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Abstract: Cheetahs are thought to exhibit low genetic variability and, as a consequence, may suffer high juvenile mortality, impaired reproduction, and susceptibility to disease. Recently, however, independent criticisms have been levelled at this body of work questioning both the analysis and the standards of evidence for lack of genetic diversity as well as the evidence that cheetahs are suffering as a consequence. O'Brien (1994a) combines a number of points in his first rejoinder to these criticisms, including novel interpretations of ecological data that we obtained and concern about their collection and analysis. Here, we first show that this interpretation is seriously flawed and that our conclusion that predation is the key source of mortality in the wild is valid. Then, extending previous criticisms and continuing to focus on this one consequence of lack of genetic variability, we discuss O'Brien et al.'s (1985) interspecific comparison of juvenile mortality in captivity by highlighting analytical problems and presenting new data on captivity.

Extrinsic Factors and Juvenile Mortality in Cheetahs

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Cheetahs are thought to exhibit low genetic variability and, as a consequence, may suffer high juvenile mortality, impaired reproduction, and susceptibility to disease (O'Brien et al. 1983, 1985, 1986, 1987; Wildt et al. 1983; Marker & O'Brien 1989; Menotti-Raymond & O'Brien 1993). The species is now regularly quoted as an example of how lack of genetic diversity affects the persistence of species. Recently, however, independent criticisms have been leveled at this body of work questioning both the analysis and the standards of evidence for lack of genetic diversity (Caughley 1994; Merola 1994) as well as the evidence that cheetahs are suffering as a consequence (Laurenson et al. 1992; Caro 1994; Caro & Laurenson 1994; Caughley 1994; Laurenson 1994; Merola 1994). It is becoming clear that a loosely strung chain of poorly established findings will no longer convince a skeptical conservation community of the importance of genetics in the fate of this species.

O'Brien (1994*a*) combines a number of points in his first rejoinder to these criticisms, including novel interpretations of ecological data that we obtained and concern about their collection and analysis. Here, we first show that this interpretation is seriously flawed and that our conclusion that predation is the key source of mortality in the wild is valid. Then, extending previous criticisms and continuing to focus on this one consequence of lack of genetic variability, we discuss O'Brien et al.'s (1985) interspecific comparison of juvenile mortality in captivity by highlighting analytical problems and presenting new data on captivity.

In the Serengeti National Park, Tanzania, cheetah cubs suffered 95% mortality before reaching independence, with predation by lions and spotted hyenas accounting for the majority (73.2%) of cub deaths whose cause of death was definitely or probably known (Laurenson 1994, Caro & Laurenson 1994). O'Brien (1994*a*) suggested that the proportion of cub deaths attributable to predation was inflated because predators kill whole litters if they discover a lair. However, examination of litters rather than cubs reveals the same pattern as analysis of individual cub deaths: 90% of litters failed to reach independence, and 66.7% of litters (n = 15) suffered known mortality due to predation (Laureson 1994). The proportion of mortality attributable to predation is unlikely to be inflated by analysis of cub deaths because other causes of mortality such as abandonment, fire, and exposure also wiped out whole litters in the lair and, in addition, nearly one third of cub deaths occurred after cubs left the lair, when partial litter mortality occurs (Laurenson 1994). Furthermore, stillborn litters, resorption of litters during gestation, and postnatal defects, which could result from low heterozygosity, were not obviously prevalent in the wild. Viable litters were quickly conceived after the loss of a litter (X = 19 days from litter loss to conception, n = 13 litters born to adult females [Laurenson et al. 1992], and just 1.6% of cubs (n = 125) were nonviable in healthy litters. Although some individual cubs may have died postnatally, before cubs were counted, the average litter size was similar to that in captivity where such deaths are observed (Caro 1994; Caro & Laurenson 1994). In summary, there is good reason to suspect that predation was the major cause of cub mortality in the wild and that the level of mortality potentially attributable to low genetic diversity was inconsequential.

O'Brien's second criticism of our data was that the observer (M. K. Laurenson) attracted predators to the lair. As discussed in our original paper (Laurenson & Caro 1994), we were sensitive to this possibility and took extensive precautions by minimizing vehicle tracks around the lair, by not entering lairs if a predator or cheetah mother was in sight, and by spending only a limited time watching lairs. Moreover, we found no difference in the number or severity of our vehicle tracks around lairs between litters that had and litters that had not been killed

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by predators. We also found no difference in the closest distance that lairs were approached when we compared litters that survived with those that succumbed to other causes. Lions and spotted hyenas in our Serengeti study site are habituated to vehicles and generally ignore them. Direct observations of predation suggested that it was usually the cheetah mother herself that gave away the position of the lair by sitting up or by being spotted entering or leaving the lair. We could find no effects of our activities on either the overall likelihood of cubs dying or the chance of their being killed by predators or being abandoned.

Our finding that predation is the key source of cub mortality in Serengeti is upheld and is not subject to the caveats advanced by O'Brien (1994*a*). In addition, cheetah biomass is inversely correlated with lion biomass across nine protected areas of sub-Saharan Africa after prey biomass is taken into account (Laurenson 1995*a*). This suggests that, after prey availability, lions are an important factor keeping cheetah numbers at low levels across the continent. The fact that cheetahs have large litters (Caro 1994) and rapid growth rates (Laurenson 1995*b*) compared to other felids corroborates the idea that predation is a key source of mortality because these are predicted life-history responses to high extrinsic juvenile mortality (Promislow & Harvey 1990).

More generally, low genetic variability can theoretically result in elevated juvenile mortality rates (Falconer 1981). In support O'Brien et al. (1985) found higher rates of juvenile mortality among captive cheetahs than captive ungulates and rodents. There are a number of problems with this comparison. In species depauperate in genetic variability for a number of generations (such as cheetahs; Menotti-Raymond & O'Brien 1993), deleterious recessive alleles may have been purged and juvenile mortality may be no greater than in outbred species (Templeton & Read, 1983). The comparison of O'Brien et al. (1985) is inappropriate because rates of mortality vary dramatically across orders in the wild and in captivity (Caughley 1994). Loudon (1985) found lower mortality rates in captive ungulates than in carnivores, which might explain O'Brien et al.'s (1985) finding. In the more appropriate comparison, Loudon (1985) found a lower rate of juvenile mortality in cheetahs than in other carnivores (24% for cheetahs versus an average of 33% for 17 carnivore species in total, or 33% for 9 felids) and ranked cheetahs second lowest among felids.

Mortality rates in captivity can be affected by differences in management technique. In cheetahs, juvenile mortality has been declining since the first captive birth in 1956 and more consistently since 1977. This decline is similar to that shown by other less homozygous felids. The figure of greater than 30% quoted by O'Brien (1994*b*) spans 30 years when mortality rates were still high. If mortality was primarily caused by intrinsic factors resulting from a supposed lack of genetic diversity, then there should be little improvement over time. More likely, management experience and increased communication between breeding facilities has lowered juvenile mortality. Furthermore, juvenile mortality and breeding success vary considerably between institutions; mortality to 6 months of age ranged from 14 to 47% at 11 North American breeding facilities which produced more than 5 litters over 21 years (Marker and O'Brien, 1989; Marker-Kraus and Grisham 1993). Again, such variation points to extrinsic sources of mortality. O'Brien et al. (1985) calculated an average mortality rate for cheetahs across facilities but used data from only single facilities, particularly the National Zoo in Washington, D.C., for several species (11 out of 16 ungulate species; Ralls et al. 1979). Thus O'Brien's unbalanced comparisons could have also disfavored cheetahs if the National Zoo had generally low rates of mortality. In contrast, Loudon's (1985) comparison collated records across facilities for each species, used larger samples, and employed a more appropriate comparison group.

When populations are compared in terms of juvenile mortality to examine the consequences of low genetic diversity, causes of mortality are more important than rates. We have detailed these in the wild and have found potential deaths due to genetic defects to be relatively unimportant. In captivity animals, the principal causes of mortality are also unrelated to low genetic variability. First, declining rates and variation between institutions point to extrinsic causes. Second, an analysis of data from three successful breeding institutions in North America (Fossil Rim Wildlife Center, Texas, $n = 82$ cubs born; White Oak Plantation, Florida, $n = 46$ cubs; Wildlife Safari, Oregon, $n = 30$ cubs) showed that 48.5% of 33 cub deaths could be attributed to abnormal maternal behavior. Felids in general require seclusion and quiet when they have cubs, and mothers are prone to neglect or eat cubs when disturbed or stressed (Husain 1966; Marma & Yunchis 1968). Of cheetah cub deaths, 24.2% were due to husbandry or hand rearing, 6.2% to disease, and 18.2% to stillbirths or abnormal development. Thus, 78.8%—the overwhelming majority—of cub deaths can be explained by extrinsic factors, whereas 3.8% of 158 cubs born may have been affected by congenital defects (Lindburg & Worley 1993). Interestingly, one of the six abnormal cubs in our sample had an inbreeding coefficient of 0.25, and four had coefficients of 0.19. All other cubs had coefficients of 0 or were offspring of parents known to be unrelated over at least two generations (Marker-Kraus 1989-1992). This suggests that congenital abnormalities are an infrequent source of juvenile mortality among unrelated parents (Hedrick 1987).

In short, lack of heterozygosity can theoretically affect juvenile mortality, but in cheetahs empirical evidence does not support the idea that juvenile mortality is caused primarily by genetic problems in the wild or in captivity, is overly high in captivity, or is threatening the

species' existence (Caro 1994). Because lack of genetic variation may exert no more than a minor influence on juvenile mortality in this species, it seems ill-advised to continue to cite the cheetah as a species suffering from this consequence of low genetic diversity.

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Cheetah Extinction: Genetics or Extrinsic Factors?

The debate addressed by Laurenson et al. (*Conservation Biology* 9:1329-1331) is interesting and important because the cheetah is indeed considered a "classic case" of low genetic variation and subsequent inbreeding depression. Unfortunately, the controversy appears to pivot around the perpetuation of a false dichotomy: that either genetics *or* extrinsic factors are likely to drive cheetahs to extinction.

It is simply not possible to separate genetic factors from environmental ones when addressing concerns over small population persistence. From the standpoint of population dynamics, inbreeding depression does not manifest solely (or even mostly) as congenital birth defects, monstrous abnormalities, or rampant reproductive failure. Rather, inbreeding depression operates through subtle modifications in birth and death rates that *interact* with other factors to increase extinction probability (e.g. Soulé 1987; Mills & Smouse 1994).

I do not question the field techniques or analysis of the Laurenson et al. research. I only suggest that both morphology and behavior—including parental care and defense against predators—span a range of phenotypic expression that surely has some genetic component. To the extent that behaviors and morphology have a genetic component, it is inevitable that genotypic changes leading to decreased fitness (inbreeding depression) could affect population persistence.

Again, I am not arguing that "genetic factors are the biggest threat to cheetahs." Laurenson et al. have aired vital issues to be weighed in evaluating conservation threats. However, their arguments do not reject the hypothesis that genetic factors join with environmental and predation factors to decrease vital rates in cheetahs. Though a simple dichotomy makes for an aesthetically pleasing debate, a view toward interactions between genetics and environmental, behavioral, and demographic factors would move us further toward helping small and isolated populations. To say this another way, I quote from the last sentence of the article commonly cited (or mis-cited) as advocating the position that genetic concerns are of minimal importance in small wild populations (Lande 1988:1459, emphasis mine): "The immediate practical need in biological conservation for *understanding the interaction of demographic and genetic factors in the extinction of small populations* therefore may provide a focus for fundamental advances at the interface of ecology and evolution."

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Clearcut Consolidation a Status Quo Solution

John Hagan's editorial (*Conservation Biology* 9:975-976) provided some insightful comments regarding conservation biology and value systems of scientists. Specifically, I appreciate his comments about different interest groups "learning how to listen to each other." I also agree with his point that a scientist may have values, but these should not affect the results or conclusions from scientific studies. However, I am upset by some of his other statements, especially regarding clearcuts.

I do agree that many bird species, and guilds of birds, respond positively to logging, including clearcuts. I suggest, however, that it is the challenge of conservation biology to think beyond the individual timber sale and to consider the landscape perspective. In this regard, we see that the proportion of logged land exceeds unmanaged lands in unprecedented levels. It may be prudent to ask, what are the limiting factors in a system? Even if biodiversity is increased by logging, what is the native diversity at the landscape level? How has this been altered? Unfortunately, misrepresentation is pervasive and clouds attempts to find common ground and, more importantly, an understanding of what conservation biology is about.

I commend Hagan in his attempt to minimize fragmentation by consolidating clearcuts. I believe, however, that it is inappropriate for conservation biologists to accept the status quo (such as unsustainably higher timber volumes) that has been shown to degrade ecological systems. Instead of arguing for consolidation of logging, conservation biologists should argue for a lower, more sustainable level of cut. This is not an "environmentalist's" value judgment, but rather an interpretation from hundreds of scientific studies.

Our field is just beginning to blossom into intelligent management practices. I continually look for some vision in the agencies responsible for managing our lands. This vision is not there. As conservation biologists we need to create that vision and provide a model for future management.

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