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Veterinary Guidelines for the Cheetah European Endangered Species Programme (EEP)

Fachgebiet

Conservation Medicine

DIPLOMARBEIT

Zur Erlangung der Würde einer
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1 Foreword

The aim of "Veterinary Guidelines for the Cheetah European Endangered Species Programme (EEP)" is to provide the zoo vet or anyone else who is responsible for the health of an EEP cheetah population a manual of the most important issues, diseases and diagnostic methods. Therefore the authors tried to give an overview of the most important health issues concerning cheetahs on the one hand and to be as concise and clear as possible on the other hand. For more detailed information please consult the literature stated in the reference section.

The authors especially wish to thank the authors of the Cheetah SSP health chapter[1] because their manual acted as the starting point for this manual for the European cheetah population.

General guidelines for veterinary medical programs and veterinary hospitals can be obtained from the AAZV website.[2]

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2 Introduction

2.1 Taxonomy and distribution

Order: Carnivora

Family: Felidae

Subfamily: Acinonychinae

Genus: Acinonyx

Species: Jubatus

Subspecies[3–5]:

- *Acinonyx jubatus hecki* (Northwest African Cheetah, Saharan Cheetah)
- *Acinonyx jubatus jubatus* (Southern African Cheetah)
- *Acinonyx jubatus raineyi* (East African Cheetah)
- *Acinonyx jubatus soemmeringii* (Northeast African Cheetah)
- *Acinonyx jubatus venaticus* (Asiatic Cheetah, Iranian Cheetah)

name		origin		meaning
english	cheetah	“chita”	Hindu	spotted one
genus	acinonyx	“akinetos” and “onyx”	Greek	unmovable claws
species	jubatus	“jubatus”	Latin	having mane or crest

Table 1: Nomenclature of the Cheetah species.[3,4]

2.2 Biology

The cheetah is the fastest land animal with a maximum speed of approximately100 km/h and acceleration from 0 to 80 km/h in just 3 strides.[5,6] Therefore its whole body is build rather for speed than for power and shows various adaptions in its musculosceletal system.[5,7–9] It has a lightweight skeleton with a small skull and long foot and leg bones.[5] The M. serratus ventralis

may have the ability to translate the scapula along the rib cage to lengthen the effective foot length even further.[7] The muscle fibers in fore and hind limbs as well as in extensors and flexors show a different composition according to the greater propulsion of the hind limb[10] and the enzyme activities reflect its eligibility for anaerobically based exercise.[11] The M. longissimus is built in a way to allow strong and quick extension of the spinal column and to increase its stiffness during running.[10] Its large eyes are positioned in a way to achieve maximum binocular vision. Needing high quantities of oxygen for high-speed chases its nostrils and sinuses are relatively large. As a sort of compromise the cheetah's jaws are weak, the canine teeth are small and it is therefore poorly equipped for fighting against larger predators than itself.[5]

In contrast to the other members of the family felidae cheetahs are not able to retract their claws.[3,12]

In some populations anatomical abnormalities are seen such as a kink in the last few caudal vertebrae, crowded lower incisors and focal palatine erosion, maybe due to a low level of genetic diversity.[5,13,14]

Older cheetahs seem to be in a poorer physical condition than younger ones, in the males maybe as a result of territorial fighting, in the females because of rearing the cubs. Also the cheetah seems to need a certain amount of physical exercise to maintain its health status leading to a poorer physical condition in individuals kept in captivity for as short a period as 30 days.[5]

Regarding body size and weight cheetahs held in captivity tend to be lighter than their wild counterparts. In the wild East African cheetahs are longer and heavier, followed by the Namibian cheetahs and the Serengeti cheetahs at last.[5]

The general anatomy of the cheetah is similar to that of domestic cats: [3,12]

Body mass:	35-40 kg[3]
Body length (without tail):	125-135 cm[3]
Tail length:	65 cm[3]
Shoulder height:	70-90 cm[3]
Age:	up to 17 years (in captivity)[12]
Full-grown:	> 49 months[5]
Sexual maturity:	2-3 years[15]
Gestation:	90-95 days[12,15,16]

Table 2: Some basic facts about cheetahs

2.3 Genetics

A population genetic survey of over 200 loci of the genome of the South African cheetah (*Acinonyx jubatus jubatus*) in 1994 revealed an extremely low genetic variability, especially in the major histocompability complex (MHC).[17,18] According to the researchers this fact may result in some undesirable consequences, e.g. difficulties in captive breeding (high risk of inbreeding), a high degree of juvenile mortality, a high susceptibility especially to viral infections and a high frequency of spermatozoal abnormalities in the ejaculate.[17]
A different publication claims that there is little evidence for inbreeding depression but other factors as habitat modification, replacement of wild prey with livestock and persecution by people are more reasonable explanations for the population decline in wild cheetahs.[19,20] The lower susceptibility to infectious diseases of free-ranging compared to captive cheetahs may indicate that next to genetic also extrinsic factors may play a part.[21]
However a different opinion is that carnivores in general show significantly lower levels of genetic variation than other mammals and the effects mentioned above may only be “artefacts of captivity”.[22] In case of the juvenile mortality another author claims that the cheetah has higher litter sizes, the highest average number of surviving cubs and the mortality is on the contrary not higher compared to other captive-bred felid species.[23]

2.4 Conservation status

At the moment the cheetah species (*Acinonyx jubatus*) is classified as "Vulnerable (VU)" by the International Union for Conservation of Nature (IUCN).
However the subspecies *Acinonyx jubatus hecki*, native to North-west Africa, is classified as "Critically Endangered (CR)" since 2008, previously classified as "Endangered (EN)" in 1996. *Acinonyx jubatus venaticus*, also known as the Asiatic Cheetah, is classified as "Critically Endangered (CR)" since 1996. It was classified as "Endangered (EN)" since 1986. Today there is only a small population left in Iran.[4,24,25]
A more recent study dating from 2011 revealed, that although for a long time it was believed that only small genetic differences exist between the various cheetah subspecies, there is new evidence that North-east African (*Acinonyx jubatus soemmerringi*), Southern African (*Acinonyx jubatus jubatus*) and Asiatic cheetahs (*Acinonyx jubatus venaticus*) have been long-term geographic isolates and hence have independent evolutionary histories.
To preserve the unique diversity between these subspecies found out in 2011 conservation plans for the cheetah should be adapted in this respect.[20]

3 Diseases

3.1 Notes

- Most cheetahs in captivity are dying due to a combination of gastritis, kidney disease and amyloidosis.[26]
- As a consequence the most important non CNS diseases are gastritis, glomerulosclerosis and amyloidosis.[26]
- The most important CNS disease is myelopathy, responsible for 25% of all deaths.[26]

3.2 Non-infectious diseases

3.2.1 Gastrointestinal diseases

Gastritis

Susceptibility	Susceptible animals: captive cheetahs Prevalence: EEP 81%, North America 99%, South Africa 99%[26–29]
Aetiology	Cause of disease: uncertain, maybe an altered immune response (alterations in the Th1:Th2 balance[30]) to bacteria (e.g. Helicobacter spp.[26–29,31,32], Gastrospirillum-like organisms[27,33]) in combination with chronic stress.[26–28,31,34]
Pathogenesis	Attention: the amount of bacteria does not correspond to severity of infection and the gastritis does not resolve after antibiotic treatment.[26,29,32]
Clinic	Clinical signs: subclinical to mild to severe gastritis: vomiting[1,27,31], regurgitation[1,27], chronic weight loss[1,31], abnormal faeces (presence of undigested meat)[1,31], dull hair coat[31]
Diagnosis	Gastritis:

	• ante-mortem: gastroscopy, gastric biopsy (at least 7 samples[I])[1,29] • post-mortem: necropsy[1,35] Helicobacter spp.: • gastric biopsy: pathohistology (lymphoplasmacytic inflammation, neutrophilic infiltration), impression smears, urease testing[1,28,29,35,36] • serum: antibodies[1,35] • faeces: PCR[1,35] • breathing air: C-urea breath test (UBT)[1,35]
Treatment	only in cheetahs with clinical signs: • "triple therapy": proton pump inhibitor (e.g. lansoprazole or omeprazole) and two antibiotics (e.g. clarithromycin and amoxicillin)[1,36–38] • feeding multiple times per day to provide adequate caloric intake[1,37] • species-specific probiotics (Lactobacillus Group 2, Enterococcus faecium)[39]
Prophylaxis	Stress reduction[1,36], dietary management (predigestion with trypsin[II], feeding of commercial cat food instead of supplemented meat-based diet[31])
Prognosis	Most therapies reduce the clinical signs and the amount of bacteria but only short-term.[1] Gastritis account for 29% of mortality in North American captive cheetahs and 40% in South African captive cheetahs.[31]

[I] Ch. Walzer, personal communication
[II] Ch. Walzer, personal communication

Focal palatine erosion (FPE)

Susceptibility:	Prevalence: 9,4% (captive: 19%, free living: 2,9%), also in animals as young as 7 months old[40], often more severe in young than in adults[41].
Aetiology:	Cause of disease: Uncertain. A genetic component may be possible as 86% of cats with FPE are from the Namibian population.[40,42] An association to mechanical pressure of the lower first molar was suspected but rejected as in 60,6% of the felids studied there was no contact between the molar and the palatine bone![40] Dietary factors, i.e. animals raised on soft commercially prepared diet, have also been considered, believing that this may cause muscle atrophy and furthermore malocclusion. As there is a high prevalence also in animals fed on whole prey diet this theory is implausible.[40]
Pathogenesis	Erosion of the oral mucosa leading to perforation of the palatine bone in advanced stages in about 40%.[40]
Clinic: Clinical signs Types	Clinical signs: In the beginning bloody-mucus nasal discharge[40], halitosis[40], sneezing[40] and coughing[40], in more severe cases also localized osteomyelitis[40], intermittent chronic septicemia[40] with associated systemic consequences finally leading to death[40].
Diagnosis	Diagnostic methods: • Oral examination • Pathology
Treatment	Changing diet[42], trimming the lower molars[40,42], eliminating irritants[42], antibiotics[40,42], surgical reconstruction[40,42].
Prophylaxis	
Prognosis	

Foreign body ingestion

- Cause of disease: Intake of enrichment items may lead to gastrointestinal obstructions.[1]
- Clinical signs: choking, vomiting, anorexia[1]

3.2.2 Renal diseases

Diagnostic auxiliaries

Because of the high prevalence of chronic kidney disease in cheetahs, **radiographs** have been used to determine the measurements of healthy kidneys to facilitate diagnosis. In the domestic cat the normal kidney should be of the length of the body of the second lumbar vertebra. In cheetahs the ratio of kidney to vertebra is about 1,81 +/- 0,14. However some cheetahs with confirmed renal failure showed the same ratio.[43]

In a **ultrasonographic** study of 21 captive cheetahs with normal urea and creatinine values various kidney parameters have been measured. Cortico-medullary distinction could be seen in all individuals and in most of them a cortico-medullary rim sign was present. The mean kidney parameters measured were a length of 63,9 +/- 5,7 mm, a height of 38,1 +/- 5,2 mm and a width of 42,1 +/- 5 mm.[44]

To facilitate the early detection of renal disease the measurement of the **endogenous creatinine clearance** (1,47 +/- 0,2 ml/min/kg body mass) may be used and should provide a reliable estimate of the glomerular filtration rate (1,59 +/- 0,17 ml/min/kg body mass). Standard renal parameters as urea and creatinine are elevated only after a substantial loss of renal function. [45]

Amyloidosis

Susceptibility	Susceptible animals: cheetahs > 1 year[26] Prevalence: EEP 48% (NA 38%, SA 82%)[1,26]
Aetiology	Cause of disease: amyloid deposition in association with chronic disease (glomerulosclerosis, nephrosclerosis, lymphoplasmacytic

	gastritis)[1,46,47] Transmission route: feces may also play a role in the transmission of AA amyloid fibrils[48]
Pathogenesis	systemic Amyloid AA deposition mainly in liver and kidney as well as adrenals, thyroid, spleen, gastrointestinal tract[1,26]
Clinic	Clinical signs: depending on the organ affected and the amount of amyloid deposition (subclinical to organ failure)[46]
Diagnosis	Amyloidosis: • Sample material: organ biopsy[1] • Pathology: organs may be enlarged, moderately firm, abnormally discoloured[46] • Histology: extracellular eosinophilic homogeneous deposits, affinity for Congo red dye, green appearance under polarized light[46,49] • Immunohistochemistry: determination of the amyloid type[46] Renal function: • early detection: ◦ Endogenous creatinine clearance[1,45] ◦ Fractional excretion of electrolytes (i.e. Na, K, P, Ca)[1] • detection of end stage lesions: ◦ BUN/creatinine[1]
Treatment	symptomatic treatment, treatment of underlying chronic disease, dietary management, stress reduction[1]
Prophylaxis	dietary management, stress reduction[1]
Prognosis	

Glomerulosclerosis

Susceptibility	Susceptible animals: cheetahs > 1 year[1,26,29] Prevalence: EEP 80% (NA 67%, South Africa 71%)[1,26,29]
Aetiology	Cause of disease: uncertain, possibly diet or metabolic changes due to chronic stress[1,50]
Pathogenesis	Progressive thickening of the glomerular membrane leads to glomerular ischemia and sclerosis.[26,27,50]
Clinic	
Diagnosis Sample collection	Glomerulosclerosis: • Sample material: kidney[26] • Pathohistology: thickened glomerular membrane[26], interstitial fibrosis[26,29], nephritis[26,29], glomerulonephritis[26,29], calcifications[26,29] Renal function: • early detection[1]: ◦ Endogenous creatinine clearance ◦ fractional excretion of electrolytes (i.e. Na, K, P, Ca) • detection of end stage lesions[1]: ◦ BUN/creatinine
Treatment	symptomatic treatment[1], dietary management[1], stress reduction[1]
Prophylaxis	dietary management[1,50], stress reduction[1,50]
Prognosis	

Oxalatnephrosis

Susceptibility	Prevalence: sporadically in the United States, first described in the EEP population in 2009[51]
Aetiology	Cause of diseases: • Oxalates and oxalate precursors ingested in sufficient quantities (e.g. certain plants, oxalic acid, ethylene glycol)[51–53] • hyperoxaluria as a rare genetic disease in cats[51,54]
Pathogenesis	
Clinic	Clinical signs: Symptoms of acute renal failure (e.g. anorexia, loss of body mass)[51]
Diagnosis Sample collection	• Blood analysis: elevated renal parameters[51] (urea, creatinine, phosphorus, calcium, potassium) • Renal biopsy[51] • Histopathology: acute tubular degeneration associated with large numbers of birefringent crystals[51], renal medullary amyloidosis[51], glomerosclerosis[51]
Treatment	symptomatic treatment[51], dietary management[51]
Prophylaxis	
Prognosis	

3.2.3 Neuromuscular diseases

(Spinal) Myelopathy and encephalopathy

Susceptibility	Susceptible animals: European cheetahs, responsible for 25% of all deaths.[1,26,29,55]
Aetiology	Cause of disease: Unknown, but stress, viral, bacterial, parasitic, genetic, nutritional-metabolic, toxic and physical causes have been

	considered. Antigens of FHV-1, BDV, CPV and CDV could not be found.[1,26,29,55–59]
Pathogenesis	Degenerative lesions of the spinal cord (bilateral symmetrical degeneration of the white matter, myelin and axonal loss) and cerebellum (myelin and axonal loss in the white substance, astrogliosis, microgliosis, degeneration of Purkinje and granular cells)[1,261,251,26,29,55–58]
Clinic	Clinical signs: ataxia, paresis Types: rapid onset of clinical signs vs. slower progressive development (stabilization and acute relapsing episodes)[1,26,29,55–58]
Diagnosis	Sample material: spinal cord[60] Pathohistology: degenerative lesions (see above) Differential diagnosis: FSE[1,26,29,55–58]
Treatment	symptomatic treatment[1,26,29,55–58]
Prophylaxis	
Prognosis	fatal[1,26,29,55–58]

3.2.4Miscellaneous non-infectious diseases

(Myelo)Lipomas

Susceptibility:	Distribution: Multiple myelolipomas of the spleen and the liver are common in cheetahs (EEP 54%, 51% NA, 13% SA)[1,26,29]
Aetiology:	Cause of disease: Unknown, but maybe due to dietary or stress-induced metabolic alterations.[1]
Pathogenesis:	Myelolipomas are benign extramedullary tumor-like nodules of bone marrow[61] and consist of mature adipose tissue and myeloid cells.[62]
Clinic:	The lesions are not clinically important but they should not be

	misdiagnosed as metastatic cancer![1,26,27]
Diagnosis:	Diagnostic methods: • Ultrasound: The hyperechogenic lesions can be found easily.[29] • Necropsy: Often found by chance at necropsy.[62]
Treatment	
Prophylaxis	
Prognosis	

Generalized mastocytosis (mast cell "tumors")

Susceptibility:	
Aetiology:	Cause of disease: Associated with insect bites[1,63]
Pathogenesis:	
Clinic:	Clinical signs: single or multiple firm raised skin masses[1,63]
Diagnosis:	
Treatment:	Do not confound with highly malignant mast cell tumors because mast cell infiltrations in cheetahs generally disappear on their own![1,63]
Prophylaxis	
Prognosis	

Exudative dermatitis with mast cell infiltration

Susceptibility:	
Aetiology:	Cause of disease: maybe allergic[1,63]

Pathogenesis:	
Clinic:	Clinical signs: discomfort, weight loss[1,63]
Diagnosis:	
Treatment:	short term corticosteroids[1,63]
Prophylaxis	
Prognosis	

Peaugres-Syndrome

Susceptibility:	Distribution: One case report about twenty seven cubs of two sibling mothers and on unrelated male born in five litters.[26,29]
Aetiology:	Cause of disease: Unknown, but genetic defect, similar to the human Menkes disease, related to a defect in copper transport proteins, is suspected.[26,29]
Pathogenesis	-
Clinic: Types	Clinical signs: In the reported case twenty six of the twenty seven born cubs died within 134 days of age. The pathological lesions included: poor hair coat, heart malformations, liver fibrosis, stunted growth, osteoporosis, encephalitis.[26,29]
Diagnosis	Necropsy[26,29]
Treatment	-
Prophylaxis	-
Prognosis	Fatal[26,29]

Leukoencephalomyelopathy

Susceptibility	Susceptible animals: only cheetahs in the SSP population, mostly over 10 years old[1,26]
Aetiology	Cause of disease: Unknown, but maybe associated with diet or medical management[1,26]
Pathogenesis	The lesions are restricted to the cerebral cortex and characterized by bizarre astrocytosis and loss of white matter.[1,26]
Clinic	Clinical signs: blindness or visual abnormalities, lack of responsiveness to the environment, behavioural change, incoordination, ataxia, convulsions[1,26]
Diagnosis	MRI: most sensitive ante-mortem diagnostic method[1] Pathohistology: typical lesions (see above)[1,26] Differential Diagnosis: FSE, CDV[1]
Treatment	symptomatic treatment[1,26]
Prophylaxis	
Prognosis	fatal[1]

Ulnar metaphysal osteochondrosis

Over a period of twenty years seven cases of ulnar metaphyseal osteochondrosis, characterized by bilateral carpus valgus conformation in captive-bred cheetahs have been documented. The cause is suspected to be familial or from dietary origin.[64]

Spontaneuously beta amyloid deposition and neurofibrillary tangles

Cheetahs spontaneously may develop neurodegenerative disease similar to human Alzheimer disease manifesting in deposition of beta amyloid and neurofibrillary tangles.[65]

Veno-Occlusive Disease (VOD)

This disease caused by fibrous occlusion of the liver's blood supply has a prevalence of up to 63% of the NA population but no European cheetahs have been affected so far.[1]

3.3 Infectious diseases

3.3.1Prions

Feline Spongiform Encephalopathy (FSE)

Susceptibility	Susceptible animals: domestic cats, captive non-domestic cats (cheetahs, pumas, ocelots, tigers, cougars): cheetahs > 4-5 years[26,66] Distribution: mainly in the UK (first reported case), some cases in France[26,29,66–68] Zoonotic potential: unknown[26]
Aetiology	Cause of disease: prions[26,66] Transmission route: feeding of bovine carcasses infected with bovine spongiform encephalopathy (BSE)[26,29,66], maybe there is also a possibility for vertical, i.e. maternal transmission[66] Incubation period: unknown, but diagnosed animals are mostly between 4-9 years of age[66]
Pathogenesis	pathological accumulation of abnormal prion protein[66]
Clinic	Clinical signs: chronic progressive ataxia (first hind limbs, later fore limbs)[26,29,66,69], postural difficulties[26,69], hypermetria[26,69], muscle tremor[26,66,69] (mainly of the head), behavioural changes (aggressiveness, anxiety)[26,29,66,69], hyperaesthesia to sounds[26,66,69], ptyalism[26,69], blindness[26,69], dilated pupils[66], prominent nictitating membranes[69]
Diagnosis	Sample material: brain[26] (forebrain to the C1 spinal cord segment[66])

	Pathohistology: vacuolation in the neuropil and neurons[26] (HE stain), amyloid plaques (Congo red stain)[66] Immunohistochemistry
Treatment	-
Prophylaxis	no feeding of infected bovine carcasses[26]
Prognosis	fatal[26], death occurs after 6-8 weeks[66]

3.3.2 Viruses

Feline Corona Virus (FCoV)

Susceptibility:	Susceptible animals: Although it has been suggested that cheetahs, due to intensive breeding and a genetic bottleneck, had become more susceptible to viral infections generally and to develop fatal FIP because of their genetic homogeneity of the major histocompability complex[70,71], the example of the North American SSP population with a FCoV prevalence of at least 50% but extremely rare outbreaks of FIP shows that FIP is very rare in cheetahs and seems to be no major concern at the moment.[1]
Aetiology:	Cause of disease: At the moment two types of FCoVs are known: Feline enteric coronavirus (FECV) and Feline infectious peritonitis virus (FIPV). Transmission route: faecal-oronasally. Asymptomatic FCoV shedders may be a great risk of infection to other cheetahs.[71–73]
Pathogenesis	Regarding to the "in vivo mutation" theory relatively harmless FECV mutates to FIPV which replicates in macrophages. Complex immune reactions between the FIPV, antibodies and the complement system lead to a disseminated vasculitis. • Effusive or "wet" form: In cats with a poor cell-mediated

	immune response the infection leads to an immune complex vasculitis causing protein-rich fluid to diffuse from the blood vessels into different compartments, e.g. into the abdominal cavity causing ascites that manifests in a distended abdomen. Furthermore pyogranuloma and fibrinous plaques on abdominal serosal surfaces can be found.[72,74] • Non-effusive or "dry" form: In cats with partial cell-mediated immunity the infection results in (pyo)granulomatous lesions in multiple tissues. The dry form may develop to the wet form when the immune system is about to collapse.[72,74]
Clinic:	Clinical signs: • FECV: asymptomatic to mild transient diarrhoea, rarely associated with chronic ulcerative colitis.[71,72,75] • FIPV (domestic cats): ◦ Effusive or "wet" form: Dyspnoea, mild pyrexia, muffled heart sounds, uveitis, keratic precipitations, changed colour of the iris[72], effusions, antibiotic non-responsive fever, anorexia, depression, lethargy, weight loss[74] ◦ Non-effusive or "dry" form: symptoms are vague, have a slow onset and depend on the organs affected.[74]
Diagnosis	• Screening for FCoV: Use both, serology and RT-PCR (5 consecutive faecal samples)[1,76] • Screening for persistent shedders: Once an individual is tested positive in the PCR it should be retested monthly (3 consecutive faecal samples) for 6 months.[1] • Diagnosis of FIP: Additionally to clinical signs, serology and PCR a FIP diagnosis should be confirmed both by histopathology (the "gold standard") and

	immunohistochemical staining of FCoV antigen in tissue.[72] Diagnostic methods: • Medical history • Clinical signs: see above • Histopathology ("gold standard", H&E staining): ◦ Vasculitis with central necrosis and perivascular infiltration with macrophages, neutrophils, lymphocytes and plasma cells is characteristic for ("wet") FIP.[72,77] ◦ Focal accumulations of inflammatory cells and fibrinous necrotic-proliferative lesions are characteristic for "dry" FIP.[72] • Pathology: Lesions similar to those of the domestic cat: fibrinopurulent pleuritis, peritonitis, vasculitis, multifocal necrosis of many organs.[71] • Antigen detection: ◦ Methods: ▪ Immunohistochemical staining (tissue): detection of FCoV. This method also cannot differentiate between FECV and FIPV. Nevertheless FIPV replicates more actively than FECV leading to higher concentrations of viral antigen as well as there are also higher concentrations of antigen in "wet" FIP.[72,73] ▪ Electron microscopy (faeces): detection of FCoV[72] ▪ Reverse transcriptase polymerase chain reaction (RT-PCR) (faeces): detection of FCoV. For cheetahs is recommended to test five consecutive faecal samples for a 90% probability of finding a FCoV shedder.[71,72]

	◦ Attention: ▪ These methods cannot distinguish between FECV and FIPV! ▪ The shedding status does not help to predict if the animal will develop the disease. ▪ Because many healthy cats also shed FCoV in their faeces it is only possible to screen for FCoV-carriers and to estimate the prevalence of FCoV in a population. ▪ There may be transient, intermittent or persistent shedders of FCoV. Mistimed, shedders may not be recognised leading to false negative results.[71,72] • Serology (antibody detection): ◦ Methods: ▪ Antibody titres (serum): detection of antibodies[72,78] ▪ Competitive ELISA (serum, effusions): detection of antibody-antigen-complexes[72,79] ◦ Attention: ▪ Serology cannot distinguish between FECV and FIPV![71,72] ▪ The presence of serum antibodies implies only a previous not a current infection with FCoV.[71] ▪ Seropositivity does not correlate with faecal shedding of FCoV.[71] ▪ Healthy individuals may have antibodies against FCoV and sick cats with the “wet” form of FIP may have low or absent antibody titres because of the formation of antigen-antibody-complexes or the loss of antibodies

	into effusions.[73] • Additional diagnostic methods: ◦ Effusions: ▪ clear yellow, viscous, fibrinous fluid ▪ modified transudate to exudate • decreased albumin-to-globulin ratio, increased protein concentration • moderate cellular content: macrophages, neutrophils (lymphocytes, plasma cells) ▪ Rivalta-Test: precipitation because of the high protein concentration: • false positives: lymphoma • false negatives: bacterial peritonitis ▪ Immunofluorescent staining of intracellular FCoV antigen in macrophages: PPV 100%, NPV 57%[72,79] ◦ Haematology and blood chemistry: ▪ lymphopenia, neutrophilia: can also be interpreted as "stress leukogramm" ▪ decreased albumin-to-globulin ratio (increased total serum protein concentration, mainly γ-globulins) ▪ increased acute phase protein concentrations ◦ X-ray: effusions (pleural, pericardial, peritoneal), hepatomegaly, renomegaly, abdominal mass lesions because of mesenteric lymphadenopathy ◦ Ultrasonography: abdominal fluid, evaluation of pancreas, liver, lymph nodes, kidney ◦ MRI: periventricular contrast enhancement, ventricular dilation, hydrocephalus

Treatment	• Healthy but FCoV positive: No specific treatment, avoidance of stress.[73] There is absolutely no reason for euthanasia only because of a positive FCoV test result without any clinical signs! • FECV enteritis: Self-limiting in domestic cats. Supportive care (fluid therapy, diet).[73] • FIP: Domestic cat: Relatively high doses of immunosuppressive and anti-inflammatory drugs may slow down the progression of the disease. An effective antiviral treatment against FIP has not been found yet.[73]
Prophylaxis	Prophylaxis: • Try to keep potential carriers (e.g. feral cats) away. • The safety and efficacy of an intranasal administered vaccine producing local immunity is still under discussion in domestic cats and therefore there is no recommendation for the vaccination of cheetahs.[12,73] Management implications: • At the moment identification of all shedders seems to be impractical with the available methods. Therefore eradication of the virus from a population seems unreasonable especially when restricting the choice of potential breeding pairs.[71] • It may be a more feasible approach to manage a population as endemically infected with FCoV. Therefore individuals tested positive should not automatically be excluded from exchange between institutions. In case of genetically valuable animals the use of artificial reproduction techniques should be considered.[1,27,71] • Persistent shedders should be removed from breeding

	facilities, housed away from other susceptible animals and kept as exhibit animals only.[1]
Prognosis	Fatal once FIP has developed

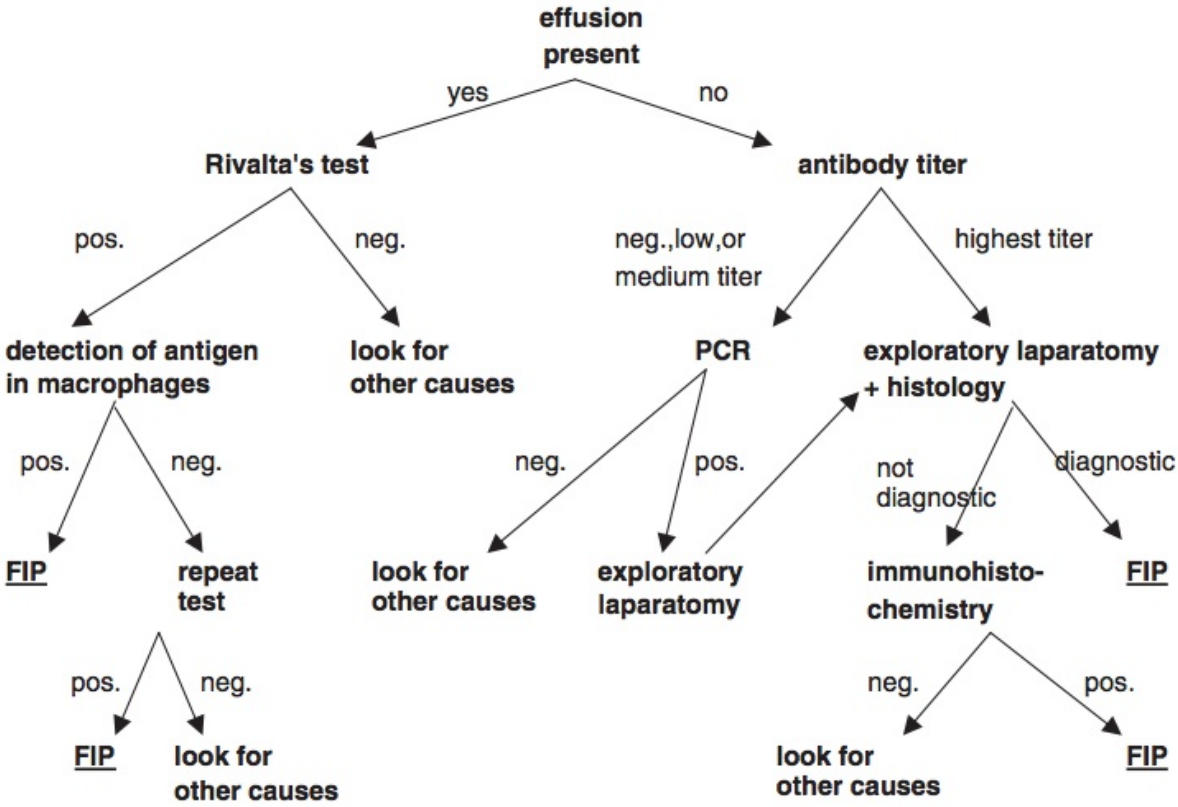

Abbildung 1: Algorithm for the diagnosis of FIP in domestic cats[73]

Feline Herpes Virus (FHV), Rhinotracheitis

Susceptibility:	Distribution: widespread in the SSP population, possibly ubiquitous[1]
Aetiology:	Cause of disease: Cheetah Herpes Virus (ChHV), a strain of Feline Herpes Virus-1 (FHV-1)[1,12,29,80] An unusual reaction in form of ulcerative and eosinophilic dermatitis in chronic carriers suggests a propensity of cheetahs for a Th2-

	dominant response to FHV that is suspected to be heritable or a stress response.[81,82] Transmission route: direct contact but transmission by flies, animal handlers or feed containers may also be possible[80]
Pathogenesis	In neonatal cubs inadequate passive transfer of antibodies together with infection by their own mother may be the main problem. In adult cheetahs confinement stress together with severe virus challenge may contribute to FHV infections despite regular vaccination.[80,81]
Clinic:	Clinical signs: listlessness[80], sneezing[1,29,80], watery eyes[1], nasal discharge[80,83], ocular discharge[80,83], salivation[80], anorexia[80], ulcerative rhinitis[80,83], ulcerative conjunctivitis[29,80,83], Pneumonia[80] Types: • neonatal cubs: o < 2 weeks: often develop the worst and most persisting lesions![1] o a small percentage dies from acute infection, mostly from FHV-associated pneumonia[1] o some may develop severe and persistent lesions as corneal ulcers/scars[1], chronic keratitis[1], blindness[1], prolapsed third eyelids[1], chronic epiphora[1], ulcerative dermatitis[1,84,85] • > 1 month: mostly self-limiting[1] • chronic carriers: o all infected animals become chronic carriers of FHV and some of them may have a recrudescence![1] o seldom severe ulcerative (and eosinophilic[82]) dermatitis or non-resolvable ocular signs (prolapsed

	third eyelids, corneal scars) develop[1]. Ulcerative and eosinophilic dermatitis expresses itself as erythematous, ulcerated plaques primarily on the face and forelegs, sites that are exposed to lacrimal and salivary secretions.[82]
Diagnosis	• Sample collection: blood serum[80], conjunctival[1], nasal[1,80] or oropharyngeal[1] swabs, conjunctival biopsies[1] • Antibody detection: indirect fluorescent antibody test (IFA)[80], unfortunately no differentiation between infection with field or vaccine virus Antigen detection: • PCR on swabs or biopsies followed by sequencing[1] (recommended). Treatment with corticosteroids may increase the detection of low level or latent shedders![1] • cytopathic effects (CPE) on host cells[80], electron microscopic examination of cell culture fluid[80], direct fluorescent antibody test[80]
Treatment	• Antibiotics (e.g. amoxicillin, doxycycline) for prevention of secondary bacterial infection.[1] The symptoms should disappear after 2 weeks.[80] • Interferon may be helpful.[1] • Antiviral drugs: o Acyclovir and such are not recommended! Besides they may carry some risk of bone marrow suppression, especially in young cubs![1] o Eamciclovir seems to be useful in domestic cats and may be used safely in cheetahs[1,III]

[III] Haefele, personal communication

	• Treatment of the eyes: Some products seem to be painful (e.g. triflurindine), but compounded preparations of idoxyuridine or cidovovir may be useful.[1] • Lysine as a dietary supplement may reduce lesions as well as shedding of the virus.[1] • Cryotherapy may be an effective treatment of skin lesions.[1,82]
Prophylaxis	Prophylaxis: • Unfortunately immunity acquired after vaccination is relatively short-lived and together with viral latency effective prophylaxis against FHV can be difficult.[1,80] • Nevertheless vaccination of females before breeding and 2-3 weeks before cubbing may enhance colostral antibody transmission.[1] • Stress reduction[1] Management implications: • Evidence of FHV in a cheetah population should be no reason against movement between institutions. Populations should be managed as infected and efforts should be made to minimize virus transmission with other susceptible species (e.g. Pallas cat).[1] • Because cheetahs may shed the virus for a unknown long time it is recommended to keep them isolated for at least 7 days (or longer if possible) after all lesions and symptoms have cleared.[1] • The virus is susceptible to high temperatures and a dry environment[80] and should probably be viable in the environment for less than 72 hours.[1] • During an outbreak recording of the progression and lesions

	developed is important, as well as minimizing the contact to susceptible animals and implementation of maximum hygiene methods for people in contact with infected animals (separate clothes, boots, gloves and tools, foot baths, care taking for the affected animals at the end of the day or keepers solely working with sick animals).[1] • In collections with repeated or severe cases it may be useful to separate the cubs from their mothers and to hand raise them to protect them from infection[1] or help cubs already infected to recover.[83]
Prognosis	self-limiting disease to chronic, persistent lesions[1,80]

Feline/Canine Parvo Virus (FPV/CPV) (Feline Panleukopenia)

Susceptibility:	Distribution: Due to vaccination parvovirus infections have been reduced greatly.[12] Most cases are caused by CPV-2b because vaccination at the moment only uses FPV strains.[1,12,86]
Aetiology:	Cause of disease: especially CPV-2b, but also FPV[1,86] Transmission route: direct contact, fomite, aerosols[12]
Pathogenesis	Features: highly contagious, persists for at least 1 year in the environment[12]
Clinic: Types	Clinical signs: • anorexia[12], depression[12], vomiting[12], mild diarrhea[1,86,87], mild necrotizing enteritis[1,86], dehydration[12], leukopenia[12], neonatal deaths in cubs[1] • cubs of exotic felids may also develop cerebellar hypoplasia and hydrocephalus[12]
Diagnosis	Sample collection: blood serum[1], faeces[1]

	Antibody detection: rising antibody titres in paired serum samples[1] Antigen detection: ELISA[1], virus isolation[1], electron microscopy[1,871], real-time PCR[1] (faeces)
Treatment	Supportive: antibiotics for prevention of secondary bacterial infection[12], fluid therapy[12] for treatment of fluid loss due to diarrhoea, nutritional support[12]
Prophylaxis	Vaccination (see also 5.1.3)
Prognosis	mild to neonatal deaths

Feline Leukemia Virus (FeLV)

Susceptibility:	Distribution: • Subclinical infections have been reported rarely in captive cheetahs of the SSP population. There is only one known case of viral-associated lymphoma in a Namibian cheetah.[1,88] • The CCF tested over 100 wild Namibian cheetahs and did not find one case of FeLV. Of over 100 cheetahs kept in captivity tested 4 appeared to be seropositive. Therefore FeLV does not seem to be endemic in the wild in Namibia but on the other hand cheetahs are susceptible to this disease. Furthermore cheetahs seem to be particularly susceptible to some viral infections like FeLV because of reduced levels of MHC diversity and they tend to develop exuberant immune responses to some infections rather than immunosuppression.[88] • In domestic cats this disease is distributed worldwide with varying seroprevalence.[89]
Aetiology:	Cause of disease: Feline Leukemia Virus (FeLV), a retrovirus which

Incubation period	produces the enzyme reverse transcriptase that helps to infiltrate the infected cells in form of a provirus that is inserted the host cell genome.[89] Additional stress (e.g. relocation, concurrent disease, immunosuppressive drugs) may aid to development of disease.[88] Transmission route: • Saliva[89], nasal secretions[89] and blood[88] (e.g. via grooming[88,89], sharing of water and food[88,89] or fighting[88,89]) are most important. • Transplacental, lactational and venereal transmission is less important.[89]
Pathogenesis	The virus replicates in the oropharynx and after that is disseminated throughout the body to the bone marrow. Is the bone marrow infected persistently epithelial structures like salivary and lacrimal glands are infected via white blood cells and platelets.[89]
Clinic:	Clinical signs (domestic cats): • Infected felids show nonspecific signs of immunodeficiency[12] like anorexia[89], weight loss[89] and depression[89]. • Cheetahs may react exuberantly to an infection rather than to appear immunosuppressed.[88] • In domestic cats the clinical signs vary from ocular, gastrointestinal, respiratory, urogenital, neurologic to orthopaedic symptoms.[89] • In domestic cats the most common forms of neoplasia include mediastinal, multicentric and alimentary lymphomas.[89] Types: • Abortive: Successful elimination of the infection[89] • Progressive: Development of clinical illness and persistent viremia[89]

	• Regressive: Antigen negative and lower transiently positive real-time PCR results[89] • Latent: Transiently antigen positive and persistently positive real-time PCR results[89] Regressive and latent FeLV infections may be activated by administration of glucocorticoids or other immunosuppressive drugs![89]
Diagnosis Sample collection	Cheetahs: • Necropsy: In a cheetah findings included enlargement of all lymph nodes[88], splenomegaly[88] and hepatomegaly[88]. Domestic cats show neoplastic lesions[89] and lesions of nonneoplastic diseases.[89] • Histology: effacement of lymph node architecture[88], marked infiltration of liver and spleen by malignant lymphocytes[88] as well as in kidney, lung, tonsil, salivary gland, thyroid, trachea and bone marrow. • Immunohistochemistry: stains for B- and T-cell markers[88] Domestic cat and cheetahs: • ELISA[88,89] (serum or whole blood, plasma, saliva, tears): positive before infection of bone marrow but only after a delay of to 2 weeks after the onset of viremia.[89] Mainly domestic cat: • Haematology: nonregenerative anemia[89], lymphopenia, neutropenia, thrombopenia[89], increase in red blood cells or macrocytosis[89] • Bone marrow examination: erythrodysplasia[89] • Biochemistry: Azotemia[89], hyperbilirubinemia[89], increased activity of liver enzymes[89]

	• Urine: Bilirubinuria[89], proteinuria[89] • Imaging: mass lesions depending on the organs affected[89], pleural effusions[89], obstructive intestinal patterns[89] • Cytology (tissues, peripheral blood smears, effusions, cerebrospinal fluid): malignant lymphocytes.[89] • IFA (neutrophils, platelets): only positive, if bone marrow is infected.[89]
Treatment	Domestic cat: • Reverse transcriptase inhibitor AZT: effect in domestic cats is questionable.[89] • Interferon[89] • Chemotherapy: for domestic cats with neoplasia[89] • Supportive therapy: hematinic agents, vitamin B_{12}, folic acid, anabolic steroids, erythropoietin, blood transfusion[89] • Immunosuppressive therapy: may help with autoagglutinating haemolytic anaemia but may activate virus replication.[89]
Prophylaxis	• Because of the scarcity of this illness there is no need for vaccination at the moment besides they have close contact with feral cats or other potentially infected wild felids.[1,88,90] • Nevertheless testing blood sera of all cheetahs and isolation of infected animals is recommended![1,88,90] • As well as in domestic cats it is recommended that fomites like water bowls and litter pans should not be shared by seropositive and –negative cats.[89] • In the only confirmed case of FeLV-associated lymphoma the virus was most probably transmitted via saliva through the fence separating two cheetahs because the risk of transmission was thought to be low at that time. Maybe therefore housing

	of known seropositive animals next to seronegative animals should be omitted.[88]
Prognosis	• In the only confirmed case of FeLV-associated lymphoma in non-domestic felids the cheetah died within three months after the first clinical signs.[88] • Persistently viraemic domestic cats die within 2 to 3 years.[89]

Feline Immunodeficiency Virus (FIV)

Susceptibility:	Susceptible animals: Cheetahs seem to be infected only rarely with FIV[1], e.g. there is a lower incidence in cheetahs (about 20%) compared to lions (about 73%) maybe due to different life-style (very social vs. solitary). Also it seemed to be that these lentiviruses evolve independently within their host species, as there is no relationship between lentivirusus of pumas (PLV) and domestic cats (FIV).[91] However a more recent study revealed that pumas and bobcats in Southern California share a FIV strain.[92] So cross-species transfer of lentiviruses may be implausible but possible under certain circumstances.[91,93] Distribution: The virus seems to be restricted geographically in cheetahs, as no serum antibodies could be found in Namibia, Serengeti and Kruger National Park populations.[91]
Aetiology:	Cause of disease: Feline immunodeficiency virus (FIV)[91] Transmission route: direct contact (e.g. fighting, sexual contact, from mother to offspring)[92]
Pathogenesis:	FIV in domestic cats causes a lethal disease due to destruction of T-lymphocytes making the host incapable of dealing with infections. Interestingly such impairment could not be documented in non-domestic felid species.[91]

Clinic:	Clinical signs: No reported signs in cheetahs.[1,91]
Diagnosis:	Serology
Treatment	
Prophylaxis	
Prognosis	

Canine Distemper Virus (CDV)

CDV is widespread in cheetahs in some regions! Serum antibodies could be confirmed in Namibian cheetahs and in European cheetahs. So far there is no evidence for clinical disease. Vaccination would be possible but because of the low risk it is not recommended at the moment.[1]

Cowpox

Susceptibility:	Susceptible animals: Carnivora of the family Felida, non-human primates, cows, exotic herbivores (giraffes, okapis, elephants, rhinos, llamas, alpacas, edentates)[63,90,94,95] Hosts: wild rats, voles, mice[63,90,95]
Aetiology:	Cause of disease: Cowpox-virus (orthopox-virus) Transmission route: direct contact, biting Incubation period: 1-3 weeks[90]
Pathogenesis	
Clinic:	Dermal form: ulcerated skin lesions (possibly only visible on the face unless the hair is parted), oral lesions[95] Pulmonary form: rejection of food, lethargy, fever, frequent breathing, paroxysmal cough, cyanosis of mucous membranes, wheezing with open jaws[94]

Diagnosis	Sample collection: lungs, pleural exsudate, skin[94,95] Diagnostic methods: • Virusisolation (lungs, pleural exsudate, skin)[94,95] • Transmission Electron Microscopy[90,94] • Serology[90,95] • PCR[90] • Pathology: mucosa of the respiratory tract reddened with petechia, lung oedema, lungs filled with exsudate of gelatinous consistency or fibrin, large volumes (up to 3 litres) of pleural exsudate[94] • Histology: Intracytoplasmatic inclusion bodies[90,95]. Lungs: plethora, infiltration of alvealoar tissue with cells, exsudate. Liver, kidney, spleen: plethora. Brain: edema[94]
Treatment	Antibiotics against secondary bacterial infections[90,95]
Prophylaxis	• Control of wild rodents[90] • In case of an outbreak: keep potentially infected animals separated from healthy animals, preferably in an environment easy to clean and desinfect[90,95] • Vaccination: Smallpox vaccine did not seem to lead to a significant response of the cats' immune system[95]
Prognosis	• Dermal form: depends on severity of infection, recovery to death[95] • Pulmonary form: fatal (death after clinical signs for 5-8 days)[94]

Miscellaneous viral diseases

- In an US breeding facility probably the first outbreak of astroviral diarrhea has been reported in a group of cheetahs. All animals recovered after a few days treatment with

bismuth subsalicylate tablets.[96]

3.3.3Bacteria

Clostridium perfringens

Susceptibility:	
Aetiology:	Cause of disease: Gram positive, anaerobic, spore-forming bacteria Clostridium perfringens.[97]
Pathogenesis	The bacteria produce enterotoxins which insert themselves in the intestinal epithelium causing the formation of blebs and alter the ion fluxes and permeability of the membranes causing damage in the epithelium. This leads to enhanced secretion of fluids into the lumen and to dying and sloughing of epithelial cells.[97]
Clinic:	Clinical signs: Chronic, intermittent to continuous, bloody, mucoid diarrhoea without evidence of systemic illness or weight loss, occasionally tenesmus[97]
Diagnosis Sample collection	Diagnostic methods: • Pathohistology (endoscopic colon biopsy): colitis with the presence of spiral bacteria[29,97] • Anaerobic faecal cultures[97]
Treatment	Tylosin, metronidazole, psyllium fiber[97]
Prophylaxis	
Prognosis	

Haemobartonella felis (Mycoplasma haemofelis)

Susceptibility:	Susceptible animals: In domestic cats the prevalence may be up to

	25%. For a long time this bacteria has not been found in non-domestic felids. Clinical disease in non-domestic felids may be underreported or non-domestic felids may be more resistant to this disease than domestic cats.[98] In the meantime there have been reports on haemoplasma infections in a variety of cat species such as Lynx, European wildcat and African lions.[99,100] 2013 there has been the first report of a haemotropic mycoplasma infection in a free-ranging cheetah in Namibia.[99]
Aetiology:	Cause of disease: Gram negative bacteria Haemobartonella felis (Mycoplasma haemofelis), genus mycoplasma, appears rod-, ring- or coccoid-shaped in blood smears.[98] Transmission route[98]: • vectors: blood-sucking arthropods like fleas and ticks • vertical: in utero, during parturition, through lactation • horizontal: infected blood, iatrogenic by blood transfusion or infected needles
Pathogenesis	The bacteria are epicellular on red blood cells and cause, depending on the severity of the infection, regenerative or non-regenerative anemia.[98]
Clinic:	Domestic cats[98]: • Clinical signs: Anorexia, depression, lethargy, weakness, splenomegaly, cyclic fever, pale mucous membranes, icterus, weight loss and ultimately death • Infections with H. felis is commonly associated with FIV, FeLV and FIP. Non-domestic felids[98]: • Clinical signs: Infection seems to be rare and associated only

	with little or no clinical disease at all. The first reported infected cheetah lived almost four years after the initial sampling and may therefore not have been impaired by the infection.[99]
Diagnosis	Diagnostic methods[98]: • Complete blood count • Peripheral blood smears (different stains are used e.g. acridine orange and Giemsa) • Immunofluorescence • Western Immunoblot analysis • PCR (recommended)
Treatment	At the moment it is unknown if non-domestic cats need treatment at all.[98]
Prophylaxis	
Prognosis	

Anthrax

Susceptibility: Distribution	Susceptible animals: Primarily ruminants, humans, primates and ocassionally other species, e.g. cheetahs[90,101–103] Distribution: worldwide, enzootic in Namibia, Botswana, Simbabwe[90] Zoonotic potential: yes! If an animal is found dead under suspicious circumstances and blood is draining from its body orifices or there is bloating of the abdomen in ruminants or the head in carnivores do not open the body before taking special precautions to avoid spreading of the agent.[104]
Aetiology:	Cause of disease: Gram-positive bacteria Bacillus anthracis[90], genus bacillus, rod-shaped, endospore-forming

	Incubation period: 3-5 days[90] Transmission route: percutaneous[90,104], perorally[90,101–104], aerosol[90,104], direct contact[104]
Pathogenesis	
Clinic: Types	Clinical signs: • Cheetahs: tachypnoe[102], vomiting[102], apathy[102], death[29] • Humans: cutaneous ulcers[102], intestinal anthrax[102], pulmonary anthrax[102]
Diagnosis	• Blood smears[102] • Cultivation[102] • Ascoli-reaction[102] • PCR[102] • Pathohistology: lung edema[102], bloodtinged hydrothorax
Treatment	Antibiotics (penicillin[90], tetracyclin[90]), antitoxic treatment (chloroquine[90])
Prophylaxis	Vaccination[105]. Unfortunately the naturally acquirement of antibodies is uncommon in cheetahs.[106]
Prognosis	Fatal, if not treated[90,104]

Miscellaneous bacterial diseases

- Bartonella (agent of the “cat-scratch-disease”) infections mainly asymptomatic in felids have also been reported in cheetahs.[107,108]
- There has been a report on probable cases of ehrlichiosis in cheetahs resting upon inclusion bodies in lymphocytes, characteristic clinical signs and responsiveness to enrofloxacin and imidocarb treatment.[109]
- There is also a reported case of Bronchopneumonia after Pasteurella spp. infection within the EEP population.[29]

- Campylobacter spp. and Salmonella spp. are regularly isolated in cases of diarrhoea.[29]

3.3.4Fungi

Dermatophytes ("Ringworm")

Susceptibility:	Zoonotic potential: Zoonosis, exposure especially likely from hand-reared cubs.[1]
Aetiology:	Cause of disease: Microsporum spp., Trichophyton spp.
Pathogenesis	
Clinic:	Clinical signs: hair loss[1,1101,1091,1081,1061,1051,1031,1021,1001,110], scarring[110] Types: • subclinical[110] • mild and superficial[110] • highly inflammatory reaction with extensive scarring and hair loss[1,110], formation of granulomas[110]
Diagnosis	Sample collection[110]: • skin: scrape samples near the edges of the rings with a scalpel • hair: plucking • skin samples and hairs should be mixed to 10-15% KOH Sample transportation[110]: in a dry packet Diagnostic methods[110]: • Wood lamp: infected hairs may fluoresce, but do not have to • Light microscopy: Be aware that up to 50% are false negative! • Culture (colony pigmentation, texture, morphological structure) • Serology: often difficult • Molecular biology: mostly PCR based
Treatment	Similar to the domestic cat:

	Griseofulvin (may be used but can cause side effects[111], see also 6.1), itraconazole, terbinafine. The effectiveness of lufenuron was not substantiated in control studies.[112] Topical treatments include lime sulphur (1:6), 0,2% enilconazole rinses and 2% miconazole/chlorhexidine shampoo.[112]
Prophylaxis	
Prognosis	

Miscellaneous mycotic diseases

- Systemic candidiasis[113] (incidental finding after euthanasia of an geriatric individual)
- Cryptococcus neoformans infection[114–116] (granulomas in the lung, cryptococcal meningoencephalitis).

3.3.5Parasites

Toxoplasmosis

Susceptibility:	
Aetiology: Incubation period	Cause of disease: Toxoplasma gondii[117], additional stress (worms, weaning, etc.) may enhance the risk for development of disease.[1] Transmission route: in utero, faeces, infected intermediate hosts (mice, meat)[1]
Pathogenesis	
Clinic:	Clinical signs and types: • subclinical • acute: anaemia, retinitis, iritis, hepatitis, blindness, central nervous disorders, respiratory distress, diarrhea[1] • primary acute disseminated toxoplasmosis: rapidly progressive pyrexia[117], tachypnea[117], abdominal effusion[117],

	hepatomegaly[117]
Diagnosis	Diagnostic methods: • Faeces analysis (flotation): oocysts[1] • Histopathology[1] • Serology • Immunohistochemistry[1,117] • PCR[117]
Treatment	
Prophylaxis	
Prognosis	

Dirofilaria immitis

Susceptibility:	Susceptible animals: canids, felids, ferrets Zoonotic potential: Worms may reach the pulmonary artery, die and may cause pulmonary emboli and granuloma, leading to clinical signs as coughing and dyspnoea. Distribution: Tropical to warmer regions[118]
Aetiology:	Cause of disease: Dirofilaria immitis Transmission route: Sting of mosquitoes infected with Dirofilaria immitis, transplacental transmission of microfilariae. Larval development within mosquitoes is only possible at temperatures above 17 °C. Incubation period: 6-9 months, microfilariae can survive for years within the definitive host[118]
Pathogenesis	Life cycle: • Intermediate host (mosquito): ingestion of microfilariae with blood meal, development of larvae L3 within 10-14 days

	• Definitive host: Mosquito sting, larvae L3 enter through skin wound, development of larvae L4 in skin, migration to muscles and subcutaneous tissue, development to immature adults within 60-70 days, migration to pulmonary arteries 3-4 months post infection, maturation to adults, production of microfilariae; accidental migration to brain and eye possible[118]
Clinic:	Clinical signs (domestic cat): • Infection may be subclinical or resulting in clinical signs as coughing, breathing difficulties, retching, vomiting (unrelated to feeding), anorexia, weight loss, lethargy as far as sudden deaths. • Clinical signs may be associated with arriving of immature adults in the pulmonary arteries or death of the adult worms.[118]
Diagnosis Sample collection	Diagnosis may be difficult: • Antigen tests may fail because of low worm numbers present or the inability of test systems to detect immature or male worms. • Antibody tests for domestic cats may be used for screening purposes. • Post-mortem: white, slender worms, 12-30 cm in length may be found in Aa. pulmonales, heart, V. cava caudalis[118]
Treatment	see prophylaxis
Prophylaxis	Ivermectin, Moxidectin, Milbemycin at standard feline doses in endemic areas[12], e.g. 0,1-0,2 mg/kg monthly[1,IV]
Prognosis	

[IV] S. Citino, personal communication

Miscellaneous parasitic diseases

- Massive infections with ascarids (Toxascaris spp., Toxocara spp.) are frequently seen despite to regular deworming.[12,29]
- A stomach worm, Ollulanus tricuspis, caused vomiting, gastritis and weight loss in captive South African-born cheetahs.[119]
- Also lungworms (Aelurostrongylus spp.) are detected regularly in faeces, sometimes leading to pneumonia.[29]
- In the wild cheetahs may be infected with blood protozoans such as Hepatozoon spp. and Theileria spp.[120,121] In 2010 a novel Babesia species, Babesia lengau sp. nov. has been described in South African cheetahs with a prevalence of 28,5%.[122]
- In the wild the prevalence of Sarcoptes mite infection in cheetahs (12,77% in the Masai Mara) is associated with the climatic conditions on one hand and on the other hand with the prevalence of mites in the prey species, e.g. Thomson gazelles and wildebeest. Therefore a prey-to-predator parasitic infestation scheme is suspected.[123,124]

3.4 Miscellaneous pathological findings

- Cutaneous[29]
    - invasive fibrosarcoma on the neck
    - focal eosinophilic granuloma on the nose
- Gastrointestinal[29]
    - suppurative gastritis
    - biliary adenocarcinoma
    - pancreatic cysts
- Respiratory[29]
    - focal bronchoalveolar carcinoma
- Urogenital[29]

- chronic cystitis
- paraovarian cysts
- uterine fibroleiomyoma[125]

3.5 Stress response

The non-invasive measurement of adrenocortical function provides an important tool in stress assessment of captive animals like cheetahs because a lot of health problems seem to be closely associated with stress.[126–128]

Radio and enzyme immnuoassays may be used to measure fecal glucocorticoid metabolites and steroids such as cortisol, corticosterone, estradiol, progesteron and testosterone.[126,127] Comparing the results of free-ranging and captive cheetahs, baseline concentrations of fecal corticoids were significantly higher in captive individuals. Furthermore the testosteron levels were lower in captive male cheetahs. In this study the same effect could not be detected in the female group as no suppression of estradiol concentrations could be measured. There is also morphologic evidence manifesting in larger corticomedullary ratios of the adrenal glands.[128]

Fecal excretion of cortisol was used also used to draw conclusions from ovarian activity in cheetahs. High cortisol levels (about 200 ng/g feces) corresponded with the most nervous individuals that may be compromised in their ovarian cycling. The reproducing females showed low to intermediate cortisol levels.[129]

The movement of animals may also result in stress. Cheetahs with low baseline corticoid concentrations showed a greater response to movement than animals with initially high baseline levels. Concentrations tended to rise in animals moved on-exhibit whereas decreased in animals changing to off-exhibit.[130]

4 Diagnosis

4.1 Standard values

4.1.1Haematology

Parameter	Value
White blood cell count (x 10^3/µl)	10,35 +/- 3,5
Red blood cell count (x 10^6/µl)	6,84 +/- 1,06
Haemoglobin (g/dl)	12,5 +/- 1,9
Haematocrit (%)	37,9 +/- 5,8
MCV (fl)	55,6 +/- 5,5
MCH (pg/cell)	18,3 +/- 1,7
MCHC (g/dl)	33 +/- 2,6
Platelet count (x 10^3/µl)	349 +/- 119
Segmented neutrophils (x 10^3/µl)	6,998 +/- 2,739
Neutrophilic bands (x 10^3/µl)	0,375 +/- 0,79
Lymphocytes (x 10^3/µl)	2,033 +/- 0,983
Monocytes (x 10^3/µl)	0,339 +/- 0,314
Eosinophils (x 10^3/µl)	0,861 +/- 0,809
Basophils (x 10^3/µl)	0,083 +/- 0,175

Table 3: Haematology values for cheetahs in captivity[12,131]

4.1.2Serum chemistry

Parameter	Value
Calcium (mg/dl)	10,6 +/- 0,8
Phosphorus (mg/dl)	5,9 +/- 1,8
Sodium (mEq/l)	157 +/- 5
Potassium (mEq/l)	4,4 +/- 0,5

Chloride (mEq/l)	122 +/- 4
Bicarbonate (mEq/l)	18,5 +/- 2,7
Carbon dioxide (mEq/l)	23,9 +/- 11,5
Osmolarity (mOsm/l)	325 +/- 14
Iron (µg/dl)	52 +/- 22
Blood urea nitrogen (BUN) (mg/dl)	36 +/- 9
Creatinine (mg/dl)	2,4 +/- 0,9
Uric acid (mg/dl)	0,2 +/- 0,2
Total bilirubin (mg/dl)	0,3 +/- 0,2
Direct bilirubin (mg/dl)	0,1 +/- 0,1
Indirect bilirubin (mg/dl)	0,2 +/- 0,2
Glucose (mg/dl)	138 +/- 40
Cholesterol (mg/dl)	197 +/- 59
Triglyceride (mg/dl)	48 +/- 41
Creatine phosphokinase (IU/l)	296 +/- 311 (?)
Lactate dehydrogenase (IU/l)	92 +/- 87
Alkaline phosphatase (IU/l)	37 +/- 54
Alanine aminotransferase (IU/l)	98 +/- 71
Aspartate aminotransferase (IU/l)	52 +/- 35
Gamma-glutamyl-transferase (IU/l)	2 +/- 3
Amylase (U/l)	1308 +/- 330
Lipase (U/l)	11 +/- 12
Total protein (g/dl)	6,7 +/- 0,6
Globulin (g/dl)	3,1 +/- 0,6
Albumin (g/dl)	3,6 +/- 0,4
Fibrinogen (mg/dl)	220 +/- 143
Total triiodothyronine (ng/ml)	74,2 +/- 40,6
Total thyroxine (mg/dl)	1,3 +/- 0,6

Cortisol (ng/ml)[132]	39,3 +/- 27,8

Table 4: Serum chemistry parameters for cheetahs in captivity[12,131]

4.2 Medical examinations

4.2.1Standard health evaluation protocol[V]

Physical examination

A thorough evaluation of the whole animal is mandatory! Furthermore focus your attention on:

- identification of the specific animal
- body weight and body condition
- coat: use a flea comb like in small animal practice
- feet: superficial ulcers may be characteristic for calicivirus infections (see also 3.3.2)[1]

Dental examination and prophylaxis

- dental formula: I 3/3, C 1/1, P 2-3/2, M 1-1[12]
- odour from the mouth?
- calculus accumulation:
    - Remove calculus as treatment as well as part of regular prophylaxis.
    - Bacterial infections of the oral cavity may lead to bacteraemia which may lead or promote eventually systemic disease!
    - After use of an ultrasonic scaler a polisher should be used to smoothen the teeth's surface to prolong renewed accumulation of calculus.
- soft tissue: papillomatous plaques (under the tongue), ulcers
- hard tissue: the mandibular molar may cause lesions on the palatine (gingiva, palatine bone) and may be rounded off or shortened prophylactically after eruption of the permanent teeth.

[V] see appendix I "Standard Health Evaluation Protocol" of the "Cheetah SSP Health Chapter"[1]

- foreign bodies: remove and treat acquired lesions as necessary[1]

Vaccination

Check of the vaccination status and if necessary application of vaccinations (see also 5.1).

Urine collection

- As in small animal practice urine can be collected in various ways, ascending in sterility:
    - expression of the bladder
    - catheterisation of the bladder
    - cystocentesis
- Urine analysis should include:
    - routine analysis
    - urine sediment[1]

Faeces collection

The faeces should be examined at least by flotation and direct smear (see also 5.2).[1]
If fecal steroid analysis is required the samples should preferably be stored frozen at -20°C. Second best is the storage at room temperature in 95% ethanol up to 14 days. Drying of the samples may result in variations in concentration of the steroid hormones but not of the androgens.[133]

Blood collection

- Blood reference values are available through the "International Species Inventory System" (ISIS)[131]
- Blood analysis should include:
    - complete blood count
    - serum chemistry
    - serum banking

- serologic testing
- testing for haemoparasites (blood smear)[1]

4.2.2State in the EEP regarding testing for infectious diseases

According to a report given by the EAZWV and the EWDA in 2002[134] EEP institutions are testing regularly for the following infectious diseases:

- No regularly testing: 79%
- Feline Leukemia Virus (FeLV) and Feline Immunodeficiency Virus (FIV): 21%
- Feline Infectious Peritonitis (FIP): 18%
- Testing for other diseases: 7%

4.2.3Quarantine and pre-shipment examinations[VI]

Health certificate

A health certificate including complete past medical history, blood values, immunizations, faecal examinations and serology results should be available before animal transport.[1]

Health examinations

Every cheetah moved between facilities or obtained from the wild should undergo a thorough health examination. The procedures recommended for differing purposes are:[1]

Sample	Procedure	Routine	Quarantine	Pre-shipment
Whole body	Identification	required	required	required
	Body weight	required	required	required
	Physical examination	required	required	required
	Vaccination	*optional*	*optional*	*optional*
	Survey radiographs	*optional*	*optional*	required

[VI] see section "Preventive medicine" of the "Cheetah SSP Health Chapter"[1]

Blood	Examination (incl. haemoparasites)	required	required	required
	FCoV, FIV, FeLV serology	required	required	required
	FPV, FHV, FCV, Toxoplasma serology	*optional*	*optional*	*optional*
	Heartworm testing	*optional*	*optional*	required
	Serum bank	required	required	required
Faeces	Faecal examination	required	required	required
	FCoV PCR	*optional*	required	required
	Culture	*optional*	required	required
Urine	Analysis	required	required	required
Head	Dental examination	*optional*	*optional*	*optional*
Abdomen	Ultrasound	*optional*	*optional*	*optional*
	Gastroscopy, gastric biopsy	*optional*	*optional*	*optional*

Table 5: Recommended health examination procedures[1]

4.2.4Quarantine period

- The quarantine period should be long enough to cover the incubation period of the most infectious diseases and should therefore be at least 30 days long. For individuals obtained from the wild or non-accredited facilities it may be even longer.
- The quarantine period should be used to monitor the animal's behaviour and health status including performing of another required tests.
- If the new institution provides a different diet the transition should preferably be made before moving the animal otherwise a gradually change to the new diet is highly recommend to avoid the animal stop eating because of the stress situation.[1]

4.3 Necropsy

All cheetahs that die should undergo a thorough necropsy including complete gross and histopathologic examination. Samples should be collected for fixation in formalin as well for freezing at -20°C.[1]

Fixation in formalin

- Tissues should be preserved in **10% buffered formalin** at a **1:10** ratio tissue to formalin.
- Sample size should not exceed **1 cm** in thickness. Exception: brain and spinal cord must be fixed in total![1]

Frozen tissues

Each tissue should be preserved in a separate freezable plastic bag and stored by at least **-20°C**.[1]

For more detailed information, including the tissues required, see appendix I “EEP cheetah necropsy protocol”.

5 Prophylaxis

5.1 Vaccination

5.1.1Preliminary considerations

- The local prevalence of a disease should be considered when deciding for or against vaccination.
- Type, serial number and source of the vaccine should be recorded carefully.
- Most vaccines are not approved for use in non-domestic species so be aware that there is a potential liability! Used in different species or delivered by a different route may potentially cause disease. In cheetahs there had been cases of vaccine-induced disease after application of FeLV and FPV vaccines.
- For security reasons killed (inactivated) vaccines should be preferred in exotic animals. Additionally use of polyvalent vaccines should be avoided.
- Be aware that some drugs (antibiotics, glucocorticoids) may interfere with the vaccination!
- When using a remote delivery system (e.g. darting an animal) one must ensure that the full dose was administered.
- Animals already showing clinical signs of an illness should not be vaccinated!
- In case of a disease outbreak all susceptible animals should be vaccinated immediately! 14-21 days later there should be a booster vaccination.[1]

5.1.2Disease susceptibility

CAV	CDV	FPV	FeLV	FHV	FCV	Rabies	Leptospirosis	Toxoplasmosis
-	+	++	+	+	+	+	+	++

Table 6: Disease susceptibility[90]

5.1.3Recommendations

Feline/Canine Parvo Virus (FPV, CPV)

General: There are different types of parvovirus:

- Feline Panleukopenia Virus (FPV): Felidae are susceptible.
- Canine Parvo Virus type 2 (CPV-2):
  - CPV-2: Virus cannot replicate in Felidae.
  - CPV-2a, CPV-2b: Antigenic types of CPV, large cats are highly susceptible.

Recommended vaccine: Killed vaccine, preferably containing CPV-2a and CPV-2b. Unfortunately there is only a CPV-2 vaccine available at the moment.

Vaccination regime:

- The EEP recommends a multivalent (Panleukopenia Virus, Rhinotracheitis Virus, Calici Virus) killed vaccine given every 2 weeks from 8 to 16 weeks of age. Cheetah cubs unfortunately do not develop sufficient antibody titres when given vaccinations only every 4 weeks.
- A booster at the age of 40 weeks is recommended.
- Depending on the risk factors boosters may be necessary from every 3 months to every 12 months.[27]

Feline Calici Virus (FCV)

General: Vaccination is recommended.

Recommended vaccine: see Parvo

Vaccination regime: see Parvo[90]. The same regime as in domestic cats can be used.

Feline Herpes Virus (FHV) (Feline Rhinotracheitis Virus)

General: Although felids are susceptible large felids only show mild symptoms or none at all.

Recommended vaccine: Killed vaccine.

Vaccination regime:

- see Parvo. The same regime as in domestic cats can be used.

- In high risk situations boosters may be required every 3 months.[90]

Canine Distemper Virus

General: The vaccination of large felids against CDV is possible but not recommended unless in high risk situations.[90]

Feline Leukemia Virus

General:

- As in domestic cats a serological test is recommended before vaccination.
- Vaccination is possible but only recommended if there is close contact with feral cats.
- In cheetahs there had been cases of vaccine-induced disease after application of FeLV vaccines![90]

Rabies Virus

General: Vaccination is recommended in areas with a high incidence of rabies in wildlife.

Recommended vaccine: Killed vaccine.

Vaccination regime: The first vaccinations should be given at 6 and 12 months of age and afterwards boostered every year.[90]

5.1.4State in the EEP

According to a report given by the EAZWV and EWDA in 2002[134] vaccination regimes against the common infectious diseases in thee EEP are carried out as follows:

- Feline respiratory disease complex (FHV, FCV): 64%
- Feline panleukopenia (FPV, CPV): 50%
- Feline leukaemia (FeLV): 29%
- Feline infectious peritonitis (FIP): 11%
- Rabies: 14%
- No vaccination at all: 32%

5.2 Endoparasites

Parasites known to infest cheetahs

Mainly ascarides (Toxocara spp., Toxascaris spp.) and strongylids (Ancylostoma spp.). Be aware that not all eggs or larva found have to be parasites of the cheetah but may be associated with the feeding (e.g. Coccicidia in whole rabbits).[12,29,135]

Faecal examination

- Interval: Should be conducted periodically on a regular basis, e.g. 4-6 times a year.[12]
- Technique: At least flotation of collected faeces. If necessary testing for lung worms or Giardia antigen may be useful.[12]
- Negative result: Be aware that a negative result does not necessarily imply that there are no parasites because of latency periods or intermittent shedding.
- Positive result: Because of resistance problems a treatment should only be conducted after positive test results to avoid unnecessary treatments.

Treatment

The following drugs can be used in cheetahs in the following dosages:

Drug	Parasite class	Dosage
Pyrantel	nematodes	3-5 mg/kg p.o., for 3-5 days[1]
Fenbendazole	nematodes	5-10 mg/kg p.o., single application or for 3-5 days[1,135]
Ivermectin	nematodes, heartworms	0,2 mg/kg s.c./p.o.[1] 0,1-0,2 mg/kg[1] (or 10 mg/individual)[135] monthly for ascarid elimination or heartworm prophylaxis[VII]
Praziquantel	cestodes,	5,5-6,6 mg/kg s.c./p.o., single application

[VII] S. Citino, personal communication

	trematodes	higher doses as needed (e.g. Spirometra spp.)[1]
Sulfadimethoxine	coccidia	50 mg/kg SID p.o./parenteral[1]
Trimethoprim	coccidia	15 mg/kg BID or 30 mg/kg SID p.o.[1]

Table 7: Treatment options for endoparasites in cheetahs[1,135]

5.3 Heartworms

Treatment

Ivermectin, Moxidectin, Milbemycin at standard feline doses in endemic areas[12] (e.g. 0,1-0,2 mg/kg monthly[1,VIII])

5.4 Ectoparasites

Ectoparasites known to infest cheetahs

- Fleas
- Lice
- Ticks
- Mites: Cheyletiella spp., Otodectes spp., Notoedres spp., Sarcoptes spp., Demodex spp.
- Flies (including myiasis)[1]

Treatment

The following agents can be used in cheetahs with dosages similar to domestic animals without apparent side effects:[1]

	Fipronil	Methoprene	Imidacloprid	Lufenuron	Nitenpyram	Permethrin	Ivermectin

[VIII] S. Citino, personal communication

Fleas	x	x	x	x	x	x	(x)
Lice	x		x			x	x
Ticks	x					x	x
Mites	x					(x)	x
Flies		x				x	(x)

Table 8: Treatment options for ectoparasites in cheetahs.[136]

6 Management

6.1 Drugs potentially causing adverse reactions

- **Griseofulvin**[1]: According to[111] Griseofulvin can be used in cheetahs against Microsporum spp. infections. Not only because there is a reported case of drug-induced anaemia treated animals should be monitored carefully.
- **Metronidazole**[1]: According to[137] metronidazole may cause DNA damage in lymphocytes.
- **Zuclopenthixol acetate**[1]: Zuclopenthixol alone and in combination with other drugs causes inappetence, ataxia, extra pyramidal reactions, akathisia and prolapse of the third eyelid and should not be used in cheetahs! As a tranquilizer use perphenazine enanthate at 3.0 mg/kg instead.[1,138] (see also 6.2.5)
- **Haloperidol**[1]: The use of the long acting tranquilizer Haloperidol may result in extrapyramidal effects.

6.2 Sedation & Anaesthesia

6.2.1Preparation

- No feeding for 8-24 hours[1,12] and no water for 6-12 hours[1,12] to avoid emesis and aspiration pneumonia during induction and recovery period.[12] In hot conditions or in individuals with specific diseases water may be withheld for a shorter time span.[1]
- A venous access is mandatory for the possibility of fast drug administration or IV fluid supply!
- For drug administration an area as calm, small and safe as possible would be ideal.
- The calmer the animal the lower the drug doses are required and the smoother will be the induction.

Intubation

- Intubation material should be ready for every procedure before induction of anaesthesia to maintain the airways and to be ready for supplemental oxygen supply when needed (e.g. apnoea after propofol administration).[12]
- For procedures longer than 20 minutes the patient should be intubated with an appropriate sized endotracheal tube.[12]
- Topical anaesthesia of the larynx is not required necessarily.[1]

6.2.2Monitoring

- General: responsiveness to stimuli, muscle tone[1], position of the eyeball (depending on drugs used)
- Respiratory system: respiration rate and type, pulse oximetry, capnography[1]
- Cardiovascular system: colour of mucous membranes, pulse rate and type, blood pressure, electrocardiogram[1]
- Body temperature: Monitoring of the body temperature is important because both hypothermia and hyperthermia may occur![1]

Hypothermia

Causes: low environmental temperature, prolonged surgical procedures[1]

Treatment: use of forced air thermal heaters[1], warm water enemas, warmed IV fluids, body warmth, gloves filled with hot water

Hyperthermia

Causes: convulsions, pre-anaesthetic excitement, high environmental temperature, exposure to direct sunlight[1]

Treatment: especially severe hyperthermia (inner body temperature > 40,6 °C) has to be treated aggressively: water immersion, cold water enemas, IV fluids (colloids), antibiotics[1]

6.2.3Injectable anaesthetics drug combinations

The following drugs may be applied intramuscular unless specified otherwise:

drug dose	partial antagonist dose
0,2-1 mg/kg ketamine IV	**---**
2,5 mg/kg ketamine + **0,05-0,07 mg/kg** medetomidine	**--- +** **0,3 mg/kg** atipamezole
3,0 mg/kg ketamine + **0,03 mg/kg** medetomidine + **0,3 mg/kg** butorphanol	**--- +** **0,15 mg/kg** atipamezole + **0,3 mg/kg** naltrexone
0,035 mg/kg medetomidine + **0,15 mg/kg** midazolame + **0,2 mg/kg** butorphanol	**0,175 mg/kg** atipamezole + **0,03 mg/kg** flumazenil + **0,2 mg/kg** naltrexone
5 mg/kg ketamine + **0,02 mg/kg** dexmedetomidine	**--- +** **0,1 mg/kg** atipamezole
5-10 mg/kg ketamine + **0,5-1,1 mg/kg** xylazine	**--- +** **0,1 mg/kg** atipamezole
3-4 mg/kg ketamine + **0,75-1,5 mg/kg** xylazine + **0,03-0,04 mg/kg** midazolam	**--- +** **0,1 mg/kg** atipamezole + **0,03 mg/kg** flumazenil or **0,1 mg/kg** sarmazenil
3-5 mg/kg tiletamine-zolazepam	**0,03 mg/kg** flumazenil or **0,1 mg/kg** sarmazenil
1,6 mg/kg tiletamine-zolazepam + **0,03 mg/kg** medetomidine	**0,03 mg/kg** flumazenil or **0,1 mg/kg** sarmazenil + **0,15 mg/kg** atipamezole
1,3-1,5 mg/kg tiletamine-zolazepam + **0,013(?)-0,15 mg/kg** medetomidine + **1,3-1,5 mg/kg** ketamine	**0,03 mg/kg** flumazenil or **0,1 mg/kg** sarmazenil + **0,75 mg/kg** atipamezole
1,0-1,3 mg/kg tiletamine-zolazepam + **0,4-0,52 mg/kg** xylazine + **1,6-2,1 mg/kg** ketamine	**0,03 mg/kg** flumazenil or **0,1 mg/kg** sarmazenil + **0,1 mg/kg** atipamezole
0,5-4 mg/kg propofol IV	---

Table 9: Overview of appropriate injectable drug combinations for cheetahs[1,90]

Antagonists

- Naltrexone: 1 mg naltrexone per 1 mg butorphanol[139]
- Atipamezole: 5 times the medetomidine dose[139]
- Sarmazenil, flumazenil: There is no difference between these two antagonists, the use of both is recommended.[140]

Ketamine + medetomidine

If reversals are abnormally long (i.e. >20 minutes) the atipamezole dose may be increased to **0,5 mg/kg**.[139]

Tiletamine-zolazepam

- Avoid in cats with known or suspected renal disease![1]
- In a few cases cheetahs stopped breathing 60-90 minutes after administration of tiletamin-zolazepam and did not respond to doxapram![139,141]
- Prolonged recoveries may be possible. Flumazenil can be used to antagonize the zolazepam fraction.[1,12]

Combinations with ketamine

- The use of ketamine alone is not recommended in cheetahs! It may be necessary to treat resulting seizures with benzodiazepines (diazepam, midazolam).[1]
- Avoid in cats with known or suspected renal disease![1]
- Ketamine cannot be antagonised! Therefore you have to wait for at least 30 minutes after ketamine administration before you may antagonise the other components of the drug combination.

  Otherwise the animal will experience a recovery phase solely under influence of ketamine! This may result in uncontrolled body movements possibly combined with severe hyperthermia which can lead to injuries or in the end to the death of the animal.[139]

Xylazine

- Possible urine contamination during electroejaculation.[1]
- Avoid xylazine in late term gestation![1]

Propofol

- (Rapid) administration may result in apnoea! Apply oxygen before administration and intubate as quickly as possible to maintain sufficient oxygen supply.[1,12]

6.2.4Inhalation anaesthesia

- For prolonged procedures inhalation anaesthesia is recommend after induction with injectable anaesthetics.[1]
- Isoflurane[1] is the recommend but sevoflurane and halothane also can be used safely[12].
- Normally spontaneous respiration with occasional assisted respiration is sufficient[1] but ventilation may be useful because of subclinical hypoxia.[12]

6.2.5Tranquilizers

Drug	Dosage
Diazepam	0,5-2,0 mg/kg p.o. SID-TID (also for long term use)
Acepromazine	0,5-1,0 mg/kg p.o.
Perphenazine enanthate	3,0 mg/kg i.m. (long acting for 5-7 days!)

Table 10: Tranquilizers for use in cheetahs[1] (see also 6.1)

6.2.6Analgesics

NSAIDs

Drug	Dosage	Comment
Meloxicam	0,1-0,2 mg/kg p.o/i.m. SID	p.o. for repeated treatments, 0,2 mg/kg for single application

Carprofen	1,2 mg/kg p.o. SID	
Etodolac	6 mg/kg SID	

Table 11: NSAIDs for use in cheetahs[1]

Opioids

Drug	Dosage	Comment
Butorphanol	0,2-0,4 mg/kg s.c./i.m.	κ-agonist and μ-antagonist
Fentanyl	50 μg/h s.c. or 100 μg/h patch	μ-agonist, short term post-op
Morphine	0,1 mg/kg epidurally	Administer 45 min pre-op for hind limb orthopaedic
Tramadol	2,0-2,5 mg/kg p.o. BID	μ-, κ-, δ-agonist, short term and long term

Table 12: Opioids for use in cheetahs[1]

7 Nutrition

7.1 Potential sources of infectious diseases

- Poultry carcasses: Avian influenza[1]
- Bovine carcasses: Feline spongiform encephalitis (FSE)[26]
- Carcasses generally (especially deer): Toxoplasma cysts. Consider freezing carcasses for 48 hours.[1]
- Raw meat: Salmonella, Clostridium. Consider cooking the meat before feeding to cubs.[1]

7.2 Mineral supplements, trace elements and vitamins

Calcium phosphorus imbalance

A well-balanced calcium phosphorus balance (approximately 1:1) should be maintained.[16]

- **Calcium deficiency**[1]:
  - Causes: Absolute deficiency or relative due to
    - Phosphorus excess: Food items like muscle meat, liver and heart contain relatively too much phosphorus compared to calcium.
    - Vitamin D3 deficiency: Cheetahs, like other felids, probably cannot synthesize enough vitamin D3 by themselves through sunlight exposure and therefore need sufficient vitamin D3 in their diet.
  - Consequences: Osteodystrophy (developmental bone malformation)
- **Calcium excess**[1]:
  - Consequences: Osteochondrosis dissecans (developmental deformity of the forelegs), Angular limb deformities (in cubs calcium excess may not be the only reason!), Enlarged joints, Splayed feet, Stunted growth
  - Treatment/Prophylaxis: calcium phosphorus balanced diet with sufficient vitamin D3

Copper

- **Copper deficiency**[1]:
  - Causes: copper deficient diet
  - Consequences:
    - lateral head tremor, ataxia, partial collapse, loss of balance, paralysis of the hind limbs, staggering gait
    - cubs: fatal respiratory distress
    - pregnant females: severe long bone deformities in cubs
  - Treatment/Prophylaxis:
    - acute: dietary copper usually reverses any symptoms
    - chronic: chronic deficiency may lead to permanent effects

Vitamin A

- **Hypovitaminosis A**:
  - Causes: Pre-formed vitamin A is an essential nutrient for felids and therefore must be provided in the diet. Because it is a fat-soluble vitamin it is not necessary to supplement it on a daily basis.[1]
  - Consequences: In domestic cats hypovitaminosis A may lead to skeletal malformations eventually leading to deafness and N. facialis paralysis because of narrowing of the foramina. A deficiency may also be related to neurologic, ophthalmologic, dermatologic and respiratory failure.[142,143]
- **Hypervitaminosis A**[1]:
  - Causes: Vitamin A excess in the diet due to feeding of organ meat, especially liver, along with commercial diets.
  - Consequences: Accumulation of vitamin A to toxic levels may lead to skeletal malformations, fractures, internal haemorrhage, enteritis, conjunctivitis, reduced liver and kidney function in growing animals.

Phytoestrogens

Commercial food diets containing to much soja proteins may lead to fertility problems due to included phytoestrogens.[16]

7.3 Diet composition

The main component of a cheetah's diet should be 1,5-2,5 kg of fresh meat, such as beef, horse[144], veal, sheep or goat. At least once a week whole carcasses, such as rabbit, guinea pig, chicken and other small animals, should be fed. At times also liver, heart and kidney can be given.[16] Be aware that unsupplemented muscle meat is nutritionally imbalanced and therefore appropriately supplemented chunk meat may be given instead.[145]

Some institutions feed a commercial diet which have the advantage of adequate supplementation of minerals, trace elements and vitamins but to the detriment of variation in texture and composition of the food.[145]

A study comparing the feeding of a meat-only diet (supplemented beef) or a whole prey diet (whole rabbit) revealed that neither of them alone is suitable to provide appropriate nutrition to captive cheetahs. The whole prey diet leads to a higher food intake but nevertheless a lower energy intake. It provides higher cholesterol levels, low taurine levels but a well-balanced mineral intake. There may be a risk of hypervitaminosis A when exclusively fed whole prey. The meat-only diet provides a higher protein:fat ratio leading to higher serum urea levels and a higher zink concentration. Because of a low calcium:phosphorus ratio the risk of metabolic bone disease may be increased.[146]

Pay attention to feed only first quality food as cheetahs are susceptible to fall ill because of ruined food.[16]

A fast day a week can be maintained.[144]

7.4 Hand-rearing of cubs

Reasons for hand-rearing

Hand rearing may be necessary because of the inability of the mother to raise its offspring itself (death, running out of milk, abandonment of the cubs because of external stress factors).[16]

Milk composition

Analysis of the milk of two captive-bred cheetahs showed the following composition per kg milk:

- 99,6 g protein (34,2 g caseins, 65,3 g whey proteins)
- 64,8 g fat (no short chain fatty acids but substantial levels of uneven carbon chain fatty acids)
- 40,21 g lactose (whereas only small amounts of oligosaccharides, glucose, galactose and fructose)

The milk compositions variied little among the individual cheetahs and the protein sequences were similar to those of lions and domestic cats.[147]

Be aware that despite of high digestibility commercially prepared milk replacers may not be perfectly balanced in terms of nutritient concentrations and ratios (e.g. containing high concentrations of a number of minerals whereas no vitamin D3) compared with domestic cat milk.[148]

Daily weight gain

Mother-reared cubs show a weight gain about 45 g/day[149] similar to a reported daily weight gain of 40-50 g achieved by suckling by a domestic cat[16]. Hand-reared cheetah cubs exhibited a daily growth rate about 27-32 g[149,150] pre-weaning, but increased to a post-weaning rate of 55g per day.[150]

7.5 Appetite stimulants

- Benzodiazepines
- Vitamin B
- Feeding whole or live prey[1]

8 Reproduction

8.1 Contraception

The GnRH analogue deslorelin, initially developed as an ovulation-inducing agent in mares, may be used in male and female cheetahs for contraception.[151]

A subcutaneous implant with a dose of 6 mg can be used for achieving reversible contraception in male cheetahs for about a year.[151] Because of males being fertile for about 6 weeks after administration they should be kept separated from cyclic females during that period.[152]

Also females may be treated with the same implant resulting in contraception as well lasting for about a year. Cheetahs may undergo oestrous and may be attractive for males a few days after treatment, but mating could not be observed.[152,153]

8.2 Breeding

Female

The cheetah is polyoestrous, seasonal in the wild, possibly the whole year in captivity and ovulation is almost always induced. There are also periods of anoestrus unreleated to season.[16,154–156] The cheetah's oestrus cycle is of comparatively short, ranging from 7 to 21 days, with oestrus lasting 2-10 days.[15,16] Gestation lasts about 94 days, non pregnant luteal phases about 53 days.[15] After birth the female's next oestrous may be 9-10 months later.[16]

For successful oestrus induction in the female keep females and males separately or at least separate them some time before breeding. Oestrus is induced in the female through acoustical and olfactorial stimulation by the male. A more intensive stimulation may be obtained if female and male are allowed in their enclosures only in turns.[16,155] Keeping behaviourally incompatible females together in pairs instead of keeping them solitary, as they normally would live in the wild, may lead to suppressed ovarian cyclicity.[15,157,158]

The status of sexual hormones can be checked by testing of faecal samples.[16]

Another important factor is the sympathy between the breeding partners sometimes leading to fixed pair bonds. Is there no sympathy it may also lead to disinterest, aggression or even no cycle in the female at all.[16]
In the wild females may mate with more than one unrelated male within an oestrus cycle. In a study 43% of the litters with more than one cub, the siblings had more than one father.[159]

Male

Juvenile cheetah's ejaculates display poorer sperm motility and forward progressive status, lower seminal volume and fewer total motile sperms than adult and aged individuals. The ejaculate of captive cheetahs shows higher volume but lower sperm density.[160] The cheetah reaches its peak in semen production at the age of eight years.[161] Spermatogensis itself takes place throughout the year regardless the season.[160]

Breeding

The female should start breeding between 2-4 years of age, the male between 3-10 years. In an analysis of 35 years of breeding data the oldest femals was 10,5 years old.[16,162]
Sexual interest in the male turns out by unrest, erections, characteristic calls and picking up the scent of the female.[16]
The female's signs of oestrous vary individually. In prooestrous vaginal discharge may be seen with difficulty, in oestrous they are rolling themselves, rubbing their cheek and body on dead objects, drizzling urine, putting the tail to the side and also give characteristic calls and pick up the scent of the male.[16]
For breeding the female and the male should be allowed together for at least one hour, increasing the time together every time until their behaviour allows them to stay together definitely.[16]
The male inspects the anogenital region of the female until the female allows the copulation. Copulation takes place 3-5 times a day. Unfortunately the sperm quality of cheetahs is not that good but keeping competing males may enhance the chance for successful copulation.
If the breeding partners likes each other you should keep them together for about 3 weeks to

give them another possibility for copulation if the female comes into oestrous again.[16]

8.3 Non-invasive pregnancy detection

A study proposes fecal progestagen measurement as a useful method for non-invasive pregnancy detection and monitoring in cheetahs. The fecal progestagen concentrations remain low until copulation, start to increase 3-4 days after the last copulation and remain high during the pregnancy until parturition. The progestagen composition does not differ significantly during the course of pregnancy.[163]

8.4 Neonates

Preparations for giving birth

30-35 days after copulation the female should be kept alone again. At least two boxes should be provided for the female to cast its young. This gives the female the possibility to choose and also to move to another stand later on.[16]

Giving birth

About 10 days before birth 4 pairs of teats become clearly visible. Normally giving birth will last 1,5-2,5 hours but it may take up to 11 hours. Litter size may range from 1-8 cubs, mostly between 3-4 young.[16] Older females seem to produce smaller litter sizes. The sex ratio at birth is usually balanced.[162]

The female should not be disturbed after birth and should also have the possibility to use its outside enclosure to take a break from its young.[16]

Development of the cubs

New-born cheetahs are blind as other cats, have long fur with blurred spots and are still able to retract their claws. Their weight may vary from 250-700 g, in the mean 450 g, mainly depending on litter size.[16]

Young cheetahs develop quite fast:

- Between 2-14 days they open their eyes and gain their full vision during day 16-20.[16]
- With 3 days they start to crawl, walk after two weeks, leave the box after 20-25 days and start to play with their siblings.[16]
- The development of the characteristic crests starts with about 20 days. The crest is lost again after 5-6 months on the back and after about a year in the neck.[16]
- At 1 month of age young cheetahs are able to climb but they loose this skill about 4 months because the claw's sheath is formed back.[16]
- In captivity about 70% of the cubs survive the weaning period.[162]
- They young will be accepted to stay with their mother for about a year's time.[16]

Diet

The female suckles the young for 3-6 months. The first solid food uptake starts with about one month of age, regularly after 45-50 days.[16]

Serum administration

If a deficiency in the passive immunity is suspected serum can be given orally or subcutaneously. The best option is to collect serum from the mother but if not possible the serum of a different adult cheetah living longer than one year in the same environment can be used. To remove bacteria the serum should be filtered.
Dosage (alternatively):

- orally 2-5 ml per feeding for 3-5 days
- subcutaneously 150 ml/kg splitted over several days[1]

Dental development

- Incisivi: 16-18 days
- Canini: 25 days
- Premolars: 31 days[16]

Weight gain

- Birth: 450 g (250-700 g)[16]
- 4 weeks: 1,8-2,0 kg[16]
- 8 weeks: 3,8-4,2 kg[16]

Birth weight may vary because of sex, gestation length and the amount of inbreeding.[149] In free-living cheetahs continuous growth of the cubs required a maternal food intake of at least 1.5 kg/day.[164] In the first 40 days growth seems to be linear and cubs show a daily weight gain about 45 g when mother-raised, compared to about 27 g hand-reared. The neonatal growth rate may be influenced by birth weight, gestation length, parity and the mean litter size during the first 40 days.[149]

8.5 Assisted Reproductive Technologies

Assisted Reproductive Techologies (ART) provide an accessory tool next to natural breeding. Fields of application range from pairing of behaviourally incompatible but genetically suitable individuals, breeding of aged individuals that have not reproduced before, storage of genetic material (cryopreservation) or avoidance of transfers of living animals only for breeding to reduce stress and cost.[165]

Artificial insemination (AI) is the ART technique most used in cheetahs. Unfortunately it is often applied too late to be successful, that is in femals 6 years of age or older. At the moment the success rate is as low as 30% after stimulation with exogenous gonadotropins mostly resulting in singleton litters only.[165]

Nevertheless in cheetahs success in stimulation of follicular development and ovulation induction with the aid of gonadotropins as eCG and hCG is comparatively higher than in other felid species because in cheetahs it does not lead to ovarian hyperstimulation resulting in excessive oestrogen production detrimental to reproductive success. A possible reason for the cheetah's relatively high AI success rate may be due to the frequently quiescent state of the ovaries allowing the ovaries to be more responsive to gonadotropin stimulation.[15,158]

Older female cheetahs mostly have ovaries still responsive to gonadotropins, producing

normal hormon levels and develop follicles suitable for fertilisation. Low reproductive success of older femals may therefore more likely be the result of pathologies as uterine endometrial hyperplasia. As a result their follicles may be suitable for in vitro fertilisation and embryo transfer.[166]

8.6 Population management

As the annual report of the Conservation Breeding Specialist Group (CBSG) of 2009 stated, at present neither the European (EEP) nor the North American (SSP) cheetah populations are sustainable on their own. As a result either imports of wild cheetahs or animals held in captivity in Africa which are not suitable for release into the wild are necessary for "freshing up" the gene pool as well as further discussion about the exchange of individuals between the EEP and the SSP populations.[165,167]

List of abbrevations

AAZV	American Association of Zoo Veterinarians
AI	Artificial insemination
ART	Assisted Reproductive Technologies
BID	twice a day
TID	three times a day
CAV	Canine Adenovirus-1
CBSG	Conservation Breeding Specialist Group
CCF	Cheetah conservation fund
CDV	Canine Distemper virus
CPV	Canine Parvo virus
EAZWV	European Association of Zoo and Wildlife Veterinarians
eCG	Equine chorionic gonadotropin
EEP	European Endangered Species Programme
EWDA	European Wildlife Disease Association
FCoV	Feline corona virus
FECV	Feline enteric corona virus
FeLV	Feline Leukemia virus
FHV	Feline Herpesvirus
FIPV	Feline infectious peritonitis virus
FPV	Feline Parvo Virus/Feline Panleukopeniavirus
H&E	Haematoxylin and eosin
hCG	Human chorionic gonadotropin
IFA	Immunofluorescent antibody testing
i.m./IM	intramuscular
IUCN	International Union for Conservation of Nature
i.v./IV	intravenous
MHC	Major histocompability complex
MCH	Mean corpuscular haemoglobin

MCHC	Mean corpuscular haemoglobin concentration
MCV	Mean corpuscular volume
MRI	Magnetic resonance imaging
NA	North America
NPV	Negative predictive value
PPV	Positive predictive value
RT-PCR	Reverse transcriptase polymerase chain reaction
p.o./PO	per os
SA	South Africa
s.c./SC	subcutaneous
SID	once a day

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Appendix I: EEP cheetah necropsy protocol

EEP CHEETAH NECROPSY PROTOCOL

Date		Prosector		Location	
Conditions					

Cheetah Name		ARKS ID		Other ID	
Captive		Free-Ranging		Hunted	
EEP	(Yes) (No)	Studbook-Number		Studbook-Name	
Sex		Date of Birth/Age		Offspring	
Date of Death		Time of Death		Cause of Death	
Date of Necropsy		Time of Necropsy		Degree of decomposition	
Total Weight (kg)		*dead*	*alive*	*including coat*	*without coat*
History	Enclosure structure: Group composition: Nutrition: Vaccinations: Deworming: Clinical signs: Clinical disease: Other remarks:				

Gross Examination Worksheet

General Condition	(nutritional condition, physical condition)
Orifices	(eyes, nares, mouth, anus, genitals)

Skin	
Fur	

Musculoskeletal System	(bones, joints, tendons, muscles)
Body cavities	(serosa, fat stores, abnormal fluids)
Hemolymphatic System	(spleen, lymph nodes, thymus)
Respiratory System	(nasal cavity, larynx, trachea, lungs, regional lymph nodes)
Cardiovascular System	(heart, pericardium, great vessels)
Digestive System	(oral cavity, oesophagus, stomach, intestines, liver, pancreas, mesenteric lymph nodes)
Urinary System	(kidneys, ureters, urinary bladder, urethra)
Reproductive System	(gonads, uterus, vagina, penis, prepuce, accessory glands, mammary glands, anal glands, placenta)
Endocrine System	(adrenals, thyroid, parathyroids, pituitary)
Nervous System	(brain, spinal cord, peripheral nerves)
Sensory Organs	(eyes, ears)
Preliminary Diagnosis	

Notes:

Clinical Pathology

Laboratory Studies	Date	Sample	Examination	Result
Serology				
Blood chemistries				
Hematology				
Bacteriological cultures				
Viral cultures				
Parasitological screen				
Fungal cultures				

X-Rays

Positioning	Date	Result

Other examinations:

Fixed Tissue Check List

Preserve the following tissues in 10 % buffered formalin at a ratio of 1 part tissue to 10 parts formalin.
Tissues should be no thicker than 1 cm (excluding brain and spinal cord – fix in total).
Include sections of all lesions and samples of all tissues on the required tissue list.

✓	Required tissues (Formalin)	Tissue sampling procedure
	Adrenal glands	Both entire glands with transverse incision
	Brain[1]	In Toto – see below!
	Diaphragm	Representative section
	Eyes	Leave intact
	Gastrointestinal tract	3 cm long sections of oesophagus, stomach (cardia, antrum and pylorus), duodenum, jejunum, ileum, cecum, colon, rectum and omentum. Open carefully along the long axis
	Heart	Longitudinal section including atrium, ventricle and valves from both right and left heart
	Kidney	Sections from both kidneys including cortex, medulla and pelvis
	Liver	Sections from 3 lobes with capsule and gall bladder
	Lungs	Sections from several lobes including a major bronchus
	Lymph nodes	Cervical, anterior mediastinal, bronchial, mesenteric and lumbar with transverse cut
	Pancreas	Sections from 2 areas, 1 including central ducts
	Parathyroids + Thyroid	Leave glands intact
	Peripheral nerve[2]	In separate container with topographic name
	Reproductive tract[3]	Entire uterus and ovaries with longitudinal cut into lumen. Entire testis with transverse cut, entire prostate with transverse cut
	Skeletal muscle[2]	Cross section of M. biceps brachii and M. quadriceps femoris
	Skin	Full thickness of abdominal skin and lip
	Spinal cord[1]	Sections from cervical, thoracic and lumbar cord in separate containers
	Bone	Cross section Femur w . bone marrow and distal femur including growth plate (Epiphysis)
	Spleen	Cross section including capsule
	Thymus	Representative section
	Tongue	Cross section near tip including both mucosal surfaces
	Trachea	Representative section
	Urinary bladder/ureter/urethra	Cross section of bladder and 2 cm sections of tubular structures

[1] A very careful preparation of these tissues is required!
Remove the entire brain place small frontal section (max 25% of Brain) at – 20 or lower if possible and place rest in toto in formalin.
Method description for spinal cord removal under field conditions:
Separate the spinal column from the remaining carcass; remove the paravertebral soft tissues and muscles. Transect the spinal column at the level of the intervertebral discs into approximately 15-cm-long segments. Do not confuse the individual segments in order to preserve an accurate description of the lesion distribution. Insert the provided metal blade carefully laterally to the spinal cord and into the spinal canal and move it dorsally and ventrally within the canal transecting the segmental nerves. It should be attempted to separate the dura mater from the epidural attachments in order to remove the spinal cord with the intact dura. Following this circumferential preparation, the spinal cord is grasped at one and with forceps and pulled out of the spinal canal while carefully removing persisting attachments. If possible, the spinal cord should be grasped by the dura mater to reduce artefacts. Repeat the process for each segment. The part grasped by the forceps is unsuitable for histologic examination, but should be frozen at -20 °C or more if available. The cranial part of each spinal cord segment is marked with a small incision and stored in formalin (use separately marked containers!).
[2] Place peripheral nerve and muscle tissue on a clean cardboard piece so that it adheres before submerging in the formalin.

[3] For semen assessment in male cheetahs follow the guidelines provided by the IZW!

Frozen tissue checklist

Preserve the following tissues in separate freezable plastic bags by at least – 20 °C.
Include sections of all lesions and samples of all tissues on the required tissue list.

✓	Required tissues (Frozen)	Tissue sampling procedure
	Brain[1]	left frontal lobe/ olfactory bulb
	Eye chamber liquid	Aspirated from camera anterior
	Femur with marrow	Freeze 1/2 part of the femur
	Gastrointestinal tract	3 cm long sections of oesophagus, stomach (cardia, antrum and pylorus), duodenum, jejunum, ileum, cecum, colon, rectum and omentum. Samples from stomach contents, ingesta and feces in separate small containers
	Heart	Longitudinal section including atrium, ventricle of left and right heart and septum
	Kidney	Sections from both kidneys including cortex, medulla and pelvis
	Liver	Sections from 3 lobes with capsule
	Lungs	Sections from several lobes including a major bronchus
	Skeletal muscle	Cross section of M. biceps brachii and M. quadriceps femoris
	Spinal cord*	Sections from cervical, thoracic and lumbar cord in separate plastic bags
	Spleen	Cross section including capsule
	Urine sample	Aspirated from intact urinary bladder

Neonatal Necropsy Protocol

Please follow the adult protocol in addition to the following:
a) Examine of malformations (Cleft palate, deformed limbs)
b) Access hydration (tissue moistness) and evidence of nursing (milk in stomach)
c) Fix umbilical stump and surrounding tissues
d) Determine if breathing occurred

Shipping Tissues

Please obtain proper CITES and Export Permits before shipping tissues!

For further information please contact

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Summary

The aim of “Veterinary Guidelines for the Cheetah European Endangered Species Programme (EEP)” is to provide the zoo vet or anyone else who is responsible for the health of an EEP cheetah population a manual of the most important issues, diseases and diagnostic methods. Therefore the authors tried to give an overview of the most important health issues concerning cheetahs on the one hand and to be as concise and clear as possible on the other hand.

Zusammenfassung

Das “Veterinärmedizinische Handbuch für das Europäische Erhaltungszuchtprogramm (EEP) für Geparden” soll Zootierärzten oder anderen Personen, die für die Gesundheit von Geparden im Rahmen des Europäischen Erhaltungszuchtprogramms verantwortlich sind, als Nachschlagewerk für die wichtigsten Problemfelder, Krankheiten und diagnostischen Möglichkeiten dienen. Daher soll einerseits ein umfassender Überblick über mögliche Problemfelder der Gesundheit von Geparden gegeben werden, andererseits wurde versucht so klar und präzise wie möglich zu bleiben.