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# Loss of Genetic Diversity in Wild Populations

Shawn Larson  
Seattle Aquarium  
United States

## 1. Introduction

The importance of genetic diversity within populations has been debated since the study of genetics began. There are two major camps: the classical school (genetic variability is low within species) and the balanced school (genetic variability is high within species; Avise, 1994). This debate was brought to a head when O'Brien et al., 1985 suggested that population viability problems within the cheetah (*Acinonyx jubatus*) could be linked to low genetic diversity. Since then there has been a significant increase in conservation genetics studies within other species focusing on genetic variability and its importance in conservation, particularly within those populations that have passed through significant population bottlenecks (Coulson et al., 1999; Hoelzel, 1997; O'Brien et al., 1985; O'Brien et al., 1996; Wildt et al., 1987). In addition to the classical and balanced genetic variability school debate, there has been criticism of the use of neutral genetic markers in conjunction with fitness related indices to suggest inbreeding depression (Hedrick & Kalinowski, 2000; Lynch, 1996). The reasoning is that since neutral markers are by definition not affected by natural selection then they cannot be causally linked to inbreeding indices (Avise, 1994). However, since they are not affected by natural selection they may actually indicate a more realistic picture of overall potential genetic diversity not yet affected by selective pressure following population bottlenecks than those regions affected by selection (Amos & Balmford, 2001; Avise, 1994). The premise here is that the total genetic variation lost following such events may be estimated and that any loss of genetic diversity may negatively affect long term population adaptability and viability because future conditions and stochastic events cannot be predicted (Avise, 1994). Due to the rapid increase in the study of genetic variation within wildlife populations there is a growing body of evidence linking fitness variables with both qualitative and quantitative genetic methods, suggesting meaningful trends that may affect population survival and viability (Amos & Balmford, 2001).

Populations that experience bottlenecks are thought to lose genetic diversity through genetic drift and inbreeding (Charlesworth & Charlesworth, 1999; Crnokrak & Roff, 1999; Hedrick & Kalinowski, 2000; Lacy, 1997; Lynch, 1996; Ralls et al., 1988). Small population size may lead to inbreeding where related individuals produce offspring (Eldridge et al., 1999; Lynch, 1996; Slate et al., 2000). Inbreeding may lead to the buildup of deleterious recessive genes, termed inbreeding depression, that may cause decreased fecundity, increased mortality, slowed growth, developmental defects, increased susceptibility to disease, decreased ability to withstand stress, and decreased ability to compete (Lacy, 1997). The cost of inbreeding to fecundity in small populations has been documented by several studies within the Felidae

(O'Brien et al., 1985; O'Brien et al., 1987; O'Brien et al., 1996; and Wildt et al., 1987). These studies revealed significantly decreased genetic variation in isolated wild felid populations and inbreeding depression measured by increased sperm abnormalities, decreased reproductive hormone levels and difficulty breeding in captivity (O'Brien et al., 1985; O'Brien et al., 1996; and Wildt et al., 1987). In addition, other work has linked inbreeding within several wild species with increased mortality, decreased fecundity, and slowed growth (Crnokrak & Roff 1999; Ralls et al., 1988; Slate et al., 2000).

Inbreeding and the potential effects on population viability are important because populations that experience a bottleneck are thought to suffer from decreased genetic variation for hundreds of thousands of years (in the absence of immigration) due to the low locus mutation rate that prevails at most genetic locations or loci ( $10^{-8}$  to  $10^{-5}$  mutations per year; Driscoll et al., 2002; Lande & Barrowclough, 1987; Lynch, 1996). Recovery of lost genetic variation in the absence of immigration is the inverse of the mutation rate (Driscoll et al., 2002; Nei et al., 1975). Small populations that retain relatively low genetic variability over several generations run the risk of fixation of alleles. Most individuals are assumed to contain unique deleterious genes at several loci, all subject to chance fixation in a small founder population (Lynch, 1996). The probability of fixation of deleterious alleles in founder populations of 30 or fewer individuals is close to the alleles' initial frequencies in the population because genetic drift overwhelms natural selection, and may lead to relatively rapid extinction of the population even in the absence of any stochastic events (Lynch, 1996; Shaffer, 1981).

Genetic studies of small populations that regularly contain fewer than 100 breeding individuals, or effective population size ( $N_E$ ), suggest that these populations are extremely vulnerable to the loss of genetic variation (Lacy, 1997; Lynch, 1996). Low diversity combined with inbreeding depression increase a small population's vulnerability to extinction from stochastic events (Lacy, 1997; Lynch, 1996). Population sizes of at least 1000 are suggested to protect against the fixation of deleterious genes and a breeding population of 10,000 adults is suggested to protect adaptive genetic variation (Lynch, 1996). Unfortunately, these population sizes are usually not found in endangered species or those that have been fragmented by human activities. The fixation of deleterious alleles, genetic drift, the loss of adaptive genetic variation and inbreeding depression all dramatically increase a small population's vulnerability to extinction from stochastic events (Charlesworth & Charlesworth, 1999; Crnokrak & Roff 1999; Fowler & Whitlock 1999; Hedrick & Kalinowski 2000; Lacy 1997; Lynch 1996; Ralls et al., 1988).

Inbreeding depression is defined in general terms as a reduction in fitness of inbred offspring, but its specific effects are highly variable and depend on how the genotypes interact with the environment (Fowler & Whitlock, 1999; Hedrick & Kalinowski, 2000). Current theory is that inbreeding depression is more severe in stressful environments because of reduced genetic plasticity to adapt to changes or stressors (Dahlgard & Hoffman, 2000; Hedrick & Kalinowski, 2000; Miller, 1994). Inbreeding depression causes an overall decrease in population growth (decreasing  $N_E$ ) due to either decreased birth rate or increased mortality or a combination of the two.

Documenting the effects of inbreeding can be difficult in wild and captive populations of vertebrates due to long generation intervals and/or the inability to control for varying environmental conditions. Consequently, many studies have employed laboratory species

as models to study inbreeding depression under controlled conditions. Studies done using the fruit fly, *Drosophila melanogaster*, have shown significant relationships between inbreeding, changing environmental conditions, and population viability (Bijlsma et al., 1999, 2000; Dahlgard & Hoffman, 2000; Miller, 1994). Specifically, inbred lines of *Drosophila* suffered 20–50% higher extinction rates compared to outbred lines when challenged with stressful environmental conditions such as high temperatures or exposure to ethanol (Bijlsma et al., 1999, 2000). These studies suggest inbred populations may not respond as well as outbred lines to stress.

When vertebrate individuals are “stressed” (e.g., exposed to adverse environmental conditions including short-term events such as human disturbance, severe weather, food restrictions, or exposure to contaminants or parasites), the hypothalamic–pituitary–adrenal (HPA) axis between the brain and the adrenal gland mediates the stress response (Norris, 1996). Physical, psychological, chemical, and other stressors trigger the release of corticotrophin-releasing hormone (CRH) in the hypothalamus leading to the release of adrenocorticotrophic hormone (ACTH) in the pituitary, which in turn mediates the release of the adrenal glucocorticoids (GCs) from the adrenal glands (Norris, 1996). The major GCs include cortisol and corticosterone, and it is the circulating levels of these hormones that produce the physiological and behavioral response to stress (i.e., fight or flight response). Most animals will secrete primarily one of the GCs (either cortisol or corticosterone) in response to stress. Birds, amphibians, and reptiles generally secrete corticosterone as their major glucocorticoid. Mammals secrete both, with cortisol tending to dominate, while fish generally secrete cortisol (Idler & Truscott, 1980; Norris, 1996).

The cost of elevated GCs is well documented. Observations include a decrease in overall health with reduced individual growth, changes in metabolism, delayed repair of tissues or healing, and immunosuppression resulting in an increased incidence of disease in stressed animals (Astheimer et al., 2000; Gorbman et al., 1983; Johnson et al., 1992; Norris, 1996; Rivier & Rivest, 1991). Chronically high basal GC levels also may negatively affect reproductive potential by depressing reproductive hormone levels (Brann & Mahesh, 1991; Moberg, 1991; Norman, 1993; Kosowska & Zdrojewicz 1996).

Genetic load is the longer term effect of loss of fitness over time due to an increase in detrimental mutations becoming fixed creating an overall loss of genetic variation for future adaptation (Kirkpatrick & Jarne 2000). Genetic load may be documented as low estimates of fitness related variables when compared to other populations that have not experience bottlenecks or have no evidence of loss of fitness associated with loss of genetic diversity.

Genetic rescue is a management technique that has been used to manage critically endangered populations with high genetic load. This strategy uses unrelated individuals from one population that are introduced into the population with apparent low fitness. This introduction of new genetic material acts to reduce genetic load by lowering or eliminating the frequency of detrimental gene variants in the population. This has been used successfully in captive settings and within wild populations (Hedrick & Fredrickson, 2010). There are clearly recognized guidelines for the successful use of genetic rescue as a conservation management strategy (Hedrick & Fredrickson, 2010). They are as follows: Evidence of low fitness; the existence of a closely related donor population from the same species and from similar habitat; experimental data in a captive situation such as successful mating, good

survival of progeny and molecular data; a translocation protocol; a detailed monitoring plan and a commitment for long term management (Hedrick & Fredrickson, 2010).

## 2. Case studies of loss of genetic diversity and subsequent loss of fitness

### 2.1 Sea otters

Sea otters, *Enhydra lutris*, were once abundant across their range in the north Pacific Rim from northern Japan to the central Pacific Coast of Baja California, Mexico (Lensink, 1962; Kenyon, 1969; Riedman and Estes 1990). During the 18th and 19th centuries, the Pacific maritime fur trade eliminated or greatly reduced sea otter populations throughout this area, eventually resulting in 11 scattered populations after protection in 1911, with a combined population totaling approximately 1% of the original abundance estimated to be at one time approximately 100,000 animals (Lensink, 1962; Kenyon, 1969).

By the 1970's, few sea otter populations had recovered to pre-exploitation levels, with the majority of historic sea otter habitat vacant along the west coast of North America from Prince William Sound, Alaska, southward to California (Estes, 1990; Kenyon, 1969; Riedman & Estes, 1990). In an effort to re-establish sea otter populations throughout their former range, management authorities made several translocations from the 1950's through the 1970's (Jameson et al., 1982). In total, 715 otters were captured at Amchitka Island in the Aleutian chain and Prince William Sound, Alaska, and then released at various unoccupied habitats in Alaska, British Columbia, Washington and Oregon (Jameson et al., 1982). The translocations to Washington, Oregon and the Pribilof Islands included only animals captured at Amchitka, and only the Washington effort was successful (Bodkin et al., 1999; Jameson et al., 1982). The translocations to Southeast Alaska and British Columbia (off the west coast of Vancouver Island) included a mix of Amchitka and Prince William Sound animals, and both were successful (Bodkin et al., 1999; Jameson et al., 1982). In spite of these successful translocation efforts, sea otter populations today remain fragmented with extant populations geographically separated and, in most cases, reproductively isolated (Bodkin et al., 1999).

The remaining sea otter populations constitute three subspecies: Russian (*E.l. lutris*), Northern (*E.l. kenyoni*), and Southern (*E.l. nereis*) based on skull morphometrics (Wilson et al., 1990) spread among remnant and translocated populations throughout the former range. Investigation into the remaining neutral genetic variation post exploitation found only half the original genetic variation left within sea otter populations (Larson et al., 2002b). Genetic diversity estimates for all extant populations were similar, with expected heterozygosities ranging from 40% within California to 47% within Southeast Alaska and the allelic richness (mean number of alleles corrected for sample size) ranging from 3.20 in Prince William Sound to 3.70 in Amichitka and Washington (Larson et al., 2002a).

This low diversity evident throughout sea otter populations make distinguishing significant genetic differences between subspecies difficult because there are so few alleles available for differentiation. Studies of mitochondrial DNA (mtDNA) have found little genetic support for distinguishing the Northern and Southern subspecies (Cheney, 1995; Cronin et al., 1996; Larson et al., 2002a; Sanchez, 1992; Scribner et al., 1997). However, population level comparisons using hypervariable nuclear (microsatellite) genetic markers have indicated some phylogeographic structure among contemporary sea otters. Cronin et al., 2002 found

stock differences in Northern sea otters using microsatellite markers: a Southwest stock including the Aleutian Islands and Kodiak Island; a Southcentral stock including Prince William Sound, the Kenai Peninsula and Cordova; and a Southeastern stock including the Alexander Archipelago. In addition, Larson et al. (2002a) found significant genetic differentiation using seven variable microsatellite nuclear markers among three remnant sea otter populations: Amchitka in the western Aleutian Islands of Alaska (a United States Endangered Species Act (ESA) listed stock), Prince William Sound in southcentral Alaska, and Southern sea otters from the single California population (also an ESA listed group).

Do sea otters suffer inbreeding depression because of their loss of genetic diversity and population fragmentation and resuction? Stress as measured by circulating adrenal glucocorticoids (GC's) was found to be significantly negatively correlated with heterozygosity in sea otters (Larson et al., 2009). This significant relationship was found at both the individual and population level for corticosterone, but only at the individual level for Cortisol (Larson et al., 2009).

These results are strong evidence that genetic diversity may be a significant determinant of how individuals and thus populations are able to respond to and handle stress—from exposure to disease agents or contaminants to dealing with various levels of food availability or even short-term severe storm events. To fully evaluate the biological meaning of relative levels of circulating GCs requires an understanding, specific for each species, of the role and the affinity of the binding proteins, the cell surface and nuclear receptors to GCs, and ultimately the role of GCs through differential gene transcription. The relative importance of cortisol vs. corticosterone on gene transcription varies widely within mammals (Gayrard et al. 1996, Tanigawa et al. 2002), and the effect of either GC on sea otter physiology has not been investigated.

If corticosterone levels do relate to low genetic diversity and diminished ability to cope with environmental stress, then these relationships could also suggest a fitness repercussion from a population bottleneck and may serve as an indicator of inbreeding depression. One potential consequence of higher circulating GCs is reduced immune response (Norris 1996). California sea otters historically have had the lowest population growth rate of any sea otter population as well as the highest reported incidence of disease within adult animals, those that make up the breeding population (Gerber et al., 2004; Hanni et al., 2003; Kreuder et al., 2003; Miller et al., 2004; Thomas & Cole, 1996). In addition, a high incidence of infectious diseases among this population suggests a weakened immune system or immunosuppression (Gerber et al., 2004; Hanni et al., 2003; Kreuder et al., 2003; Miller et al., 2004). It seems at least conceivable that the low genetic diversity and corresponding high corticosterone levels in this population have played a role in susceptibility to infectious disease via immunosuppression. The higher incidence of disease eventually leads to elevated mortality of prime-aged animals and low overall population growth rates.

Many extant sea otter populations appear to be in a precarious balance. Because they inhabit coastal areas, they are often in contact with humans and can suffer negative interactions associated with fishing activities and exposure to shoreline and near-shore pollution sources. Add to these difficulties the potential for negative effects associated with the loss of genetic diversity and buildup of deleterious alleles called genetic loading, and we may improve our understanding of why some sea otter populations fail to thrive, like the threatened California population.

## 2.2 Florida panther

The American mountain lion, cougar or puma (*Puma concolor*) once was widely distributed throughout North, Middle and South America. There are currently seven recognized subspecies based on genetic data: *P. c. cougar*: North America, *P. c. corryi*: Florida, *P. c. costaricensis*: Central America, *P. c. capricornensis*: eastern South America, *P. c. concolor*: northern South America, *P. c. cabreræ*: central South America, and *P. c. puma*: southern South America (Culver et al., 2000). The cougar subspecies in southern Florida swamplands, *P. c. corryi*, is the only subspecies exclusively referred to as “panther” and is the only subspecies listed as critically endangered by the IUCN (2008 IUCN red list) and endangered by the ESA (Endangered Species List 1967). The Florida panther has been restricted to a small area that includes the Big Cypress National Preserve, Everglades National Park, and the Florida Panther National Wildlife Refuge, which is thought to represent only 5% of its historic range. The number of living Florida panthers was estimated to be approximately 100 in 2010 (Johnson et al. 2010). Overhunting was responsible for the initial population bottleneck in Florida panthers which created a small, isolated population that became inbred.

The population was found to have several problems associated with reproduction (cryporchidism in males, high neonate mortality), as well as shared genetic abnormalities such as heart defects and kinked tails (Roelke et al., 1983). This prompted a program to protect and enhance the genetic diversity of the remaining panthers through a program of genetic rescue. In 1995, conservation managers translocated eight unrelated female pumas from Texas to increase depleted genetic diversity, improve population numbers, and reverse indications of inbreeding depression (Hedrick, 1995). The effect of the genetic rescue was dramatic. Since the introduction of those few unrelated females the panther numbers have increased threefold with the panther population growth rate increasing from 0 to 12% annually, genetic heterozygosity has doubled, individual survival and fitness measures have improved, and inbreeding correlates declined significantly. (Hedrick & Fredrickson, 2010; Johnson et al. 2010). The remarkable success of the genetic rescue experiment within Florida panthers makes it a model for other genetic rescue programs. However even with the recovery of the Florida panther after the influx of new genes the population continues to struggle with issues related to habitat loss, and disease (Johnson et al. 2010).

## 2.3 Mexican wolf

The Mexican wolf (*Canis lupis baileyi*) is a an endangered subspecies of the grey wolf (*Canis lupus*) (Leonard et al, 2005). The population was driven to critically low numbers because of hunting pressure resulting in their extinction in the United States and only a few small isolated populations in Mexico by 1980. These small, isolated populations were thought to have been negatively affected by inbreeding resulting in zero population growth due in large part to reduced reproductive rate and relatively high levels of mortality. Based on the population fragmentation and low growth rate this wolf was listed as endangered by the ESA in 1976 (Hedrick & Fredrickson, 2010). The surviving individuals were eventually caught and placed in captivity. It is thought that all Mexican wolves surviving today are from descended three captive lineages produced by from a total of seven founders caught between 1960 and 1980 (Hedrick et al., 1997). The captive wolves were kept and bred both within founder lineages and between. These captive groups were monitored for several

fitness related traits to determine effects of inbreeding and out-breeding. Initial fitness improvements were significantly higher numbers of pups in each litter within the groups with crossed lineages (Hedrick & Fredrickson, 2010). In 1998 a pure lineage group was reintroduced into the wild. Then in 2009 a group from another lineage was introduced resulting in the first genetically mixed progeny in the wild (Hedrick & Fredrickson, 2010). Almost immediately the population rate started growing with the average litter size doubling and a significant decline in the measured inbreeding coefficients (Hedrick & Fredrickson, 2010). Based on these reproductive indices (litter size and inbreeding coefficients in pups) there appeared to be successful genetic rescue in wild Mexican wolves but after only two distinct lineages were allowed to interbreed (Hedrick & Fredrickson, 2010).

### **3. Case studies of loss of genetic diversity and little subsequent loss of fitness**

#### **3.1 Northern elephant seals**

There are examples in nature where populations that have experienced bottlenecks and consequently have very little measurable genetic variation that seem to suffer no ill effects from inbreeding depression and grow rapidly. One case in point is the northern elephant seal (*Mirounga angustirostris*) which was exploited for its blubber in the 19th century resulting in only 10-30 remaining individuals (Hoelzel, 1997). The population recovered quickly, numbering approximately 127,000 individuals in 1991 (Hoelzel, 1997). Genetic analysis using a variety of markers revealed little genetic variation (Bonnell & Selander, 1974; Hoelzel, 1997). Furthermore, a study of pre-exploitation northern elephant seal genetic diversity compared to contemporary seals, revealed a significant loss of genetic diversity (55% loss, Weber et al., 2000). Even though the northern elephant seal recovery has been remarkable, recent data suggests that they may be experiencing some ill effects such as decreased reproductive fitness. Paternity success of male northern elephant seals was lower than expected when compared to observed copulations and compared to the average paternity success of the southern elephant seals, *M. leonine*, which did not experience the severe exploitation similar to the Northern elephant seal (Hoelzel, 1997).

#### **3.2 Canadian moose**

Another case study of loss of genetic variation within bottlenecked populations and dramatic recovery is that found within the moose, *Alces alces*. In the late 19th and early 20th century, six moose were translocated from mainland Canada to two areas of Newfoundland (Broders et al., 1999). The population in 1999 was estimated to be approximately 150,000 with more than 400,000 harvested since their introduction (Broders et al., 1999). Average genetic diversity as measured by microsatellite heterozygosity over all populations was 33%, with the loss of heterozygosity from the three founder events (two translocations and one natural colonization) ranging from 14%-30% (Broders et al., 1999). The cumulative loss of heterozygosity from a translocated population which then seeded another by natural colonization was 46% (Broders et al., 1999). All the moose populations suffered a significant loss of diversity but have not exhibited inbreeding depression as indicated by population growth rates, although the long term viability of the population and individuals remains unknown (Broders et al. 1999).

#### 4. Conclusion

Genetic studies of small populations that regularly contain fewer than 100 breeding individuals suggest that these populations are extremely vulnerable to the loss of genetic variation (Lacy, 1997; Lynch, 1996). Low diversity combined with inbreeding depression increase a small population's vulnerability to extinction from stochastic events (Lacy, 1997; Lynch, 1996). Population sizes of at least 1000 are suggested to protect against the fixation of deleterious genes and a breeding population of 10,000 adults is suggested to protect adaptive genetic variation (Lynch, 1996). Unfortunately, these population sizes are usually not found in endangered species or those that have been fragmented by human activities such as sea otters, Florida panthers and Mexican wolves. The fixation of deleterious alleles, genetic drift, the loss of adaptive genetic variation and inbreeding depression all dramatically increase a small population's vulnerability to extinction from stochastic events (Charlesworth & Charlesworth 1999; Crnokrak & Roff, 1999; Fowler & Whitlock, 1999; Hedrick & Kalinowski, 2000; Lacy, 1997; Lynch, 1996; Ralls et al., 1988). In an increasingly fragmented world where wildlife individuals have little opportunity to regularly migrate freely between isolated populations to maintain geneflow and thus overall effective population size, it becomes imperative for management policies to encourage genetic diversity. If the ultimate goal is to maintain maximal diversity within wildlife populations to ensure maximal potential to respond to environmental changes then management decisions may need to be based on maintaining genetic diversity rather than maintaining unique populations such as subspecies. Genetic rescue may require the mixing of subspecies and thus the loss of unique subspecific characteristics. Managers must decide what is most important to preserve, the species with maximal genetic diversity for future stochastic changes or a genetically unique race that may be negatively affected by isolation and inbreeding and incapable of responding to future challenges. That ultimately may be the cost of maintaining genetically healthy populations, the loss of genetically unique groups and adaptive variation.

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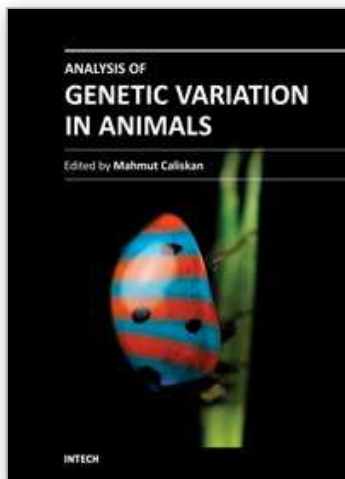
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## **Analysis of Genetic Variation in Animals**

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Analysis of Genetic Variation in Animals includes chapters revealing the magnitude of genetic variation existing in animal populations. The genetic diversity between and within populations displayed by molecular markers receive extensive interest due to the usefulness of this information in breeding and conservation programs. In this concept molecular markers give valuable information. The increasing availability of PCR-based molecular markers allows the detailed analyses and evaluation of genetic diversity in animals and also, the detection of genes influencing economically important traits. The purpose of the book is to provide a glimpse into the dynamic process of genetic variation in animals by presenting the thoughts of scientists who are engaged in the generation of new idea and techniques employed for the assessment of genetic diversity, often from very different perspectives. The book should prove useful to students, researchers, and experts in the area of conservation biology, genetic diversity, and molecular biology.

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Unit 405, Office Block, Hotel Equatorial Shanghai  
No.65, Yan An Road (West), Shanghai, 200040, China  
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Phone: +86-21-62489820  
Fax: +86-21-62489821

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