Animal Conservation (2000) **3**, 145–153 © 2000 The Zoological Society of London Printed in the United Kingdom

Genetic management of chondrodystrophy in California condors

Katherine Ralls[1], Jonathan D. Ballou[1], Bruce A. Rideout[2] and Richard Frankham[3]

[1] National Zoological Park, Smithsonian Institution, Washington, DC 20008, USA
[2] Zoological Society of San Diego, Post Office Box 551, San Diego, California 922112, USA
[3] Department of Biological Sciences, Macquarie University, NSW 2109, Australia

(Received 14 June 1999; accepted 12 November 1999)

Abstract

Five out of 169 fertile California condor (*Gymnogyps californianus*) eggs laid in captivity have exhibited chondrodystrophy, a lethal form of dwarfism. Pedigree records indicate that this chondrodystrophy, like similar conditions in chickens, turkeys and quail, is probably inherited as an autosomal, recessive allele. We estimate that the frequency of this putative allele is about 9%. This high frequency is probably due to a founder effect. We consider three management options for the allele: ignoring it, eliminating it by selection and minimizing its phenotypic manifestation by avoiding matings between possible carriers. We recommend minimizing its phenotypic expression because an unacceptably large proportion of condors (up to 78 out of 146) would be prevented from breeding under a selection strategy designed to eliminate the allele. We predict that many captive populations will prove similar to the California condor population in that it will prove inadvisable or impractical to select against one or more deleterious alleles detected in the population.

INTRODUCTION

California condors (*Gymnogyps californianus*), the largest New World vulture, once ranged over much of southern North America. They may have been restricted to the west coast after the extinction of the Pleistocene terrestrial megafauna left the species reliant on the carcasses of large marine mammals for food (Emslie, 1987). After Europeans arrived, human impacts such as shooting, egg collecting, poisoning of wolves and other predators, and lead poisoning led to a precipitous decline of the condor population (Snyder & Snyder, 1989) that culminated in 1987, when the last wild condors were brought into captivity (Wallace & Toone, 1992).

The captive population was founded by 14 individuals. DNA fingerprinting indicated that the captive individuals could be divided into three clans, in which birds within each clan were more closely related to each other than to individuals within the other clans (Geyer *et al*., 1993). Fortunately, California condors have bred well in captivity, probably because zoos had extensive experience with Andean condors (*Vultur gryphus)*. The first individuals were reintroduced to the wild in California in 1992. At the end of 1998, the population consisted of 146 individuals: 98 captive birds, 26 wild birds in California and 22 in Arizona (Mace, 1999).

The captive flock has produced five severely deformed embryos that exhibited chondrodystrophy and died near the time of hatching. Chondrodystrophy is a form of dwarfism caused by a disorder of the growth plates of the long bones in which linear growth is impaired while mineralization and appositional growth appear normal (Thorp, 1994). Chondrodystrophy in birds can be caused by a variety of factors such as mycoplasma infections and dietary deficiencies, particularly of manganese (Thorp, 1994). However, the lethal nature of the chondrodystrophy in condors and its pattern of occurrence are consistent only with a genetic origin. Many forms of inherited chondrodystrophy are known in humans (McKusick, 1998) and other vertebrates. Chondrodystrophy is inherited as a single autosomal recessive in chickens (Lamoreux, 1942; Landauer, 1965; Sullivan, 1994), quail (Collins, Abplanalp & Yoshida, 1968; Hermes *et al*., 1990) and turkeys (Asmundson, 1944; Gaffney, 1975; Nestor, 1978; Stout & Buss, 1979).

We describe the chondrodystrophic embryos produced by the condor population and infer the genetic basis of this trait from the population's pedigree. We consider three possible ways of managing the deleterious allele: ignoring it, eliminating it by artificial selection and minimizing its phenotypic expression. Finally, we make

All correspondence to: Katherine Ralls

recommendations for the genetic management of chondrodystrophy in the condor population.

METHODS

Each affected embryo received a complete necropsy. All tissues were fixed in 10% neutral-buffered formalin and processed routinely. Paraffin embedded tissues were sectioned at 5–7 μ and stained with haematoxylin and eosin.

Data on fertility and hatchability of all condor eggs produced since 1988 were derived from the annual *Captive breeding production* reports generated by R. Mesta (pers. comm.). Pedigree data were derived from the California condor studbook (Mace, 1999) in SPARKS (ISIS, 1996) format. Studbook data were complete and up-to-date through to the end of February 1999. The pedigree analysis was based on the animals alive on that date and included both captive and reintroduced birds.

To evaluate the impact of different potential management regimes on the condor population, we calculated several measures of genetic diversity within the population by pedigree analyses, using GENES software, version 11.92 (Lacy, 1999) and METAMK software, version 5.2 (Ballou, 1998). These genetic parameters were:

1. Mean kinship (MK): this value, calculated for every living member of the population, is the average kinship between that individual and all members of the population (including itself). Typically, living founders are excluded in the calculation of mean kinships. We included founders in our analysis because we were interested in how their removal from the breeding population under different selection strategies against the chondrodystrophy allele might affect overall levels of genetic diversity.
2. Average mean kinship: mean kinship averaged across all living individuals measures the overall level of relatedness (as measured by kinship) in a population and is used to calculate the proportion of gene diversity (GD) retained (retained GD = 1 – average MK).
3. Founder genome equivalent (FGE): the number of equally represented founders, with no loss of founder alleles, that yield the same retained gene diversity as is in the population. This can also be interpreted as the number of newly caught unrelated animals (i.e. founders) that would be needed to obtain the diversity in the population. This measure is directly related to mean kinship and gene diversity:

$$GD = 1 - 1/(2FGE).$$

4. Founder alleles: the number of founder alleles still in the population assuming that each founder possessed two unique alleles.

Lacy (1995) provides a more detailed discussion of these genetic parameters.

RESULTS

The chondrodystrophic embryos

All of the affected embryos died or were euthanized around the time of hatching. The most characteristic gross lesion was severe shortening of the legs (about 60% of the normal length), with variable twisting and/or curvature of the long axis (Fig. 1). Two embryos also had mild mandibular brachygnathism (shortening of the mandibular beak) and another had marked hydrocephalus. Histologically, the growth plates exhibited disorganization of the zone of proliferation with degeneration and necrosis of chondrocytes. The levels of 32 pesticides, 38 herbicides, 10 carbamate insecticides and polychlorobiphenyls in the liver of one affected embryo were within normal limits (data not presented).

Genetic analysis

Four of the five abnormal embryos (egg numbers 793, 894, 496 and 597) were produced by a single pair: male studbook number 27 (*Cuyama*) and female studbook

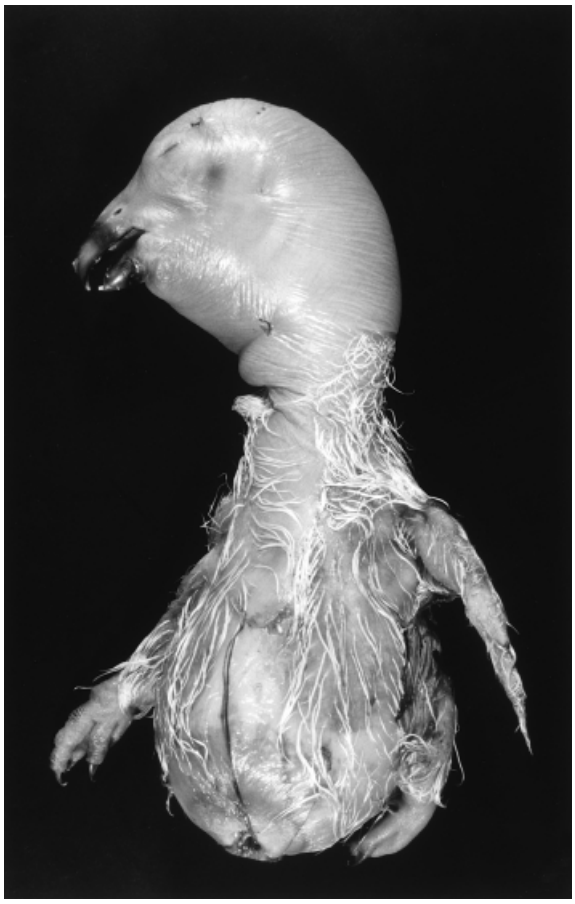

Fig. 1. Photograph of a chondrodystrophic California condor embryo (RP5860), showing the extremely shortened and curved legs (note that the shape of the right leg follows the contour of the body wall), marked oedema of the head and neck, and relatively normal wing length. Normal legs would be more than 50% longer than the legs of this embryo, extending well beyond the edge of the photograph.

number 31 (*Cachuma*). The fifth case was a sibling of male 27, studbook number 41. The hatchability of the fertile eggs produced by 27 and 31 was relatively low (74%) but not statistically different when compared to other pairs (mean 88%; $P = 0.08$, χ^2 test, $n = 205$, d.f. = 1). The pair often laid a second, or even a third egg when their previous egg was removed for artificial rearing, so they nevertheless produced 19 fertile eggs from 1989 to 1997. To date they have been the most productive pair of captive condors.

If we assume that male 27 and female 31 are heterozygotes for the putative recessive chondrodystrophy allele, i.e. have the genotype Dd where d (dwarf) is the lethal allele, we expect that 25% of their offspring will be homozygotes (with genotype dd) and die of chondrodystrophy. The observed frequency of chondrodystrophy in condors closely matches this expectation: four out of 19 fertile eggs produced by this pair (21%) displayed the trait. A frequency of 21% is significantly different from the 50% expected for a dominant trait ($P = 0.01$, χ^2 test, $n = 19$, d.f. = 1). The trait cannot be sex-linked or sex-limited as the affected embryos were of both sexes (female: pathology numbers RP5053, RP6364, RP5860. male: RP5232. unknown: 20509), while all affected individuals would be females (because females are the heterogametic sex in birds) if the allele were a sex-linked recessive.

We therefore assumed that chondrodystrophy in condors is caused by an autosomal, recessive lethal allele (Fig. 2). If so, both parents of male 27 (numbers 4 and 8) must have been heterozygotes, because they produced a chondrodystrophic embryo (number 41). The parents of female 31 were numbers 3 (*SSM*) and 12 (*AC8*). This pair produced six chicks, all of which were normal. The probability of producing six normal offspring if both were heterozygous is only 18%, so we assumed that only one of them was heterozygous for the putative deleterious allele and assigned each of them a 50% probability of carrying the allele. The founders involved (numbers 3, 4, 8 and 12) belong to two of the three clans identified by DNA fingerprinting (Geyer *et al.*, 1993).

We can estimate the frequency of the allele by calculating the probability that each of the living individuals carries the allele and summing over all individuals. This approach does not assume Hardy–Weinberg proportions and measures the frequency of the allele after selection against the five affected chicks. Under our assumptions, the only living sibling of male 27, number 23, has a 67% probability of carrying the allele, the 14 living offspring of 27 × 31 have a 67% probability and the offspring of 23 have a 33% probability. The siblings of 31 have a 50% probability, the half siblings of female 31 (her mother, number 12, also produced offspring with male 7 and male 21) a 25% probability, and crosses between half siblings and siblings a 12.5% probability. If both parents of 31 were heterozygotes, many of these probabilities would be higher. At the end of 1998, there were 146 living condors, 78 of which were potential carriers of the deleterious allele. However, given our assumptions, the frequency of the allele in the current population is only 8.8%, with 95% confidence limits of 3.5–14.2%. (We calculated the confidence limits for our estimate by running 50 000 gene drop simulations on the condor pedigree assuming the founder genotypes in Fig. 2, with a 50% probability of either number 3 or number 12 being heterozygous.) If the frequency of the allele is 8.8%, we would expect to see the trait in less than 1% of the fertile eggs (0.088^2) in a randomly mating population. As the condor population is not randomly

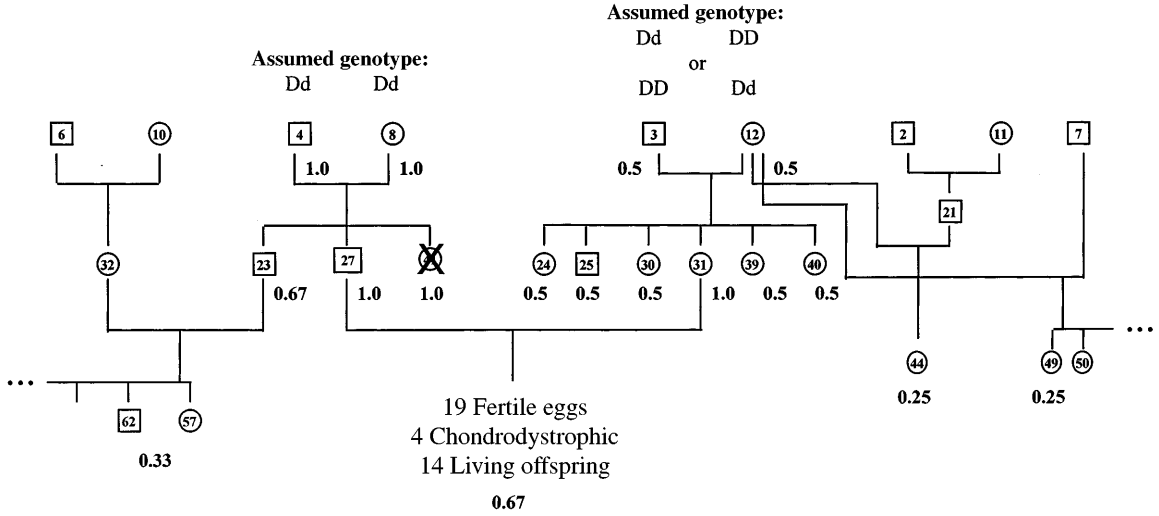

Fig. 2. Portion of the California condor pedigree showing the probability that individuals are carriers of the putative chondrodystrophic allele. Studbook numbers are shown in the circles (females) and squares (males). X marks affected chick number 41. Except for number 41, which was homozygous, numbers under the birds show the probability of being a carrier (heterozygote for trait) assuming the genotypes shown for founders 4, 8, 3 and 12 (assuming that either 3 or 12 was a heterozygote). The deformed embryos were 41 and four of the offspring of 27 × 31. One of the offspring of 27 × 31 died from causes unrelated to chondrodystrophy.

mating but is managed to avoid inbreeding, the expected proportion of homozygotes is even lower. However, because the known carriers came from two of the three clans, and the population is managed to avoid inbreeding, there could be additional undetected carriers. If so, we are underestimating the frequency of the allele in the population.

We consider three possible ways of managing the deleterious allele: ignoring it, eliminating it by artificial selection and minimizing its phenotypic expression by avoiding matings between potential carriers.

What will happen if we ignore the allele and maintain the current genetic management strategy?

Changes in the frequency of the putative chondrodystrophy allele will be determined both by natural selection eliminating copies of the allele and by the breeding strategy. The standard Species Survival Plan breeding strategy used in the USA is to minimize mean kinship among the members of a captive population (Ballou & Lacy, 1995). This is accomplished by mating individuals based on mean kinship, beginning with the individuals with lowest mean kinships, until the desired number of pairs is attained. Efforts are also made to avoid mating closely related individuals when choosing pairs. The remaining individuals with high mean kinship scores are not allowed to reproduce during that breeding cycle. However, because the condor population is still very small and rapid population growth is a high priority (Ralls & Ballou, 1992), the current breeding strategy is to choose new pairs to minimize mean kinship as far as possible but to pair all birds not reintroduced to the wild. This is the same strategy as that used in the black footed ferret reintroduction program (Russell *et al*., 1994). If all pairs produce similar numbers of offspring, the frequency of the deleterious allele will vary only slightly over time under this breeding strategy, and will be reduced only by natural selection.

Captive pairs are likely to produce more young than wild pairs because the first egg produced by a pair is often removed for artificial rearing in the hope that the pair will lay a second egg (Wallace & Toone, 1992). However, artificially increasing the reproductive rate of the captive pairs would not affect the frequency of the deleterious allele unless it was more common among the captive pairs than the wild pairs. (None of the wild birds have formed pairs as of the 1999 breeding season but this may occur soon as some males have exhibited courtship displays.)

Once the captive population reaches the target size of 150 individuals (U.S. Fish and Wildlife Service, 1996), it will be maintained at that size and managed to minimize mean kinship. Thus, individuals with lower mean kinship will be bred more frequently than individuals with higher mean kinship. If condors that are possible carriers of the deleterious allele have, on average, lower mean kinship than the rest of the population, the frequency of the allele would be likely to increase. However, the mean kinship of condors that are possible carriers is, on average, higher than the average mean kinship in the population (average MK for carriers is 0.059 relative to 0.053 for the entire population; Fig. 3). Thus, the frequency of the allele would probably decrease

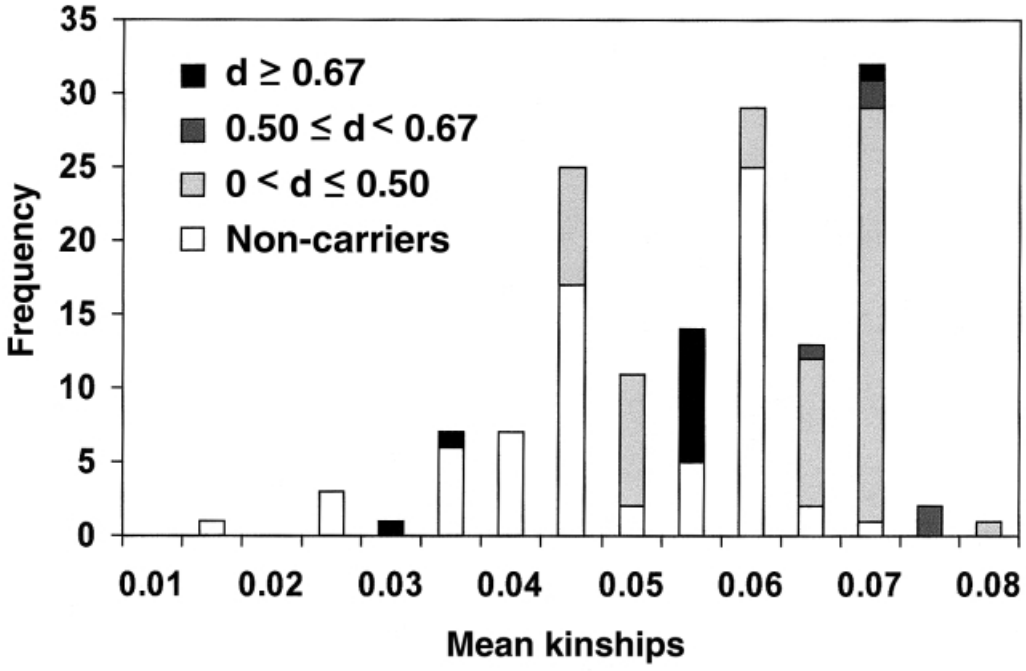

Fig. 3. Distribution of mean kinships in the 1998 condor population showing the mean kinship of potential carriers relative to non-carriers. Shaded areas show mean kinships of birds with different probabilities (*P*) of carrying the deleterious allele. Individuals with high mean kinship are more likely to be carriers of the allele than individuals with low mean kinship.

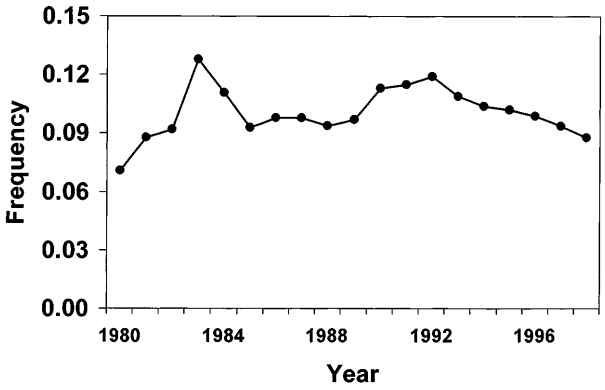

Fig. 4. Change in the frequency of the chondrodystrophy allele in the condor population from 1980 through the end of 1997

under this management plan. A plot of the changes in the frequency of the putative allele in fact shows a declining trend in the frequency of the allele over the last 6 years (Fig. 4).

There are two birds with relatively low mean kinship that have a high probability of carrying the allele (Fig. 3). These are numbers 27 and 23. The mean kinship of these two birds is relatively low because they are the only offspring of two dead founders, numbers 4 and 8 (Fig. 2). Although 27 is a known carrier and has produced 14 living offspring, his mean kinship is still only 0.030 compared to the average mean kinship of 0.053. In contrast, his mate 31 has a higher than average mean kinship of 0.068, largely because her siblings have produced more than 27 offspring in addition to the 14 she has produced. The relatively low mean kinship values of 27 and 23 suggests that they would be preferentially bred relative to other carriers and probably would reduce the degree to which the frequency might otherwise decline.

Over time, the mean kinship breeding strategy will result in decreased variance of mean kinship values and, eventually, approach equalization of mean kinship values among animals in the population. At this point, all animals are genetically similar enough to no longer warrant preferential breeding of some animals to the exclusion of others (at least on the basis of mean kinship alone). Changes in the putative chondrodystrophy allele frequency at this point will be determined solely by natural selection against homozygous recessive genotypes. The change in allele frequency would be at a rate of $-q^2/(1+q)$ per generation under random breeding, where q is the frequency of the allele (Crow & Kimura, 1970). With q currently at 0.088, the expected decline in the allele frequency due to mortality of homozygotes alone would be $-0.088^2/1.088 = -0.007$, or a decline of less than 1% over the next generation. However, the actual decline in allele frequency will probably be even slower, because part of the condor population will be maintained in captivity. Genetic management of captive populations reduces levels of homozygote production below those expected under random mating and thus provides the allele with greater protection against natural selection.

What will happen if we select against the allele?

An alternative breeding strategy would be to intentionally select against the deleterious allele by removing known or possible carriers from the reproductive population, as shown in Table 1. We could select against the allele at various intensities. For example, we could remove only numbers 27 and 31 from the breeding population. Or, we could select against the allele more strongly by removing all individuals with at least a 67% probability of carrying the allele. These would include numbers 27 and 31, all their offspring and number 27's sibling, number 23. The most intense form of selection would be to remove all individuals that are possible carriers of the allele. However, the benefits of removing carriers from the population (i.e. reducing the frequency of the chondrodystrophy allele and hence the probability of producing deformed chicks) must be weighed against the costs, which can be measured in terms of reduction in population size (i.e. loss of demographic potential) and loss of genetic diversity. Removal of individuals from under-represented gene lines, even though they are carriers, will reduce the overall level of genetic diversity in the population.

Because there are so many possible carriers of the allele, removing 27 and 31 from the breeding population would not appreciably reduce the frequency of the allele (Table 1), nor would there be much cost in terms of loss of either demographic potential or genetic diversity. Imposing more intense forms of selection against the allele would require removing substantial numbers

Table 1. The costs and benefits of selecting against the putative allele

Scenario	Number remaining	Costs				Benefits
		Number removed	Gene diversity	FGE	Number of alleles	Allele frequency
Current (keep all birds)	146	0	94.58	9.22	12.87	0.088
Excluding 27 & 31	144	2	94.56	9.19	12.87	0.082
Excluding ≥ 67% carriers	134	12	94.30	8.77	12.33	0.063
Excluding ≥ 50% carriers	129	17	94.31	8.79	12.33	0.056
Excluding ≥ 25% carriers	79	67	92.42	6.59	9.69	0.008
Excluding ≥ ALL potential carriers	68	78	92.29	6.48	8.9	0

Costs are both demographic (loss of potentially breeding animals) and genetic (number of alleles = number of founder alleles still remaining in the population based on gene drop analysis of 10 000 simulations; gene diversity = proportion of wild gene diversity retained; FGE = founder genome equivalents; Lacy, 1999).

of individuals from the breeding population and would significantly reduce the size and genetic variation of the population. The genetic effects of sequentially removing birds in order of decreasing probabilities of carrying the allele are shown graphically in Fig. 5(a). Although the frequency of the allele steadily decreases, so does the genetic diversity retained in the population. We would have to remove 32 birds to reduce the frequency of the allele in the population by 50% (Fig. 5(a)).

The above strategy selects against the allele without any consideration being given to the genetic value (mean kinship) of the probable carriers. Since carriers differ in their mean kinship (Fig. 3), an alternative strategy would be to select against high probability carriers while simultaneously considering their genetic value. This might allow selection against the allele without reducing the genetic diversity of the population. For example, bird 31 is a known carrier with a high mean kinship, while bird 27, also a known carrier, has a low mean kinship. Thus a selection strategy might select against 31 but leave 27.

Using the METAMK software (Ballou, 1998), we explored this approach by sequentially removing carrier birds from the population considering both their mean kinship and carrier probability. We worked through the birds beginning with those with the highest mean kinship. Among birds with similar mean kinship values, the birds with higher carrier probabilities were removed first. This process was continued until all 78 possible carriers were removed from the population, leaving the 68 non-carriers as the only remaining birds. As each bird was removed, the program calculated the resulting gene diversity and allele frequency. The results are shown in Fig. 5(b).

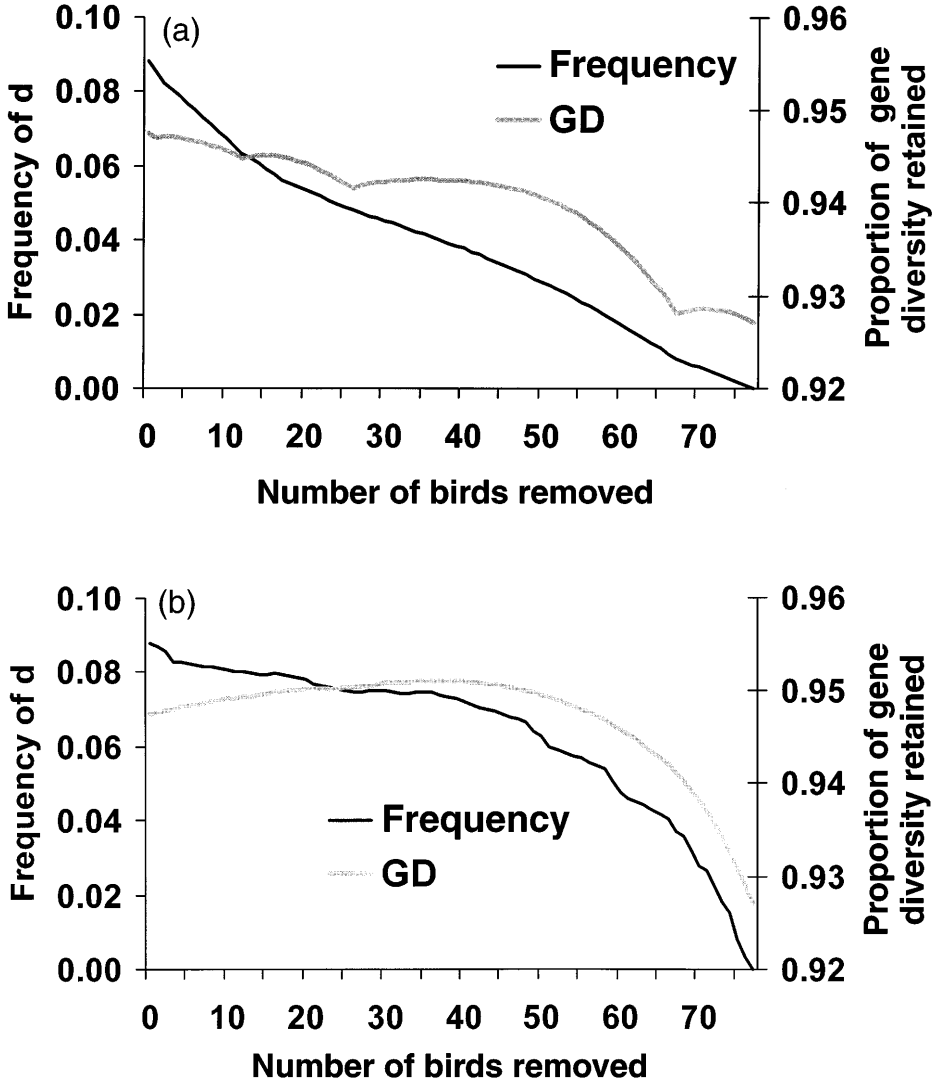

Fig. 5. Genetic effects of two methods of selection against the putative allele. (a) Reduction in frequency of the putative chondrodystrophy allele (d) (left axis, black line) and change in gene diversity (GD) (right axis, grey line) as potential carriers are sequentially removed from the population in order of the probability that they are carriers (individuals with the highest probability of being carriers are removed first); (b) reduction in allele frequency and change in gene diversity as potential carriers are sequentially removed from the population in order of mean kinship (individuals with the highest mean kinship are removed first).

By selectively removing carriers with high mean kinship, the gene diversity in the population can actually increase as the allele frequency decreases. The decline in allele frequency is slow, however, since most carrier birds with high mean kinship have a low probability of being carriers. Eventually, however (after removing about 55–60 birds), even genetically valuable carrier birds are removed, causing the gene diversity to drop sharply.

As birds are removed, the gene diversity initially increases then drops. Consider the point (at 56 birds removed) that the gene diversity returns to its initial level (94.7%). At this point, the allele frequency has dropped to about 5.5%. These results indicate that at no genetic cost (in terms of gene diversity) we can reduce the allele frequency by about 40% – from 8.8% to 5.5%. The demographic costs (56 birds removed from a population of 146 birds), however, would be substantial.

What will happen if we minimize phenotypic expression of the allele?

The current breeding strategy already reduces the production of homozygotes by avoiding pairings between siblings and other closely related individuals. Thus, the results of further reducing the production of homozygotes would be very similar to those of maintaining the current breeding strategy: the frequency of the deleterious allele will vary only slightly over time, declining slightly in frequency. We could further reduce the production of homozygotes by avoiding pairings between the offspring of animals with a high probability of being carriers, e.g. offspring of numbers 4 and 8 and those of numbers 3 and 12. Furthermore, if a pair produces a chondrodystrophic embryo, production of further homozygotes could be avoided by breaking up the pair and re-pairing them with non-carriers.

DISCUSSION

Abnormalities of skeletal growth and development can have genetic, toxic, infectious, or nutritional causes (Romanoff & Romanoff, 1972: 153–191). However, the pattern of occurrence, phenotype and lack of toxins in the affected embryos strongly suggest that chondrodystrophy in California condors, like similar conditions in other birds, is due to an autosomal, recessive lethal allele. We estimate that the frequency of this putative allele is about 9%.

The relatively high frequency of this deleterious allele is probably due to a founder effect because at least three of the 14 founders must have carried the allele. Founder effects that increase the frequency of deleterious recessive alleles are common in human populations, such as the Afrikaner population of South Africa or the Amish community in Pennsylvania, USA, that were founded by a small number of individuals (Diamond & Rotter, 1987). Relatively high frequencies of deleterious recessives have also been described in a number of captive animal populations that were founded by a small number of individuals (Laikre, 1999). Examples include diaphragmatic hernia in golden lion tamarins (Bush *et al*., 1980), blindness in wolves (Laikre, Ryman & Thompson, 1993), albinism in bears (Laikre *et al*., 1996), gingival hyperplasia in silver foxes (Dyrendahl & Henricson, 1959) and hairlessness in red-ruffed lemurs (Ryder, 1988; Nobel, Chesser & Ryder, 1989). The widespread occurrence of inbreeding depression in captive populations also suggests that many of these populations harbour substantial numbers of deleterious recessive alleles (Ralls, Ballou & Templeton, 1988).

Thus, it is extremely likely that the condor population carries a number of other deleterious alleles that have not yet been detected. For example, the population appears to produce a relatively high proportion of malpositioned eggs and at least one of the female lineages tends to produce extremely small (though viable) eggs. We could not afford to select against multiple deleterious alleles given the very small size of the condor population. Fortunately, natural selection will slowly reduce the frequency of lethal alleles. Unfortunately, natural selection will not be strong enough to eliminate many deleterious alleles with less serious effects, because genetic drift is often stronger than natural selection in small populations (Hedrick, 1994).

Should we select against the putative allele for chondrodystrophy in the condor population? Making a decision about this question requires consideration not only of the genetic and demographic costs of selection but of the current phase of the condor breeding program and the goals of program managers. The development of a captive breeding population can be divided into three phases: the founding phase, during which the population is initiated; the growth phase, during which the population rapidly increases to the final size desired by its managers (the target population size); and the carrying capacity phase, during which the population is maintained at its final size (Ralls & Ballou, 1992). Management concerns, including the emphasis placed upon genetic management of the population, change as the population progresses through these phases. The condor population is still in its growth phase, with about 98 birds in captivity and a target population size of 150 birds. Management emphasis during the growth phase is normally on rapid population growth until the target population size is attained. Rapid growth minimizes both the loss of genetic diversity in the captive population and the likelihood that the population will become extinct due to the stochastic factors associated with small size. As the population nears its target size, the selection of breeding individuals is increasingly based on genetic measures and all individuals are no longer allowed to breed. Removal of individuals for reintroduction generally begins as the population nears its capacity phase. The condor program is unusual in that reintroduction began during the growth phase due to other pressing concerns, such as the need to preserve habitat and uncertainty over the availability of financial resources for reintroduction in future years. Currently, almost all chicks (about 20 per year) are released to the wild,

although some are returned to the captive population due to undesirable behaviour in the wild. Birds are being released in California and Arizona, and program managers hope to initiate releases at two additional locations in the near future. This early emphasis on reintroduction has slowed the growth of the captive population and removing possible carriers of the chondrodystrophy allele from the population would slow its growth even more.

We analyzed the pedigree of the entire population. However, only 98 birds are in captivity with the remainder in the wild. If we were to remove all of the 78 possible carriers, we would have to bring 31 birds back into captivity and prevent them from breeding. Although it is clear that the demographic impacts of preventing more than half of the total population from breeding would be substantial, it is difficult to predict the exact impact due to uncertainty about long-term survival rates and the future fecundity of the wild birds (no breeding has occurred in the wild as yet). Considering only the captive population, there are now 98 birds, of which 51 appear to be non-carriers. If none of the chicks were reintroduced to the wild, a captive population of 98 birds growing at the current growth rate of 10–14% per year (this is the growth rate of the total population; all reproduction is in captivity and most mortality is in the wild) would reach the target size of 150 in about 5 years, while a captive population of 51 birds would take about 12 years to reach the same size. However, these projections are unrealistic because most chicks are currently released to the wild rather than maintained in the captive population.

At this time, given the program's emphasis on producing chicks for reintroduction and the high demographic cost of selection, we recommend minimizing the phenotypic expression of the allele rather than selection against it. The pair 27 and 31 has already been broken up and none of the other current pairs has a high probability of producing a homozygote. If another chondrodystrophic embryo is produced, we should re-pair its parents and re-evaluate the genetics of the trait based on the new data. However, some selection against the allele may be appropriate at a later date as the captive population enters its capacity phase. If selection is undertaken, we recommend removing possible carriers with high mean kinship rather than removing the birds with the highest probability of being carriers. As the captive population enters the capacity phase, managers might also consider mating high probability carriers with each other or with known carriers to determine which birds actually carry the allele.

Managers of captive populations tend to assume, often with very little evidence, that deformities that appear in a population are genetic and initiate programs to select against the defect. Verifying that a trait is genetically determined, and its mode of inheritance, can be statistically extremely difficult depending on the specific pedigree involved. We were fortunate in this case because chondrodystrophy is a known lethal recessive in other birds and enough chicks were produced to strongly support our assumptions. However, another advantage of delaying selection against the putative allele is that this strategy allows time for the accumulation of more evidence that the chondrodystrophy in condors does indeed have a genetic basis and possibly even the development of some diagnostic method to distinguish carriers from non-carriers.

The decision to select against a trait should only be made after careful analysis of the pedigree and the effects of various selection strategies must be evaluated in the light of their effects on overall levels of genetic diversity in the population. For example, Laikre *et al*. (1993) conducted a detailed statistical analysis of the trait for hereditary blindness in wolves and concluded that the trait could be reduced without seriously affecting founder allele survival. However, we predict that many captive populations will prove similar to the California condor population in that it will be inadvisable or impractical to strongly select against one or more deleterious alleles detected in the population.

Acknowledgements

We thank Cyndi Kuehler and Mike Mace, past and present keepers of the California condor studbook, and Robert Mesta, U.S. Fish and Wildlife Service, for providing us with the data necessary to conduct our analyses. We also appreciate the contributions of the clinical veterinarians and pathologists at the Zoological Society of San Diego and the Los Angeles Zoo.

REFERENCES

Asmundson, V. S. (1944). Inherited shortening of the long bones in the turkey. *J. Hered*. **35**: 295–299.

Ballou, J. D. (1998). *METAMK software: mean kinship analyses for two populations with gene flow*. Washington, DC: National Zoological Park.

Ballou, J. & Lacy, R. C. (1995). Identifying genetically important individuals for management of genetic diversity in captive populations. In *Population management for survival and recovery:* 76–111, Ballou, J. D., Gilpin, M. & Foose, T. (Eds). New York: Columbia University Press.

Bush, R. M., Montali, R. J., Kleiman, D. G., Randolph, J., Abramowitz, M. D. & Evans, R. F. (l980). Diagnosis and repair of familial diaphragmatic defects in golden lion tamarins. *J. Am. Med. Assoc*. **l77**: 858–862.

Collins, W. M., Abplanalp, H. & Yoshida, S. (1968). Early embryonic chondrodystrophy in Japanese quail. *J. Hered*. **59**: 248–250.

Crow, J. F. & Kimura, M. (1970). *An introduction to population genetic theory*. New York: Harper & Row.

Diamond, J. M. & Rotter, J. I. (1987). Observing the founder effect in human evolution. *Nature, Lond*. **329**: 105–106.

Dyrendahl, S. & Henricson, B. (1959). Hereditary gingival hyperplasia of silver foxes. *Vet. Bull*. **29**: 658–659.

Emslie, S. D. (1987). Age and diet of fossil California condors in Grand Canyon, Arizona. *Science* **237**: 768–770.

Gaffney, L. J. (1975). Chondrodystrophy: an inherited lethal condition in turkey embryos. *J. Hered*. **66**: 339–343.

Geyer, C. J., Ryder, O. A., Chemnick, L. G. & Thompson, E. A. (1993). Analysis of relatedness in the California condors from DNA fingerprints. *Mol. Biol. Evol*. **10**: 571–589.

Hedrick, P. W. (1994). Purging inbreeding depression and the probability of extinction: full-sib mating. *Heredity* **73**: 363–372.

Hermes, J. C., Abbott, U. K., Johnston, E. & Owens, M. (1990).

A new chondrodystrophy mutation in Japanese quail (*Coturnix japonica). J. Hered.* **81**: 222–224.
ISIS (1996). SPARKS: single population animal record keeping system software. Apple Valley, MN: International Species Inventory System.
Lacy, R. C. (1995). Clarification of genetic terms and their use in the management of captive populations. *Zoo Biol.* **14**: 565–577.
Lacy, R. C. (1999). *GENES: pedigree analysis software*. Chicago, IL: Chicago Zoological Society.
Laikre, L. (1999). Hereditary defects and conservation genetic management of captive populations. *Zoo Biol*. **18**: 81–89.
Laikre, L., Andren, R., Larsson, H. O. & Ryman, N. (1996). Inbreeding depression in brown bear, *Ursus arctos*. *Biol. Conserv*. **76**: 69–72.
Laikre, L., Ryman, N. & Thompson, E. A. (1993). Hereditary blindness in a captive wolf (*Canis lupus*) population: frequency reduction of a deleterious allele in relation to gene conservation. *Conserv. Biol.* **7**: 592–601.
Lamoreux, W. F. (1942). Hereditary chondrodystrophy in the fowl. *J. Hered.* **33**: 275–283.
Landauer, W. (1965). Chondrodystrophy of the fowl. Observations on penetrance and expressivity of Lamoreux's chondrodystrophy. *J. Hered.* **56**: 209–214.
Mace, M. (1999). *California condor studbook*. Escondido, CA: San Diego Wild Animal Park.
McKusick, V. A. (1998). *Mendelian inheritance in man*, 12th edn. Baltimore: The Johns Hopkins University Press.
Nobel, S. J., Chesser, R. K. & Ryder, O. A. (1989). Inbreeding effects in a captive population of ruffed lemurs. *J. Hum. Evol.* **5**: 283–291.
Nestor, K. E. (1978). Hereditary chondrodystrophy in turkeys. *Poultry Sci.* **57**: 577–580.
Ralls, K. & Ballou, J. D. (1992). Managing genetic diversity in captive breeding and reintroduction programs. *Trans N. Am. Wildl. Nat. Res. Conf.* **57**: 263–282.
Ralls, K., Ballou, J. D. & Templeton, A. (1988). Estimates of lethal equivalents and the cost of inbreeding in mammals. *Conserv. Biol.* **2**: 185–193.
Romanoff, A. L. & Romanoff, A. J. (1972). *Pathogenesis of the avian embryo: an analysis of causes of malformations and prenatal death*. New York: Wiley-Interscience.
Russell, W. C., Thorne, E. T., Oakleaf, R. & Ballou, J. D. (1994). The genetic basis of black-footed ferret reintroduction. *Conserv. Biol.* **8**: 263–266.
Ryder, O. A. (1988). Founder effects and endangered species. *Nature, Lond.* **331**: 396.
Snyder, N. F. R. & Snyder, H. (1989). Biology and conservation of the California condor. In *Current ornithology, volume 6:* 175–267. Dennis, M. P. (Ed.). New York: Plenum Press.
Stout, J. T. & Buss, E. G. (1979). Identification of a 16–17-day lethal factor in a chondrodystrophic stock of turkeys. *J. Hered.* **70**: 62–64.
Sullivan, T. W. (1994). Skeletal problems in poultry – estimated annual cost and descriptions. *Poultry Sci.* **73**: 879–882.
Thorp, B. H. (1994). Skeletal disorders in the fowl: a review. *Avian Path*. **23**: 203–236.
U.S. Fish and Wildlife Service (1996). *Recovery plan for the California condor*. Portland, OR: U.S. Fish and Wildlife Service.
Wallace, M. & Toone, W. (1992). Captive management for the long-term survival of the California condor. In *Wildlife 2001: populations:* 766–744. McCullough, D. & Barrett, R. (Eds). London: Elsevier Applied Science.