Christie D, Blyde P, Hartley W. Thiamine deficiency in cheetah.

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Thiamine Deficiency in Cheetah

Abstract

Western Plains Zoo, Dubbo has had several incidences of sickness and death in Cheetah, *Acinonyx jubatus*. These incidences are now thought to have resulted from Thiamine deficiency. The events leading to this conclusion, the various other options pursued, and the preventative measures taken will be discussed. The implications for the management of other collections and the incidence of disease outbreak in Cheetah will also be discussed.

by Peter Christie, Dr David Blyde and Dr William Hartley.

Introduction

Western Plains Zoo has held cheetah since 1978 . During this time five litters containing 12 cubs have being born. Of these, nine cubs survived to independence. During the same period eight animals died, three were cubs less than four weeks old, the remainder being adults. This scenario of what appears as an unsatisfactorily high mortality rate is one which is repeated in many institutions holding cheetah around the world.

Table One. Cheetah Births at Western Plains Zoo

CHEETAH BIRTHS AT WESTERN PLAINS ZOO

LITTER	DATE OF BIRTH	SEX/NAMES	DATE OF DEATH	CAUSE
1.	1 Oct 1986	0.0.2	11 Oct 1986	Mismothering
2.	24 Jan 1987	1.0.0	4 Feb 1987	Mismothering
3.	11 Jun 1987	3.0.0 Tsavo Mikumi Kibo		
4.	22 Oct 1988	2.0.0 Nakuru Ovambo		
5.	20 Nov 1989	3.1.0 Kariba Asante Etosha Malindi	30 Mar 1991 (1.0 Kariba)	Thiamine deficiency

For example, the AAZPA Cheetah SSP in it's summary states that due to low fecundity and high mortality the current Nth American population is not self sustaining. Due to this unsatisfactory state significant research work is being conducted on the SSP population in the following areas: Reproduction, Nutrition, Behaviour, Mortality, genetics, and Infectious diseases with an aim of improving the reproductive and mortality rates.

The Australasian ASMP similarly has relied on continued importation to maintain sufficient numbers in captivity.

Western Plains Zoo deaths are summarised below:

Table Two. Summary of Cheetah Deaths, Western Plains Zoo

Death Type	No of Animals
Euthanasia	1.0
Mismothering	1.0.2
Disease	3.1
Total	5.1.2

Prior to discussion of the case history of the Western Plains Zoo deaths it is appropriate to discuss aspects of cheetah biology and disease occurrence at other institutions.

Genetic problems

Research by Stephen O'Brien, David Wildt and Mitchell Bush suggest that the Cheetah has lost it's genetic variation. That is as a result of intensive inbreeding generations ago, each cheetah appears to be nearly identical with every other Cheetah. Their electrophoretic survey of De Wildt Cheetah indicated no variation in any of the 52 proteins examined, i.e. each cheetah was electrophoretically identical with every other Cheetah.These researchers collaborated their results with skin graft experiments and examination of morphological variation, which confirmed a level of genetic monomorphism that is unprecedented in any outbred mammalian species.

These researchers hypothesised that the reason for this apparent monomorphism is that at some time in the past the species went through an extreme bottleneck: a severe population reduction, followed by inbreeding, which diminished the gene pool by the chance loss of alleles. The apparent consequences of such genetic uniformity to the species include: difficulty in captive breeding, high levels of infant mortality, high frequency of spermatozal abnormalities and apparent vulnerability to coronavirus associated FIP.

Meltzer, Coubrough and van Dyk found that spermiograms of the Cheetah at De Wildt showed a high abnormality count, despite a good motility rating in most cases. These abnormalities were mostly primary, and are therefore associated with lowered fertility in other species. They also found the species is vulnerable to disease and has a high infant mortality.

Disease occurrence.

There are currently four main suspects associated with Cheetah death: Feline Infectious Peritonitis (FIP), Copper deficiency, Kidney and Liver related diseases and Vitamin A toxicity.

Many institutions have experienced difficulty in diagnosis of disease outbreaks in their cheetah populations. Some more notable ones are discussed.

van Dyk of De Wildt also reports cheetah as susceptible to copper deficiency, demonstrated by a loss of co-ordination of hindquarters, resulting in a swaying gait.

Charles Vallet of Montepellier Zoo reported that three cubs of six months age developed a paresis of the posterior limbs causing a serious impediment to walking. Dietary deficiency was suspected and treated with alternate daily injections of calcium and Tridocelan (Vitamins B_1, B_2 and B_{12}) for twelve days resulted in rapid improvement and resolution of the problem.

Evermann reported on the Wildlife Safari Park, Oregon which had one of the most successful cheetah breeding programs in the world. In 1983 a pair of Cheetah on loan died from Feline Infectious Peritonitis (FIP). Although FIP seldom kills more than 10% of infected animals within six months at Safari

Park 13 cheetahs had died including three adults of FIP related disease. This epizootic (an epidemic amongst animals) was the most extreme response to an FIP viral infection so far reported in any species. During the following two years another six animals died due to FIP. Other FIP epizootics have occurred in cheetah breeding compounds in Ireland, Canada and Namibia. The lack of resistance to such a disease is explained by apparent monomorphism at the major histocompatibility complex (MHC) which would limit the species repertory of defences.

Gandras and Enke reported on the death of Cheetah at Krefield Zoo in Germany where four cheetah died in a two year period all suffering from symptoms including apathy, food refusal, staggered gait, loss of balance, cramping/fits and paralysis. They suspected that a degenerative disease of the liver was the cause of death brought about by some unknown agent. They hypothesised that viruses, bacterial diseases, toxic agent in food or parasites, repeated anthelmintics and other agents could have been responsible.

van de Werken (1967) conducted a survey on captive cheetah covering some 279 animals kept in 44 zoos in the period 1945 to 1967. The average longevity in captivity for 153 animals over the period of the survey was three years and five months with only 24% of cheetah surviving to 4 and a half years of age. Liver disease was found to the be the most significant cause of death.

Snodgrass reported on illness in cheetah at Fossil Rim in 1985 when 2.1 died due to Feline Infectious Peritonitis and in 1988 when several cubs became ill displaying acute hind limb weakness and unco-ordination. Tests were undertaken for FIP, FE, LV, and Toxoplasmosis, Lymes Disease, Vitamin A, D, E, and selenium levels. All results were normal. Further tests revealed levels of copper deficiency lower than that found in the wild. Within four weeks of treatment for copper deficiency dramatic improvements occurred in the affected cubs.

Setchell and others conducted a histopathological evaluation of more than 100 cheetah livers and concluded that the relatively high concentrations of phytoestrogens from soybean protein present in the commercial diet fed to cheetahs in North American zoos may be one of the major factors in the decline of fertility and in the etiology of liver disease in this species. Gosselin and others also investigating veno-occlusive disease of the liver in Cheetah found that high levels of Vitamin A in the diet of captive bred animals may also contribute to liver disease.

Diet

The above cases indicate the importance of diet to the health and well being of Cheetah. Various diets have being fed to Cheetah. At Western Plains Zoo this has included:

1978 - 1985	8-10lb meat a day plus chickens and other carcass meats. Vitamin E and calcium powder on all food.
1985 - 1986	2 - 2.5 kg meat a day plus chickens and other carcass meats. Vitamin E (6gms), Hi-Phos (3Gms) powder on all food.
1986 - 1987	2 - 2.5 kg meat a day plus chickens and other carcass meats. Vit E Plus (6gms), SA37 (6gms), Ca Co_3 (9gms) powder on all food.
1987 - 1989	AS above plus half tablet betamin (thiamine) per animal once per week.
1989 - present	Betamin tablet deleted in favour of Thiamine powder.

In other zoos cheetah diet has included:

Meats:	Donkey, horse, beef, chicken, rabbit, deer, beaver and commercially prepared diets.
Supplements:	Powdered milk, brewers yeast, vitamin-mineral salts, steamed bone flour, Vitamins A, D3, E, B1, B12, copper,

lactogen, hyphos, bone meal, entrails, cod liver oil, Pervinal, protein, multivitamin powder, Lipo-plus capsule, general vitamins/minerals.

Feline Infectious Peritonitis (FIP)

First described in 1966 in the USA. It is a slowly progressive and fatal disease of young cats, and sometimes older ones also, caused by a coronavirus. Natural route of infection is unknown. Signs include fever, depression, loss of appetite, gradual weight loss, distension of the abdomen, diarrhoea and vomiting. No vaccine available.

Case History of deaths

Table 2. CHEETAH DEATHS AT WESTERN PLAINS ZOO

NAME	DATE OF DEATH	AGE	CAUSE
Zulu	13 Feb 1985	7 years	Thiamine deficiency
Hapurah	15 Feb 1985	3 years	Thiamine deficiency
D186	11 Oct 1986	10 days	Mismothering
D286	11 Oct 1986	10 days	Mismothering
D187	4 Feb 1987	11 days	Mismothering
Octavia	2 Oct 1990	7 years	Nephritis
Kariba	30 Mar 1991	16 months	Thiamine deficiency
Hymah	26 Jan 1992	10 years	Degenerative renal failure

Plus 6.3.0 Cape Hunting Dogs in 1985.

1. At Western Plains Zoo in February 1985 two adult male cheetah died as well as 6.3 Cape Hunting Dogs. In addition the other three cheetah held at the time showed identical symptoms but managed to survive. Various analysis of the illness and deaths were conducted. The illness and death which occurred was initially thought to be lead (or some other toxic agent) poisoning. Yards were searched for car batteries and other sources of contamination. Water, vomit, blood and tissue samples were analysed for a variety of substances including lead, toxins, organochlorins and organophosphates. No definite diagnosis of the causal agent was given at the time.

2. On 11 October 1986 two ten day old cubs could not be found in their enclosure and are suspected of being eaten by the mother. These were the first litter for the mother and concern mounted for the survival of the cubs following heavy rain and hail. The mother had shown varying degrees of maternal knowledge and it is suspected that she may not have sort adequate shelter during bouts of inclement weather.

3. On 4 February 1987 on 11 day old male cub was found dead in a night den upon a routine check of his progress. The cub was emaciated and appeared to not have received sufficient nourishment. The mother's nipples upon inspection had become encrusted and contaminated possibly through excruding of milk without sufficient suckling from the young to maintain clean nipples.

4. On 2 October, 1990 a six year old female cheetah Octavia died following bouts of scouring over several months before developing a severe cough. She was relocated to the Veterinary Quarantine Centre on 25.9.90, continued to deteriorate. Histopathology indicated death can be

attributed to Nephritis (Kidney failure).

5. On 30 March 1991 a sixteen month old male was found apparently drowned in the exhibit moat. The previous day he was observed to have mild hind limb unco-ordination. He was given 1.8 ml (150 mg) Vitamin B_1 by dart. Histopathology of brain tissue revealed thiamine deficiency. It is though the animal disorientated and lacking limb co-ordination wandered into the moat and drowned. Presence of blue green algae in water tested for and found no evidence.

6. On 26 January 1992 a ten year old male was euthanased following deterioration in condition. This animal had a periodic history of condition loss, inappetence and lack of co-ordination stemming from the 1985 disease outbreak. Hymah suffered from degenerative renal failure.

Case history of Illness

1. Female Masai survived the illness of February 1985. She became known for her lethargy tended to obesity and appeared to have significant hearing loss.

   In April 1987 Masai appeared more dull and lethargic than normal. On the third day she was observed to take a fit of some 20 second duration. A five day treatment of Multibex and Clorphenicol resulted in a full recovery.

2. Male Gus appeared dull and lethargic on 12.2.86. This was successfully treated with Multibex 5 ml im. Appeared ataxic and given 150 mg of Vitamin B_1 on 29.3.91 and again on 5.4.91 and 24.4.91.

4. Male Claudius displayed lethargy, inactivity, neck stiff and arched and was taken to VQC 23.11.89. Responded well to treatment of Thiamine and returned to enclosure.

   Another bout of Thiamine deficiency was successfully diagnosed and treated by analysis of the level of the enzyme thiamine pyrophosphate in May 1991.

5. Female Marah displayed lameness and unco-ordination in hind limbs accompanied by some swelling in muscle above hock joint in February 1985. This episode was repeated in February 1986 and treated with Multibex. 5 ml im.

   Marah also exhibited inappetence, lethargy, vomiting, depression, un-co-ordination and distended abdomen following drenching with Ivermectim in March 1989.

   In April 1990 Marah exhibited lethargy, anorexia, regurgitating, slight wobbliness in hind legs. Thiamine injected by dart.

Discussion

Blood serum has analysed of Western Plains Zoo Cheetah for a variety of infectious diseases. They have recently tested negative for Feline Immunodeficiency Virus and Feline Leukemia Virus. They have tested positive for Toxoplasmosis. Tests conducted by the National Institute of Health USA indicated three of the five samples submitted tested positive for feline coronavirus. (Nakuru, Ovambo and Octavia testing positive).

The implications of this positive test for coronavirus is not definitive in that the precise relationship between all the coronavirus is unknown.

As we have shown Western Plains Zoo has had disease outbreaks typical of many institutions holding cheetah. We have been able to eliminate some of the more commonly suspected diseases such as FIV and FeLV and also quite possibly FIP.

So where does this leave us in diagnosing our outbreaks.

Following the 1985 episode all Cheetah illnesses have being treated with

Initially Multibex and later Vitamin B_1 to compensate for thiamine deficiency. Thiamine deficiency is now suspected of being the primary problem in all subsequent illnesses and is considered to be highly likely as the primary agent responsible for the 1985 deaths.

Studdert and Labuc reported on thiamine deficiency in domestic cats and dogs being fed fresh minced meat which contained sulphur dioxide as a preservative.

It would appear the diet fed to Cheetah at Western Plains Zoo has at varying times been deficient in Thiamine due to treatment with Sulphur Dioxide as a preserving agent. This has been compounded by the ineffective supplementation of the diet with thiamine.

At one time it was thought that chickens fed as part of the diet contained a high level of thiaminases due to being grain fed. Thiamine supplementation was given with the chickens. When a shortage in the supply of the chickens occurred the cheetahs missed out on the regular dosage of thiamine and several animals became effected.

Thiamine is a necessary dietary nutrient for most vertebrates. Central nervous disorders due to thiamine deficiency occur in many mammals. Clinical cases of thiamine deficiency in cats have been associated with the feeding of canned commercial cat foods or fish. Thiamine deficiency leads to haemorrhages in the periventricular grey matter in the brain. Treatment with thiamine (100 - 250 mg by intravenous or subcutaneous administration) usually brings about gradual recession of clinical signs, with complete recovery in a few days. Animals with severe neurologic damage may not recover.

Major signs of Thiamine deficiency include: anorexia, vomiting, weight loss, dehydration, paralysis, prostation, abnormal reflexes, convulsions, cardiac disorders, ventral flexion of neck.

Conclusion

Cheetah should be fed a wholesome diet in captivity using carcass meats free of preservative and including items such as rabbit, chicken and red meats. A balanced vitamin powder should be added with extra Vitamin B_1. Calcium should also be added to balance the calcium/phosphorus ratio.

In response to the possible risk of infectious diseases transmission between institutions an Animal Movement Protocol for the Australasian region has being developed for adoption by participating ASMP institutions.

Animal Movement Protocol - Cheetah

A 30 day quarantine period is mandatory prior to the movement of cheetah from one institution to another.

FIP testing is not currently available in Australia, therefore the following should be done.

Prior to shipment:

1. Animal to be vaccinated with killed Feline Enteritis (FE), Feline Rhinotracheitis (FR) and Feline Calicivirus (FC) within last twelve months.
2. Animal to be tested for Toxoplasmosis. Two test to be done three weeks apart looking for both IgG and IgM.
3. Animal to be tested for Feline Immunodeficiency Virus (FIV) using ELISA test.
4. Animal to be tested for Feline Leukemia Virus (FeLV) using both ELISA and IFA tests.
5. Animal to be tested for internal parasites.
6. Animal to be weighed upon departure.

Minimum standards for crating of Cheetah are IATA standard Container Requirement 11.

A complete set of records for the animal to be sent with the animal. A duplicate set to be sent to the receiving Zoo's Record Officer for distribution.

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