GENETIC EVIDENCE FOR PHILOPATRY IN A COLONIALLY NESTING SEABIRD, THE FAIRY PRION (*PACHYPTILA TURTUR*)

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ABSTRACT.—Philopatry is the selective return of individual birds to breed close to the site of their own hatching. This phenomenon can greatly influence the operation of evolutionary forces within and among populations of a species. Detection of philopatry normally involves long-term mark, release, and recapture studies. As an alternative, we have applied restriction enzyme analysis of mitochondrial DNA to a colonially nesting seabird species, the Fairy Prion (*Pachyptila turtur*), as a test of philopatry. As the mitochondrial genome is strictly maternally inherited, each colony in a philopatric species should have a unique combination of mitochondrial haplotypes. Twenty-one prions taken from one colony all had identical mitochondrial genomes, and we argue that juveniles as well as experienced adults return to the colony to breed. Philopatry within this colony does not explain the lack of mtDNA sequence variation. The most likely explanation is the recent occurrence of either a bottleneck or founder event within the colony probably involving a maximum of four females and an unspecified number of male prions. Two additional colonies of prions were not genetically homogeneous and did not have significantly different combinations of mitochondrial DNA lineages. It was not possible to confirm the existence of philopatry in these colonies. *Received 4 September 1990, accepted 18 February 1991.*

AMONG the myriad of breeding sites potentially available, the majority of birds chose to breed close to the site of their own hatching (Greenwood and Harvey 1982). This is true for both migratory and nonmigratory species (Greenwood 1987), although it does not apply to species occupying unpredictable habitats such as inland Australia. The amount of philopatry displayed by a species will determine, in part, the degree of genetic subdivision between populations that modulates the operation of evolutionary forces such as random mutation, genetic drift, and natural selection.

Estimating the degree of philopatry exhibited by avian species in the field has invariably involved long-term mark and recapture studies of large numbers of individuals. We applied restriction-site analysis of the mitochondrial DNA (mtDNA) of breeding adults sampled from three Australian prion colonies to test for philopatry.

Mitochondrial DNA is a maternally inherited genetic tag (Lin et al. 1990) recoverable from all individuals. Unlike nuclear DNA, its components are not subject to recombination between generations and its nucleotide sequence is highly polymorphic among individuals (Avise et al. 1987). Together, these characteristics mean that maternally related groups of males and females are highly likely to share mitochondrial genomes with the same nucleotide sequence. Groups of animals that are not maternally related are unlikely to share identical mitochondrial genomes. For example, Avise and Nelson (1989) found that geographically continuous populations of the Seaside Sparrow (*Ammodramus maritimus*) on the Atlantic and Gulf coasts of eastern North America possessed different sets of mitochondrial lineages.

The Fairy Prion (*Pachyptila turtur*, Procellariidae) is a small, tube-nosed seabird distributed around the 40°S convergence in the oceans of the southern hemisphere. It is widespread at sea around Australasia, and in the southern Atlantic and Indian oceans. The species breeds in dense colonies from August to February on offshore islands around southern Australia and New Zealand and on the Chatham, Snares, and

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Antipodes islands (Lindsey 1986). Australian Fairy Prions use their colonies as foraging bases outside the breeding season. Although banding studies of individuals in New Zealand suggest fidelity to island colonies and the formation of relatively stable pair bonds (Harper 1976), such studies have not been performed on Fairy Prions breeding in Australia.

In a species that is not in a state of flux, and which has geographically discrete breeding groups or colonies, philopatry should leave an unambiguous stamp on the pattern of mtDNA sequence variation among and within colonies. This pattern will have developed over evolutionary time by the independent stochastic creation and extinction of mtDNA lineages in each breeding group (Avise et al. 1984). Each philopatric colony will have its own combination of mtDNA lineages that is significantly different to that possessed by another philopatric colony. These unique combinations may consist of as few as one or as many as thousands of lineages, depending on long-term effective size of each colony.

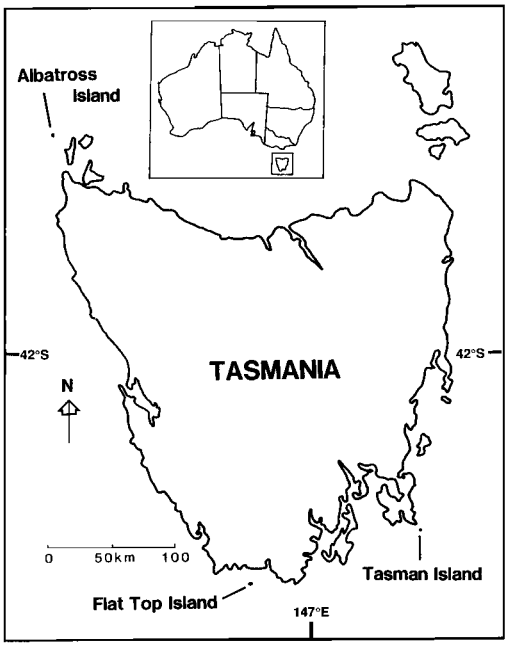

Fig. 1. Locations of Albatross, Flat Top, and Tasman islands, from which prions were sampled for this study.

Materials and Methods

We collected prions from Flat Top Island (43°38'S, 146°23'E, $n = 19$), Tasman Island (43°14'S, 147°56'E, $n = 21$), and Albatross Island (40°23'S, 144°39'E, $n = 21$) during the southern summer breeding season of 1988–1989 (Fig. 1). Males and females were taken arbitrarily from accessible nesting sites on each island. One Antarctic Prion (*Pachyptila desolata*) was sampled from Macquarie Island (52°S, 158°E). The liver was dissected from each bird and stored in liquid nitrogen. Mitochondrial DNA was extracted from thawed liver samples by a modification (Ovenden et al. 1988) of the method of Chapman and Powers (1984).

The mtDNA samples were digested with two 5.33 class restriction enzymes (*Ava* I, and *Ban* I) and seven 6.0 class restriction enzymes (*Bam* HI, *Bgl* I, *Cla* I, *Hin* dIII, *Nco* I, *Pst* I and *Xho* I). Each sample was digested with up to a tenfold excess of enzyme to ensure complete cleavage, but otherwise we followed the supplier's (New England Biolabs, U.S.) directions. Restriction fragments were radiolabelled with alpha-^{32}P or ^{35}S-deoxycytosine triphosphate using the exonuclease and polymerase activity of the Klenow fragment of DNA polymerase I (Ovenden et al. 1989). Fragments were visualized by autoradiography in dried 1.4% agarose gels. Fragment sizes were estimated by comparison with the mobility of fragments of known size produced by a *Hin* dIII digest of bacteriophage lambda DNA.

Samples that produced unique fragment patterns with a particular enzyme were assigned a unique letter. Lettering began with *A* and proceeded through the alphabet. No attempt was made to assign consecutive letters to the most similar restriction patterns. In all cases, the number of fragments in the pattern was assumed to be equal to the number of restriction sites in that genome for that enzyme. The relative gain or loss of restriction sites between samples was determined by the additive loss or gain of appropriately sized fragments. The haplotype, or mitochondrial genotype, of each bird was a summary of letters corresponding to the fragment patterns produced from that sample for each of the nine restriction enzymes.

We estimated sequence divergence between pairs of mitochondrial genomes, with standard errors, by the maximum likelihood method of Nei and Tajima (1983). We also estimated the average sequence divergence between individual genomes from different colonies and between genomes from the same colony (Nei and Jin 1989). The standard errors of these estimates produced by this method are free of the bias introduced by the nonindependence of the individual pairwise divergence measurements. Student's *t*-test was used to test the null hypothesis that the mean sequence divergence between genomes collected from one colony was significantly different from that from another colony.

We compared observed and expected haplotype frequencies to detect possible population subdivision among island colonies, by the Chi-square test. A Monte-Carlo test was used to determine the significance

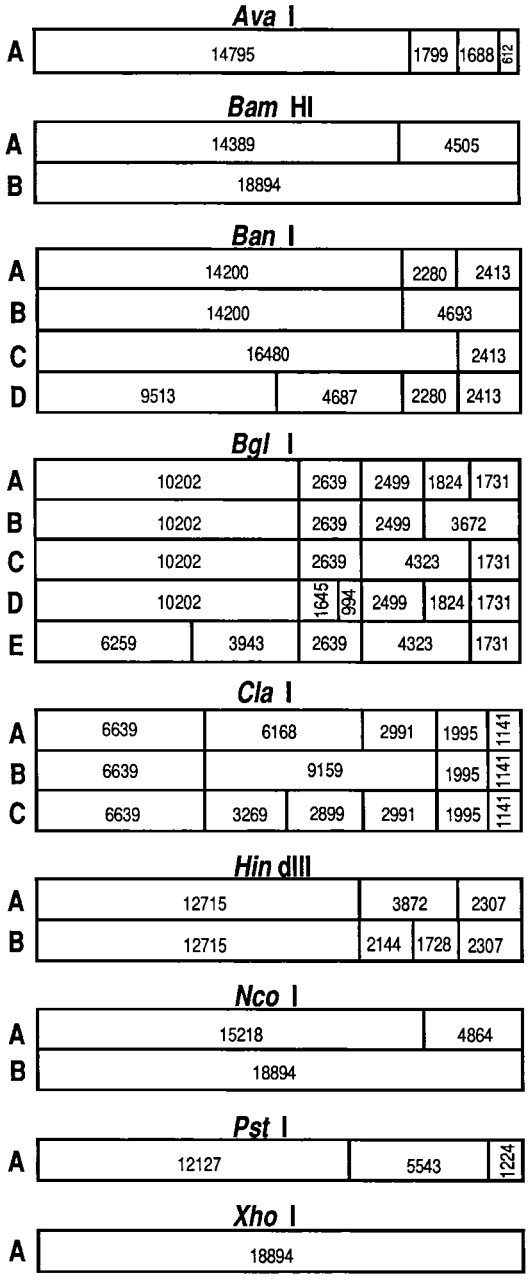

Fig. 2. The fragment composition of the restriction site morphs identified among Fairy and Antarctic prion mitochondrial genomes by nine restriction enzymes. Fragment sizes were scaled so that their sum is equal to the overall mean genome size of 18,894 nucleotide pairs. Fragments were placed on the linear mtDNA maps to show restriction site gains or losses between them. The relative position of the fragments was not determined absolutely.

of the Chi-square value (Roff and Bentzen 1989), as expected class sizes were often <5. As population subdivision causes localized excesses of homozygotes, a gene diversity analysis on groups of prions was done by taking nucleotide substitutions (Takahata and Palumbi 1985) as alternate alleles. The statistic calculated (G_{ST}) can be interpreted as the proportion of overall genetic variation that can be attributed to the presence of subpopulations among which gene flow is limited. The significance of the G_{ST} value was assessed against 1,000 pseudo-G_{ST} values obtained from random rearrangements of the raw data (Palumbi and Wilson 1990). If population subdivision is present, the true G_{ST} value will be greater than 95% of the pseudo-G_{ST} values. The cladistic relationship between prion haplotypes was explored using MacClade (version 2.1, Wayne Maddison and David Maddison, Harvard University).

RESULTS

We identified 1–5 morphs from 9 restriction enzymes among the 62 prion mitochondrial genomes (Fig. 2). The Antarctic Prion sample was not analyzed with *Nco* I or *Xho* I. It was assigned the Fairy Prion common morph (A) for each of these enzymes. The approximate sum of the sizes of restriction fragments from the mtDNA from the Fairy Prion was 18,900 nucleotides.

We scored eleven haplotypes among the 62 prions in our survey of 33 restriction sites (Table 1). Each haplotype was defined by the presence of between 26 to 30 of these sites. Twenty-one restriction sites were present in all haplotypes, nine were present in one haplotype only, and three sites (*Ban* I site 1, *Bgl* I site 1, and *Nco* I site 1) were present in more than one—but not all—haplotypes. All prions collected from the breeding site on Albatross Island possessed haplotype 1. This haplotype was found in 7 birds from Flat Top Island and 12 from Tasman Island (Table 1). The remaining 12 individuals from Flat Top Island had four different haplotypes (2, 3, 4, and 5). Four birds from Tasman Island shared two of these haplotypes (2 and 5). Haplotypes 3 and 4 were found only on Flat Top Island. The remaining five birds from Tasman Island possessed haplotypes found only within this colony.

Fairy Prion haplotypes 2 and 4–10 differed by one restriction-site gain or loss from the most common haplotype 1 (Fig. 3). The Antarctic Prion (haplotype 11) differed by three restriction-site gains or losses from Fairy Prion haplotype 5. Fairy Prion haplotype 8 was one restriction-site mutation from either haplotype 5

TABLE 1. Numbers of prion haplotypes scored from each of three island colonies. Each haplotype is composed of the morph designations for the restriction enzymes *Ava* I, *Bam* HI, *Ban* I, *Bgl* I, *Cla* I, *Hin* dIII, *Nco* I, *Pst* I, and *Xho* I.

Haplotype	Locality: Albatross (n = 21)	Flat Top (n = 19)	Tasman (n = 21)	Total
Fairy Prion				
1 AAAAAAAAA	21	7	12	40
2 AAAAAABAA	0	1	2	3
3 AABBABBAA	0	2	0	2
4 AAAABAAAA	0	2	0	2
5 AAACAAAAA	0	7	2	9
6 AACAAAAAA	0	0	1	1
7 ABAAAAAAA	0	0	1	1
8 AAACAABAA	0	0	1	1
9 AABAABAAA	0	0	1	1
10 AAADAAAAA	0	0	1	1
Antarctic Prion				
11 AADECAAAA				1

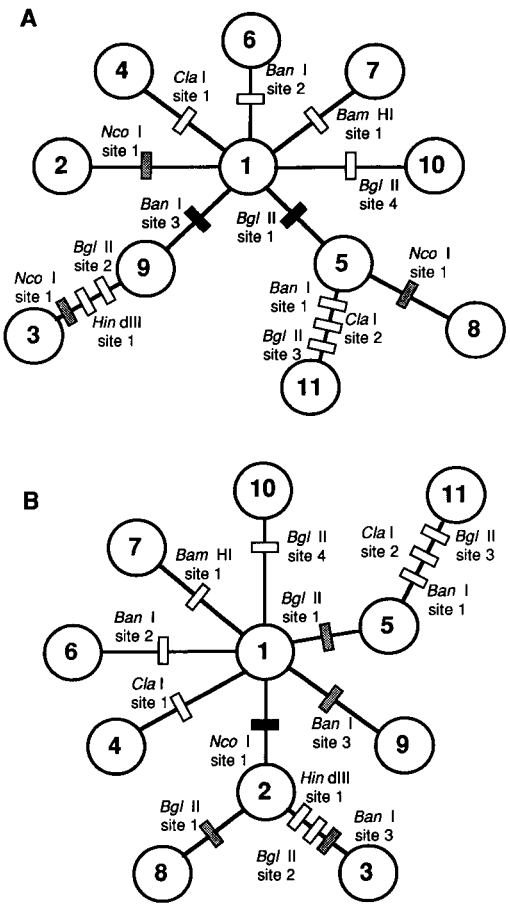

Fig. 3. The two most parsimonious, unrooted networks (A, B) describing the relationship between ten Fairy (no. 1–10) and one Antarctic Prion (no. 11) haplotypes. Labelled bars across lines joining haplotypes indicate the relative gain or loss of restriction sites. Sites that were present or absent in only one haplotype are represented by an open bar. Sites that were present or absent in more than one, but not all, haplotypes are represented by a shaded bar if they appear in the network more than once and by a solid bar if they appear only once. *Bam* HI site 1 was lost by *Bam* HI morph A. *Ban* I site 1 was gained by *Ban* I morph D, and *Ban* I sites 2 and 3 were lost by morphs C and B, respectively. *Bgl* I morph B lost *Bgl* I site 2, while morph C lost site 1. *Bgl* I site 4 was gained by morph D and site 5 was gained by morph E. *Cla* I morph B lost *Cla* I site 1 and morph C gained site 2. *Hin* dIII site 1 was gained by morph B, while *Nco* I site 1 was lost by morph B (Fig. 2).

(Fig. 3A) or haplotype 2 (Fig. 3B). Fairy Prion haplotype 3 was three restriction-site mutations from either haplotype 9 (Fig. 3A) or haplotype 2 (Fig. 3B).

The mean (±SD) sequence divergence among the mitochondrial genomes from 19 prions from Flat Top Island was 0.51 ± 0.25%. This was significantly different from the genomes sampled from the Tasman Island colony (0.28 ± 0.15%, $t = 3.86$, $t[P = 0.05, \text{df} = 38] = 2.02$, two-tailed). The magnitude of mtDNA sequence divergence on Flat Top Island sample differed from Tasman Island because of the exclusive occurrence of two birds that possessed the divergent haplotype 3 within the Flat Top Island sample.

The net mtDNA sequence divergence between birds from the Flat Top and Tasman island colonies was not significantly different from zero (0.0087 ± 0.0192%). The frequencies of haplotypes sampled from the two islands were also not significantly different ($\chi^2 = 13.36$, $P = 0.05 \pm 0.0195$). Finally, the amount of overall mitochondrial gene diversity that was due to genetic subdivision among island colonies (G_{ST}) was 0.17. This value does not provide evidence for a lack of gene flow between island colonies as it was greater than only 55.4% of 1,000 pseudo-G_{ST} values.

The mitochondrial genome of the Antarctic Prion was similar to those of the Fairy Prion. One Fairy Prion haplotype (no. 3) was as different from the remaining Fairy Prion haplotypes (range of estimated sequence diversity (±SE), 1.00 ± 0.59% to 1.70 ± 0.80%) as was the

Antarctic Prion (1.05 ± 0.62% to 1.78 ± 0.84%). The carcasses of the two prions that had haplotype 3 were not morphologically different from the remaining 17 birds collected from Flat Top Island (Barton 1989). We estimated the sequence divergence between the Antarctic Prion haplotype and Fairy Prions haplotype 3 at 2.54 ± 1.03%.

DISCUSSION

All 21 birds from Albatross Island had the same mtDNA haplotype. Although this cannot be taken to indicate absolutely that all individuals on the island are homogeneous for the mitochondrial genome sampled, we believe that this haplotype must predominate in the colony. If all birds we examined were part of a single panmictic population and the haplotypes were present in the entire population in ratios represented in the total sample examined here, the probability of choosing 21 birds all with haplotype 1 is only 0.00014.

There are approximately 10,000 breeding pairs of Fairy Prions on Albatross Island (N. Brothers unpubl. data). Prions forage over many hundred square kilometers of the open ocean and it is likely that the foraging range of prions from the three colonies sampled in this study would overlap. Within their foraging range, individuals would encounter other breeding colonies of prions, as well as unused potential breeding sites. mtDNA is strictly maternally inherited and the best explanation for the current exclusive occurrence of haplotype 1 among the male and female prions sampled from the Albatross Island population is strict philopatry of both sexes with no immigration from genetically distinct colonies. It is likely that juvenile prions chose mates from other juveniles encountered on the island. There is evidence that visual (Serventy et al. 1989), auditory (V. Bretagnolle pers. comm.), and olfactory cues (Grubb 1974) are used by seabirds to guide their return to specific colonies. Analysis of the mtDNA of prions from other island colonies in western Bass Strait, Black Pyramid Island for example, is necessary to test the hypothesis of island—as opposed to regional—philopatry.

The mtDNA profiles provide no information on philopatry of birds from the colonies on Flat Top and Tasman islands. It is, however, unlikely that the birds from these two southerly islands behave differently from their conspecifics on Albatross Island. They are presumably philopatric. Our sampling regime, in terms of either numbers of individuals or numbers of restriction sites surveyed per genome, may have been inadequate to identify significant differences in mtDNA profiles, especially if the colonies have not experienced bottlenecks, founder events, or the completion of stochastic lineage sorting. Both colonies are larger than the Albatross Island colony, which would slow the process of lineage sorting. However, from the length of time each island has been isolated from mainland Tasmania, the colonies may be older than the colony on Albatross Island. Some evidence that philopatry operates within each of the colonies is given by the significant difference in the magnitude of mtDNA sequence divergence within each colony. Different amounts of mtDNA sequence variation in each colony would not accumulate if a large amount of gene flow occurred between them.

We suggest that the observed mitochondrial genome homogeneity within the Albatross Island colony indicates philopatry. Philopatry does not, however, necessarily cause homogeneity of haplotypes. There are at least four possible explanations for haplotype homogeneity within the Albatross Island colony: a founder effect, a bottleneck event, stochastic sorting of matriarchial lineages, and selection.

A *founder event* occurs when a new population is established from the offspring of a few individuals in a range remote to the original population. The Albatross Island colony must be relatively young and may have experienced a recent founder event. From 30,000 to at least 8,000 years B.P., the sea level around Tasmania was at least 100 m lower than it is today. During that time Albatross Island would have been an elevated area well inland from the coast and unsuitable habitat for prions. In contrast, Flat Top and Tasman islands are surrounded by deep, precipitous waters, and any land link between them and the Tasmanian mainland at lower sea level would have been of shorter duration and fractured earlier than one between the mainland and Albatross Island.

If the prion colony on Albatross Island was established recently, the mtDNA haplotypes of the colony would probably not be homogeneous unless the haplotypes of the founding females were identical. Assuming that the frequency of haplotype 1 in the original population was the same as it is now, it is likely that the founding group consisted of no more than four females and their mates. With strict philo-

patry and abundant resources, only a few dozen generations would have been required to reach the current colony size.

An alternative explanation for the observed homogeneity of mitochondrial genomes on Albatross Island could be a recent bottleneck event. A *bottleneck event* is similar to a founder event, except that a new population is produced from a few individuals within the range of the original population, which is assumed to be extinct now. For all of the prions to have the same mtDNA, the maximum number of genetically identical female prions that survived the bottleneck to repopulate the colony may again have been four or less.

The derivation of an entire population from a small number of individuals can lead to reduced genetic variation, not only in the mitochondrial genome but also in nuclear genes. Nei et al. (1975) have shown that this will occur if the population does not expand rapidly after a founder or bottleneck event. Extreme philopatry can also lead to the loss of genetic variability because of random genetic drift in a finite population (Greenwood 1987). It may be possible to assess the degree of inbreeding of the prion colony on Albatross Island by a survey of heterozygosity at allozyme loci and by measurements of fitness that could include fecundity estimates, hatching rates, longevity, and morphological variation.

Theoretically, monophyly or homogeneity of mtDNA lineages can occur within a population in the absence of either founder or bottleneck events. Avise et al. (1984) calculated that the processes of stochastic lineage extinction and creation in a stable-sized population at carrying capacity could lead to monophyly in $4n$ generations, where n is the female population size. The generation time, or the mean age of a breeding adult for Fairy Prions is ca. 5 yr. The female population size for the Albatross Island colony is ca. 10,000. Assuming complete philopatry, 200,000 yr would be needed to homogenize the mtDNA lineages. This argument, therefore, is not supportable given the short history of the colony.

Natural selection operating directly on the mtDNA or its gene products is another way of removing genetic variation in a population. It has been suggested that inefficiency of mitochondrial genome repair and replication in somatic cells during the lifetime of an individual is responsible for aspects of aging and death (Linnane et al. 1989). Members of maternal lineages that have mtDNA less susceptible to this type of decay during the life of a somatic cell may be more long-lived and hence leave more offspring than other lineages. Although there are many good reasons why selection may operate on the mitochondrial genome, the concept has not been widely accepted (MacRae and Anderson 1988).

The range of intraspecific mtDNA variation for Fairy Prions is similar to that found in other vertebrate species. However, interspecific genetic variation between the Fairy and Antarctic prions is low compared with measurements made between other congeneric avian species. The range of diversity measurements between the Antarctic and Fairy prion haplotypes (0.011–0.025) is below that for *Anas* (0.004–0.088), *Aythya* (0.025–0.043), *Melospiza* (0.026–0.030) and *Dendroica* (0.031–0.055) (Kessler and Avise 1985). Future analyses of mtDNA may explain this low interspecific genetic divergence.

Based on our finding of mtDNA homogeneity in a single colony of Fairy Prions, we suggest that juveniles that were hatched on the island and experienced adults that bred at least once in the colony return continually to the colony to breed. The presence of philopatry alone cannot explain the lack of mtDNA sequence variation in the colony. The most likely explanation is the recent occurrence of either a bottleneck or founder event within the colony, possibly assisted by selection for mitochondrial genomes, which increased the fitness of birds within certain maternal lineages. The bottleneck or founder event may have involved a maximum of only four females and an unspecified number of males. The other two colonies of prions examined appear not to have experienced such severe bottleneck or founder events. Confirmation that philopatry operates within these two colonies will rely on the detailed analysis of the mtDNA from large numbers of individuals. Finally, because the amount of mtDNA sequence divergence between species of prions is low, the phylogeny of the genus could be investigated by applying cladistic and phenetic analysis to the presence and absence of restriction sites in their mtDNA.

ACKNOWLEDGMENTS

We than Jenny Jarrett and Wayne Kelly for assistance in the laboratory. The NAVAIDS section of the Tasmanian Department of Transport and Neil Smith assisted in the collection of samples.

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