FORUM ARTICLE

Managing genetic diversity in threatened populations: a New Zealand perspective

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Abstract: Genetic diversity allows a population to adapt genetically to a changing environment or to buffer it against stochastic events such as harsh weather or disease outbreaks. Genetic diversity is therefore an important consideration in the development of management strategies for threatened populations around the world, with the possible exception of New Zealand, where species recovery programmes tend to focus on increasing population size while neglecting the maintenance of genetic diversity. Many of New Zealand's threatened species have relatively low genetic variation and consequently may still be at risk in the long-term due to reduced resilience even if the effects of introduced predators were eliminated. The three main factors affecting genetic diversity – genetic drift, inbreeding and population subdivision – are processes that potentially impact on many of our locally threatened species, but their effects tend to occur over a considerably broader timescale than ecological effects, and as such are much more difficult to detect and ultimately to justify additional resource spending towards. Our message is that genetic management of New Zealand threatened species should not take priority over other management concerns such as controlling predators or improving habitat quality, but it needs more attention than it currently receives. We recommend that genetic diversity be a fundamental component in long-term management strategies for threatened species, and that such strategies are made explicit within the New Zealand Department of Conservation's current species recovery plans so that the persistence of biodiversity becomes of key importance, as opposed to current approaches that seek solely to maximise representation.

Keywords: conservation genetics; disease risk; extinction risk; genetic drift; genetic variation; inbreeding

Introduction

The defining issue surrounding the conservation of threatened species is that they have undergone a gradual or sudden decrease in numbers, increasing the risk of extinction. Controlled intervention can either eliminate the factors causing the decline, such as removal of introduced predators or habitat restoration, or attempt to slow the decline by increasing the size of the threatened population through captive breeding or translocation to less stressful habitats. The resulting genetic structure of such a bottlenecked population may also have important consequences to its long-term viability and therefore should be of concern to conservation managers. One consequence of small population sizes is that the frequency of inbreeding can increase, which can lead to the immediate loss of fitness (i.e. decreased survival or reproductive success, termed 'inbreeding depression'). A second but entirely different consequence is the loss of genetic variation, which can reduce the potential of populations to adapt to new challenges in their environment such as infectious diseases or climate change (Wallis 1994; Frankham et al. 2002; Keller & Waller 2002). Although eliminating the agent of decline should always take priority, both inbreeding depression and loss of genetic variation can also lead to an increased risk of extinction. Of the two, inbreeding depression tends to receive more attention because its effects are more obvious and immediate, while the consequences of loss of genetic variation are subtle and may take many generations to realise.

In a recent paper, Jamieson et al. (2006) argued that species recovery programmes in New Zealand tend to focus on increasing population size or number without having clear management guidelines in place to prevent inbreeding or maintain genetic diversity. That paper specifically documented the occurrence of inbreeding depression, and managing its impacts, in New Zealand threatened species. This forum article focuses on the loss of genetic diversity, with the aim of providing a synthesis of well-known and generally agreed upon causal links between genetic

diversity and population viability (including the contexts of island translocations and disease-risk management) from a New Zealand perspective; it is not meant to be an exhaustive review of the current literature. We end by highlighting some upcoming concerns in the management of genetic diversity in New Zealand. We consider that our 'target audience' is New Zealand managers involved in developing reintroduction strategies and implementing species recovery plans, plus ecologists with limited background in population genetics. We concentrate on avian conservation genetics, where there are already well-documented issues of concern in New Zealand, and which are the focus of our current research. Our overall aim is to provide an overview of the criteria used to justify spending limited conservation resources in New Zealand on the genetic management of threatened populations and outline where genetic issues are of relevance to species-based conservation.

Why preserve genetic diversity?

Species recovery programmes should aim to preserve a significant degree of the genetic diversity of a population and thus the potential for that population to adapt to a changing environment or to buffer it against stochastic events such as catastrophic weather or disease outbreaks (Lacy 1987). As future environmental changes and selective pressures are unpredictable, maintaining significant genetic variation is an important safeguard to ensure species are able to withstand and survive perturbations. The less genetic variation, the slower natural selection can operate, which has negative consequences for future adaptation. The importance of maintaining genetic diversity is therefore increasingly recognised as a key component in the development of management strategies for threatened populations around the world (Amos & Balmford 2001).

Two primary metrics are used to quantify genetic diversity in a population. The first, allelic diversity, is the simplest measure of genetic diversity as it describes the number of alleles present at a given genetic locus. The second, observed heterozygosity (*H*), is the proportion of individuals in a population that are heterozygous at a particular locus. In evaluations of genetic diversity, DNA markers are normally employed to measure allelic diversity and heterozygosity at a number of loci across the genome. It is assumed that these loci are representative of the genome as a whole, so that the number and frequency distribution of alleles detected can be used to compare populations, or to monitor changes over time. The most commonly used marker for measuring genetic diversity and gene flow between populations is highly variable microsatellite DNA (Frankham et al. 2002).

Although the relationship between genetic diversity and adaptive potential at the population level is well understood, the relationship between genetic variation and fitness at the level of the individual is more controversial (Pemberton 2004). Some recent studies have reported that individual heterozygosity measured with as few as 5–10 microsatellite markers is correlated with key components of individual fitness such as survival, fecundity, disease resistance, and lifetime reproductive success or recruitment (Coltman et al. 1999; Slate et al. 2000; Acevedo-Whitehouse et al. 2003; Foerster et al. 2003; Marshall et al. 2003; Markert et al. 2004). Currently, it is thought that such heterozygosity–fitness correlations found in large, outbred populations reflect linkage of microsatellite loci to functional loci that affect fitness and have little do with inbreeding, whereas such correlations in small inbred populations are more likely to reflect genome-wide heterozygosity levels and hence indicate genuine cases of inbreeding depression (Balloux et al. 2004; Pemberton 2004; Slate et al. 2004), although evidence provided from endangered species is surprisingly scarce (C.E. Grueber and I.G. Jamieson, unpubl. data).

While measuring the relationship between individual fitness and genetic diversity may be problematic, there is little doubt that significant loss of genetic diversity at the population level can have serious long-term consequences for the population. We briefly review the three main factors that can cause loss of genetic diversity: genetic drift, inbreeding and population subdivision.

Effects of genetic drift

Genetic variation can decline in populations because alleles are lost due to random genetic drift, defined as random fluctuations in allele frequencies across generations due to stochastic processes (Lacy 1987). The founder effect is a special case where the genetic composition of a new population may change if it originates from a small sample of a source population of individuals, such as occurs during the colonisation of an island (Frankham et al. 2002). The loss of genetic diversity due to drift is ultimately countered by mutation, which creates new alleles. However, mutations are rare events that are unable to counteract the relatively rapid pace of genetic drift characteristic of small, isolated populations (Lacy 1987; Frankham et al. 2002).

Genetic drift may seem of little concern in conservation biology because it mostly affects selectively neutral or near-neutral alleles. Alleles that have adaptive value in the current environment are unlikely to be lost due to drift (unless the population is very small), because natural selection will tend to maintain those alleles (Lande 1999). However, it is possible in small populations for harmful alleles to become 'fixed' by chance through the process of genetic drift, hence reducing population fitness (Hedrick & Kalinowski 2000). This is a possible explanation for the generally low egg fertility and hatching rates observed in genetically impoverished populations of the New Zealand endemics the takahē (*Porphyrio*

hochstetteri) and the kākāpō (*Strigops habroptilus*) (Jamieson et al. 2003, 2006; Roberston 2006).

In order to lose a large fraction of its genetic variation by genetic drift, a population must remain small for several generations. Following such loss, genetic variation can be restored by natural immigration, human-assisted translocation, and/or mutation, although an isolated population must regain and maintain a large size for a significantly long period of time (in the order of hundreds of generations) for mutation alone to restore genetic variation to pre-bottleneck levels (Lande 1999). By contrast, low immigration rates in the order of one or two individuals per generation from genetically variable populations may be sufficient to prevent further loss of variation due to drift (Wang 2004).

Effects of inbreeding

Loss of genetic variation due to drift can occur without any inbreeding, although the two processes often go hand in hand. Inbreeding is normally defined as mating between relatives. Individuals are considered related if they share at least one ancestor at any level in a pedigree. The level or extent of inbreeding is measured by the inbreeding coefficient *f*, which is the probability that two alleles at a given locus are homozygous by descent. Homozygosity by descent requires that the same allele was present in one copy in a common ancestor and passed through two separate lineages to come together through the mating of related individuals to form a homozygote. Thus homozygosity by descent differs from homozygosity by non-descent – cases where two copies of the same allele come together by chance alone (Frankham et al. 2002). It is therefore pertinent that homozygous genotypes can occur commonly in outbred individuals, but generally at a lower frequency than in inbred individuals. Pedigree information is required to identify whether an individual is likely to be homozygous due to descent or non-descent. Inbreeding does not alter the frequencies of alleles in a population like drift does, but redistributes them, resulting in an increase in homozygosity (a decrease in heterozygosity), and thus a loss of genetic diversity at the individual level (Keller & Waller 2002). This increase in homozygosity is likely to also lead to the expression of deleterious recessive alleles that are otherwise masked by dominant functional alleles in outbred individuals, causing a reduction in fitness. Therefore inbreeding has both immediate (inbreeding depression) and long-term (loss of adaptive potential) effects on population fitness (Frankham et al. 2002; Keller & Waller 2002). Looking at it another way, genetic drift has a greater impact on overall genetic diversity due to loss of alleles, while inbreeding increases the likelihood that the alleles that are present will be homozygous rather than heterozygous.

The frequency of inbreeding increases in threatened populations due to the small effective population size and a subsequent deficit of potential mating partners. Even if matings occur at random, a small population size will inevitably lead to matings between relatives (Frankham et al. 2002). Close inbreeding is preventable in captive breeding populations, but has not been closely managed in some captive breeding programmes in New Zealand in the past (e.g. blue duck – *Hymenolaimus malacorhynchos*, J. Wilcken and I. Fraser, unpubl. data; Otago skinks – *Oligosoma otagense,* Connolly 2005). Inbreeding is much more difficult to manage in wild populations. Since many animals evolved incest-avoidance mechanisms (Pusey & Wolf 1996), close inbreeding may not be a problem in threatened populations as long as there are sufficient numbers of unrelated breeders. The deliberate translocation of endangered takahē between islands is one of the few cases where close inbreeding in a free-ranging population is managed (Jamieson & Wilson 2003; Grueber & Jamieson 2007; Wickes & Crouchley 2008). Prevention of close inbreeding and maximising genetic diversity through intensive management is also underway in free-ranging kākāpō (Robertson 2006; Neill 2008).

Effects of population subdivision and migration

Subdividing a threatened population into several smaller ones – whether through indirect processes such as habitat fragmentation or through direct management activities such as the founding of small populations in captivity or on offshore islands – can have important implications for the genetic management of the species. The benefits of subdividing a highly threatened population include reduced risk from stochastic environmental events and reduced propagation of diseases, by effectively reducing population density and permitting the quarantine of subsets of the population if needed (Jones 2004). However, by subdividing a population, the effective sizes of the resultant sub-populations are reduced, thus increasing the speed at which genetic drift and inbreeding can have adverse effects on the long-term genetic diversity of the individual populations and the species as a whole.

Often the need to safeguard threatened populations against immediate threats, disease, predation and so forth is more pressing than the long-term negative genetic impacts of population fragmentation. Regardless, the immediate benefits of subdivision must be weighed against the potential for genetic problems in the long term. The manifestation of such problems will depend to a certain extent on the life-history characteristics of the individual species. Equivalent numbers of two species translocated to the same island could show very different rates of loss of genetic variation due to inherent differences in population growth rates. For example, all else being equal, translocated populations of saddlebacks (*Philesturnus carunculatus*) and robins (*Petroica australis*) are expected to lose much less genetic variation due to genetic drift and inbreeding compared with takahē and kākāpō populations (Taylor et al. 2005). Similarly, differences in degree of isolation or habitats' carrying capacities (e.g. islands versus mainland

populations) have also been shown to impact rates of loss of genetic variation in these species (Boessenkool et al. 2007; Taylor et al. 2007). For example, modelling has shown that South Island saddleback (*Philesturnus c. carunculatus*) populations successfully established on small offshore islands are expected to show considerably higher rates of loss of genetic variation if left unmanaged relative to those established on larger islands (Taylor & Jamieson 2007).

In true metapopulations, where exchange between sub-populations is minimal, human-assisted translocation can substitute for natural migration. Migration can aid the maintenance of genetic variation in the metapopulation, as it increases the effective population size by connecting sub-populations (Newman & Tallmon 2001). This can disperse rare or novel alleles throughout the population, increasing overall genetic diversity. Often very little migration is required for a significant increase in genetic diversity. One (reproducing) migrant per generation, which has been suggested as a rule-of-thumb for threatened population management (Mills & Allendorf 1996), is, according to computer simulation, a viable management option for threatened species (Wang 2004). Such an approach may circumvent most effects of population subdivision, while minimising the stress and expense associated with translocation. Such a programme has been proposed for managing genetic diversity in island populations of takahē (Grueber & Jamieson 2007).

At the other extreme, deliberate crosses between individuals from populations that have been separated for thousands of years can break down locally adapted gene complexes, resulting in outbreeding depression (Frankham et al. 2002; Edmands 2007). Therefore, unless a population is exhibiting severe inbreeding depression or is on the verge of extinction, crosses between strongly divergent populations or subspecies should be avoided.

Managing genetic diversity during translocation events

New Zealand managers have largely focused on removing immediate threats of introduced predators by translocation to island refuges or captive breeding. Which individuals are selected for translocation or captive breeding depends on the population characteristics that a manager intends to maximise. Haig et al. (1990) evaluated the benefits associated with six different selection criteria for choosing captive pairs of Guam rail (*Rallus owstoni*) for release back into the wild. These criteria included selection based on reproductive fitness (choosing the most fecund individuals), three selection options based on genetic management (maximising allozyme diversity, maximising allelic diversity, and equalising founder representation) and a random-selection option. Selecting the most fecund breeders may maximise the number of offspring produced, but can have several negative effects on the genetic structure, and ultimately survival, of the population (Haig et al. 1990). Some of the disadvantages of choosing the most fecund animals include: (1) the possibility of selecting for traits that are more suited to breeding and survival in the captive environment than the natural environment; and (2) potential loss of unique or rare alleles from the less fecund breeders and a decrease in genetic variation and evolutionary potential of the overall population.

The simulations performed by Haig et al. (1990) indicated that, where pedigree data were available, translocated animals should be selected by merit of their founder representation in the population at large, rather than by fecundity. This means individuals are chosen that best represent the diversity of the original founders to the source population. Jones et al. (2002) used these considerations in the management of the endangered whooping crane (*Grus americana*). The founders of the original population were identified and microsatellite analysis was used to calculate founder similarity coefficients based on DNA profiles and band sharing. The resulting DNA-based studbook provided information on previously unknown genetic relationships between founders of the whooping crane population. It also aided in the selection of individuals for translocation by equalising founder-allele frequencies, thus reducing losses in genetic variation (Jones et al. 2001). The Kakapo Recovery Programme has similarly employed genetic markers to assess paternity in order to limit additional breeding opportunities of overly successful males, as well as increase mating opportunities of under-represented lineages (Robertson 2006).

Genetic variation and disease risk management

One reason frequently given for managing genetic diversity in threatened species is to reduce the impact of disease, as levels of immunity may decline with inbreeding and loss of genetic variation (Frankham et al. 2002; Keller & Waller 2002). This relationship is firmly established in theory and has been supported in laboratory research, but its application to disease risk management of endangered species might understandably be viewed with cynicism when results of laboratory studies get overextended. For example, Spielman et al. (2004) showed that highly inbred populations of *Drosophila* with reduced allozymic variation were significantly less resistant to two types of bacterial infection relative to outbred populations. The relevance of these specific results to threatened species in general, however, is questionable, given the unnatural levels of inbreeding in some of the treatment populations (average inbreeding coefficient of 0.99 derived from 20–35 generations of sib–sib matings).

Other treatment populations with more realistic levels of inbreeding (f = 0.04–0.10) showed no significant reduction in disease resistance (Spielman et al. 2004, fig. 1). Based on their laboratory results, Spielman et al. (2004) went on to recommend that conservation managers minimise exposure of inbred or threatened populations to pathogens, and take precautions when moving animals between zoos or between fragmented populations. Such recommendations add nothing new to current best-practice wildlife management protocols; one assumes that procedures for minimising the risk of exposing New Zealand endemic species to infectious diseases, whether inbred or not, are already in place.

It should also be noted that susceptibility to disease is likely to be influenced by many factors other than just genetic variation, such as sociality, population density, climate, and proximity to likely vectors (Daszak et al. 2000; Harvell et al. 2002; Altizer et al. 2003). For example, West Nile virus has recently had devastating effects on local populations of American crows (*Corvus brachyrhynchos*), even though crows are widespread and common, and unlikely to have suffered from any recent or historical genetic bottlenecks (Eidson et al. 2001). Avian malaria was introduced to Hawai'i two centuries ago, but the devastating effect it had on Hawai'i's endemic avifauna occurred primarily after the introduction of its main mosquito vector *Culex quinquefasciatus* (Fonseca et al. 2000; van Riper et al. 2002). Avian malaria can have disastrous effects on any naïve host it encounters, not just those with reduced genetic variation. It therefore makes sense that recent surveys of the prevalence of avian malaria in New Zealand have been undertaken with respect to the expanding distribution of *C. quinquefasciatus* (Tompkins & Gleeson 2006), and not necessarily with regard to threatened species with low genetic variation, in order to quantify the risk associated with the spread of this disease.

Regardless of the above caveats, the increased risk of extinction associated with disease agents and small populations appears to be real (de Castro & Bolker 2005). Other than some compelling examples in captive populations (e.g. Ross-Gillespie 2007), there are only a few well-documented cases from natural populations showing increased susceptibility to pathogens or decreased immune response with increased homozygosity, but such examples are increasing (Coltman et al. 1999; Acevedo-Whitehouse 2003; Reid et al. 2003; Pearman & Garner 2005; Tompkins et al. 2006; Whiteman et al. 2006). For example, island populations of the Galápagos hawk (*Buteo galapagoensis*) with low levels of genetic diversity had higher parasite abundances and lower antibody levels than island populations that were more genetically diverse (Whiteman et al. 2006). Measures of immune functions are markedly higher in the cosmopolitan red-crowned parakeet (*Cyanoramphus novaezelandiae*) than in the endangered island endemic Forbes' parakeet (*C. forbesi*), as well as being higher in naturally occurring hybrids of the two species (Tompkins et al. 2006).

Recent research has also focused on the genetic variation of major histocompatibility complex (MHC) loci, which are thought to play a major role in disease resistance in vertebrates (Potts & Wakeland 1990; Sommer 2005). In New Zealand, Miller and Lambert (2004) showed that the black robin (*Petroica traversi*) had low variation in MHC genes compared with its more abundant congener, the South Island robin (*Petroica australis*). Given that one of the study populations of South Island robin (Motuara Island) had also gone through a short but severe bottleneck, Miller and Lambert (2004) concluded that MHC variation may only be eroded when population size is at a low level for a substantial period of time, as was the case of the black robin (Ardern & Lambert 1997). A preliminary survey did not find any evidence that black robins suffer from increased susceptibility to pathogens, but Miller and Lambert (2004) noted that the population could still be vulnerable to new pathogens. A similar study is underway to assess MHC variation in kākāpō (Robertson 2006). We believe that growing knowledge about the role of genetic variation in promoting disease resistance (for a recent review, see Sommer 2005) is reason in itself to pay greater attention to maximising or maintaining genetic variation in threatened populations.

Where to from here?

In the short term, the greatest risk of extinction to threatened populations in New Zealand continues to come primarily from introduced predators. Genetic effects tend to operate on a considerably broader timescale than ecological effects, and as such are much more difficult to detect. Therefore, even if conservation measures succeed at controlling predators, the loss of genetic variation could still compromise a population's resilience since it may be less able to respond to selection pressures. Many of New Zealand's endangered species now persist in small numbers on offshore islands or 'mainland island' sites where introduced predators have been eradicated and controlled, or in isolated patches of alpine, forest or wetland habitats. This presents research opportunities to investigate how genetic factors might influence the processes affecting population persistence.

In the short term, differences in genetic diversity between individuals may manifest as variable reproductive success. In the long term, the effects of drift, inbreeding, and population subdivision may all act to reduce evolutionary potential of small populations, and managers need to consider these factors in their recovery programmes. These concerns, which come under the broad umbrella of conservation genetics, have had a relatively low profile in New Zealand (for reviews see Wallis 1994; Lambert 1995; Lambert & Millar 1995) despite our threatened species

showing lower than average levels of genetic diversity compared with threatened species elsewhere, based on both microsatellite DNA (see table 1 in Jamieson et al. 2006) or minisatellite DNA (see table 2 in Robertson 2006). This situation may be changing as the Department of Conservation (DOC) has recently identified the loss of genetic diversity in our threatened species as a research topic of national priority (DOC 2003/04). In a recent email survey conducted by one of us (IGJ), 27 of 32 group leaders of DOC's recovery programmes stated they would like to have further information relating to the potential detrimental effects of inbreeding and loss of genetic variation. Although the majority of respondents worked with threatened bird species (14), several other recovery programmes – involving bats (2), fish (2), frogs (1), lizards (2), insects (2) and plants (4) – were also dealing with issues of inbreeding and loss of genetic variation. One specific question that requires further research is whether conservation managers should promote outbreeding by sourcing individuals for translocation from multiple sites or populations, or whether isolated populations should be kept genetically distinct. This is now becoming an extremely important issue to resolve as there is increasing demand from community-led biodiversity restoration initiatives for supply of avian species into predator-controlled sites.

In summary, genetic management of New Zealand threatened species should not take priority over other management concerns such as controlling predators or improving habitat quality, nor should it be seen as a panacea for recovery programmes (Robertson 2006; Jamieson 2007). However, in order to safeguard the long-term resilience of populations, genetic issues do require more attention than they currently get. We support IUCN recommendations that genetic diversity should be a fundamental component in long-term management strategies for threatened populations. We therefore recommend that such strategies be made explicit within DOC's current recovery plans such as those recently completed for takahē (Wickes & Crouchley 2008) and kākāpō (Neill 2008), so that the persistence of biodiversity becomes of key importance, as opposed to approaches that seek solely to maximise representation.

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References

Acevedo-Whitehouse K, Gulland F, Greig D, Amos W 2003. Disease susceptibility in California sea lions. Nature 422: 35.

Altizer S, Harvell D, Friedle E 2003. Rapid evolutionary dynamics and disease threats to biodiversity. Trends in Ecology and Evolution 18: 589–596.

Amos W, Balmford A 2001. When does conservation genetics matter? Heredity 87: 257–265.

Ardern SL, Lambert DM 1997. Is the black robin in genetic peril? *Molecular* Ecology 6: 21–28.

Balloux F, Amos W, Coulson T 2004. Does heterozygosity estimate inbreeding in real populations? Molecular Ecology 13: 3021–3031.

Boessenkool S, Taylor S, Tepolt C, Komdeur J, Jamieson IG 2007. Large mainland populations of South Island robins retain greater genetic diversity than offshore island refuges. Conservation Genetics 8: 705–714.

Coltman DW, Pilkington JG, Smith JA, Pemberton JM 1999. Parasite-mediated selection against inbred Soay sheep in a free-living island population. Evolution 53: 1259–1267.

Connolly J 2005. A comparison of the "quality" of wild and captive Otago skinks (*Oligosoma otagense*): are captive stocks of potential use in conservation? Unpublished MSc thesis, University of Otago, Dunedin, New Zealand. 172 p.

Daszak P, Cunningham AA, Hyatt AD 2000. Emerging infectious diseases of wildlife—threats to biodiversity and human health. Science 287: 443–449.

de Castro F, Bolker B 2005. Mechanisms of disease-induced extinctions. Ecology Letters 8: 117–126.

Department of Conservation 2003/04. Science Counts: National Priorities & Research Strategies. Wellington, N.Z.

Edmands S 2007. Between a rock and a hard place: evaluating the relative risks of inbreeding and outbreeding for conservation and management. Molecular Ecology 16: 463–475.

Eidson M, Komar N, Sorhage E, Nelson R, Talbot T, Mostashari F, McLean R, West Nile Virus Avian Mortality Surveillance Group. 2001. Crow deaths as a sentinel surveillance system for West Nile virus in the northeastern United States, 1999. Emerging Infectious Diseases 7: 615–620.

Foerster K, Delhey K, Johnsen A, Lifjeld JT, Kempenaers B 2003. Females increase offspring heterozygosity and fitness through extra-pair matings. Nature 425: 714–717.

Fonseca DM, LaPointe DA, Fleischer RC 2000. Bottlenecks and multiple introductions: population genetics of the vector of avian malaria in Hawaii. Molecular Ecology 9: 1803–1814.

Frankham R, Ballou JD, Briscoe DA 2002. Introduction to conservation genetics. Cambridge, Cambridge

University Press. 617 p.
Grueber CE, Jamieson IG 2007. Quantifying and managing the loss of genetic variation in a free-ranging population of takahe through the use of pedigrees. Conservation Genetics doi:10.1007/s10592-007-9390-3.
Haig SM, Ballou JD, Derrickson SR 1990. Management options for preserving genetic diversity: reintroduction of Guam rails to the wild. Conservation Biology 4: 290–300.
Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, Ostfeld RS, Samuel MD 2002. Climate warming and disease risks for terrestrial and marine biota. Science 296: 2158–2162.
Hedrick PW, Kalinowski ST 2000. Inbreeding depression in conservation biology. Annual Reviews in Ecology and Systematics 31: 139–162.
Jamieson IG 2007. Has the debate over genetics and extinction of island endemics truly been resolved? Animal Conservation 10: 139–144.
Jamieson IG, Wilson GC 2003. Immediate and long-term effects of translocations on breeding success in Takahe *Porphyrio hochstetteri*. Bird Conservation International 13: 299–306.
Jamieson IG, Roy MS, Lettink M 2003. Sex-specific consequences of recent inbreeding in an ancestrally inbred population of New Zealand takahe. Conservation Biology 17: 708–716.
Jamieson IG, Wallis GP, Briskie JV 2006. Inbreeding and endangered species management: Is New Zealand out-of-step with the rest of the world? Conservation Biology 20: 38–47.
Jones CG 2004. Conservation management of endangered birds. In: Sutherland WJ, Newton I, Green RE eds Bird ecology and conservation: a handbook of techniques. Oxford, Oxford University Press. Pp. 269–301.
Jones KL, Glenn TC, Lacy RC, Pierce JR, Unruh N, Mirande CM, Chavez-Ramirez F 2002. Refining the whooping crane studbook by incorporating microsatellite DNA and leg-banding analyses. Conservation Biology 16: 789–799.
Keller LF, Waller DM 2002. Inbreeding effects in wild populations. Trends in Ecology and Evolution 17: 230–241.
Lacy RC 1987. Loss of genetic diversity from managed populations: interacting effects of drift, mutation, immigration, selection, and population subdivision. Conservation Biology 1: 143–158.
Lambert DM 1995. The new science of molecular ecology. New Zealand Journal of Ecology 19: 93–96.
Lambert DM, Millar CD 1995. DNA science and conservation. Pacific Conservation Biology 2: 21–38.
Lande R 1999. Extinction risks from anthropogenic, ecological, and genetic factors. In: Landweber LF, Dobson AP eds Genetics and the extinction of species. Princeton, New Jersey, USA, Princeton University Press. Pp. 1–22.
Markert JA, Grant PR, Grant BR, Keller LF, Coombes JL, Petren K 2004. Neutral locus heterozygosity, inbreeding, and survival in Darwin's ground finches (*Geospiza fortis* and *G. scandens*). Heredity 92: 306–315.
Marshall RC, Buchannan KL, Catchpole CK 2003. Sexual selection and individual genetic diversity in a songbird. Proceedings of the Royal Society of London B (Suppl.) 270: S248–S250.
Miller HC, Lambert DM 2004. Genetic drift outweighs balancing selection in shapingpost-bottleneck major histocompatibility complex variation in New Zealand robins (Petroicidae). Molecular Ecology 13: 3709–3721.
Mills LS, Allendorf FW 1996. The one-migrant-per-generation rule in conservation and management. Conservation Biology 10: 1509–1518.
Neill E 2008. Kakapo recovery plan – 2006–2016. Wellington, Department of Conservation.
Newman D, Tallmon DA 2001. Experimental evidence for beneficial fitness effects of gene flow in recently isolated populations. Conservation Biology 15: 1054–1063.
Pearman PB, Garner TW 2005. Susceptibility of Italian agile frog populations to an emerging strain of *Ranavirus* parallels population genetic diversity. Ecology Letters 8: 401–408.
Pemberton J 2004. Measuring inbreeding depression in the wild: the old ways are the best. Trends in Ecology & Evolution 19: 613–615.
Potts WK, Wakeland EK 1990. Evolution of diversity at the major histocompatibility complex. Trends in Ecology & Evolution 5: 181–187.
Pusey A, Wolf M 1996. Inbreeding avoidance in animals. Trends in Ecology & Evolution 11: 201–206.
Reid JM, Arcese P, Keller LF 2003. Inbreeding depresses immune response in song sparrows (*Melospiza melodia*): direct and inter-generational effects. Proceedings of the Royal Society of London B 270: 2151–2157.
Robertson BC 2006. The role of genetics in kakapo recovery. Notornis 53: 173–183.
Ross-Gillespie A, O'Riain MJ, Keller LF 2007. Viral epizootic reveals inbreeding depression in a habitually inbreeding mammal. Evolution 61: 2268–2273.
Slate J, Kruuk LEB, Marshall TC, Pemberton JM, Clutton-Brock TH 2000. Inbreeding depression influences lifetime breeding success in a wild population of red deer (*Cervus elaphus*). Proceedings of the Royal Society of London B 267: 1657–1662.
Slate J, David P, Dodds KG, Veenvliet BA, Glass BC, Broad TE, McEwan JC 2004. Understanding the relationship between the inbreeding coefficient and multilocus heterozygosity: theoretical expectations

and empirical data. Heredity 93: 255–265.
Sommer S 2005. The importance of immune gene variability (MHC) in evolutionary ecology and conservation. Frontiers in Zoology 2: 16 doi:10.1186/1742-9994-2-16.
Spielman D, Brook BW, Briscoe DA, Frankham R 2004. Does inbreeding and loss of genetic diversity decrease disease resistance? Conservation Genetics 5: 439–448.
Taylor S, Jamieson IG 2007. No evidence for loss of genetic variation following sequential translocations in extant populations of a genetically depauperate species. Molecular Ecology 17: 545–556.
Taylor S, Jamieson IG, Wallis GP 2007. Historic and contemporary levels of genetic variation in two New Zealand passerines with different histories of decline. Journal of Evolutionary Biology 20: 203–2047.
Taylor SS, Jamieson IG, Armstrong DP 2005. Successful island reintroductions of New Zealand robins and saddlebacks with small numbers of founders. Animal Conservation 8: 415–420.
Tompkins DM, Gleeson DM 2006. Relationship between avian malaria distribution and an exotic invasive mosquito in New Zealand. Journal of the Royal Society of New Zealand 36: 51–62.
Tompkins DM, Mitchell RA, Bryant DM 2006. Hybridization increases measures of innate and cell-mediated immunity in an endangered bird species. Journal of Animal Ecology 75: 559–564.
van Riper C III, van Riper SG, Hansen WR 2002. Epizootiology and effect of avian pox on Hawaiian forest birds. Auk 119: 929–942.
Wallis GP 1994. Population genetics and conservation in New Zealand: a hierarchical synthesis and recommendations for the 1990s. Journal of the Royal Society of New Zealand 24: 143–160.
Wang J 2004. Application of the one-migrant-per-generation rule to conservation and management. Conservation Biology 18: 332–343.
Whiteman NK, Matson KD, Bollmer JL, Parker PG 2006. Disease ecology in the Galápagos Hawk (*Buteo galapagoensis*): host genetic diversity, parasite load and natural antibodies. Proceedings of the Royal Society of London B 273: 797–804.
Wickes C, Crouchley D 2008. Takahe recovery plan – 2007–2012. Department of Conservation, Wellington, New Zealand.

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