Ecological and Genetic Factors in Conservation: A Cautionary Tale

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During the last decade, genetic problems potentially faced by small populations have constituted a central topic in conservation biology (*1*). Genetic theory predicts that inbreeding between members of small populations will reveal deleterious recessive alleles, which may be manifested in lowered fecundity, high infant mortality, and reduced growth rates that could eventually drive a population to extinction (*2*). In addition, loss of heterozygosity may reduce a population's ability to respond to future environmental change, such that the probability of extinction is increased or, at best, opportunities for evolution are limited (*3*). Consequently, genetic considerations play a central role in identifying risks to wild and captive populations (*4*).

The effects of inbreeding and loss of genetic diversity on the persistence of populations in the real world are, however, increasingly questionable (*5*). Although inbreeding results in demonstrable costs in captive (*6*) and wild situations (*7*), it has yet to be shown that inbreeding depression has caused any wild population to decline (*8*). Similarly, although loss of heterozygosity has detrimental impact on individual fitness, no population has gone extinct as a result. In the absence of such empirical data, circumstantial evidence is often marshalled to support the importance of genetic factors driving wild populations to extinction [for example, (*9*)]. One key example used in such arguments has been the cheetah because it is depauperate in genetic variation (*10*) and has poor survival prospects in the wild (*11*).

Specifically, a genetic survey of 55 cheetahs from southern Africa demonstrated a complete absence of genetic variation at each of 47 allozyme loci (*10*). Two-dimensional gel electrophoresis of 155 proteins from six animals revealed a percentage polymorphism of 3.2% and average heterozygosity of 0.013, both far lower than other Felidae sampled (*12*) and lower than other mammalian populations, which averaged 14.7% polymorphisms and 0.036 heterozygosity (*13*). Subsequent work in East Africa, mostly in the Serengeti ecosystem, Tanzania, detected only two allozyme polymorphisms in an electrophoretic survey of the products of 49 genetic loci (*14*). Additional evidence of depauperate variation came from 14 reciprocal skin grafts performed between pairs of unrelated cheetahs (*15*). Eleven grafts were accepted and three showed slow rejection, in marked contrast to skin of domestic cats, which was rejected by cheetahs within 2 weeks of the operation. These results suggested that the major histocompatability complex (MHC), a highly polymorphic group of tightly linked loci in vertebrates that is responsible for cell-mediated rejection of allogenic skin grafts, was unusually invariate in cheetahs.

As homozygous loci may expose deleterious recessives, O'Brien *et al.* (*15*) suggested that juvenile mortality should be high in cheetahs and cited elevated rates of juvenile mortality in captivity in comparison with other exotics [but see (*16*)]. They also reasoned that species-wide homozygosity would make populations and the species more susceptible to extinction from pathogens: If one member was unable to mount an effective immune response to a pathogen, the whole population would be similarly vulnerable. Examining a case study of disease sweeping through a successful felid breeding colony of 42 cheetahs in Oregon, O'Brien *et al.* noted that 43% [or 60%, (*17*)] died from coronavirus-associated diseases, including feline infectious peritonitis, while none of the lions developed symptoms. Rightly, the authors noted that such mortality was consistent with but was not necessarily the consequence of genetic uniformity, and in their subsequent papers were properly cautious in linking their genetic findings to the conservation problems faced by cheetahs such as low population density compared to other carnivores (*18*) and poor breeding performance in captivity (*19*). Nevertheless, a considerable secondary conservation and evolutionary literature, as well as the popular press, has uncritically assumed that lack of genetic variation is the cause of the cheetah's plight in the wild and in captivity [for example, (*20*)]. Now, in light of new evidence that has emerged from a long-term study of cheetah reproduction in the wild, we reexamined the potential consequences of genetic homozygosity for this species.

Laurenson (*21*) radio-collared female cheetahs in the Serengeti, relocated them regularly in their 800-km^2 home ranges, and thereby pinpointed the timing of births and location of lairs. Soon after a female had given birth, Laurenson entered lairs to count and weigh the cubs while the mother was known to be away hunting. Regular monitoring of the family showed that cubs suffered from extremely high mortality in the first weeks of life such that only 36 out of 125 cubs (29%) emerged from the lair at 2 months of age. By the time cubs reached independence over a year later, only 5% had survived. Other long-term studies of large and medium-sized felids have yet to document mortality in the lair, but comparative mortality estimates between emergence and independence average 50% as opposed to 80% for cheetahs (*22*).

Direct observation of lairs and circumstantial evidence surrounding cub disappearances in many instances enabled the causes of mortality to be determined. Predation was by far the most important cause (35.5 out of 48.5 cubs; one litter size was unknown but estimated as 3.5, the mean size); four cubs were abandoned by their mothers when prey was scarce, seven died of fire and exposure, and two may have been inviable. Lions were responsible for all of the observed instances of predation in the lair and, with spotted hyenas, were responsible for most of the predation in this and parallel studies conducted in the same ecosystem (*23*). Stringent checks ruled out the possibility that mortality was influenced by visits to the lair or intensive observation schedules (*24*). Elsewhere in sub-Saharan Africa, large carnivores may also be important in depressing cheetah populations. Analysis suggests that across protected areas cheetah densities are low where lion densities are high and vice versa once the effects of prey biomass in the range 15 to 60 kg have been removed (*25*). Predation on young cubs is therefore a strong candidate for explaining why cheetahs have low population densities in comparison with lions and spotted hyenas in many areas of Africa.

These findings suggest that genetics may have been overemphasized in relation to the plight of cheetahs. First, only two of the observed cub deaths in the lair could have been attributable to genetic defects. Second, neonatal mortality in the first days of life before cubs were examined was probably low because observed litter sizes were similar to those reported at birth in captivity. Third, elevated juvenile mortality in utero in this species seems improbable because mothers reproduced extremely rapidly following the loss of an unweaned litter. Fourth, the high numbers of females breeding and rapid rates of litter production imply that neither the reproductive anatomy or physiology of either sex is functionally compromised as a result of genetic monomorphism (*26*). Finally,

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wild cheetahs tested seropositive to a number of infectious agents or microparasites including feline coronavirus (32% to 62%), herpesvirus (44%), feline immunodeficiency virus (22%), and toxoplasmosis (69%) (*17*, *27*), and captive cheetahs produced antibodies after vaccination with modified live feline panleukopenia, herpes, and calci viruses (*28*). Similarly, only 60% (that is, not nearly all) of captive cheetahs succumbed to feline infectious peritonitis in Oregon. All of these studies demonstrate a variability in individuals' responses to pathogens and show that some cheetahs' immune systems can recognize and mount an immune response to a range of agents. While lack of variation at the MHC leaves a species potentially vulnerable to disease, as yet there is no evidence that a disease has circumnavigated the immune defenses of all cheetahs. With hindsight, it is easy to understand why exciting genetic results were invoked to explain low population density of cheetahs, but predation on cubs is clearly more important in natural populations.

What of cheetahs' poor reproductive performance in captivity—can genetic problems account for their poor breeding success? The key problem preventing the North American cheetah population from being self-sustaining is failure of females to conceive (*19*). However, a physiological survey of 68 captive females shows almost no anatomical or physiological impairment of reproductive function (*26*). Instead, marked differences in the success of institutions in breeding cheetahs suggests that husbandry practice may be crucial, and difficulties in detecting estrus, and perhaps inappropriate social conditions may act as impediments to mating (*29*). Juvenile mortality is of lesser import in preventing the captive population from increasing (*19*). Moreover, in response to a partially open-ended questionnaire, zoos ascribed much of their juvenile mortality to poor husbandry (10 of 37 mentions), maternal neglect (10 cases), and cannibalism (5), all unconnected to homozygosity. Congenital defects (5), disease (4), and stillbirths (3) played a lesser role (*30*). Disease and juvenile mortality are secondary to other factors in preventing the captive population from expanding.

Genetic considerations are clearly important in the management of captive populations but may only be relevant to free-living populations in limited circumstances because they impact populations on a slower time scale than environmental or demographic problems (*8*, *31*). Indeed, there is widespread agreement that the environmental consequences of human disturbance present the greatest challenge to most populations in the wild (*32*), and these usually occur at a far swifter rate than inbreeding. Rapid declines in populations due to poaching [for example, rhinoceroses and elephants (*33*)], habitat fragmentation [primates, birds, and bees (*34*)], decimation by exotics [birds (*35*)], and pollution [crayfish (*36*)] attest to this. Among populations less subject to anthropogenic influence, such as those of the checkerspot butterfly, extinctions still result from environmental rather than genetic causes (*37*). Even in natural or reintroduced populations exhibiting reduced genetic variation, population growth and persistence may be little affected (*38*). Species that have undergone a demographic bottleneck such as the California sea otter or Great Indian rhinoceros (*39*) do not necessarily show reduced genetic variation, and in those that do, the number of deleterious recessives will depend on how fast the bottleneck occurred because they will have been purged not fixed if decline was slow.

In practical terms, the cheetah case history highlights the necessity of carrying out detailed ecological studies of endangered species in order to determine environmental causes of population decline (*40*). Studies collecting ecological data require a longer time to complete than those collecting genetic samples and are labor intensive but may be the key to understanding and hence preventing population extinctions.

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