Kat PW. 1993. Genetics of the cheetah: What we know and how we should use this knowledge. Swara:13.

Keywords: Acinonyx jubatus/cheetah/conservation/disease/genetic uniformity/genetics/population genetics

Abstract: The number of people the author met that are conversant with the genetics of cheetahs is amazing. The wisdom of programmes that propose to curve a variety of ills merely by increasing genetic variation, however, are unconvincing, and have largely resulted from a lack of understanding of complexities involved.

## Genetics of the cheetah:

by Pieter W. Kat

## What we know and how we should use this knowledge.

S A GENETICIST, I AM AMAZED at the number of people I meet Late that are conversant with the genetics of cheetahs. Through a plethora of articles in the popular press over the past ten years, an astonishing variety of people have not only become cognizant that this species is rather unusual genetically, but these same people are now also proposing a diversity of 'solutions' to this 'problem'. Although these calls for action are well-meaning, I am far from certain that these proposals have the best interests of the animals in mind, largely because there may be only just beginning to gather the kind of information necessary to glean insight into the genetics of large mammal species, and until we have much more of this kind of knowledge, we should he itate to tinker unintelligently. Cheetahs have long held our fascination as the fastest of all terrestrial animals, and have always made popular exhibits at 200 s.

However, the species apparently did not breed well in captivity, and captive populations had to be regularly augmented with individuals caught in the wild. When reproductive physiologists such as David Wildt at the National Zoo in Washington DC began to make assessments of cheetahs, they noted that infant mortality levels were high, and that males exhibited low levels of testosterone coupled with a very high frequency of nonfunctional, aberrant sperm. Such abnormal reproductive traits can be characteristic of animals that have been highly inbred, and the next step was to examine a number of cheetahs genetically. These studies, performed in large part by Steve O'Brien at the US National Cancer Institute, proved that cheetah individuals were all remarkably similar to each other, again indicative of a high level of inbreeding, which leads to an erosion of genetic variability.

To determine the level of genetic identity among cheetahs, O'Brien and his colleagues next performed an ingenious experiment: rather than only examining variability in genetic markers, they tested the relationships among unrelated cheetahs by challenging their immune system. The genetics of immune resistance are complex and variable, as our

immune systems constantly have to meet challenges from a number of pathogens in the environment. Central to this resistance, however, is the ability of the immune surveillance system to be able to differentiate self from an invader. So well tuned is this system that fetuses have to be carefully sequestered from the mother's immune system. Similarly, organ transplants have to be meticulously chosen to best match donor and recipient, and supplemented immunosuppressive drugs. The experiment performed by O'Brien's group consisted of a series of skin transplants, where each cheetah received a small patch of skin from another cheetah, a domestic cat and as a control, itself. All cheetahs rejected the cat transplant, but all transplants from other cheetahs were accepted, indicating that the immune system could not differentiate the transplant from the cheetah's own tissues: such acceptance again indicates a very high level of genetic resemblance.

This observation caused concern. If cheetahs were all so genetically similar to each other, did that imply that a disease would affect all individuals in the same fashion, possibly causing very high mortality? The genetics of disease resistance are still imperfectly known, but it stands to reason that the diversity of responses among individuals of our own species to a disease such as the flu, or measles, or chickenpox indicates that genetic differences among us could influence how well we deal with disease. Shortly after the skin transplant experiment, these fears were realized when an outbreak of feline infectious peritonitis caused high mortality among cheetahs in a US breeding colony. This incident was worrisome for other cheetah populations, captive and free-ranging: could an epidemic of this or a similar disease cause sudden and significant mortality among these animals?

Once it was established that there was very little genetic variation among captive cheetahs in the US, which were all of southern African origin, O'Brien and his team travelled to East Africa to sample captive and freeranging cheetahs there. Once again, David Wildt observed a high incidence of sperm abnormalities and low testosterone levels, and

O'Brien found very low levels of genetic variation. What was immediately apparent, however, was that eastern African cheetahs were genetically different from southern African conspecifics, and this led to widespread speculation that genetic variability could be increased by hybridizing cheetahs from these two localities.

This, then, is the basis for the arguments to crossbreed cheetahs. The wisdom of programmes that propose to cure a variety of ills merely by increasing genetic variation, however, are unconvincing, and have largely resulted from a lack of understanding of complexities involved. Species are generally composed of populations that differ genetically to varying extents from each other. Some species, such as African Wild Dogs and Grant's Gazelles, show very large genetic differences among populations sampled from different parts of the geographic range. These differences are likely to be adaptive to some extent, and this diversity is important because, as has been pointed out many times before, such variation is the raw material of evolution. Species depauperate in genetic variability are thought to be compromised in their potential to evolve. Nevertheless, management plans should avoid misguided programmes to increase levels of genetic variability through crossbreeding individuals drawn from such dissimilar populations.

Also, is this crossbreeding really necessary from a management point of view? Do we really have sufficient information to propose that wild eastern and southern African cheetahs are not reproducing well, or are especially vulnerable to decimation by disease? Could the disease problems and reproductive difficulties that have been observed among captive populations be related to some extent to factors associated with captivity? It is my opinion that we cannot yet claim to have a sufficient understanding of these issues, and that there is a necessity to perform a variety of studies before we propose drastic measures such as crossbreeding and releasing hybrid cheetahs. Such studies would require a joint effort by behaviourists, geneticists and veterinarians. As Karl Ammann has pointed out, caution rather than emotion should guide our decisions in this instance, and a well-balanced plan of action is needed.

ιž

## STATE IS SELECTION OF THE SELECTION OF T



1993 SEPT/OCT VOL.16 NO.5

**EDISTU** 

Cheetahs in Namible Ecyptrand Tanzania