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A Serosurvey of Viral Infections in Lions (*Panthera leo*), from Queen Elizabeth National Park, Uganda

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ABSTRACT: Serum samples from 14 lions (*Panthera leo*) from Queen Elizabeth National Park, Uganda, were collected during 1998 and 1999 to determine infectious disease exposure in this threatened population. Sera were analyzed for antibodies against feline immunodeficiency virus (FIV), feline calicivirus (FCV), feline herpesvirus 1 (feline rhinotracheitis: FHV1), feline/canine parvovirus (FPV/CPV), feline infectious peritonitis virus (feline coronavirus: FIPV), and canine distemper virus (CDV) or for the presence of feline leukemia virus (FeLV) antigens. Ten lions (71%) had antibodies against FIV, 11 (79%) had antibodies against CDV, 11 (79%) had antibodies against FCV, nine (64%) had antibodies against FHV1, and five (36%) had antibodies against FPV. Two of the 11 CDV-seropositive lions were subadults, indicating recent exposure of this population to CDV or a CDV-like virus. No lions had evidence of exposure to FeLV or FIPV. These results indicate that this endangered population has extensive exposure to common feline and canine viruses.

Key words: Canine distemper virus, feline calicivirus, feline coronavirus, feline herpesvirus 1, feline immunodeficiency virus, feline parvovirus, *Panthera leo*, Uganda.

As in other parts of sub-Saharan Africa, Uganda's large carnivore populations have undergone a substantial decline over the last five decades (Bauer, 2003). Lions (*Panthera leo*) play an integral role in the ecological dynamics of savanna ecosystems and are critical to the Ugandan economy as a tourist attraction. A census conducted in Queen Elizabeth National Park (QENP) from 1997 to 1999 estimated that only 210 lions resided in the park (M. Driciru, unpublished). In addition to threats from poaching of lions or their prey and habitat loss, recovery of lion populations may be further compromised by infectious diseases, such as occurred in the 1994 canine distemper virus (CDV) epidemic in the Serengeti lion population (Roelke-Parker et al., 1996). Serosurveys of other African lion populations have indicated exposure to CDV, feline immunodeficiency virus (FIV), feline calicivirus (FCV), feline herpesvirus 1 (feline rhinotracheitis: FHV1), feline/canine parvovirus (FPV), and feline infectious peritonitis virus (feline coronavirus: FIPV) (Olmstead et al., 1992; Spencer and Morkel, 1993; Hofmann-Lehmann et al., 1996; Osofsky et al., 1996; Roelke-Parker et al., 1996; Packer et al., 1999). Knowledge of what infectious agents may threaten the QENP lions is important to understanding the impact of disease on population viability and the risk of translocating animals.

Our serosurvey of the lion population in the northern savanna sector of QENP was conducted to establish baseline knowledge of endemic viruses. The study was conducted in the Katwe (313 km^2) and Katunguru (433 km^2) regions of QENP (29°45′E–30°15′E and 0°30′S–0°15′N) in the southwestern region of Uganda astride the four districts of Kasese, Bushenyi, Kaborole, and Rukungiri. The techniques used to locate lions included random searches, tracking, the presence and behavior of vultures and prey animals, and responses to audio calls, as well as reports from tourists, field assistants, researchers, tour guides, local residents, and park workers. During the 1998–1999 study period, 14 QENP lions were immobilized: nine adults (>4 yr), four subadults (2–4 yr), and one cub (0–1 yr). Age

TABLE 1. Seroprevalence of antibodies against feline and canine viruses in 14 Ugandan lions from the Queen Elizabeth National Park sampled from 1998 to 1999. Antibodies to feline immunodeficiency virus (FIV), feline calicivirus (FCV), feline herpesvirus 1 (FHV1), feline parvovirus virus (FPV), feline infectious peritonitis virus (FIPV), and canine distemper virus (CDV) and antigens of feline leukemia virus (FeLV) were surveyed; n = number of lions tested.

	Number of seropositive lions by age group			
Virus	Adults (n=9)	Subadults (n=4)	Cubs (n=1)	Total (n=14)
FIV	7	3	0	10 (71%)
CDV	9	2	0	11 (79%)
FHV1	5	3	1	9 (64%)
FCV	9	2	0	11 (79%)
FPV	4	1	0	5 (36%)
FIPV	0	0	0	0
FeLV	0	0	0	0

was determined by documented knowledge of birth year or was estimated by examining the wear of the teeth and the size and condition of the individual. Lions were anesthetized using a combination of Zoletil® (250 mg tiletamine-HCl and 250 mg zolazepam-HCl; VIRBAC Nederland BV, Berneveld, The Netherlands; 1.3–1.5 mg/kg, IM) and Zalopine® (10 mg/ml medetomidine hydrochloride; Orion Farmos Corporation, Espoo, Finland; 0.1 mg/kg, IM), administered intramuscularly using Dan-Inject darts (Dan-Inject SA, Private Bag X402, Skukuza, 1350, South Africa) and a Dan-Inject dart gun (Model JM Special; Smith, Gelsenkirchen, Germany) from a distance of 10–25 m. Medatomidine was reversed using Antisedan® (5 mg/ml atipamezole hydrochloride; Orion Farmos Corporation) at a dose of 3 mg of atipamezole for every 1 mg of medetomidine used. Half of the dose was administered intravenously and half intramuscularly. Blood was collected via cephalic, saphenous, or femoral venipuncture into serum separator tubes and centrifuged. Serum samples were stored in liquid nitrogen. A physical exam was performed on each animal.

Serum samples were evaluated at the New York State Diagnostic Laboratory at Cornell University in Ithaca, New York, USA, for antibodies against FIV, FCV, FHV1, FPV, and CDV and for FeLV antigens. The following methods were used: virus neutralization test (VNT) to detect CDV, FCV, and FHV1 antibodies; both Western blot and an enzyme-linked immunosorbent assay (ELISA) to detect FIV antibodies; ELISA to detect FeLV antigen; and hemagglutination inhibition/2-mercaptoethanol (HAI/2-ME) to detect FPV IgG antibodies (Harrison et al., 2004; Munson et al., 2004). Feline coronovirus (FCoV or FIPV) antibodies were determined by indirect immunofluorescence (IFA) at the Washington Animal Disease Diagnostic Laboratory, Pullman, Washington, USA (Harrison et al., 2004; Munson et al., 2004).

The serosurvey revealed that lions in QENP are exposed to most of the selected viruses (Table 1). Two lions with antibodies against CDV were subadults, indicating that CDV or a CDV-like virus was in the region during their lifetime (within 2–4 yr). The cub with antibodies against FHV1 was 7–9 months old, indicating that these antibodies were acquired from viral exposure and not passively acquired. The mother of this cub was seropositive for FHV1 as well as FIV, CDV, FCV, and FPV.

Four lions had healed skin lesions on the face, limbs, and body from moving through burned grassland areas. One lioness had an acute 15–20-cm-long, 7-

cm-deep laceration along the thorax. Most lions had scars presumably from intra- and interspecific aggression. Only two of 14 lions had evidence of poor health. One subadult was emaciated and anemic (hematocrit=22%) and had multiple footpad ulcers on both front feet. This animal had antibodies against FIV, FHV1, and FCV, but not CDV, FIP, or FPV. The cub had severe stomatitis and was one of a litter of five cubs that became progressively emaciated, three of which subsequently died. It is not known whether the clinical signs in the cubs were caused by a viral infection; however, the cub that survived had antibodies against FHV1, but not FCV.

The high prevalence of antibodies against CDV in the Ugandan lion population is similar to that found in Serengeti lions after the 1994–1995 epidemic (Roelke-Parker et al., 1996). The occurrence of antibodies in 2–4-year-old QENP lions in 1999 suggests a recent exposure to CDV or a CDV-like virus. Because antibodies against CDV usually protect against disease and the majority of lions had antibodies, this population may be protected currently; however, as a greater portion of the population becomes naïve, an epidemic could occur, and the QENP lion population may be too small to recover.

The high proportion (71%) of QENP lions with antibodies against FIV is similar to that reported for lion populations in the Serengeti National Park/Ngorongoro Crater in Tanzania (79%) and Kruger National Park (KNP) in South Africa (83%) (Olmstead et al., 1992). In contrast, fewer Botswana lions (25.8%) and no Namibian lions tested to date have been FIV-positive, indicating that not all African lion populations are infected (Spencer et al., 1992; Spencer and Morkel, 1993; Osofsky et al., 1996). Packer et al. (1999), noted that in the Serengeti National Park and in the Ngorongoro Crater, rates of FIV infection did not vary significantly across years for either adult or immature lions. This suggests that the virus is endemic to those populations. No specific health effects of FIV have been identified to date in wild lions despite the high prevalence of infection (Olmstead et al., 1992; Osofsky et al., 1996; Packer et al., 1999). Although a decreased CD4:CD8 T-cell ratio or absolute T-cell numbers have been observed in some FIV-infected captive lions (Spencer et al. 1995; Bull et al., 2003), all but one FIV-infected lion in our study appeared healthy.

Many QENP lions (64%) had antibodies against FHV1, as have other free-ranging lion populations (Spencer, 1991; Spencer and Morkel, 1993; Roelke-Parker et al., 1996; Packer et al., 1999). Though captive lions may develop clinical disease (Wack, 2003), no signs have been reported in free-ranging lions. The high proportion (79%) of QENP lions with antibodies against FCV was similar to the prevalence (70%) in Tanzanian lions (Olmstead et al., 1992) that are in close proximity. In contrast, southern African lions in KNP and Etosha National Park had no evidence of FCV exposure (Spencer, 1991; Spencer et al., 1992; Spencer and Morkel, 1993). Although FCV may be endemic in some areas, a longitudinal serosurvey of Tanzanian lions (Packer et al., 1999) suggested that FCV occurs in epidemics. No clinical disease due to FCV infection has been reported. The QENP lions also have been exposed to FPV, as is evident in the 36% antibody prevalence. However, as with FCV, no clinical signs of FPV infection have been observed in wild lions, although diarrhea, vomiting, and leukopenia occurred in FPV-infected captive lions (Studdert et al., 1973; Mochizuki et al., 1996). Clinical signs in wild lions may be missed in populations not under close observation, so lack of pathogenicity should not be assumed. Unlike the Serengeti lion population (Hofmann-Lehmann et al., 1996; Roelke-Parker et al., 1996; Packer et al., 1999), QENP lions had no evidence of exposure to FIPV. On the

other hand, the FeLV-negative status of the QENP population was similar to that of lions from the Serengeti National Park and Ngorongoro Crater in Tanzania, Hluehluwe/Umfolozi Park in South Africa, and Etosha National Park and Bushmanland/Caprivi regions of Namibia and Botswana (Osofsky et al., 1996; Packer et al., 1999). This provides further evidence that FeLV is not present in wild felids (Munson et al., 2004).

Although antibodies against some viruses provide protection against infection, FIV, FHV1, and rarely FCV and FPV infections (Steinel et al., 2000; Lenghaus et al., 2001) can persist in seropositive hosts, and virus can be shed from asymptomatic carriers. Therefore, prior to any translocation, lions from both origin and destination sites should be screened for evidence of these viruses to prevent introduction into naïve populations.

This study contributes to ongoing efforts to understand the ecology of carnivore viruses in African ecosystems. Continued monitoring of Ugandan lions is recommended to determine the causes of mortality, assess the morbidity caused by viral infections, and detect the emergence of an epidemic. Monitoring for FIV and CDV is of particular importance in order to understand their pathogenicity in lions and dynamics of these viruses within the ecosystem.

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