Endemic and epidemic diseases, viruses and parasites Impacting lion (*Panthera leo*) populations

Last updated 8 November 2011

Introduction

Diseases can be classified as endemic or epidemic dependant on their persistence in a population. Although lion populations can be affected by high mortality over brief periods caused by epidemic viruses, endemic viruses can be constantly prevalent and are thought to exhibit low pathogenicity [1].

Epidemic disease risks for animals living in fragmented small populations become significantly higher as contact with human and domestic animal populations become more frequent and as a result of alterations in microclimate and landscape ecology. The tools to predict, prevent and respond to these risks are not well established in conservation management.

Co-infection by more than one pathogen can change the expected transmission rates and virulence of a disease [2] whilst also promoting parasite infection. Environmental perturbations can also change the effect on the host by the pathogen/s [3]. 100% of extant lion populations are infected with at least one, and most with multiple pathogens, often with multiple strains of those pathogens.

Different populations, even those geographically close to each other, carry different viruses [4]. Plans for corridors between isolated wildlife populations to promote gene-flow might also include the unwanted consequence of spreading diseases between sub-populations and promoting co-infections. This is especially of concern where one population may be naive to a disease carried by another and as such, have no acquired immunity to it. Many infections can persist in seropositive hosts and asymptomatic carriers can continue to transmit, or shed, the virus [5-6]. Translocation of shedding individuals into a susceptible population thus could entail serious consequences for overall population health [3].

Given the lack of data on pathogens infecting lions in the greatest percentage of lion populations, the consequences of infection upon the host species, the alterations to transmission and virulence of these diseases through combination of the viruses and their subtypes in a single host or how these combined pathogens respond to stochastic disease outbreaks, it should be considered that using any extant wild populations as a source for reintroduction programs should involve extreme caution.

It should also be noted that the pathogenicity and course of disease progression of many of the feline pathogens discussed in this section has only been carefully described among domestic cats. However, post-mortem studies of mortalities among exotic felids in zoos indicate that among many viral diseases at least, there are parallel consequences of infection. In addition, the occurrence of a particular disease among free-ranging lion populations is by necessity based on serum antibody presence, which is by definition retrospective – the animal survived the challenge. In only few cases, as perhaps in the Canine Distemper Virus outbreak among lions in the Serengeti in 1994, is progression of a disease recorded with any scientific rigour in a wild lion population.

Mostly, lions simply “disappear” from study populations. This is especially true of lion cubs, the most disease susceptible among the population. Carcasses, even if found intact by field biologists, cannot be given the careful post-mortem analysis to determine cause of death as such biologists neither have the means nor the expertise to do so. Multiple infections by a diversity of pathogens as is common among lions will also cloud a clear diagnosis of mortality. It is entirely likely that lions, like many other wildlife species, eventually succumb to an overload rather than a single pathogen.

What has become clear is that lion populations, even in protected areas like Kruger National Park (bovine tuberculosis) and the Serengeti (canine distemper), are fragile and susceptible to introduced diseases in addition to those that we assume occur naturally in their environment. The high number of individuals infected with the several strains of lion Feline Immunodeficiency Virus now identified is worrisome, as it has belatedly been recognized that this virus erodes immune competence over time as similar among domestic cats. Some strains seem more virulent than others, and individuals infected with multiple strains are potent sources for the evolution of new FIV viral strains.

Disease threats to wild animals have long been recognized and now become ever more important as we deal with small and isolated wildlife populations. The increasing rate of lion population declines has many causes, and we need to pay careful attention to pathogens as contributory agents in the future. We cannot medicate and inoculate wild lions. But we can more carefully identify sources of diseases from domestic animals and ensure they do not increasingly spread to wild carnivores.

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Further reading

Distinguishing epidemic waves form disease spillover in a wildlife population (pdf)
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Climate Change and Animal Disease (pdf)
Summers BA (2009) Veterinary Pathology 46 (6): 1185-1186

Endemic diseases

Feline Herpesvirus (FeHV)

Feline herpesvirus 1 (FeHV - 1) has been reported in free-ranging wild felids in Europe, Asia, North America, and Africa [1-6]. FeHV – 1 permanently infects the host causing feline viral rhinotracheitis (FVR), a disease of the upper respiratory system. It grows in the nasal, oral and conjunctival mucous membranes and intravaginal instillation of the virus has led to vaginitis and congenitally infected offspring [6]. In domestic cats the nasal discharge leads to the sense of smell becoming severely diminished, causing inappetence. Whilst loss of appetite is dangerous in all cats, it is especially so in the young. Additionally, secondary bacterial infections can occur due to the damage caused to tissues [7]

FeHV is highly prevalent in all free-ranging lion populations tested so far (67% in Etosha National Park, 91% in Kruger NP and 99% or 100% in Serengeti NP, Ngorongoro Crater, Lake Manyara region and Central Kalahari region) [4, 8-10]. FeHV however is thought to be innocuous to survival or to reproductive success in infected lions, but high prevalence throughout different populations makes it difficult to compare such parameters among infected and uninfected hosts [8].

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Further reading:

Feline Herpesvirus (pdf)
Gaskell R, Dawson S, Radford A, Thiry E (2007) Veterinary Research 38: 337-354

Feline Immunodeficiency Virus (FIV)

FIV is a significant cause of disease in domestic cats (FIV_{fca}) producing AIDS-like pathology characterized by CD4 depletion, immune suppression and death [1-2]. Species-specific strains of FIV were demonstrated in non-domestic cat species [3-5]; however general opinion was that FIV_{ple}, widespread in African lions, was benign. [3, 6-7].

Recent studies, including the first to study the complete genome sequence of the provirus from some FIV_{ple} subtypes, have shown that infected lions exhibit declines in CD4+ subsets, reductions of the CD4+ / CD8+ ratio, reduction of CD8+βhigh cells and expansion of the CD8+βlow subset [8-12] parallel to observations with HIV infection, strongly suggesting an immunological cost of FIV infection to lions. FIV_{ple} subtype E, recorded in populations from the Okavango Delta in Botswana, has been shown to be more similar to FIV_{fca} than to FIV_{ple} subtype B indicating a possible pathogenesis similar to that seen in domestic cats including a possibly as yet un-sequenced strain for the env gene [8-9, 13].

The extent of recombination among the six FIV_{ple} subtypes within free-ranging lions is not yet known, but studies of Serengeti lions (where up to 93% of adult lions are infected) have shown that 43% of individuals are multiply infected with FIV_{ple} subtypes A, B and C allowing opportunities for recombination and possible evolution of more virulent strains [14]. FIV_{ple} negative populations have been confirmed in Etosha NP [7] although it is possible that they are infected with an as yet un-sequenced subtype.

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The Evolutionary Dynamics of the Lion *Panthera leo* Revealed by Host and Viral Population Genomics (pdf)
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Brennan G, Podell MD, Wack R, Kraft S, Troyer JL, Bielefeldt-Ohmann H, VandeWoude S (2006) Journal of Clinical Microbiology 44 (12) 4345 – 4352

Seroprevalence of Selected Infectious Agents in a Free-Ranging, Low-Density Lion Population in the Central Kalahari Game Reserves in Botswana (pdf). Ramsauer S, Bay G, Meli M, Hofmann-Lehmann R, Lutz H (2007) Clinical and Vaccine Immunology: 808–810


Epidemic diseases

Bovine Tuberculosis (bTB)

Mycobacterium bovis, the causative agent of bovine tuberculosis (bTB) was introduced in the southern part of Kruger National Park [1] by domestic cattle. The disease has spread northwards through infected African buffalos (*Syncerus caffer*) with serious concerns for the viability of the Kruger lion population as well as the development of the Greater Limpopo Trans-frontier Conservation area [1-2]. Aside from the clinical effects of bTB leading to greater mortality in affected populations the prevalence of the disease within Kruger has also been shown to drive social change with lower lion survival and breeding success with more frequent male coalition turnover and consequent higher infanticide [1].

Elsewhere, among the Serengeti lions, 4% of animals tested were seropositive for bTB [3]. The disease is also present in reserves adjacent to Kruger NP including Hluhuwe-iMfolozi [1]. bTB also occurs among buffalos in Queen Elizabeth NP, Uganda, although so far not observed among resident lions [4], and also among lechwe (*Kobus leche*) in Zambia's Kafue NP [5]. Lions in Mozambique's Niassa reserve have tested negative for the disease (Colleen Begg, pers comms. 2011)

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Canine Distemper (CDV)

Canine Distemper Virus (CDV) affects both free-living and captive carnivores worldwide, and is now recognised as a cause of large-scale epidemics in felids [1-2]. Primarily transmission is by aerosol transfer or contact with bodily exudates containing the virus. Clinical symptoms mainly affect the respiratory, gastro-intestinal and central nervous systems, causing grand mal seizures and myoclonus with mortalities usually occurring due to encephalitis or pneumonia [1-3].

Lion populations in the Serengeti-Mara ecosystem experienced two major CDV epidemics in 1994 and 2001, with the disease originating in domestic dogs [3-4]. The 2001 outbreak in the Ngorongoro Crater caused 35% mortality among its small population of lions [4], while the 1994 outbreak that hit the Serengeti National Park lions, spreading North to Kenya's Maasai Mara National Reserve infected 85% of lions causing mortalities in a third of the 3000 strong population [3]. Several other carnivore species were also affected and a single CDV variant was found circulating in lions, spotted hyenas, bat-eared foxes, and domestic dogs, suggesting extensive inter-specific transmission [3, 5-6].

Possible explanations for such high mortality rates of infection in these outbreaks include the emergence of a particularly virulent strain of CDV, repeated introduction due to multi-host disease spill-over and climate extremes, coinciding with both outbreaks, which created conditions exacerbating the immunosuppressive effects of infection, which may otherwise have been tolerated in isolation [7-10].

CDV antibodies have also been detected in Central Kalahari lions and in Uganda's Queen Elizabeth National Park [11-12]. CDV has been, and continues to be, present across much of Southern Africa, in its domestic dog and wild carnivore populations [13-17]. Lions in Mozambique's Niassa reserve have tested negative for the disease (Colleen Begg, pers comms. 2011)

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Cleaveland S, Appel MG, Chalmers WS, Chillingworth C, Kaare M, Dye C (2000) Veterinary Microbiology 72: 217–27.

The Canine Distemper Epidemic in Serengeti: Are Lions Victims of a New Highly Virulent Canine Distemper Virus Strain, Or Is Pathogen Circulation Stochasticity To Blame? (pdf)
Guiserix M, Bahi-Jaber N, Fouchet D, Sauvage F, Pontier D (2007) Journal of the Royal Society Interface. 4: 1127-1134.

Phylogenetic Evidence for Canine Distemper Virus in Serenegti's Lions (pdf)
Harder TC, Kenter M, Appel MJG, Roelke-Parker ME, Barrett, T, Osterhaus ADME. (1995) Vaccine 13: 521–523.

Canine Distemper in Terrestrial Carnivores: A Review (pdf)
Deem SL, Spelman LH, Yates RA, Montali RJ (2000) Journal Zoo Wildlife Medicine 31**:** 441-451.

The Conservation Relevance of Epidemiological Research Into Carnivore Viral Diseases in the Serengeti (pdf)
Cleaveland S, Mlengeya T, Kaare M, Haydon D, Lembo T, Laurenson MK, Packer C (2007) Conservation Biology 21: 612–622.

Feline Parvovirus (FPV)

Also known as feline infectious enteritis (FIE) or feline panleukopenia, feline parvovirus (FPV) is an acute, enteric, viral infection of domestic and exotic felines caused by a single stranded DNA virus, from the *Parvoviridae* family [1]

Parvovirus is very stable in the environment and indirect transmission can spread rapidly. In naïve populations this can be the cause of high mortality [2]

Clinical symptoms include depression, vomiting and diarrhoea. Ataxia (tremors and jerky movements) may also ensue due to cerebellar hypoplasia which will be more noticeable in young cubs [1].

Antibody titers for FPV are highly prevalent in Serengeti populations (75%) but less so in the nearby Ngorongoro Crater population (27%). Other locations with published results include Laka Manyara region (60%), Kruger National Park (84%), Etosha National Park (0%) [3] and Central Kalahari Game Reserves (0%) [4] .

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Further reading:

Fatal Infection with Feline Panleukopenia Virus in Two Captive Wild Carnivores (*Panthera tigris* and *Panthera leo*) (pdf – purchase required)
Duarte MD, Barros SC, Henriques M, Lobo-Fernandes T, Bernardino R, Monteiro M, Fevereiro M (2009) Journal of Zoo and Wildlife Medicine 40 (2) 354-359

Specific identification of feline panleukopenia virus and its rapid differentiation from canine parvoviruses using minor groove binder probes (pdf – purchase required) Decaro N, Desarioa C, Lucentea MS, Amoriscoa F, Campoloa M, Eliaa G, Cavallia A, Martellaa V, Buonavoglia C (2008) Journal of Virological Methods 147 (1) 67-71

Feline Calicivirus (FCV)

Feline Calicivirus (FCV) has been well documented among domestic felines; more so than exotic felids. Nevertheless, this highly prevalent pathogen has been found to have infected wild members of the Felidae [1-3], and the nature, severity and clinical signs associated with FCV in exotic felines are similar to those reported in domestic cats [3].

FCV is related to upper respiratory tract diseases and can be identified by rhinitis, pneumonia, fever, lameness and oral ulcerations [3-4]. The FCV strain usually causes temporary infection and appears to be harmless in most cases [5]. However, upon recovery, the infected host may still act as an asymptomatic carrier [5]; potentially infecting those who are susceptible to the disease.

Outbreaks of the virus have been reported in different lion populations residing in Africa. The prevalence rate has fluctuated in each population, with FCV being absent from the small, isolated Crater population in Ngorongoro [1,5], to occurring at low prevalence in Botswana [2], to being highly prevalent in the Serengeti Plains [1]. This suggests that the FCV infection occurs in populations of high density, with outbreaks increasing as the size of the susceptible host population grows [5].

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[3] Harrison TM, Sikarskie J, Kruger J, Wise A, Mullaney TP, Kiupel M, Maes RK (2007). Systemic calicivirus epidemic in captive exotic felids. Journal of Zoo and Wildlife Medicine 38: 292-299 (pdf – purchase required)

[4] Radford AD, Coyne KP, Dawson S, Porter CJ, Gaskell RM (2007). Feline calicivirus. Veterinary Research 38: 319-335 (pdf)

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Norovirus in captive lion cub (*Panthera leo*). (pdf)
Martella V, Campolo M, Lorusso E, Cavicchio P, Camero M, Bellacicco AL, Decaro N, Elia G, Greco G, Corrente M, Desario C, Arista S, Banyai K, Koopmans M, Buonavoglia C (2007) Emerging Infectious Diseases 13: 1071-1073.

A strain of calicivirus isolated from lions with vesicular lesions on tongue and snout. (pdf – purchase required)
Kadoi K, Kiryu M, Iwabuchi M, Kamata H, Yukawa M, Inaba Y (1997) New Microbiologica 20 (2): 141-148.

A serosurvey of viral infections in lions (*Panthera leo*), from Queen Elizabeth National Park, Uganda. (pdf)
Driciru M, Siefert L, Prager KC, Dubovi E, Sande R, Princee F, Friday T, Munson L (2006). Journal of Wildlife Diseases 42: 667-671

Infectious diseases of wild mammals. (e - book)
Williams ES, Barker IK (2000). 3rd ed. USA: Iowa State University Press. 288 – 291

Antibody response of lions inoculated with inactivated calicivirus vaccine experimentally prepared. (pdf – purchase required)
Kadoi K, Kiryu M, Inaba Y (1998) New Microbiologica 21 (2): 147-151.

Feline Coronavirus (FCoV)

Feline Coronavirus is an upper respiratory infection not dissimilar to FHV, which is transmitted by various routes; faecal, oral and possibly aerosolized. It can infect domestic animals such as dogs and in cats has been known to develop into the more pathogenic feline infectious peritonitis [1]. It has also been found to be the cause of high mortality in domestic kittens [2].

Like many of the other epidemic diseases (such as parvovirus or calicivirus) found in serological studies of lions there have been no consistent signs of clinical disease for FCoV [3-7].

Unlike the endemic diseases, FCoV is an epidemic disease, and thus has different implications for wild lion populations. Epidemic diseases by nature briefly sweep through a population often inflicting a high mortality rate however, due to this high mortality and a lack of further susceptible hosts the initial bursts are short lived. The disease will then return through the population at a later date once there are sufficient numbers of susceptible individuals, known cases of this include coronavirus, parvovirus and calicivirus in the Serengeti lion population [6]. FCoV has been found in varying levels of population infection throughout African lion populations [3-5].

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Feline Infectious Peritonitis (FIP)

Feline Infectious Peritonitis is a viral disease caused by some strains of feline coronavirus (FCoV) either by mutation of the virus or by an aberration of the immune response. Cats with weak immune systems such as young or old cats and those infected with feline leukaemia virus (FeLV) are at particular risk from developing FIP from FCoV.

An intense inflammatory reaction occurs around vessels in the tissue; often in the abdomen, kidney or brain. The virus is unique in that antibodies actually assist the infection of white blood cells of the FIP virus. Clinical FIP is progressive and almost always fatal. Symptoms include inappetance, weight loss, depression, anemia, fever and roughening of the fur. Fluid may also accumulate in the abdomen causing difficulty in breathing when accumulation becomes significant. [1]

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Potential Disease Threats Not Yet Identified in Free Ranging Lion Populations

Feline Leukaemia Virus (FeLV)

Feline leukaemia virus (FeLVs) can be classified into three subgroups: A, B and C and are pathogenic retroviruses that induce proliferative, degenerative and immunosuppressive disorders [1-2]. The disease can be transmitted via saliva (ie, grooming each other), through nasal secretions or across the placenta from a queen to its developing foetuses. [3]

This disease is a cancer of the blood cells known as lymphocytes. The main systems to be affected are lymphatic, immune and nervous however all other body systems can be affected due to secondary infections. Initial symptoms include – Lymphadenomeagly (enlarged lymph nodes), rhinitis, persistent diarrhoea, gingivitis and progressive ataxia (the ''wobbles''/collapsing) [2]

In cases of infected animals, several antiviral agents have been proposed. Unfortunately, administration of a 'reverse inhibitor AZT' does not appear to clear viremia in most felines. The prognosis for infected animals is guarded and the majority die within in 2 – 3 years. [3]

Non symptomatic individuals can harbour a latent infection in a dormant state within bone marrow and may be reactivated if the immune response is compromised or removed. The latent viral infection is eliminated over time however evidence suggests that virus shedding may occur from a queen to her young through milk [4]. Whether latently infected individuals can shed the virus by other means is undetermined.

To date no evidence of the virus infecting wild lions has been discovered although clinical signs may be missed in populations not under close observation, so lack of pathogenicity should not be assumed. Seronegative samples have so far been collected from populations including Queen Elizabeth National Park, Uganda [5], Serengeti National Park, Ngorongoro Crater and the Lake Manyara area in Tanzania [6-7], various locations in Botswana [8] and Zakouma National Park, Chad [9]

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[2] Fujino Y, Ohno K, Tsujimoto H (2008) Molecular pathogenesis of feline leukaemia virus-induced malignancies: Insertional mutagenesis. Veterinary Immunology and Immunopathology 123: 138-143 (pdf)

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Feline Monocytotropic Ehrlichiosis

Ehrlichia is a tick-borne obligately intracellular bacterium of the family *Rickettsiaceae* that infect white blood cells causing monocytotropic ehrlichiosis [1].

Common clinical signs include anorexia, lethargy, weight loss, vomiting or diarrhoea, pale mucous membranes, joint pain, lymphandenomeagly and dyspnoea (shortness of breath) (2) Non-regenerative anaemia or thrombocytopenia (low platelet count) and low white blood cell counts have been observed in laboratory blood tests.

Whilst seropositive results for the antigens in African lions have not been identified results from some captive wild felids and domestic cats have been [4].

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Parasites: Parasitic infection may be endemic or epidemic and clinical symptoms of infection may or may not be prevalent, possibly suggesting some level of genetic adaptation to some parasitic infections.

Haemoprotozoans

Babesia spp.

Babesia is a tick-borne intracellular erythrocytic haemoprotozoan parasite causing babesiosis. The disease infects red blood cells with symptoms characterized by haemolytic anaemia [1]. Only infected ticks which carry the disease from feeding on blood from an infected animal transmit the disease. Infected ticks may pass on the infection to the next generation through eggs [2]

Babesia leo has been isolated in lions and is morphologically similar to *B. felis* found in domestic cats and wild pumas (*Felis concolor*) [1]. However clinical babesiosis has not been recorded in lions except in one specific case. "Elsa" the lion made famous by Born Free was reported to have died of babesiosis following release into an area where she was severely mauled by presumed infected wild lions. It is speculated that the severe stress caused by the release protocol negatively impacted her immunocompetence as the reason why she succumbed to an infection which other lions have been shown to tolerate [3]

56 free-ranging lions and 25 captive lions in South Africa and Swaziland were tested finding that 28 (50%) of the wild lions and 12 (48%) of captive lions tested positive for the parasite. Of those that tested positive some were infected with *B. felis*, some with *B. leo*, some with both whilst others were infected with unidentified *Babesia* spp. [1].

Two outbreaks of canine distemper virus (CDV) in Serengeti lion populations 1994 and in the Ngorongoro Crater population in 2001 resulted in high mortality whilst a further, at least, five outbreaks of CDV between 1976 and 2006 occurred without clinical signs or measurable mortality suggesting CDV was not necessarily fatal. A common event for the two periods of high mortality was extreme drought conditions with widespread die-off in herbivore species, and in particular Cape buffalo (*Syncerus caffer*). After the resumption of the rains heavy tick infestation of compromised buffalo populations led to unusually high *Babesia* infection in lions which were already immunocompromised by CDV infection. *Stomoxys* flies also flourished in the rains following the Crater drought, causing pruritic skin ulcers in lions, further compromising them. Biotic and abiotic factors had converged to create a "perfect storm" resulting in unprecedented mortality in lions [4]. "Such mass mortality events may become increasingly common if climate extremes disrupt historic stable relationships between co-existing pathogens and their susceptible hosts." [4]

Policies of fire suppression allow grass to grow taller resulting in increasing tick survival rates and raising the chances of infected ticks passing on the disease [5].

Prophylactic treatment of the disease is possible although dosages for wild animals are yet to be determined. Extrapolation of information on dosages for domestic animals may be possible [3].

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Babesia leo N. Sp. from Lions in the Kruger National Park, South Africa, and Its Relation to Other Small Piroplasms. (pdf – purchase required)
Penzhorn BL, Kjemtrup AM, Lopez-Rebollar LM, Conrad PA (2001) Journal of Parasitology 87 (3) pp. 681-685.

Trypanosoma spp.

Trypanosomes are a unicellular parasitic flagellate protozoa transmitted by tsetse flies (*Glossina* spp.).

Between July and September 1985 113 lions were examined for blood parasites in the Serengeti National Park and 10 from the Ngoro Ngoro Crater in Tanzania. 28% of Serengeti lions and 0% of Ngoro Ngoro crater lions carried *trypanosoma* sp. of parasites. Higher incidence of infection was found in lions utilising the Serengeti woodlands of highest concentrations of tsetse flies suggesting transmission is most likely from the flies. With some positive results discovered in non-tsetse areas it has been suggested that mechanical transmission is also possible through predator-prey interactions of infected prey animals [1].

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Further reading

Patterns in age-seroprevalance consistent with acquired immunity against *Trypanosoma brucei* in Serengeti lions (pdf)
Welburn S, Picozzi K, Coleman PG, Packer C (2008) PLoS Neglected Tropical Diseases 2(12): e347. Doi:10.1371/journal.pntd.0000347

Theileria spp.

Theileria is a genus of parasitic protozoan that belongs to the phylum *Apicomplexa* and is closely related to *Plasmodium*. *Theileria* are transmitted by ticks. The life cycle within cats is similar to Babesia spp except the infectious sporozoites invade leucocytes as well as erythrocytes. In lymphocytes, the sporozoites undergo shizogony and multiply, then invade erythrocytes. Erythrocytic shizogony is rare or absent in cats. Infected erythrocytes are then ingested by ticks where they undergo gamogony and fertilization within the midgut, then invade the salivary gland cells. Here, sporogony occurs, resulting in sporozoites that are transmitted to the cat when the tick feeds.

Clinical signs in domestic cats include Clinical signs in cats include fever, weight loss, lethargy, anorexia and lymphadenopathy. [1]

Between July and September 1985 113 lions were examined for blood parasites in the Serengeti National Park and 10 from the Ngoro Ngoro Crater in Tanzania. 100% of lions in both locations carried *Theileria*-like sp. piroplasms [2].

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Hepatozoon spp.

Hepatozoon is a genus of *Apicomplexan* protozoa which incorporates over 300 species of obligate intraerythrocytic parasites. Hepatozoonosis results when an animal eats an infected tick rather than a bite from one. Feline hepatozoonosis is associated with muscular pathology and is often reported in conjunction with a retroviral disease [1].

Between July and September 1985 113 lions were examined for blood parasites in the Serengeti National Park and 10 from the Ngoro Ngoro Crater in Tanzania. 100% of lions in both locations carried *Hepatozoon* sp. of parasites [2]

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Endoparasites

Endoparasites can exist in one of two forms: intercellular (inhabiting spaces in the host's body) or intracellular (inhabiting cells in the host's body). Intracellular parasites, such as bacteria or viruses, tend to rely on a third organism which is generally known as the carrier or vector. The vector does the job of transmitting them to the host. Intercellular parasites include protozoa, trematoda (flatworms), cestoda (tapeworms), nematode (roundworms), acanthocephalan (thorny-headed worms) and arthropoda.

> *"*Numerous reports of African lion (*Panthera leo*) endoparasites originate from zoological parks and managed game reserves without indication of the lions' origins, their diets or prey, or their movements. Reports are sparse on parasites of free-ranging wild lions of eastern Africa. *Taenia regis*, *Taenia gonyamai*, and *Taenia simbae* were found in lion collections from the Serengeti [1] and *Spirometra* sp. was frequent in these lions [2].
>
> Accounts of parasitism in free-ranging lions from other areas of Africa, particularly southern Africa, are more numerous. *Ancylostoma paraduodenale* has been found in lions in Northern Rhodesia [now Zambia] [3] as have *Lagochilascaris major* in the Congo [4], *Echinococcus felidis* in Northern Transvaal [5], and *Echinococcus granulosus felidis* in Transvaal [6]. *Trichinella spiralis*, *Dirofilaria sudanensis*, *Linguatula serrata*, *Linguatula nuttalli*, *Cylicospirura* sp. [6] and *Schistosoma mattheei* [7] were reported from Kruger National Park. An individual lion from Northern Rhodesia was infected with *Pharyngostomum cordatum*, *Galoncus perniciosis*, *Gnathostoma spinigerum*, *Mesocestoides* sp., *Dipylidium* sp., *Ollulanus tricuspis*, *Toxascaris leonina*, *Physaloptera praeputialis*, *Dirofilaria acutiuscula*, *A. paraduodenale* [8], and *Taenia hydatigena* was reported from a lion in Nigeria that had originated from the Leipzig Zoological Gardens, Germany, where it had been fed raw goat meat [9].
>
> Many reports of lion endoparasites are from zoos in India, but information on lion habitat, origin, or diet is incomplete. *Sarcocysts* were found in two zoo lions [10] and *Taenia jaipurensis* was found in the intestine of a lion that died in the Jaipur Zoo [11]. *Spirometra erinacea* [12], *Ascaris felis*, *Galonchus perniciosus* [13], *T. leonine* [14], and *Parascaris felis* [15] were all reported. Both wild Gir forest lions and Indian zoo lions had *Spirometra* sp., *Toxascaris* sp., and *Ancylostoma* sp. [16-18]
>
> *Toxocara cati*, *T. leonina*, and *Spirometra* sp. were described in Australian circus lions [19] and zoo lions in central California were serologically positive for *Toxoplasma gondii* [20]. *Giardia* sp. was reported from a captive lion [21]. Two undescribed species of *Isospora* were reported from captive lion cubs in England [22]. A spurious coccidian parasite, *Eimeria felina*, was observed in a lion from the Leningrad Zoo [18]"
>
> In a survey of lions of the Serengeti nineteen different parasites were identified of the orders Protozoa, Trematoda, Cestoda, Nematoda, Acanthocephala and Arthropoda. The number of parasites recovered per lion averaged 3 (range 0 – 9) including *Eimeria* sp., *Giardia* sp., *Isispora sp.* including *I. felis* and *I. rivolta*, *Sarcocystis* sp., *Toxoplasma*-like sp. Trematoda-like sp., Anoplocephalidae, Taeniidae, *Aelurostrongylus* sp., *Ancylostoma* sp. *Capillaria* sp., *Habronema* sp., *Tococara cati*, *Trichuris*-like sp., Acanthocephala and *Demodex* sp [1].

Quote & last paragraph reference: Bjork KE, Averbeck GA, Stromberg BE (2000) Parasites and parasite stages of free-ranging wild lions (*Panthera leo*) of northern Tanzania. Journal of Zoo and Wildlife Medicine 31 (1): 560-061 (pdf)

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Further reading

Neospora caninum and *Toxoplasma gondii* in lion (*Panthera leo*) from Senegal , West Africa (pdf)
Kamga-Waladjo AR, Gbati OB, Kone P, Lapo RA, Dombou E, Chatagnon G, Baou SN, Diop PEH, Pangui LJ, Tainturier D, Akakpo JA (2009) Asian Journal of Animal And Veterinary Advances 4 (6): 346-349

A coprological survey of intestinal parasites of wild lions (*Panthera leo*) in the Serengeti and the Ngoro Ngoro Crater, Tanzania, East Africa (pdf)
Müller-Graf CDM (1995) Journal of Parasitology 81 (5): 812-814

Epidemiology of an intestinal parasite (*Spirometra* spp.) in two populations of African lions (*Panthera leo*) (pdf)
Müller-Graf CDM, Woolhouse MEJ, Packer C (1999) Parasitology 118: 407-415

Parasites in captive carnivores at the animal orphanage in Kenya (pdf - purchase required)
Vincent O, Francis G (2007) The Kenya Veterinarian 31 (2): 59-63

Prevalence of *Bartonella* infection in wild African lions (*Panthera leo*) and cheetahs (*Acinonyx jubatus*) (pdf - purchase required)
Molia S, Chomel BB, Kasten RW, Leutenegger CM, Steele BR, Marker L, Martenson JS, Keet DF, Bengis RG, Peterson RP, Munson L, O'Brien SJ (2004) Veterinary Microbiology 100: 31-41

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Further Reading

Metabolic bone disease in lion cubs at the London Zoo in 1889: the original animal model of rickets (pdf)
Chesney RW, Hedberg G (2010) Journal of Biomedical Science 17 (Suppl 1): S36

Predicting disease dynamics in African lion populations (pdf)
Craft ME (2008) PhD thesis

Sub-occipital craniectomy in a lion (*Panthera leo*) with occipital bone malformation and hypovitaminosis A (pdf – purchase required)
Shamir MH, Shilo Y, Fridman A, Chai O, Reifen R, Miara L (2008) Journal of Zoo and Wildlife Medicine 39 (3): 455-459

Infectious diseases subdue Serengeti lions (pdf – purchase required)
Dybas CL (2009) BioScience 59 (1): 8-13