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REVIEW OF DISEASE EPIDEMICS IN FREE RANGING WILDLIFE IN KENYA 1991-1997

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Bien que le Kenya héberge un grand nombre d'espèces sauvages, la rapide croissance de la population humaine conduit à des conflits pour l'espace et l'accès aux ressources. L'augmentation des interférences faune domestique-faune sauvage conduit à de nouveaux risques de transmission d'agents pathogènes.
Depuis 1991 et la réorganisation du Service Kényan de la Nature, plusieurs épidémies sont apparues chez la faune sauvage. Cet article les passe en revue et met l'accent sur l'efficacité de l'épidémiosurveillance et du système de suivi des épidémies.
Il s'agit de la maladie de Carré dans l'écosystème Mara-Sérengeti, de la gale des guépards au même endroit, de mortalité sur les petits flamants des lacs Nakuru et Bogozia et de la peste bovine dans les parcs nationaux de Tsavo et de Nairobi.

INTRODUCTION

Four major disease outbreaks have been well documented in the last 5 years. Surveillance for diseases in free-ranging wildlife in Kenya has been passive (i.e no active search for the disease unless symptoms reported). Active surveillance is only done once confirmatory diagnosis has been arrived at. Reporting of diseases has mostly been by park administration and non-KWS staff working inside the park eg. hotel administration, tour drivers and visitors. Disease syndromes not manifesting overtly in high mortality or other noticeable symptoms can be assumed to often go unnoticed when passive surveillance is relied on. The effect of such syndromes to wildlife population dynamics has not been investigated but may be one of the factors causing persistence of below capacity population numbers in areas like the Tsavos and Meru national parks.

DISEASE DESCRIPTION

1. CANINE DISTEMPER IN THE MARA-SERENGETI ECOSYSTEM

An epidemic caused by a pathogenic morbilivirus closely related to canine distemper virus (CDV) emerged abruptly in the lion population in the Serengeti National Park, Tanzania in early 1994 , resulting in fatal neurologic disease characterized by grand *mal* seizures, ataxia, and myoclonus. The prevalence of CDV-antibodies rose from 0% in October 1993 to 85% by August 1994. In late 1994 the epidemic spread north to affect lions in the Maasai Mara national reserve, Kenya. The mortality rate in the Serengeti was estimated at 30%. All necropsied lions had histopathologic and immunohistochemical evidence of CDV infection. The virus was isolated from a lion with neurologic disease (Roelke-Parker et. al., 1995).
The lions in the Serengeti side of the ecosystem were well known and could be identified individually. The information on the Maasai Mara was not accurate because there was no previous surveillance going on in the lion prides before the outbreak occurred in the Tanzanian side. Thus the mortality and morbidity rates in the Maasai Mara ecosystem were estimates based on anecdotal reports from sighting of carcassess and cases of lions showing myoclonus and ataxia by tour drivers and security personell operating within the park.
Reports of the disease on the Maasai Mara side were received from the Tanzanian side of the ecosystem. Cross-border warnings of diseases in this area has been informal but effective.

2. MANGE IN THE CHEETAH (*ACINONYX JUBATUS*) IN THE MAASAI MARA CONSERVATION AREA

An outbreak of a skin disease manifesting mainly in alopecia and debilitation was reported in an area just outside the Maasai Mara game reserve. Infection by mites of the species *Sarcoptes scabiei* was confirmed to be the cause of the condition.
The area affected was in the Lemek/Aitong group ranch area (35 13' E 1 12'S) which borders the game reserve to the east and south with a total area of 400km^2. The area had an estimated 25 resident cheetah, 12 of which were confirmed to have the mites. Three animals were presumed to have died of the disease although this was not confirmed. Treatment with Ivermectin (Ivomec- Merck and Co. Rahway, New Jersey 07065, USA) at 0.3ug/kg body weight was attempted with good results.
Immuno-suppression either by stress or by infection by another pathogen was suspected to have predisposed the animals to infection by the mite which is endemic in the area. Seventy percent of the affected cheetah also tested positive to antibodies cross-reactive with feline immuno-deficiency virus proteins.
Reports of the condition were received from camp owners and tour drivers who reported having seen severely affected animals. The disease was well progressed in particular individual animals by the start of the monitoring period. Monitoring for disease was done by week long monthly visits to the area by veterinary teams from Nairobi, a distance of 400km. This was continued for two and a half years by the end of which all the treated

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animals had recovered and no new infections were recorded. Occasional reports of the disease have been received recently but have been controlled by immobilization and treatment.

3. RINDERPEST IN TSAVO AND NAIROBI NATIONAL PARKS

An outbreak of a disease causing high mortality in buffalo in the Tsavo National park was reported to the veterinary unit by both the park administration and hotel management in November 1994. The clinical signs were weakness, inappetance, oculo-nasal discharge, diarrhoea, reluctance to move from water, increased bellicosity and death. Initial laboratory testing did not detect rinderpest virus antigen in the agar gel immunodiffusion test or recover rinderpest virus in cell culture. However, all the serum samples collected from sick buffaloes had high titers of specific antibodies to rinderpest virus in both virus neutralizing test and competition ELISA. The presence of these in unvaccinated animals together with precipitating antibodies which are usually found in the early stages of a primary immune response to the virus indicated that the animals had recently been infected with the field strain of rinderpest virus (Kock et.al., 1995).

In May 1994, the KWS veterinary unit had received reports from the park administration of a disease in the lesser kudu (*Tragelaphus imberis*) manifesting in blindness caused by corneal opacity resulting from keratitis, lachrymation and death. Samples taken from animals found moribund at the time were misplaced before appropriate virological and bactriological tests could be performed. However, electron microscopic examination of some formalin fixed tissues detected morbillivirus-like particles in the lymph node of a dead kudu. (Kock et.al. 1995). The timing of this finding was interesting in that it came after rinderpest was confirmed in the buffalo population.

A disease manifesting the same clinical signs as rinderpest was discovered by the veterinary unit in eland (*Taurotragus oryx*) in Nairobi National Park (KWS and Veterinary unit headquarters located at the park gate) in October 1996. The disease was confirmed in the laboratory by agar gel immuno-diffusion test within two weeks of the first report. The disease later spread to the buffalo (*Syncerus caffer*) population and eventually died down after four months. Mortality in eland was estimated at 25% and 10% in the buffalo population. No other susceptible species was found to get infected by the virus even though all the animals were grazing together. The outbreak in The Nairobi National Park was diagnosed early and the monitoring was much easier due to the proximity of the park to the laboratory. This was the only outbreak diagnosed from the occasional surveillance done by the veterinary unit.

4. FLAMINGO DISEASE EPIDEMIC IN LAKE BOGORIA AND LAKE NAKURU

An outbreak of disease occurred in the lesser flamingo (*Phoeniconaias minor*) in Kenya resulting in more than 30,000 deaths over six months in 1993-1994. The disease spread between lakes along the Rift Valley, concentrating primarily on Lake Bogoria (0^0 11' - 0^0 20'S 36^0 07'E). Coincidental to the outbreak was an algae bloom the toxins of which were suspected to be causative. The main algae were in the cyanobacteria group of the genera Spirulina and Synechocystis. A cyanotoxin, microcystin, which has been known to cause death in animals on a worlwide basis was found in filtered water from the lake (Unpublished KWS report). Discrete necrotic lesions were noted in spleen and liver and a variety of bacteria recovered in pure culture from visceral organs, including *Escherichia coli*, *Pseudomonas aeruginosa*, and *Proteus* spp. Histolopathological evaluation revealed chronic and often life threatening lesions of mycobateriosis, primarily involving spleen, liver, with rare pulmonary lesions. Typical hepatic lesions associated with algae toxicosis were not seen in any of the birds. It was suspected that the algae bloom allowed the proliferation of potentially pathogenic bacteria in such numbers that the birds were unable to mount appropriate immune response and were overwhelmed by infections (Kock et.al.,1995).

Past records indicate that flamingo mortality has occurred occasionally over the years but because of poor data collection and storage this information was not readily available at the time of the outbreak. An ecological monitoring system for the Rift valley lakes is currently in the pipeline and a strong veterinary input is envisaged.

DISCUSSION

In the four epidemics reviewed, initial reports were by the park administration, tour drivers and hotel/camp owners and rarely from direct surveillance by the veterinary personnel. The result of this kind of reporting system is that response to disease outbreaks has been a "fire-fighter" response. There is need for a formal surveillance system capable of acting as an early warning system for health issues in free-ranging wildlife.

Surveillance of disease in free-ranging wildlife is difficult due to inaccessibility of most conservation areas. Restraint of wildlife is expensive and in cases of some endangered species sometimes detrimental to herd cohesion and therefore the survival of the species. An effective surveillance system should be simple, cheap and causing minimum disturbance to the animals.

The existing structure at KWS can be utilized to set up a formal reporting system on health of wildlife populations. Decentralization of the veterinary services with veterinarians being located within the conservation areas although expensive offers the best solution to the problem of inaccessibility. In addition, rangers designated for biodiversity issues need to be trained to perform some para-veterinary duties. Some of the rangers have qualifications in animal health which places them in a good position to perform these duties. The para-veterinary training should include assessment of population health, health data collection and recording and simple techniques of sample collection, storage and transportation. Parameters for population health should be simplified and summarized in a form that can be used by rangers who frequently patrol the conservation areas. Initial information received from rangers indicate that they notice a lot of health associated incidents in wildlife populations which go un-reported. Once tapped, this information will contribute a lot to assessment of health in free-ranging wildlife populations and the effect of diseases to population dynamics.

Tour drivers and hotel administration are also in constant view of the animals and they could be a valuable source of information. Trained rangers would be important links to these and could routinely collect this information and record it. Cross-border harmonization of disease surveillance should be encouraged in border areas as the animals know no boundaries. A formal link between the wildlife veterinarians and their domestic counterparts needs to be built up as many of these diseases cross-infect at the domestic-wildlife interface. In areas such as the Maasai Mara, vaccination of domestic dogs against the common disease pathogens has been found to be a necessary conservation measure, protecting endangered wild carnivores against these pathogens and vice-versa.

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