frozen at −70 C until tested. The amount of available serum limited the number of serologic tests that could be conducted, so all tests were not performed on every animal.

Sera from 1992–93 was tested for FPV, FHV1, and feline calicivirus (FCV) antibodies by indirect immunofluorescent antibody tests at the Department of Virology, MEDUNSA, Republic of South Africa (Spencer and Burroughs, 1991); sera from 1993–98 were tested by serum neutralization tests (FHV1 and FCV) or hemagglutination inhibition assays (FPV) at the New York State Veterinary Diagnostic Laboratory, Ithaca, New York, USA, because these tests were no longer available at the laboratory at MEDUNSA. The FPV assays used in this study also detect antibodies against CPV2. All sera were tested for CDV neutralizing antibodies against the Onderstepoort stain of CDV at the New York State Veterinary Diagnostic Laboratory. Antibodies against FIV were measured by Western blot at the National Cancer Institute, Frederick, Maryland, USA, by using an FIV antigen isolated from a domestic cat (Olmstead et al., 1992). Feline coronavirus antibodies were detected by indirect immunofluorescence, and FeLV antigens were detected by enzyme-linked immunosorbent assay at the Washington Animal Disease Diagnostic Laboratory in Pullman, Washington, USA (Evermann et al., 1988). These laboratories were selected because they are used by the American Zoo and Aquarium Association Cheetah Species Survival Plan to test captive cheetahs in the US.

RESULTS

The overall prevalence of viral antibodies in juvenile and adult cheetahs and prevalence by age group and sex are presented in Table 1. The geographic and temporal pattern of CDV seropositivity are depicted in Figures 1 and 2a. The first CDV-seropositive animal was a 4-yr-old male sampled in January 1993; however, the test was categorized as suspicious and may not have been a true positive. This was the only positive animal until December 1995. Thirteen of the 17 CDV-positive animals sampled between 1995 and 1998 were cubs or juveniles, including six 7- to 8-mo-old animals sampled in January and February 1997.

The geographic and temporal distribution of FCoV/FIPV-seropositive animals are depicted in Figures 1 and 2b. The youngest animal with antibodies to FCoV was 6 mo old. None of the five cubs and juveniles sampled between 1994 and 1996 had FCoV antibodies.

Temporal and geographic distribution of FPV-, FHV1-, and FCV-seropositive animals are depicted in Figures 1 and 2c–e. Serum from juveniles or cubs was not available to test for FPV or FCV between 1994 and 1996, so it cannot be determined if these viruses are endemic. None of the 39 free-ranging cheetahs tested had FIV antibodies, and none of the 78 free-ranging cheetahs tested for FeLV had antigen. Of six cubs tested for CDV, FHV1, and FCoV, one cub had CDV antibodies and one cub had FHV1 antibodies, but no cub had FCoV antibodies. Of the five cubs

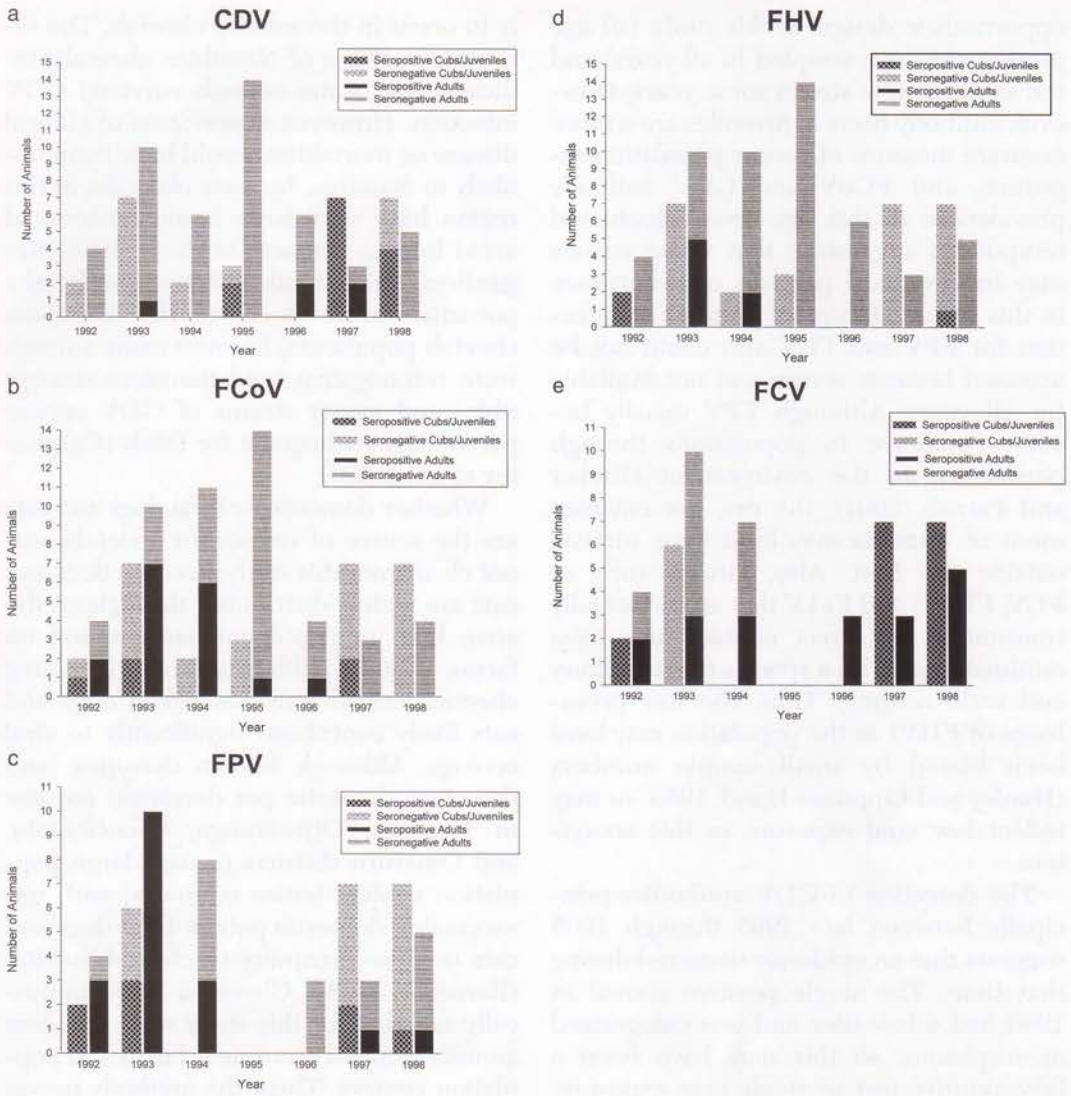

FIGURE 2. Temporal patterns of seropositivity in 99 free-ranging Namibian cheetahs. (a) Canine distemper virus (CDV), (b) feline coronavirus (FCoV; also known as feline infectious peritonitis virus [FIPV]), (c) feline panleukopenia virus (FPV; the test also detects canine parvovirus), (d) feline herpesvirus (FHV1), and (e) feline calicivirus (FCV).

tested for FPV and FCV, two littermates had FPV antibodies and three littermates had FCV antibodies.

DISCUSSION

This study disclosed widespread exposure of free-ranging Namibian farmland cheetahs to common feline and canine viruses (or antigenically similar viruses) that are known to cause serious clinical disease in captive cheetahs. Because the regions sampled are typical of the habitat of most Namibian cheetahs outside the Etosha National Park, these results most likely are representative of the Namibian population as a whole. Both sexes had similar exposure to most viruses, which reflects the equally large home ranges of males and females in Namibia (Marker et al., 2003a). Detection of seropositive animals differed among years, although conclusions drawn from these patterns should consider the

opportunistic design of this study (all age groups were not sampled in all years) and the small sample size in some years. However, antibody titers in juveniles are a more accurate measure of recent population exposure, and FCoV and CDV antibody prevalences in this age group fluctuated temporally, suggesting that these viruses may have cyclical patterns of occurrence in this region. Temporal patterns of infection for FPV and FCV also could not be assessed because serum was not available for all years. Although FPV usually becomes endemic in populations through persistence in the environment (Barker and Parrish, 2001), the dry, hot environment of Namibia may limit virus survival outside the host. Also, viruses such as FCV, FHV1, and FeLV that are principally transmitted by direct contact may have minimal impact on a species that is solitary and wide ranging. Thus, the low prevalence of FHV1 in the population may have been biased by small sample numbers (Hanley and Lippman-Hand, 1983) or may reflect low viral exposure in this ecosystem.

The detection of CDV antibodies principally between late 1995 through 1998 suggests that an epidemic occurred during that time. The single positive animal in 1993 had a low titer and was categorized as suspicious, so this may have been a false-positive test or single case exposure. This period of seropositivity closely follows the 1994–95 CDV epidemic in free-ranging felids in the Serengeti ecosystem (Roelke-Parker et al., 1996) and the 1995 CDV epidemic in African wild dogs (*Lycaon pictus*) in Chobe National Park, Botswana (Alexander et al., 1996). This geographic pattern suggests that a CDV pandemic occurred in sub-Saharan Africa in the mid-1990s. Whether CDV has the potential to be as fatal in cheetahs as in lions (*Panthera leo*) is unknown. Many deaths during the CDV epidemic in the highly social Serengeti lions were attributable to intraspecific trauma within prides as a result of infection. This cause would be less likely to occur in the solitary cheetah. The seropositive status of Namibian cheetahs indicates that some animals survived CDV infection. However, observation of clinical disease or mortalities would have been unlikely in Namibia, because cheetahs in this region have very large home ranges and avoid human contact (Marker, 2000). Regardless, CDV should still be considered a potential future threat to the Namibian cheetah population, because many animals were seronegative (and therefore susceptible) and newer strains of CDV appear particularly pathogenic for felids (Carpenter et al., 1998).

Whether domestic or feral dogs and cats are the source of viruses for cheetahs was not clear from this study, because dogs and cats are widely distributed throughout the area, both in population centers and on farms. While wildlife reservoirs (including cheetahs) could exist, domestic dogs and cats likely contribute significantly to viral ecology. Although human densities (and therefore domestic pet densities) are low in Namibia, Otjiwarango, Grootfontein, and Omaruru districts contain large population centers (cities or towns) with unvaccinated domestic pets or feral dogs and cats in close proximity to cheetah habitat (Barnard, 1998). Cheetahs opportunistically sampled for this study were problem animals trapped on or near farms and population centers. Thus, the antibody prevalences detected in this population may be higher than in the free-ranging population at large, because of the proximity of study cheetahs to domestic animals. On the other hand, antibody-positive cheetahs were found throughout the study region without a clear association with population centers. This widespread distribution was not unexpected because of the large home ranges of both male and female cheetahs on Namibian farmlands (Marker, 2000) and the presence of domestic dogs and cats on farms throughout the region. Only FHV1 and FPV antibody-positive animals appeared confined to certain districts, but only Otjiwarango contained a dense pop-

ulation center. The small sample numbers and biased sample collection (problem animals) should temper any conclusions based on proximity to human habitation. Antibody-positive and antibody-negative animals coexisted in the same territories, even those with FPV exposure, which typically occurs through a contaminated environment (Barker and Parrish, 2001). The low population density and solitary nature of cheetahs in this region (Marker, 2000) would provide little opportunity for viral transmission during active infections. Future studies should determine if transmission increases in holding facilities.

Negative FIV test results are likely true negatives because FIV has not been reported in either domestic or free-ranging cats in Namibia (Olmstead et al., 1992), and the same testing methods have detected FIV antibodies in cheetahs from other regions (Brown et al., 1993). Negative FeLV test results also are likely true, because FeLV antigens have only been detected in four of more than 200 Namibian cheetahs tested to date, and these animals were in close contact and were previously exposed to FeLV-positive domestic cats (Marker et al., 2003b). Even if the low sample numbers for this study failed to detect some diseased animals (Hanley and Lippman-Hand, 1983), the need for prolonged close contact for FeLV transmission would limit spread within the population at large because of the large home ranges and solitary behavior of cheetahs.

The intent of mapping seropositive animals in this study was to identify seropositive and seronegative areas to assist in translocation decisions. Feline herpesvirus 1, FCoV, FPV, and CPV have long, unpredictable periods of viral shedding, even in the presence of serum antibodies. Therefore, translocating antibody-positive cheetahs carries the risk of contaminating new environments and imperiling the indigenous carnivores. Also important to consider is the risk of exposure to immunologically naive cheetahs when translocated from uninfected to infected environments. Stress from capture, confinement, and transport may increase susceptibility to viral infections or cause recrudescence in chronically infected animals. Together these risks advocate strict quarantine of cheetahs before translocation. Serologic testing of cheetahs during quarantine and before translocation would be ideal, but in many cases is not feasible. When possible, cheetahs that have evidence of previous exposure to FCoV, FHV1, or FPV should be isolated during holding, and all pens and cages thoroughly sanitized before housing new cheetahs. By applying strict quarantine protocols during translocation and restricting the movement of infected animals, further spread of viral infections to free-ranging cheetahs can be minimized.

Vaccination of wild animals to prevent viral infection should be approached with caution because some vaccines developed for domestic pets cause disease when administered to other species (Williams, 2001). Furthermore, vaccination disrupts normal host-pathogen relationships within an ecosystem, so would be unjustified without evidence that the disease caused significant mortality. Cheetahs develop antibody responses to multivalent modified-live vaccines for FPV, FHV1, and FCV (Spencer and Burroughs, 1991), but FHV and FPV infections still occur in vaccinated animals, indicating only partial immunity (Steinel et al., 2000). Therefore, quarantine and testing, rather than vaccination of free-ranging cheetahs, are the preferred methods for preventing acquisition of viral diseases during translocation. Vaccinating domestic dogs and cats (barrier vaccination) and minimizing contact between domestic pets and free-ranging cheetahs would be better management tools for reducing the risk of infectious disease to this population. Because stress may compromise disease resistance, translocation procedures should aim to limit human exposure and holding time of cheetahs before release. Ongoing surveillance in this region will be used to detect changes in viral

exposure in indigenous cheetahs as management of wildlife intensifies and human populations increase.

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