# A Brief Summary of the Incidence of Renal Amyloidosis in Captive-bred Cheetah (Acinonyx jubatus) at the Cango Wildlife Ranch in Oudtshoorn, South Africa

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#### Introduction

In the time period from December 1987 to February 2005 the Cango Wildlife Ranch in Oudtshoorn, South Africa have lost 67 Cheetah, 28 (41%)of these have been related to or as a direct result of renal amyloidosis.

Renal amyloidosis is a poorly understood phenomenon of the deposition of an insoluble proteinaceous substance (see photos) which infiltrates the medulla (the area between the inner pelvis and outer cortex) of the kidney, becomes waxy and renders the tissue non-functional and the organ begins to fail.

Renal amyloidosis is a common problem found in most captive-bred cheetah populations all over the world, it appears that in the time period (1990-1995) the disease increased in prevalence in the USA and Southern Africa from 20% to 70% where cheetah either died or were euthanased due to acute or chronic renal failure as a result of renal amyloidosis.

#### Pathophysiology

Cheetahs have a high prevalence of systemic amyloidosis in response to ANY inflammatory condition (gastritis, enteritis, colitis, hepatitis, periprostatic abcess, etc) and renal amyloidosis is an increasingly significant cause of morbidity (illness) and mortality (death) in captive cheetah populations.

Familial forms (affecting members of a closely related group of animals) are also described in Chinese Shar Pei dogs and Abyssinian cats.

Amyloidosis in cheetahs is type AA (i.e. secondary) and involves the renal medullary interstitium, where the amyloid deposits progressively strangulate the blood supply to the renal papilla leading to acute or chronic renal failure. This condition can also be exacerbated by the presence of glomerulosclerosis (progressive damage to the glomeruli which become shrunken, eosinophilic and with a reduction in cell numbers) which is also common in captive cheetah.

Renal amyloidosis is most commonly found secondary to a primary inflammatory condition called **chronic lymphoplasmacytic gastritis**.

Gastritis (inflammation of the lining of the stomach) in captive-bred cheetah is mostly associated with *Helicobacter*-like spiral bacteria in gastric gland cells, however some cheetah show gastritis without spiral bacteria, this may indicate that the pathogenesis of gastritis involves factors other than *Helicobacter* infection.

An autoimmune (disease due to an immune response of one's own cells or antibodies on components of the body) component to the disease is possible, because the inflammatory reaction is predominantly lymphoplasmacytic and orientated toward gastric glands. The same *Helicobacter* is also found in wild cheetah but they show no signs of gastritis.

### **Clinical symptoms**

Clinical symptoms of renal failure include protein loss in the urine, with accompanying weight loss, non-regenerative anemia, uremia, polydypsia (increased drinking) and polyuria (increased urination).

At the Cango Wildlife Ranch we have also seen signs of a stary hair coat, elevated urea and creatinine levels, ataxia, weakness, anorexia, dehydration, vomition and diarrhoea. The disease is prevalent in our older cats from about the age of seven years onwards. Unfortunately by the time we see these signs the renal damage is far advanced and most cats are euthanized.

### Diagnosis

Diagnosis of amyloidosis in the kidney is made on histopathology, amyloid deposits are recognised as bright amorphous eosinophilic deposits in the renal medullary interstitium, usually most prominent near the corticomedullary region. (See photos) These deposits are apple green in polarised light, using Congo red stain; and are purple with Masson's Trichome stain.

## Treatment

There is no proven successful treatment for amyloidosis, however early identification and treatment of the underlying causes (gastritis being one of the most important) can result in regression of amyloid and associated signs.

The ideal method of diagnosing gastritis is to examine the gastric wall endoscopically as well as take 10-15 gastric biopsy samples at least once annually; these are histopathologically examined for signs of *Helicobacter* and gastritis. The gastroscopy has its own risks associated with the immobilisation of the cheetah and thus is limited to an annual procedure.

Gastritis is being successfully treated at a number of institutions in the USA and South Africa with a number of different regimes; tetracycline hydrochloride 500 mg p.o. qid, metronidazole 250 mg p.o. qid and bismuth subsalicylate 300mg p.o. qid for seven days, thereafter each cheetah is maintained on 300mg bismuth subsalicylate p.o. sid for one yr, also omeprazole, metronidazole and amoxicillin for three weeks has had a dramatically therapeutic effect. A reduction in stress factors as well as aggressive treatment of gastritis seems to be causing a significant reduction in renal amyloidosis.

# Management and prevention

1.Continued surveillance to identify, control and treat causes of underlying inflammatory conditions (e.g. gastritis) is recommended †We routinely draw blood and check urea and creatinine values, however the most effective diagnostic tool is gastric biopsy evalution as discussed previously.

2. When possible, avoid potentially nephrotoxic drugs. (aminoglycosides etc)

3.Endeavour to keep stress to a minimum by:

• Providing comfortable sleeping quarters.

• Ensuring individuals in groups get on with each other and that males and females are compatible during the mating season.

• Providing natural and spacious enclosures away from other feline species who may cause sub-clinical stress.

4. Genetic homogeneity may increase a predisposition to susceptibility to infectious disease or increased propensity for the development of amyloidosis, this should be taken into consideration when matching males and females for breeding.

5. Whether diet plays a role or not has not been established yet but research is currently being done. Diet is unlikely to play a role as captive cheetah worldwide are fed a variety of diets and amyloidosis is prevalent in all groups.



Kidney sagittal section: cheetah. The base of the medulla is pale, waxy and streaks throughout the medulla because of amyloid deposition and fibrosis.

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