Bell, K. (2005). Morbidity and Mortality in hand reared cheetah cubs. Animal Keeper's Forum 7/8: 306-314.

Keywords: Acinonyx jubatus/captivity/cheetah/cub/hand rearing/mortality

Abstract: This paper does not purport to serve as a husbandry protocol (of which there are numerous), but instead examines the major problems reported during the hand rearing of cheetah cubs. Review is made of the reported causes of morbidity and mortality among cheetah cubs, including congenital defects, nutritional deficiencies, gastrointestinal disturbances, immunological dysfunction, hypothermia and infectious pathogens. In association with recommended prophylactic schedules (i.e. vaccination and parasite control), appropriate sanitation, nutrition, careful monitoring of cub health and growth rate is likely to significantly improve cub survivorship.

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Keywords: Acinonyx jubatus/captivity/cheetah/disease/prevalence/renal amyloidosis

Abstract: 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 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.

Morbidity and Mortality in Hand Reared Cheetah Cubs

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Abstract

The mortality rate of artificially reared cubs may be as high as 50% (Grisham, 1989; Kriek et al 1998) in some facilities. With only 20% of captive cheetahs known to reproduce (Marker-Kraus 1997) this mortality is a significant loss to the captive population. Noteworthy progress has been made since the earliest records of hand-rearing cheetahs, whereby mortality rates have been reduced from as high as 89% in the 1970's, to 37% in 1998 (CCF, 1998).

However, this figure is still high and evaluation of the major causes of death suggests a key role for husbandry and environmental factors in modern cub mortality. Congenital defects and stillbirths make up only a quarter of all cub deaths reported (CCF, 1998) whilst nutritional deficiencies, disease and infection, hypothermia and other husbandry-related events are among the remaining causes of death in cubs under the age of 6 months (CCF, 1998). Additionally, the role of management in so-called "natural" or "unavoidable" causes such as maternal abandonment and cannibalism, (accounting for 36% of deaths), must not be overlooked.

This paper does not purport to serve as a husbandry protocol (of which there are numerous), but instead examines the major problems reported during the hand rearing of cheetah cubs. Review is made of the reported causes of morbidity and mortality among cheetah cubs, including congenital defects, nutritional deficiencies, gastrointestinal disturbances, immunological dysfunction, hypothermia and infectious pathogens. In association with recommended prophylactic schedules (i.e. vaccination and parasite control), appropriate sanitation, nutrition, careful monitoring of cub health and growth rate is likely to significantly improve cub survivorship.

Introduction

Cheetahs were first kept in captivity over 4000 years ago by the ancient Egyptians who used tame cheetahs as hunting companions and noble gifts (Hunter and Hamman, 2003). However, despite a long history of cheetahs in zoological facilities and private animal collections, it was not until 1956 that cheetahs were bred successfully in captivity (CCF, 1998). This litter did not survive past three days of age and although the earliest report of successfully hand-reared cheetah was from a European zoo in 1960, (Encke, 1960) it took another 10 years for the zoological community to report the successful rearing of a litter by its mother (CCF, 1998). Even today, only 15 - 20% of the captive cheetah population has reproduced, contributing significantly to the disproportionate genetic representation occurring within this population (CCF, 1998; Marker-Kraus, 1997; O'Brien et al, 1985).

Cubs are removed from their mother prior to weaning for a variety of reasons, including maternal neglect, injury, disease or for training/educational functions. Lombardi et al (2001) report that 75% of hand-reared cubs between 1964 and 2000 were removed due to illness or neglect, at an average age of 7 days, and 24% of these cubs did not survive. The remaining 25% were healthy cubs, removed for educational purposes when much older (on average, 26 days) (Lombardi et al, 2001). It is not surprising that this latter proportion of cubs suffered a far lower mortality rate (4%) than cubs removed for medical reasons (Lombardi et al 2001).

Infant Mortality

Neonates lack the reserves of adult animals and may show only slight changes in disposition prior to rapidly becoming moribund (Meier, 1986). The susceptibility of neonatal cheetahs is emphasized in the literature with infant mortality as high as 89% in the 1970's and 52% in the 1980's (CCF, 1998). With greater understanding of cheetah physiology, improved husbandry practices and increased inter-facility reporting, infant mortality rates among captive cheetahs have decreased significantly since then (CCF, 1998). However, the most recent statistics show that 36.7% of cheetahs still die before reaching adulthood and 23% of cubs die within their first year of life (CCF, 1998).

Causes of death are as varied as the reasons for removal from the mother, with nearly a quarter of cubs dying of unknown causes prior to 30 days of age (CCF, 1998). The difference between what may be termed "natural" deaths (i.e. congenital defects or stillbirths) and "environment-related" deaths (i.e. maternal neglect, cannibalism, nutritional deficiencies, exposure and trauma) is stark. O'Brien *et al* (1985) have suggested that genetic homozygosity is primarily responsible for high infant mortality rates in this species, although this conclusion has been strongly debated by other authors (Caro et al, 1987; Laurenson et al, 1995). Only 5% of deaths are due to congenital defects and 20% to stillbirths (CCF, 1998). However, as much as 51% of deaths were due to "environmental" causes (CCF, 1998) and the greater part of many medical conditions may be due to environmental and husbandry issues (McManamon, 1993). It is noted that maternal neglect and cannibalism may be considered as "natural" causes of death. However, the frequency of these mortalities in free-ranging, mother-reared litters is much lower than in captivity (8.2% and 17%, respectively, in the case of maternal neglect) (Caro, 1994; Laurenson, 1994). Maternal neglect is frequently associated with stress in both free-ranging and captive cheetahs (Caro, 1994; CCF, 1998; Laurenson, 1994). Captive females exposed to predator species within sight or smell of cubbing den, or housing with other conspecifics (specifically males) are more likely to abandon or cannibalize their cubs (CCF, 1998). These findings make it more likely that environmental, rather than innate, factors underpin these common causes of infant mortality in captivity. Nonetheless, environmental factors and genetics cannot be examined in isolation and large inter-facility variation in infant mortality may be the combined result of genetic lineages and husbandry factors (Marker-Kraus, 1997).

Marker-Kraus (1997) determined that the most vulnerable age for captive cheetahs is the period between birth and one month of age. The first 6 weeks of life are particularly important, as cubs are unable to efficiently thermo-regulate, are relatively immobile, unable to pass urine or faeces without ano-genital stimulation, and commonly have an intestinal parasite burden after *in utero* transfer. Between 1956 and 1994, 21% of infant deaths were seen in cubs less than 1 month of age, and 28% in cubs less than 6 months of age, which is much higher than the values reported for other non-inbred zoo animals (Marker-Kraus, 1997). Yet, encouragingly, a recent survey of 15 North American facilities reported a mortality rate of only 19% in hand-reared cubs prior to 3 months of age (Lombardi et al, 2001).

A second period of susceptibility to disease may exist in captive situations for cubs between 4 – 7 months of age. After 4 months of age, typical vaccination schedules should be completed and this may be the time that cubs are introduced into novel environments and therefore exposed to novel pathogens. Diarrhoea is especially common in this age group (Caldwell, 2004). After approximately 7 months of age, risk of infection appears to decrease as cub resistance matures.

Congenital defects

4.1% of free-ranging cubs deaths were recorded as due to the cubs being “unviable” (Caro, 1994), presumed to suffer some form of congenital defect, which is remarkably similar to the 5% reported in captivity (CCF, 1998). Failure of cubs to develop a suckling instinct may be indicative of central nervous system dysfunction, whilst palate deformities may also result in suckling difficulties (Weilemann, 1962). Milk seen expelled from the cubs’ nose or mouth during suckling is often caused by cleft palate and the cub should be checked accordingly. Mother-reared cubs suffering congenital defects may be cannibalised prior to examination by zoological staff and as such the nature of many congenital defects is not described in the literature.

Nutritional Deficiencies

Nutritional deficits caused death in 7% of cubs born between 1829 and 1994 (CCF, 1998). Nutritional disorders are typically seen at approximately 4 to 6 months of age (i.e. the natural time of weaning) (CCF, 1998). One of the most common types of nutritional disorders is the imbalance of dietary calcium and phosphorus, commonly presenting as developmental bone malformation (osteodystrophy) due to insufficient supply of dietary calcium (CCF, 1998). Alternatively, excessive supplementation of calcium is known to contribute towards a developmental deformity of the forelegs (osteochondrosis dissecans), which is evident in forward bowing and outward turning of the paws, termed carpal valgus (CCF, 1998). The role of nutrition in metabolic bone disease is well covered in the literature. Investigation of the role of nutrition in carpal valgus of hand-reared cubs is currently underway by the author.

Post mortem examination of cubs at a South African facility suffering mortality rates in excess of 85% over a period of 18 months revealed clinical signs and lesions indicative

of congenital vitamin E and selenium deficiency (Kriek et al 1998). Concurrent *Salmonella* infections were deemed secondary in nature (Kriek et al 1998), as it is likely that nutritional inadequacies rendered the animals' immune systems incapable of mounting sufficient defence against opportunistic parasites and pathogens.

Lethargy, ataxia and ventroflexion of the head are often seen in the critical stages of thiamine deficiency, whilst seizures and periods of tachycardia or bradycardia may also be exhibited (NRC, 1986). Although no dietary analyses were reported, the characteristic symptoms and response of surviving animals to parenterally administered thiamine led to the belief that thiamine deficiency was primarily responsible for a number of cheetah deaths at one Australian facility (Christie, 1997).

Hoff (1960) reports the occurrence of head tremors in hand-reared exotic felines between 10 – 20 days of age. Lateral head tremors in association with ataxia, partial collapse, loss of balance and a staggering gait are among the known symptoms of copper deficiency (CCF, 1998; Downes, 1997) and the condition has resulted in fatal respiratory distress in some cubs (Downes, 1997). Treatment with dietary copper is usually effective but chronic deficiency can render the effects permanent (CCF, 1998; Downes, 1997). Evaluation of chicken and guinea fowl carcasses demonstrated the greater copper content of the latter, suggestive of a wild-diet component capable of providing sufficient copper to growing cubs (Downes, 1997).

Exposure and Hypothermia

Abandoned cubs may be left outside of the den, where they are exposed to environmental elements and are at risk of pneumonia and hypothermia. Pneumonia is frequently reported as a major cause of neonatal death in North American facilities (Munson, 1993). Low body temperatures are often accompanied by low blood glucose (Reid and Meier, 1996). Although the central nervous system depends on glucose as its' primary energy source, glucose requirements are reduced drastically during hypothermia (Reid and Meier, 1996). Sterile warm fluids administered per os and per rectum are efficient ways of raising the core body temperature, as well as stimulating gastrointestinal tract function (McManamon, 1993). However, during severe hypothermia, the gastrointestinal tract enzymatic systems function abnormally and as such hypothermic neonates should not be fed until core body temperature is returned to normal (Meier, 1986). In certain cases, bacterial overgrowth and bloat may result if milk formula or glucose is administered *per os,* whilst at best the formula is likely to be inadequately utilized by the cub (Meier, 1986).

Gastrointestinal Disturbance

Necrotizing enterocolitis, vomiting, ileus (gut stasis with bloat), severe parasite infestations and bacterial enteritis (e.g. salmonellosis) are the most serious of gastrointestinal disorders in neonates (Reid and Meier, 1996). Such problems can result in diarrhoea, dehydration and electrolyte imbalances, all of which can quickly become fatal in young animals (Reid and Meier, 1996; CCF, 2000).

Diarrhoea

Diarrhoea represents one of the most common health problems in captive cheetah and is particularly prevalent in cubs under the age of seven months (Caldwell, 2004). Causes of diarrhoea include ascarid infections, coccidiosis, giardia, trichomonas infection, dietary intolerance, environmental stress, enteric bacterial or viral infection, and lympho-plasmacytic enteritis (Caldwell, 2004). Identifying the cause of diarrhoea, whilst treating the symptoms (i.e. fluid therapy), is an important step in treating diarrhoea in cheetah (Caldwell, 2004).

Some commercial cat foods are known to result in chronic low grade diarrhoea in cheetahs (Bell, 2004; Caldwell, 2004) and the addition of meat to these diets may see a return to normal stool consistency. If an animal's carbohydrate absorption ability is exceeded, the fermentative activity of gut enterobacteria upon the residue may result in osmotic diarrhoea.

Often diagnosis of the cause of diarrhoea can be made from dietary evaluation, faecal floatation for intestinal parasites, faecal smears and the nature of the stool (Caldwell, 2004). Diarrhoea originating in the small intestine commonly presents as large amounts of liquid stool being passed infrequently, ranging from light orange to reddish in colour (Caldwell, 2004). A red mucous may be present around the faeces and initial segments of faeces may be relatively well formed (Caldwell, 2004). Black tarry faeces is indicative of haemorrhaging in the upper intestinal tract, since blood present in the small intestine will be digested as it passes along the gastrointestinal tract, giving the characteristic black colour.

Large intestine disorders are less responsive to treatment and present as small amounts of liquid faeces being passed frequently (Caldwell, 2004). Fresh (red) blood seen in the faeces is indicative of haemorrhaging in the lower intestinal tract (e.g. colitis). Ultrasound, abdominal palpation and endoscopies or mucosal biopsies may be required to diagnose some causes of diarrhoea. However, since these are invasive procedures, often requiring sedation, they are generally avoided due to the risks of sedation in young cubs (Caldwell, 2004).

Starvation for 12 – 24 hours is common practice and typically produces good results for diarrhoea of large intestinal origin (Caldwell, 2004; McManamon, 1993; Schumann and Schumann, 1994; Reid and Meier, 1996). Alternatively, formula may be diluted with oral electrolyte solution and the total volume decreased by 20-40% for 8 – 12 hours if diarrhoea is not severe or persistent (CCF, 1998). Dietary changes should be made gradually and food offered in small amounts frequently so as to prevent overburdening the gastrointestinal tract. Reasonable success has been seen with diets of raw and/or boiled chicken, as well as with formulas such as Iams® (Caldwell, 2004) or Hills Science Diet® Intestinal Diet© (pers. obs., 2004).

A variety of pharmaceuticals are available for the treatment of diarrhoea in cheetahs. However, indiscriminate use of antibiotics should be avoided due to the risk of bacterial resistance and disruption to normal gastrointestinal microflora (Caldwell, 2004; van Zyl, pers. comm., 2004). Stool binding agents should be used with precaution whilst gut-lining pharmaceuticals may be advisable where risk of gastric ulceration is apparent. Cheetahs appear to have a similar intestinal microflora composition to the domestic cat (Caldwell, 2004) and as such the administration of commercially available pro-biotic preparations (e.g. *Lactobacillus* supplements) to promote regrowth of commensal gut bacteria may be beneficial during recovery (McManamon, 1993; Schumann and Schumann, 1994; Reid and Meier, 1996).

Dehydration and Electrolyte Imbalances

Electrolyte and fluid replacement is essential in treating cubs with gastrointestinal disturbances as cubs are especially prone to dehydration (Caldwell, 2004; Schumann and Schumann, 1994). Signs of dehydration include skin tenting, prolapse of the nictitating membrane (third eyelid), dull coat, and lethargy (Caldwell, 2004). Sodium deficits may cause convulsions, nausea, vomiting, anorexia, dehydration, and lethargy (Meier, 1986). Potassium deficits are also seen during illnesses involving vomiting or diarrhoea and cause muscular, gastrointestinal and cardiovascular dysfunction (Meier, 1986). Conversely, excessive potassium induces symptoms such as restlessness, weakness and cardiac arrhythmias (Meier, 1986). Convulsions may also be due to calcium, glucose and/or magnesium deficiencies (Meier, 1986).

Internal Parasites

Internal parasites can cause diarrhoea (CCF, 2000) and can be passed from mother to cub whilst *in utero*, via the milk or acquired soon after birth (Reid and Meier, 1996). Cubs may be infected with *Ascariasis* and *Ancylostomasis* before birth and typically exhibit symptoms such as weakness, poor weight gain, abdominal distension, dehydration, anaemia and bloody mucoid faeces as early as 3 - 4 days of age (Meier, 1986). Whipworm infestations can be fatal and clinical symptoms of internal parasite infestations include loose, watery (sometimes bloody) faeces, vomiting, anemia, fever, dehydration, weakness, inappetence, dullness and signs of abdominal pain (Reid and Meier, 1996). Tapeworm infections are not typically life-threatening. Diagnosis includes faecal examination and/or radiography (Reid and Meier, 1996).

Constipation

Young cubs require stimulation in order to defecate and urinate. Dietary changes, inappropriate stimulation or the ingestion of foreign material may cause constipation. Meconium (the first faeces) can also cause constipation, bloat, inappetence, and abdominal distress if not passed (Reid and Meier, 1996). A warm enema can typically rectify the problem (Reid and Meier, 1996).

Intestinal blockages caused by a heavy worm burden are not uncommon in young animals. De-worming animals with a heavy parasite burden should aim to provide a graduated elimination of parasites. Large infestations that are treated with heavy doses of anti-helmintic drugs may cause intestinal obstructions as the parasites die off at the same time (van Zyl, pers. comm., 2004).

Immune System Dysfunction

Cubs may gain a degree of passive immunity by the transfer of maternal antibodies via the placenta but colostrum appears to be a more significant source of immunoglobulins (Meier, 1986). A neonatal intestine can absorb intact immunoglobulins for up to 36 hours after birth but after that the colostrum will have only local effects on the intestine (Reid and Meier, 1996). After this period, cubs are particularly vulnerable to bacterial, fungal and viral pathogens.

The thymus plays an important role in cell-mediated immunity through the maturation and release of large numbers of T-lymphocytes into circulation (Ross et al 1995). The organ is particularly important in early life as the humoral immune system is developing. Premature thymic atrophy (PTA) has been observed in increasing frequency at a South African breeding facility (Caldwell, 2004). Since diagnosis is typically only made upon post-mortem after death due to apparently non-immune related causes, PTA may represent an, as yet, unidentified problem within other facilities around the world, where post-mortems are either not performed or the thymus was not examined.

Symptoms such as chronic watery diarrhoea, wasting, vomition, suppurative pneumonia and perforating gastric ulcers have been seen in cubs that were later found to suffer from PTA (Caldwell, 2004). The cause of death in these specific cubs was peritonitis as a consequence of the perforated gastric ulcer (Caldwell, 2004). Although the gastrointestinal pathology in these cubs resulted in their deaths, the lack of functional immune system is likely to have played a significant role in the initiating pathogens successful infection, or alternatively may have mediated auto-immune mechanisms.

Diseases associated with immune suppression such as cadididasis, bacterial enteritis, pneumonia and pleuritis are also reported causes of morbidity and mortality amongst cheetah cubs (Lane, 2004). Hence, the role of immunosuppressive factors is deserving of further research and the involvement of dietary factors such as antioxidants, isoflavones and selenium in the functioning of the immune system should also be addressed. Isoflavone-induced perturbations in immunology are currently under investigation by the author.

A degree of passive immunity can be obtained from the administration of antibody-containing serum (collected from an immunocompetent, FIP and FIV negative animal) (CCF, 1998). Alternatively or in addition to this, homeopathic, nutritional (e.g. B-vitamin complexes) and herbal preparations (e.g. Moducare© Plant Sterols) can be utilised to assist immune function (Hindmarch, B., pers. comm.).

Infectious Causes

Respiratory Infection

Young mammals must breathe through their noses when suckling and therefore, a respiratory infection can be life threatening; either causing dehydration and malnutrition or depriving the cub of oxygen and carbon dioxide exchange (Reid and Meier, 1996). Feline Upper Respiratory Disease (FURD) is a highly contagious viral disease, commonly referred to as 'cat flu'. Younger animals often show more marked symptoms including sneezing and nasal discharge, followed by anorexia, depression, fever and signs of nasal and ocular ulceration, hypersalivation and coughing (Reid and Meier, 1996; Simpson, 1996). Secondary bacterial infection causes thick mucopurulent ocular and nasal discharge, loss of olfaction and blocked nasal passages (Simpson, 1996). Diagnosis is based on clinical symptoms but the virus can be isolated from oropharyngeal swabs (Simpson, 1996). Treatment includes isolation and symptomatic relief, although many felines that recover from FURD become carriers of the disease (Simpson, 1996).

Protozoan and Bacterial Infections

All carnivores are susceptible to toxoplasmosis but it appears to be more common in felids (Meier, 1986). Symptoms do not always occur but may include anemia, retinitis, iritis, hepatitis, blindness, central nervous disorders, respiratory distress and diarrhoea (Meier, 1986). Cubs with pre-existing conditions or under particular stress (e.g. worm burden, weaning) are most likely to show symptoms. Infection of felines may occur *in utero*, or from the ingestion of oocysts in contaminated faeces or eating infected intermediate hosts e.g. mice or infected sheep meat (Meier, 1986). Toxoplasmosis is diagnosed through the presence of oocysts (fertilized cells of the protozoan) in faeces and immunological tests. There is currently no satisfactory treatment for toxoplasmosis in felines.

Coccidiosis usually presents as marked diarrhoea, particularly in young animals. Infection is often associated with stress and most typical in cubs between 1 – 3 months of age (Meier, 1986). Cubs with watery, mucoid or bloody diarrhoea, dehydration and/or secondary bacterial infections should be evaluated for coccidiosis (Meier, 1986). Diagnosis is based on the presence of oocysts in the faeces and treatment usually involves the use of anti-microbial drugs and re-hydration. Salmonellosis is commonly referred to as "food poisoning" and usually presents as vomiting, diarrhoea and anorexia. Infection with *Salmonella* bacteria and toxins produced by such bacteria requires antibacterial drugs and symptomatic relief of dehydration associated with vomiting and diarrhoea.

Septicemia may cause lethargy, jaundice (yellowing of the mucous membranes), weight loss, poor nursing, fever, hypothermia, irritability, increased respiratory rate, vomiting, diarrhoea, and abdominal distention (Reid and Meier, 1996). Antibiotic therapy should

be started immediately and bacterial culture/sensitivity tests are useful in determining the most effective antibiotic regime (Reid and Meier, 1996).

Omphalitis (umbilical infection) was the underlying cause in 1.4% of felid cubs that died at San Diego Zoological Park between 1964 and 1982 (Meier, 1986). Such infections may lead to renal, musculoskeletal and cardiac complications and prognosis for recovery is guarded if there is no rapid response to therapy (Reid and Meier, 1996). Redness, pain, swelling or discharge from the umbilical stump may appear as well as joint swelling and lameness (Reid and Meier, 1996). Local and systemic antibiotics are advised and sanitation must be of the highest standard (Reid and Meier, 1996).

Feline Panleukopenia

This highly infectious virus (a.k.a. feline infectious enteritis, feline distemper and feline parvovirus) has a high rate of infection and mortality, especially amongst young, unvaccinated animals. The disease may cause sudden death, depression, anorexia, persistent vomiting and painful palpation of abdomen (Simpson, 1996). Diarrhoea may occur in the later stages and is usually yellow-brown liquid, possibly containing blood (Simpson, 1996). Diagnosis is based on vaccination status combined with clinical signs, hematological tests and detection of the virus in the faeces (Simpson, 1996). Currently there is no treatment for the virus so therapy is supportive in nature (i.e. fluid therapy and broad spectrum antibiotics to prevent secondary bacterial infection) (Simpson, 1996).

Feline Infectious Peritonitis (FIP)

The corona virus responsible for this fatal disease is similar to that causing the commonly encountered enteric corona virus (Simpson, 1996). The virus is readily destroyed by most disinfectants and transmission is thought to generally occur through contact with infected faeces or urine, rather than direct contact (Simpson, 1996). Early symptoms are often vague but may include fever, anorexia, weight loss and diarrhoea. A prolapse of the nictitating membrane is also common, but symptoms soon progress to more specific signs (Simpson, 1996). Some cats are thought to be genetically predisposed to higher sensitivity to this disease than others. There are two forms of the disease and the more common, less severe of cases is termed effusive FIP where fluid accumulates in the abdomen (Simpson, 1996). Non-effusive FIP causes granulomatous lesions on abdominal organs and often results in organ failure (Simpson, 1996). Half of the cases with non-effusive FIP display central nervous signs and eye lesions may be associated with retinitis and uveitis (Simpson, 1996). The disease is difficult to interpret on serology and biopsy collection and histopathological examination of lesions is currently the only definitive diagnosis (Simpson, 1996). Prognosis is guarded and there is no specific treatment (Simpson, 1996). FIP is a significant cause of mortality in captive cheetahs worldwide.

Growth rates

Knowledge of normal growth curves in mother-reared cubs is critical to determining optimum growth in hand-reared cubs. Departure from normal growth rates may provide an early-warning system for the detection of medical problems in hand-reared cubs (Wack et al 1991) and only small changes in weight may be significant (Meier, 1986). Recognition of potential medical problems provides the opportunity for earlier intervention, which is likely to decrease cub morbidity and mortality. Rough estimates of growth rates can be made from calculating the adult weight versus the infant weight and time taken to reach maturity (Reid and Meier, 1996). For the cheetah, this works out to be approximately 0.38kg weight gain per week (Average adult male weight taken as 40kg and average birth weight as 0.5kg).

An average growth rate of 40 – 50g/d, reported for hand-reared cubs (Pers. obs., Encke, 1960; Hall, N., pers. comm.; Lombardi et al, 2001) is consistent with the average growth rate observed in six mother-reared litters (21 cubs) at a North American facility (45g/d) (Wack et al 1991) (Fig. 1.). Whilst it may appear that no difference exists between mother-reared and hand-raised cubs (CCF, 1998; Lee, 1992) it is important to consider each individual animals' growth rate, rather than simply average growth rates for a litter or one cub over a long period of time.

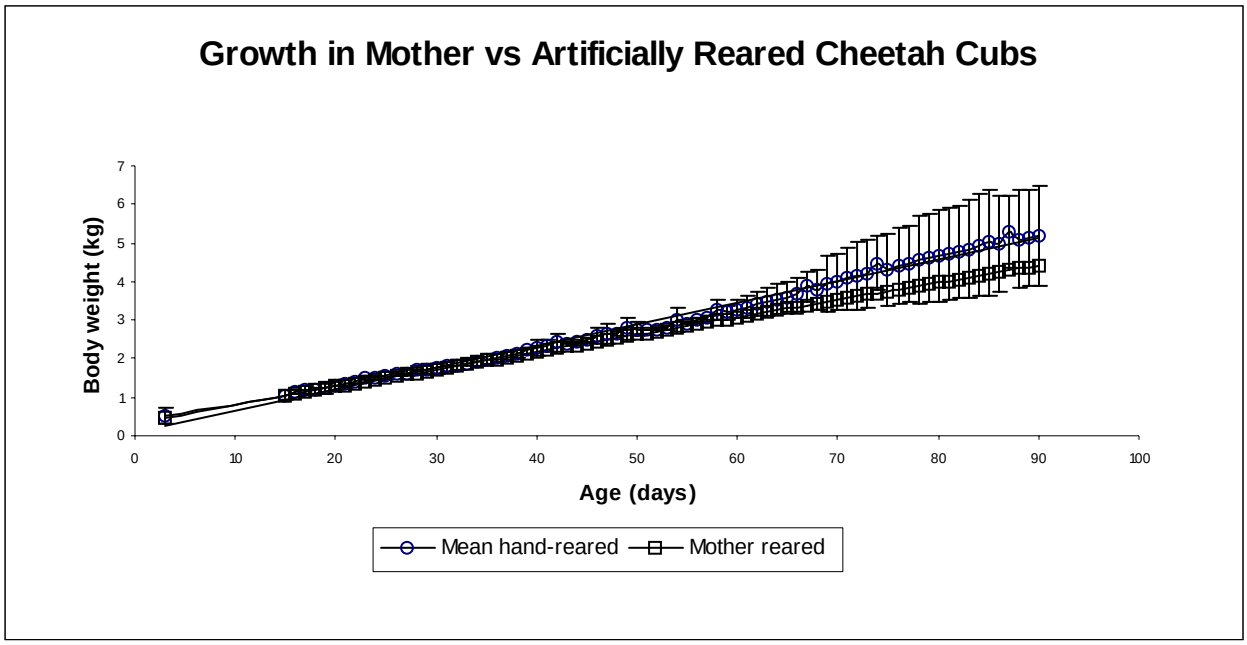

Figure 1. Growth rate of Cheetah Cubs reared artificially compared to mother-reared cubs

Hand-rearing data taken from Encke, 1960, Hall, N., pers., comm. (2004), Lombardi et al (2001), Schumann and Schumann (1994), pers. obs. (2004). Mother rearing data taken from Wack et al (1991).

Conclusion

Neonatal cheetahs removed from their mother due to illness or injury are at a weighty disadvantage, in terms of survivability, compared to healthy cubs removed for educational purposes. Even with access to specialist veterinary medicine, many cubs reach zoological nurseries in a severely moribund state, with little hope for survival. In order to reduce infant mortality rates in captive situations, focus must turn to the prevention and/or early detection of morbidity in cubs. Factors predisposing females to abandon, cannibalize or traumatize cubs must be identified and eliminated, or at least minimized. Litters born to females at risk of these behaviours should be considered for hand rearing, prior to the onset of potentially fatal problems.

Vigilant monitoring of cubs, combined with careful management of the mothers' environment should promote recognition of initial warning signs of impending medical problems. Precipitating earlier intervention is likely to result in more rapid and effective responses than therapy initiated in the later stages of a disease process. Increased cub survival rates and reduced long-term consequences of morbidity during growth and development in cubs should be the priority when rearing cheetah in captivity.

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