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REVIEW

# Albatrosses, eagles and newts, Oh My!: exceptions to the prevailing paradigm concerning genetic diversity and population viability?

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## Abstract

Numerous recent papers have demonstrated a central role for genetic factors in the extinction process or have documented the importance of gene flow in reversing population declines. This prompted one recent publication to declare that a revolution in conservation genetics has occurred. Contemporaneously with this revolution are a series of papers demonstrating long-term population persistence for several species despite having little or no detectable genetic variation. In a couple of notable cases, populations have been shown to have survived for centuries at small population size and with depleted levels of genetic variation. These contradictory results demand an explanation. In this review, I will show that these results do not necessarily fly in the face of theory as sometimes stated. The reconciliation of these two sets of observations relies on the incorporation of two major concepts. (1) Genetic factors do not act in a vacuum and it is their interaction with the environment, the strength and type of selection imposed, and the life history of the organism that determine the relative importance of genetic factors to extinction risk. (2) The relationship between molecular estimates of genetic variation and evolutionary potential, the relevance of genetic bottlenecks to adaptive genetic variation, and the nature of the stochastic process of extinction must be better integrated into expectations of population viability. Reports of populations persisting for hundreds of generations with very little detectable genetic variation provide us not only with valuable information but also with hope. However, recent studies suggest that we should not be sanguine about the importance of genetic diversity in the conservation of biodiversity.

## Introduction

Many would claim that conservation genetics has undergone a renaissance over the past decade and that the importance of genetic factors in conservation biology is unassailable (Frankham, 2005; Sarre & Georges, 2009). To some extent this is certainly true. Genetic diversity is the most fundamental unit of biodiversity upon which all other levels of biodiversity depend. Greater amounts of within-population genetic diversity have been shown to favorably impact the rate of ecosystem recovery after perturbation, be positively correlated with species diversity, increase the level of ecosystem services provided, and to increase primary productivity (e.g. Reusch *et al.*, 2005; Vellend & Geber, 2005; Cleary *et al.*, 2006; Johnson, Lajeunesse & Agrawal, 2006; Worm *et al.*, 2006; Hughes *et al.*, 2008). Thus, few would argue that genetic diversity is unimportant to conservation biology in a broad sense.

The role of within population genetic diversity to population viability is more controversial. Numerous recent papers have made a very strong case for the importance of genetic factors to population viability (e.g. Vilá *et al.*, 2003; Hogg *et al.*, 2006; O'Grady *et al.*, 2006; Pimm, Dollar & Bass Jr, 2006; Reed, Nicholas & Stratton, 2007*a*,*b*; Trinkel *et al.*, 2008; Hedrick & Fredrickson, 2010). Yet, concurrent with the influx of evidence for a central role for genetics in population viability are a multitude of papers over the past decade demonstrating the long-term persistence of populations with very low levels of genetic diversity (e.g. Groombridge *et al.*, 2000; Visscher *et al.*, 2001; Hadly *et al.*, 2003; Milot *et al.*, 2007; Babik *et al.*, 2009; Habel *et al.*, 2009;

 

Johnson *et al*., 2009). Most authors were cautious to point out that population persistence to this point does not guarantee that inbreeding will not impact future survival negatively, but all urge to some degree or other a deemphasizing of genetic concerns.

In this review, I will endeavor to unify these seemingly discordant observations concerning the importance of genetic variation to population viability. In performing the synthesis, commonalities will be uncovered that can be linked to theory and eventually to management recommendations. I begin the process with a brief review of the more widely cited reasons for believing genetic diversity is important to population viability, followed by a review of the ecological-evolutionary perspective on population viability, a review of the nine papers potentially challenging current population viability theory, and then closing with management recommendations and some brief suggestions for future research.

## Evidence for the importance of genetic diversity

### Genetic diversity and evolutionary potential

The least contentious of the potential benefits of genetic diversity is the expected correlation between genetic diversity and the adaptive potential of populations. The general acceptance of this hypothesis is two-fold. First, strong theoretical grounds exist for believing that populations with larger effective population sizes will contain more neutral genetic variation as they are less susceptible to genetic drift. Therefore, larger populations should also contain more potentially adaptive genetic variation. Second, experimentally derived populations with reduced genetic variation have consistently been shown to have lowered narrow-sense heritabilities for quantitative traits (e.g. Eisen, 1975; Weber & Diggins, 1990; England *et al*., 2003; Kristensen *et al*., 2005; Swindell & Bouzat, 2005), though some exceptions for bottlenecked populations do exist. A population with less ecologically relevant genetic diversity will be less able to successfully track changing environmental conditions and therefore is more extinction prone. Given the rate at which habitats are being changed, mostly due to anthropogenic causes, strategies for retaining genetic variation are usually considered important.

### Genetic diversity and population fitness

A relatively strong positive correlation exists between genetic diversity and population level fitness (e.g. Reed & Frankham, 2003; Reed, 2005, 2008; Johansson, Primmer & Merilä, 2007; Mattila & Seeley, 2007; Vandewoestijne, Schtickzelle & Baguette, 2008). The existence of this correlation may be due to the ameliorating effects of heterozygosity on (partially) recessive deleterious alleles, heterozygote advantage at some loci, or ineffective natural selection and high levels of random genetic drift in smaller populations during the past (Reed & Frankham, 2003). Higher fitness leads to longer persistence times for at least three reasons. (1) Populations with greater fecundity recover more quickly from environmental perturbations and therefore have less temporal variability in population size (Reed *et al*., 2003). (2) Populations with higher fitness remain closer to carrying capacity or may have a higher carrying capacity through more efficient use of resources or the ability to use a wider variety of resources (Kinnison & Hairston Jr, 2007; Reed *et al*., 2007*b*). Larger population size creates a positive feedback loop among retention of genetic variation, evolutionary potential and current fitness (Reed, 2008). (3) Populations with greater fecundity have more evolutionary potential as the response to selection depends not only on the heritability of the trait but also on the genetically determined phenotypic diversity among offspring and the number of selective deaths the population can suffer and still maintain non-negative population growth rates (Boulding & Hay, 2001; Reed, 2005).

### Genetic diversity and stress resistance

An interaction exists between inbreeding and stress (Keller *et al*., 2002; Armbruster & Reed, 2005; Pedersen, Kristensen & Loeschcke, 2005), which causes populations depauperate in genetic diversity to be especially sensitive to environmental challenges (Keller *et al*., 1994; Bijlsma, Bundgaard & Van Putten, 1999; Endels *et al*., 2007; Reed *et al*., 2007*b*). This interaction can greatly reduce population persistence times by exaggerating population declines when populations are most vulnerable to entering the extinction vortex (Fagan & Holmes, 2006; Liao & Reed, 2009). Alternatively, rather than stress, the apparent increase in inbreeding depression could be linked to the amount of phenotypic variation and the opportunity for selection in a population (Waller, Dole & Bersch, 2008), though the weight of the evidence strongly favors an interaction. Regardless of the mechanism, decreases in fitness are consistently more severe with increasing stress in more inbred populations and this negatively impacts population persistence times.

### Genetic diversity and disease resistance

Pathogens represent a powerful evolutionary force responsible for local and species level extinctions (e.g. Krkosek *et al*., 2007; Skerratt *et al*., 2007; Mitchell *et al*., 2008; Smith, Acevedo-Whitehouse & Pedersen, 2009; Sokolow *et al*., 2009). Anthropogenic introductions of pathogens and the spread of pathogens into naïve populations due to global climate change (Dobson, 2009) are expected to increase the importance of disease resistance to future population persistence. A large number of papers have shown a positive correlation between genome-wide genetic diversity (both alellic richness and heterozygosity) and disease resistance; and less consistently a correlation between heterozygosity at loci in the major histocompatibility complex (MHC) and disease resistance (e.g. Hughes & Boomsma, 2004; Spielman *et al*., 2004; Valsecchi *et al*., 2004; Pearman & Garner, 2005; Hale & Briskie, 2007; Siddle *et al*., 2007).

## Population viability: ecological-evolutionary perspective

Before discussing the nine papers that represent possible exceptions or even contradictions to current thinking on population viability, it would be beneficial to review what that thinking consists of. The ecological-evolutionary perspective on population viability (e.g. Kinnison & Hairston Jr, 2007; Reed, 2008) suggests that persistence time will be determined primarily by the mean population growth rate ($r_e$) across the relevant environmental conditions, the degree of temporal fluctuations in $r_e$, and the carrying capacity of the environment ($K$) (e.g. Shaffer, 1981; Lande, 1994; Reed *et al*., 2003; Sæther *et al*., 2005; Kinnison & Hairston Jr, 2007). The mean population growth rate and its variance together can be quantified and summarized as the stochastic or geometric population growth rate ($r_s$) (Lewontin & Cohen, 1969) and is an excellent predictor of extinction risk (e.g. Reed *et al*., 2003; Sæther *et al*., 2005; Engen *et al*., 2009). Numerous factors impact persistence times indirectly by directly affecting the stochastic growth rate or carrying capacity (see Reed, 2008 for in depth discussion). Important factors include the strength and form density-dependent population regulation, the quality and amount of habitat available, the degree of stochasticity in important environmental variables (e.g. frequency and severity of drought, variation in density and lethality of diseases and predators), the amount of demographic stochasticity (including variation in the vital rates of among individuals in a population), body size and generation length of the organism, levels of anthropogenic disturbance or harvest and mating system (Reed *et al*., 2003; Cardillo *et al*., 2005; Griffen & Drake, 2008).

Any factor (e.g. environmental stress or genetic deterioration) that causes $r_s$ to become smaller on average will reduce persistence times, though the effect of decreasing $r_s$ is only important over a fairly narrow range of stochastic population growth rates near zero (Reed, 2008). Any factor (e.g. habitat conversion or degradation) that lowers the availability of density-limiting resources will reduce $K$ and reduce persistence times (Reed *et al*., 2003; Griffen & Drake, 2008). It is critical that $K$, $r_e$ and the temporal variation in $r_e$ not be viewed as three independent factors, but as interacting factors that influence each other (Reed, 2008). For example, the evolutionary potential of a population will be affected by $K$, $r_e$ and temporal variation in $r_e$ though their impacts on the amount and type of genetic variation maintained; genetic variation will in turn impact future $r_s$ and $K$. It is important that life history, especially generation length, be considered when scaling extinction risk (O'Grady *et al*., 2008). Body size likely represents a trade-off between maximizing potential population growth and minimizing variance in population growth rates and large-bodied, long-lived species may persist with low genetic variation for extended periods even if they have already entered the extinction vortex because of the number of years necessary for demographic changes to become pernicious (O'Grady *et al*., 2008).

## Similarities among studies and potential reasons for population persistence

Table 1 presents a summary of nine papers that provide extreme examples of population persistence with low genetic diversity. Many of the examples of long-term persistence with low genetic diversity provided have several points in common with each other. These commonalities provide lessons for conservation biologists, which I discuss below.

### Environmental quality and density-dependent growth

Ellegren *et al*. (1993) describe Scandinavian beavers as reintroduced into an intact habitat, without competitors and protected from hunting. Broders *et al*. (1999) point out that the bottlenecked Canadian moose were introduced into 'ideal' sites with abundant food, no predators and no competitors. The Mauritius kestrel was driven to the brink of extinction by the use of DDT and the presence of invasive predators. This species was recovered by banning the use of DDT, rearing hatchlings in incubators, supplemental feeding of wild populations in order to double the number of clutches, predator control measures and the construction of artificial nests in order to increase carrying capacity. The Chillingham cattle live in a heavily managed environment without predators.

These examples demonstrate the power of large population growth rates to rapidly extricate populations from the extinction vortex and, with sufficient carrying capacity, to become robust populations with little short-term risk of extinction. The beavers, moose and kestrels were all reduced to very low population sizes relative to carrying capacity, providing the opportunity for very rapid density-dependent population growth. They also had moderate to very large environmental carrying capacities to sustain that growth and buffer them against future demographic and environmental stochasticity. In the case of the Mauritius kestrel and the Chillingham cattle, potential population growth rates are increased (and variance decreased) through food supplementation and other forms of population management.

Sometimes inbreeding leads to such low fitness that habitat restoration to increase growth rates cannot remedy the decline and only the infusion of new genetic material can do so (e.g. Westemeier *et al*., 1998; Hogg *et al*., 2006; Pimm *et al*., 2006; Willi *et al*., 2007; Trinkel *et al*., 2008; Hedrick & Fredrickson, 2010). For example, even though Chillingham cattle have survived for 300 years with very little genetic variation, another bovine population managed under semi-natural conditions for the past 130 years seems destined to extinction from inbreeding depression without the inducement of gene flow from another herd (Halbert, Grant & Den, 2005). This illustrates the stochastic nature of the fixation of the deleterious load of mutations under inbreeding. By chance, some populations will become fixed for such a load of deleterious mutations that they cannot rebound without genetic rescue.

 

**Table 1** Summary information for nine studies demonstrating long-term population persistence in with low levels of molecular genetic variation

| Reference | Markers | Genetic diversity | Status |
|---|---|---|---|
| Ellegren *et al.* (1993) (*Castor fiber*) | 3 minisatellite loci and five MHC loci | $H_e$ decreased >50% in both Swedish and Norwegian populations compared with Russian populations. All three populations were monomorphic at MHC loci | Norwegian population grew from <200 individuals to 50 000 individuals in 110 years. Reintroduced beaver in Sweden grew from 50 individuals to 100 000 individuals in 70 years |
| Broders *et al.* (1999) (*Alces alces*) | 5 microsatellite loci | Average loss of 22% of $H_E$ across both populations | One reintroduced population grew from six individuals to 150 000 in 90 years and another from 18 individuals to 5000 in 45 years |
| Groombridge *et al.* (2000) (*Falco punctatus*) | 12 microsatellite loci | $H_E$ has been reduced 55% compared with museum specimens | Recovered from a low of two breeding pairs in 1974 to 200 breeding pairs in 2001 |
| Visscher *et al.* (2001) (*Bos taurus*) | 25 microsatellite loci | Monomorphic for 24 of 25 microsatellite loci | Low of 13 individuals in 1947. Eighty-five individuals in 2009 |
| Hadly *et al.* (2003) (*Ctenomys sociabilis*) | Cytochrome *b* (450 bp) | Single haplotype currently exists | Estimated to have survived 950 years with a single mitochondrial haplotype |
| Milot *et al.* (2007) (*Diomedea exulans*) & (*D. amsterdamensis*) | 234 AFLPs | $H_E$ for wandering albatross 0.028 and 0.01 for Amsterdam albatross | Wandering albatross exists at 8500 breeding pairs and shows no sign of being in decline. Amsterdam albatross reduced to 5 breeding pairs in 1981 and is *c.* 22 breeding pairs currently |
| Babik *et al.* (2009) (*Triturus cristatus*) | MHC class II genes (functional and pseudogenes) and 5 microsatellite loci | Greatly reduced MHC variation & a 22.5% reduction in microsatellite $H_E$ compared with refugial population | Crested newt populations in the UK have survived in isolation from the rest of Europe for an estimated 8500 years |
| Habel *et al.* (2009) (*Parnassius Apollo vinningensis*) | 6 microsatellite loci and 16 allozymes | Current populations are monomorphic at all loci. Museum collections suggest that the population may have been monomorphic for a century | Populations underwent recent declines but seem to be rebounding currently |
| Johnson *et al.* (2009) (*Haliaeetus vociferoides*) | 36 microsatellite loci | The Madagascar fish eagle has only 39% as much $H_E$ as its closest relative the African fish eagle | The population was estimated to have been stable at *c.* 120 breeding pairs for the past 2800 years |

For each paper the reference and binomial species name is given (Reference), the number and type of molecular markers used in the study (Markers), the amount of genetic diversity present of lost in comparison to a historical or neighboring population (Genetic Diversity), and the growth of the population form its low point to most recent census or the length of time the population has persisted (Status). Expected heterozygosity is denoted as $H_E$.
MHC, major histocompatibility complex.

However, most inbred populations will not be in such dire straits. Modeling has been explicit in suggesting under what conditions inbreeding depression is likely to be important (Reed *et al.*, 2003; Robert, Couvet & Sarrazin, 2005; O'Grady *et al.*, 2006; Theodorou & Couvet, 2006; Reed, 2008). Further reductions in fitness due to an increase in the inbreeding level or from environmental deterioration have little effect on population viability until their impacts on the stochastic population growth rate are such that it approaches zero, at which time even small changes can have huge impacts on the probability of extinction (see fig. 2.1 and accompanying text in Reed, 2008). If a population is already adapted to the environment, the environment is very benign, and/or the species is colonizing a habitat with available and suitable niche space with few generalist pathogens and predators to impede its population growth – lack of genetic diversity will likely seldom be a limiting factor in their success.

In the cases presented above, populations were expected to have strong positive growth rates due to environmental conditions. Thus, these examples do not run contrary to current ecological-evolutionary theory concerning population viability. However, the Mauritius kestrel example especially, provides us with hope that some populations with even extreme inbreeding will rebound on their own, or with human intervention, as long as suitable habitat remains.

## Neutral genetic variation

Eight of the studies inferring long-term persistence despite little genetic variation rely on estimates of neutral genetic variation from molecular markers, three assayed MHC diversity in addition to neutral genetic variation, and one relied solely on a region of mitochondrial DNA to infer genome-wide levels of genetic diversity. There are a number of problems associated with these methods.

Estimates of genome-wide heterozygosity from molecular markers usually correlate poorly with estimates of genetic variation for quantitative traits (Reed & Frankham, 2001; Badri *et al*., 2007; Knopp *et al*., 2007) and microsatellite diversity may correlate only moderately with estimates of genetic diversity from single nucleotide polymorphisms (SNPs) that can be found within coding regions of genes and directly selected upon (Väli *et al*., 2008). Fundamental differences in the maintenance and production of genetic variation at neutral loci versus loci under selection suggest that the evolutionary potential of a population for ecologically important quantitative traits are unlikely to be accurately predicted from assays of neutral molecular markers. This problem is further exacerbated if the number of loci used is small.

The three studies that did not rely solely on neutral genetic markers used variation at MHC loci. The use of genes known to be under selection and to be important to a trait (disease resistance) that impacts population viability is certainly a step in the right direction. However, while many studies have shown a correlation between MHC diversity and disease resistance, many others have not. As pointed out by Acevedo-Whitehouse & Cunningham (2006), variation at MHC loci may explain only half of the variation in disease resistance and molecular studies usually focus only on a small portion of all MHC loci. Combined with the fact that populations face many other stressors in addition to pathogens, it is not surprising that populations with little or no genetic variation at a particular MHC locus have been often been found to thrive.

## Bottlenecks

Several of the studies on long-term persistence with little genetic diversity were initiated because the populations were known to have passed through a population 'bottleneck'. Unfortunately, the somewhat vague definition of a population bottleneck (i.e. a short-term restriction in the total size of a population) can apply to cases where little genetic change is expected to occur (i.e. the term bottleneck has been applied to short-term restrictions of $\geq 100$ individuals, where the loss of genetic diversity is expected to be negligible). That aside, the ability of even populations that have been severely bottlenecked to undergo rapid evolutionary change has been verified repeatedly both experimentally and during natural founder events (e.g. England *et al*., 2003; Sax *et al*., 2007), so that such occurrences should no longer be surprising. Bottlenecks may reduce genetic variation in accordance with their severity and the number of generations which they persist. However, due to the ability of selection to maintain genetic variation and purge the genetic load in inbred populations (Kristensen *et al*., 2005; Bensch *et al*., 2006; Kaeuffer *et al*., 2007; Fox, Scheibly & Reed, 2008; Demontis *et al*., 2009) as well as the potential conversion of dominance and epistatic variation to additive genetic variation during bottlenecks (e.g. Bryant, McCommas & Combs, 1986; van Heerwaarden *et al*., 2008), many populations will suffer little or no loss of quantitative genetic variation even with severe inbreeding.

The following four papers deserve special attention because of the very long persistence times involved:

Hadly *et al*. (2003) provide an interesting and impressive case of a social rodent that seems to have existed for around a 1000 years with very little mitochondrial haplotype variation in the cytochrome *b* gene. This finding is no doubt noteworthy and raises the potential that this species may have persisted at a small population size for a remarkably long time. However, the claim that genome-wide genetic variation and genetic variation for quantitative traits was 'negligible' requires quite a leap of faith. The rank correlation coefficient between mitochondrial and allozyme diversity among 97 species of animals is only a moderate 0.36 (Piganeau & Eyre-Walker, 2009). Perhaps further work will elucidate the demography and genetics of this species over the past millennia, using a more comprehensive sampling of the genome, but it would be rash to declare this population as any kind of exception based merely on mitochondrial DNA.

Milot *et al*. (2007) sample a large number of markers and a huge number of individual wandering albatrosses as well. There is no doubt that the two albatross species have little molecular genetic variation at the markers assayed and that they almost certainly inherited the low genetic variation from a common ancestor. Thus, wandering albatrosses seem to have existed for hundreds of thousands of years with low molecular genetic variation. This is very informative and I found it impressive. However, no minimum heterozygosity level for neutral genetic loci has ever been suggested to be compatible with viability. The wandering albatross exists at a population size that should provide it with long-term viability and likely an effective population size large enough that mutation can replace genetic variation faster than drift can remove it (Reed *et al*., 2003). The Amsterdam albatross is a different story, but the results of its recent bottleneck and its loss of 60% of the heterozygosity found in the wandering albatross is yet to be determined. Milot *et al*. (2007) do an excellent job of pointing out potential reasons for the ecological success of wandering albatrosses despite low genetic variation. I concur with them wholeheartedly that some species will have naturally occurring low levels of neutral genetic variation based on their life history and other factors and that this should not be cause for alarm or spark genetic management.

The results of Babik *et al*. (2009), while very interesting for a number of reasons, are not surprising from an ecological-evolutionary perspective on population viability. Crested newts from a refuge population subsequently spread

 

across Europe after the retreat of the glaciers, despite reduced levels of MHC and overall genomic genetic diversity. It is well established that species can expand their geographic ranges drastically from only a small number of founders (e.g. Pascual *et al*., 2007; Zayed, Constantin & Packer, 2007; Eales, Thorpe & Malhotra, 2008), even when not repopulating depleted ecological communities after the retreat of glaciers. Thus, while always informative, such examples do not constitute a challenge to conventional thinking on population viability as the potential for expansive openings in niche space and reduced competition can allow for rapid population growth and huge carrying capacities for establishing species.

Johnson *et al*. (2009) represents probably the biggest challenge to current ecological-evolutionary thinking concerning population viability. However, the challenge is not on the grounds of the low molecular diversity seen in the fish eagles. The heterozygosity levels in this species are much higher than in albatrosses (Milot *et al*., 2007), the *Parnassius apollo* butterfly (Habel *et al*., 2009), and many other assayed species. However, it would be a wondrous feat if this population has managed to exist at 100–120 breeding pairs for 2800 years despite environmental and genetic stochasticity. Unfortunately, the confidence intervals around the timing of the start of population decline range from 600–9000 years. To test how unusual persistence over these time intervals might be, I used the stochastic population viability models for 102 species summarized by Reed *et al*. (2003). For an eagle of this size, 600 years is *c*. 45 generations. The probability of persistence over 45 generations averaged across models scales as:

$$P_{t=45} = 8.6K^{0.26}$$

where $P$ is the probability of persistence and $K$ is the ceiling carrying capacity. Thus, if we take 120 pairs (240 adult animals) as the carrying capacity, the probability of population survival over 600 years is expected to be 36%. These models include catastrophes, whereas the genetic data for the Madagascar fish eagle suggests a stable population size during this time. Catastrophes greatly decrease persistence times of populations and also increase the importance of genetic diversity if those catastrophes are repeated bouts of drought, similar pathogens or directional environmental change (Reed, 2008). However, the probability of persistence at this carrying capacity for 2800 years ($>200$ generations) is negligible and, if true, would make this an astounding feat of population persistence.

## Summary

Most of the nine papers published in the last decade describing long-term persistence with very little genetic variation do not require any paradigm shift in the way the relationship between genetic diversity and population viability is viewed. Many of the papers describe populations that have persisted for only a relatively short time and in very benign environments, the number of loci sampled was typically small, there is little evidence that assays of neutral genetic variation correlate well with levels of potentially adaptive quantitative genetic variation, and no recommendation exists for how little molecular genetic diversity a population can have and still persist for any given length of time. In fact, I would suggest that the minimum amount of genetic variation that a population can have and still persist (or the maximum amount it can lose) is not a question that can be answered in a general sense. The answer will be highly dependent on the quality of the environment, the rate at which the environment is changing, and the nature of the changes (e.g. temporally correlated, univariate or multivariate); as well as the genetic load and the genetic architecture specific to that population in that environment for the trait(s) of ecological importance. There will be a great deal of stochasticity involved. We can, however, develop robust rules for how losses of genetic variation impact the distribution of extinction times and under what conditions genetic variation will be most or least important.

## Integration and management recommendations

1. *Population viability should never be judged on the basis of molecular genetic data alone*. Current population size, population trend and habitat quality (carrying capacity) are probably the best predictors of population viability (O'Grady *et al*., 2004). Evolutionary potential cannot be estimated from assays of neutral genetic markers. In particular, among species comparisons are likely to be unfruitful given differences in life history, the potential for selective sweeps, and different per generation mutation rates (Piganeau & Eyre-Walker, 2009). Genetic rescue should be attempted when populations show signs of inbreeding depression such as negative population growth without any apparent environmental cause, unusual susceptibility to pathogens, poor body condition or deformities despite adequate available food or lowered fecundity or survivorship compared with larger populations in similar environments.

2. *Genetic effects do not operate in a vacuum*. The extent to which the fate of new mutations is deterministic as opposed to stochastic is determined by effective population size and the distribution of mutation parameters (Lande, 1994; Reed, 2008). Effective population size is determined by the extent and quality of the habitat, the variability of the environment, life history and mating system of the organism, and the population's fitness across the range of environments it will experience. Environmental and genetic effects interact so that ecological, demographic or genetic concerns should not be considered independently. Common and often strong inbreeding–stress interactions that cause populations low in genetic diversity to be more susceptible to stressful environments, combined with increasing anthropogenic sources of stress (e.g. acid rain, introduced pathogens/predators and increased drought frequency) and current rates of environmental change means that attention must be paid to genetic factors.

3. *Populations typically retain large amounts of quantitative genetic variation following a population bottleneck*. Populations have proven to be quite resilient to population bottlenecks

(e.g. Pascual *et al*., 2007; Zayed *et al*., 2007; Eales *et al*., 2008). Introduced species typically have lower molecular genetic variation in the invasive population than in their native range, yet there is little evidence for reduced quantitative genetic variation (Dugosch & Parker, 2008). It is becoming apparent that selection can sometimes be a very powerful force maintaining ecologically important genetic variation and purging the genetic load in bottlenecked populations (e.g. Kristensen *et al*., 2005; Bensch *et al*., 2006; Kaeuffer *et al*., 2007; Fox *et al*., 2008; Demontis *et al*., 2009).

However, that does not imply that genetic variation is unimportant to population viability. Estimates indicate that failed invasions likely outnumber successes by several fold, that many invasions are typified by a long periods of little apparent growth followed by a very rapid increase, and that many invasive species do have significantly higher genetic diversity than the native populations they were derived from (Mack *et al*., 2000; Roman & Darling, 2007). This suggests that small populations often fail and that successful invasion many times follows either an influx of genetic material from another population of the same species (or hybridization with a closely related local species) or that populations must be purged of inbreeding depression before expanding.

4. *Single examples of long persistence times with low genetic variation provide hope, but research suggests that increased genetic stochasticity associated with small population size increases extinction probabilities*. The impacts of decreased genetic variation will often be small under a variety of ecologically relevant conditions. In many, probably most, cases anthropogenic influences will supersede genetic concerns. Nonetheless, genetic problems are incremental rather than representing some threshold effect and their impacts can be insidious. All things being equal, a population with genetic diversity should persist longer than one without, unless that population is already perfectly adapted and the environment in stasis. Single examples of long-lived populations with low genetic variation are inspiring and tell us what is possible. Nevertheless, they are not replicated experiments that inform us of how median extinction time is impacted by low genetic diversity. For example, some species such as the albatrosses studied by Milot *et al*. (2007) have inherited low genetic diversity from their evolutionary ancestors. Potentially, such species purged their genetic load and what genetic variation remained has been sufficient for continued adaptation and survival. Yet, we can only look at the survivors and note that here is a species that survived despite low genetic variation. We do not know how many of their ancestors might have gone extinct because they did not have enough genetic variation.

Fortunately, in the vast majority of instances, demographic, ecological and genetic recommendations are the same. Populations need to have their habitats preserved or expanded in order to increase carrying capacity and habitats should be kept as free as possible from anthropogenic harm. This helps maintain genetic diversity and also positively affects demographic and ecological threats. In some instances evolutionary genetic recommendations may be at odds with ecological recommendations. Genetic rescue is one such instance (discussed previously; see also Hedrick & Fredrickson, 2010). Another might be supplemental feeding of populations. Supplemental feeding has the ability to increase population growth through increased fecundity and decreased mortality. This increased growth has been shown to decrease temporal variation in $r$ as predicted by theory (e.g. Schoech *et al*., 2008). However, long-term persistence in supplemental feeding could potentially lead to natural selection favoring a genotype dependent on the supplemental food or unnatural densities could lead to disease outbreaks and another population crash when supplemental feeding is halted.

## Future directions

Future conservation genetic studies should focus not on demonstrating that genetics is relevant to conservation but on incorporating genetics into an applied evolutionary-ecological context. I am happy to say that this is already being done more now than ever before. Fitness should be the primary measure for population viability studies, not assays of neutral genetic variation. However, with the advent of next generation sequencing techniques, for example 454 sequencing for non-model organisms (Vera *et al*., 2008) and rapid SNP discovery (Baird *et al*., 2008), molecular measures of genetic variation will become more useful in informing the conservation decision-making process. Genomic tools will allow for more detailed reconstruction of demographic histories, the detection of loci under selection and more precise estimates of what constitutes deleterious and beneficial genetic variation, and better estimates of genome-wide heterozygosity (Kohn *et al*., 2006).

More work is needed on the extent of local adaptation and how that impacts short- and long-term genetic rescue attempts in moderately and severely inbred populations, as well as on inbreeding–stress interactions and how they affect long-term population dynamics in complex natural environments.

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