Genic variation in the yellow mongoose (*Cynictis penicillata*) in Southern Africa

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Starch gel electrophoresis was used to analyse genetic variation at 28 enzyme and protein loci, within and between eight Southern African populations of yellow mongooses Cynictis penicillata. *Thirteen loci (46%) were polymorphic in one or more populations. Mean heterozygosity* ($\bar{H}$) *was 3.4%. Allelic variation at all populations conformed to Hardy–Weinberg expectations and the mean value of Wright's* F_{IS} *coefficient (0.039) indicated no evidence of inbreeding in populations. Owing to a high proportion of 'private' alleles (polymorphic alleles occurring only in single populations), Wright's mean* F_{ST} *was exceptionally high for a mammal (0.585 for all populations, or 0.272 when samples with* $N < 3$ *were excluded), indicating significant* ($P < 0.01$) *spatial heterogeneity in allele frequencies. Independent lines of evidence suggest that low vagility in the species may contribute partly to the high* F_{ST} *result, although sampling error may also be implicated. The range of genetic distances between pairs of localities (0.000 – 0.105 for Nei's* D_N*) was typical of conspecific populations, and phenetic analysis of genetic distances failed to group populations on the basis of proposed subspecific affiliation based on morphometric evidence.*

In recent years the technique of allozyme electrophoresis has proved to be an effective tool for resolving systematic problems at both intraspecific and supraspecific levels,[1–4] as well as for characterizing the genetic structure of natural populations of plants and animals.[5] Most mammalian allozyme studies have been directed at the smaller mammals, and in particular the rodents, with relatively few studies involving species of the order Carnivora.[6] In the Carnivora, most allozyme studies have been concerned with higher order phylogenetic relationships among genera and families.[7] Data on allozyme variation in species of Carnivora are scarce (refs 8 and 9 and references cited therein), owing largely to the rarity of many carnivore species, low population densities in the wild and practical difficulties in collecting large samples of specimens.

This study is the first to examine genetic variability in a member of the family Viverridae (genets, civets and mongooses). While many viverrid species are solitary, nocturnal and difficult to collect in large numbers, the yellow mongoose *Cynictis penicillata* is a common, diurnal, communal-dwelling species which is widespread throughout (and endemic to) Southern Africa[10] and relatively easy to collect in the field. This provided an opportunity to analyse electrophoretically detectable genetic variability in and among several populations of this species. Apart from the present study, a preliminary cladistic analysis of allozyme characters in several viverrid species has recently been conducted.[62]

Previous studies of morphometric[63] and colorimetric[64] variation in *C. penicillata* resulted in the proposal of four parapatric subspecies (the species was previously considered to be monotypic).[11,12] The present study includes specimens from two subspecies, *C. p. penicillata* and *C. p. bradfieldi*. The primary purpose of this paper is to report the genetic basis of morphological differences between these two proposed phena/taxa.

It has been pointed out that tempo and mode of speciation may be predicted largely by the population genetic structure of a species.[13–15] A further aim of this paper is therefore to attempt to predict the mode and tempo of (sub)speciation in the yellow mongoose, based on the population genetic structure of the species, and to examine these predictions in the light of morphological and biogeographical evidence.

Material and methods

Thirty-five yellow mongooses from eight localities (Fig. 1) in South Africa and Namibia were shot or live-trapped between 1985 and 1988. Specimens were killed using ether or an immobilising drug consisting of tiletamine and zolazepan in equal proportions ('zoletil': Reading). Liver, kidney (and in some cases heart and serum) samples were collected and stored in liquid nitrogen, prior to later electrophoretic analysis. Voucher specimens were lodged in the Transvaal Museum in Pretoria (TM), the National Museum in Bloemfontein (NM), the Durban Natural Science Museum (DM), and the Cape Nature Conservation collection at Hartswater (HW). Locality and specimen details are given in the caption to Fig. 1.

Six specimens from Bloemfontein, collected in 1985, were analysed separately from the other specimens. Serum proteins from these specimens were analysed using polyacrylamide gel electrophoresis (PAGE) in the laboratory of the Biology Department, University of Natal (Durban), following procedures in Davis.[16] Other enzymes from liver (and in some cases kidney) homogenates from these six specimens were analysed using starch gel electrophoresis (Connaught starch: 12.5% or 13%), in the laboratory of Dr D. Gordon, Transvaal Museum, following the protocol of Selander *et al.*[46] All the other specimens ($N = 29$), which were collected later in 1987 and 1988, were analysed by starch (Sigma: 12.5%) gel electrophoresis in the laboratories of Dr I.L. Rautenbach, Transvaal Museum and the Biology Department, University of Natal (Durban). A standard individual, from the initial study of six specimens, was included in later analyses to facilitate comparison of electrophoretic results between runs.

A total of 37 presumptive structural loci were examined, of which 28 could be accurately scored in some or all specimens. (The following enzymes could not be reliably scored, owing to poor staining intensity or resolution, and were therefore excluded from this study: adenylate kinase, acid and alkaline phosphatase, octanol dehydrogenase, glucose-6-phosphate dehydrogenase, hexose-6-phosphate dehydrogenase, leucine aminopeptidase, aldolase and glycerol dehydrogenase.) Staining and buffer procedures, which were taken from Davis,[18] Selander *et al.*,[46] Shaw and Prasad,[17] Harris and Hopkinson[18] and Richardson *et al.*[60] are described in detail in Table 1.

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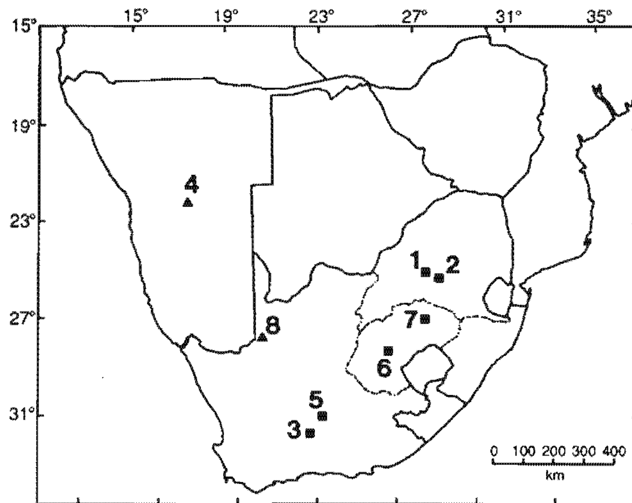

Fig. 1. Map of Southern Africa showing localities from which *C. penicillata* specimens were obtained for electrophoretic analyses. Triangles represent *C. p. bradfieldi*, and squares *C. p. penicillata*. Localities, sample sizes and specimens examined (abbreviations of institutes explained in text) are as follows: (1) Kaal Plaas (Farm), 3 km N Onderstepoort, Pretoria District, Transvaal (N = 3, TM: 39215, 39216, 39265); (2) Rhenosterfontein (Farm), 35 km E Pretoria, Cullinan District, Transvaal (N = 4, TM: 39299, 39406, 39436, 39437); (3) Karroo National Park, Beaufort West, Cape Province (N = 1, TM: 39448); (4) Windhoek, Namibia (N = 4, TM: 39937, 39947 – 39949); (5) Victoria West, Cape Province (N = 5, DM: 768 – 772); (6) Glen Agricultural College, Bloemfontein, Orange Free State (N = 15, TM: 38334, 38335, 38337 – 38339, NM: 6566 – 6568, 6576 – 6580); (7) Erfdeel (Farm), Kroonstad, Orange Free State (N = 2, NM: 6539 – 6540); (8) Riemvasmaak, 47 km NW Kakamas, Cape Province (N = 1, HW: 4784).

In order to establish that polymorphic alleles were not artifacts of electrophoretic conditions in a particular run, repeat runs were performed in the case of variable loci. In scoring polymorphic loci, different electromorphic alleles were given alphabetical designations, the most anodal being *a* and successively cathodal electromorphs being designated as *b, c, d*, etc. From the individual genotypes at each locus (including monomorphic loci in which all individuals were scored as AA), allele frequencies were determined and used to calculate measures of expected mean heterozygosity ($\bar{H}$).[19] As esterase, transferrin and prealbumin loci were scored only for the Bloemfontein sample, heterozygosity estimates for this population were calculated with and without these loci. Chi-squared, with Yates' correction for continuity,[20] and Levene's[21] correction for small sample sizes, was used to test for departures from Hardy–Weinberg equilibrium. Wright's[22] F statistics, for the partitioning of genetic variances within and between populations, were calculated from allele frequencies, as were Nei's[19] distance (D_N) and identity (I), and Roger's[23] distance (D_R) and similarity (S) coefficients between all pairs of localities. Distance coefficients were summarized by means of UPGMA (unweighted pair group method with averages[24]) phenograms.

All analyses of gene frequency data were performed using BIOSYS-1[25] on an IBM compatible personal computer with an 8087 maths coprocessor. Analyses of F statistics and genetic distances and similarities, which require more than one population, were based on 22 loci, as five loci (two esterase loci, transferrin and two prealbumin loci) were scored only in the Bloemfontein sample. F statistics were calculated for all eight localities, and for a subset of five localities having sample sizes of three or greater. This was done to detect possible artifacts due to the presence of very small sample sizes.

Table 1. Proteins and electrophoretic conditions used in analysis of genetic variation in eight populations of *C. penicillata* in Southern Africa. With the exception of FUM, GDH, SORD, GPD, LDH, PGD, PGI, PGM and XDH (which were run for about five hours), all gels were run overnight (18 hours). Apart from serum proteins (ALB, TRF, PAL), which were separated on a vertical polyacrylamide system (PAGE), all samples were run on a horizontal starch gel system.

Protein	Enzyme commission number	Locus	Tissue[1]	Buffer[2]	Source for stain[3]
Alcohol dehydrogenase	1.1.1.1.	ADH	L	5	1
Albumin		ALB	S, L	6, 7	1, 5
Catalase	1.11.1.6	CAT	L	3	2, 4
Creatine kinase	2.7.3.2	CK	L	2	2
Esterase	3.1.1.1.	EST-1,2,3	L, K	6, 8	1
Fumarase	4.2.1.2	FUM-1,2	L	3	2
Glucose dehydrogenase	1.1.1.47	GDH	L	3	3
Glucosephosphate isomerase	5.3.1.9	PGI	L	4	1
Glutamate–oxaloacetate transaminase	2.6.1.1	GOT-1,2	L	2, 3	1
Glycerol-3-phosphate dehydrogenase	1.1.1.8	GPD	L	3	1
Isocitrate dehydrogenase	1.1.1.42	IDH	L	5	1
Lactate dehydrogenase	1.1.1.27	LDH-1,2	L, K	1	1
Malate dehydrogenase	1.1.1.37	MDH-1,2	L	1, 3	1
Malic enzyme	1.1.1.40	ME	L	3	4, 6
Phosphoglucomutase	2.7.5.1	PGM	L	4	1
Phosphogluconate dehydrogenase	1.1.1.44	PGD	L	2, 4	1
Prealbumins		PAL-1,2	S	7	5
Sorbitol dehydrogenase	1.1.1.14	SORD	L	3	2
Superoxide dismutase	1.15.1.1	SOD	L	1, 3	1
Transferrin		TRF	S	7	5
Xanthine dehydrogenase	1.2.1.37	XDH	L	1, 3	1

[1] Tissues: L (liver); K (kidney); S (serum).
[2] Buffers (all from ref. 46, except for 7, from ref. 16): 1) discontinous Tris-citrate; 2) Tris-citrate I; 3) Tris-citrate II; 4) phosphate pH 6.7; 5) phosphate-citrate; 6) lithium hydroxide; 7) Tris-glycine; 8) Tris-HCl.
[3] Sources: 1) ref. 46; 2) ref. 17; 3) recipes used in laboratory of L. Robbins, Southwest Missouri State University; 4) ref. 60; 5) ref. 16; 6) ref. 18.

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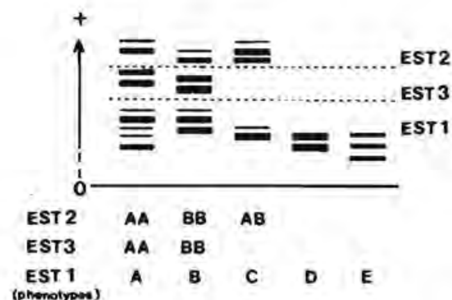

Fig. 2. Variation at three esterase loci in *C. penicillata*. EST-2 and EST-3 could be scored for only six specimens from Glen Agricultural College, Bloemfontein, while EST-1 could be scored for all specimens. The pattern indicated at the left is identical to the esterase pattern described for the raccoon and nomenclature follows these authors.[8]

Results

Electrophoretic analysis of esterases in *C. penicillata* revealed the presence of three independent loci, having a pattern of bands which resembled superficially the pattern described by Dew and Kennedy[8] for raccoons (Fig. 2). The nomenclature of Dew and Kennedy was adopted: EST-2 for the most anodal system, EST-1 for the most cathodal and EST-3 for the intermediate system. There were differences in the interpretation of esterase patterns between the present study and that of Dew and Kennedy. EST-2 was eserine-sensitive in yellow mongooses, but eserine-insensitive in raccoons. Using NASA (napthyl-as-acetate) as a substrate, EST-2 was discriminated in raccoons, whereas EST-3 was discriminated in yellow mongooses. The leading band in the five-banded pattern for EST-1 in yellow mongooses was interpreted as a separate, eserine-sensitive locus (EST-4) by Dew and Kennedy, but did not appear to be eserine-sensitive in the present study. The most anodal esterase band was interpreted as belonging to EST-1 by Dew and Kennedy, but this could not be established in the present study.

While all three esterase loci were monomorphic in raccoons, they were clearly polymorphic in *C. penicillata* (Fig. 2). EST-2 was interpreted as being monomeric as, when only the intense bands within this system were considered, the heterozygote pattern was two-banded. Two distinct, two-banded homozygotes, but no heterozygotes, were detected in EST-3. The genetic basis of variation at EST-1 could not be ascertained because of the complex nature of the different phenotypes observed. The most common phenotype (phenotype *A*: Fig. 2), had a five-banded pattern and occurred in 17 out of 30 scored individuals. Phenotypes *B* and *C* occurred in seven and four individuals, respectively, and phenotypes *D* and *E* were each observed in single individuals.

While EST-1 was scorable, and gave congruent results, using both Connaught and Sigma starch gels, EST-2 and EST-3 could be scored only on Connaught gels, owing to poor resolution of these loci when esterases were run on Sigma gels (resolution on Sigma gels was not appreciably improved by varying buffer pH, tissue type, gel strength or run conditions). As most samples were run on Sigma gels (see Material and methods), reliable data on EST-2 and EST-3 were obtained only for six specimens from Bloemfontein (which were run on Connaught starch gels).

Of the 28 loci examined, 15 were monomorphic with all samples fixed for the same allele. Allele frequencies of the 12 genetically interpretable, polymorphic loci are given in Table 2. In *C. penicillata* mean heterozygosity ($\bar{H}$) varied from 0.0% to 6.5% with a mean of 3.4% (Table 2). Allozyme variation at all loci, and in all populations, conformed to Hardy–Weinberg expectations (Table 3). The pattern of geographic distribution of electromorphs at variable loci (Tables 2 and 3) indicates a high proportion (82%) of 'private' alleles (polymorphic alleles found only in single populations). Only the GOT-1^a and GPD^a electromorphs (as well as the *A*, *B* and *C* EST-1 phenotypes) were detected in more than one population.

Individual polymorphic loci in *C. penicillata* tended to show both excesses (CAT, GOT-1, GPD) and deficiencies (ADH, GDH, PGD) of heterozygotes, as demonstrated by high negative and high positive F_{IS} values respectively, but the mean, low

Table 2. Allele frequencies, percentage polymorphism, mean heterozygosity ($\bar{H}$) and standard errors (s.e.) of $\bar{H}$ for 12 polymorphic loci in *C. penicillata* from Southern Africa. Locality codes are explained in Fig. 1.

		Localities							
		1	2	3	4	5	6	7	8
Locus	Allele	$N = 3$	$N = 4$	$N = 1$	$N = 4$	$N = 5$	$N = 11$	$N = 2$	$N = 1$
ADH	A	0.00	0.25	0.00	0.00	0.00	0.00	0.00	0.00
	B	1.00	0.50	1.00	1.00	1.00	1.00	1.00	1.00
	C	0.00	0.25	0.00	0.00	0.00	0.00	0.00	0.00
CAT	A	0.00	0.25	0.00	0.00	0.00	0.00	0.00	0.00
	B	0.00	0.00	0.00	0.38	0.00	0.00	0.00	0.00
	C	1.00	0.75	1.00	0.62	1.00	1.00	1.00	1.00
EST-2	A	—	—	—	—	—	0.33	—	—
	B	—	—	—	—	—	0.67	—	—
EST-3	A	—	—	—	—	—	0.17	—	—
	B	—	—	—	—	—	0.83	—	—
GDH	A	0.00	0.00	0.00	0.00	0.00	0.00	0.50	0.00
	B	1.00	1.00	1.00	1.00	1.00	1.00	0.50	1.00
GOT-1	A	0.00	0.00	0.50	0.00	0.00	0.00	0.25	0.00
	B	1.00	1.00	0.50	1.00	1.00	1.00	0.75	1.00
GOT-2	A	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1.00
	B	1.00	1.00	1.00	1.00	1.00	1.00	1.00	0.00
GPD	A	0.33	0.00	0.00	0.00	0.00	0.03	0.00	0.00
	B	0.67	1.00	1.00	1.00	1.00	0.97	1.00	1.00
PAL-1	A	—	—	—	—	—	0.50	—	—
	B	—	—	—	—	—	0.20	—	—
	C	—	—	—	—	—	0.30	—	—
PGD	A	0.00	0.00	0.00	0.12	0.00	0.00	0.00	0.00
	B	0.00	0.00	0.00	0.25	0.00	0.00	0.00	0.00
	C	1.00	1.00	1.00	0.63	1.00	1.00	1.00	1.00
PGM	A	0.00	0.00	1.00	0.00	0.00	0.00	0.00	0.00
	B	1.00	1.00	0.00	1.00	1.00	1.00	1.00	1.00
TRF	A	—	—	—	—	—	0.90	—	—
	B	—	—	—	—	—	0.10	—	—
Mean % *P*		4.5	9.1	4.5	9.1	0.0	18.5	9.1	0.0
$\bar{H}$		0.024	0.052	0.030	0.052	0.000	0.065	0.053	0.000
s.e.		0.024	0.037	0.030	0.036	0.000	0.032	0.037	0.000

Table 3. Table showing χ^2 values from tests for conformity of genotypic proportions to Hardy–Weinberg equilibrium in 10 polymorphic loci of *C. penicillata* from Southern Africa. All χ^2 values were non-significant ($P > 0.05$). Locality codes are explained in Fig. 1.

	Localities							
Locus	1	2	3	4	5	6	7	8
ADH	—	2.36	—	—	—	—	—	
CAT	—	0.00	—	0.06	—	—	—	—
EST-3	—	—	—	—	—	0.06	—	—
EST-2	—	—	—	—	—	2.84	—	—
GDH	—	—	—	—	—	—	0.69	—
GOT-1	—	—	0.02	—	—	—	0.00	—
GPD	0.00	—	—	—	—	0.00	—	—
PAL-1	—	—	—	—	—	0.86	—	—
PGD	—	—	—	0.21	—	—	—	—
TRF	—	—	—	—	—	0.00	—	—

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Table 4. Results of the analysis of Wright's F statistics for each variable locus for *C. penicillata* from eight localities in Southern Africa. Double asterisks indicate significance of χ^2 tests of F_{ST} at the 1% level. χ^2 values were determined from the equation: $\chi^2 = 2NF_{ST}(k-1)$, with $(k-1)(s-1)$ degrees of freedom, where N is the total population size, k is the number of alleles at a locus, and s is the number of populations.

Locus	F_{IS}	F_{IT}	F_{ST}	χ^2
ADH	1.000	1.000	0.344	39.4**
CAT	-0.481	-0.063	0.282	32.4**
GDH	1.000	1.000	0.467	27.3**
GOT-1	-0.714	-0.103	0.356	25.2**
GOT-2	—	1.000	1.000	70.0**
GPD	-0.441	-0.048	0.273	18.9**
PGD	0.529	0.654	0.265	30.2**
PGM	—	1.000	1.000	58.0**
Mean	0.039	0.601	0.585	301.5**

positive F_{IS} value (Table 4: 0.039) indicates an overall balance between the numbers of heterozygotes and homozygotes in individual populations. The high positive mean F_{IT} value (Table 4: 0.601), however, indicates a greater number of homozygotes than would be expected when data are pooled for all populations. F_{ST} values obtained for both five- and eight-population analyses indicate that between 27.1% (five-population analysis: results not shown) and 58.5% (eight-population analysis: Table 4) of genetic variability in *C. penicillata* is accounted for by inter-group differences. The much higher F_{ST} figure obtained for the eight-population analysis results from the 'fixation' of private alleles in single-individual populations at the GOT-2 and PGM loci (Tables 2 and 4). There was significant heterogeneity at all variable loci, as indicated by significance ($P < 0.01$) of chi-squared values calculated for F_{ST} statistics (Table 4).

Yellow mongoose populations differed very little from one another genetically: I varied from 0.900 to 1.00, and S varied from 0.922 to 0.998 (Table 5). No clear genetic groups were distinguished from UPGMA phenograms of D_N and D_R (Fig. 3), although the single-individual populations of Karroo National Park and Riemvasmaak were somewhat different from the other populations owing to the apparent 'fixation' of polymorphic alleles in these populations, as discussed above.

There did not appear to be any correlation between geographic and genetic (D_N) distances, as evidenced by the geographic plot of genetic distances between localities in Fig. 4. Although the Windhoek population was geographically distant from the South African populations, D_N values between Windhoek and South African populations (0.008 – 0.066) were similar in magnitude to values between localities which were very close together (such as Kaal Plaas and Rhenosterfontein in the Transvaal: D_N = 0.012: Fig. 4). Since the Windhoek and Riemvasmaak populations represent the subspecies *C. penicillata bradfieldi*, and all the other populations represent *C. p. penicillata*, the present data indicate an absence of electrophoretically detectable genetic differences between morphologically defined subspecies of *C. pencillata*.

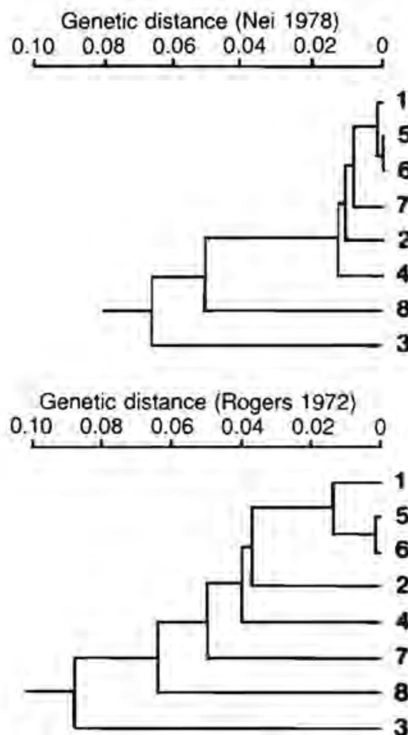

Fig. 3. UPGMA phenograms of Nei's[19] (above) and Roger's[23] (below) genetic distances among eight populations of *C. penicillata* in Southern Africa. Co-phenetic correlation coefficients 0.945 and 0.888, respectively.

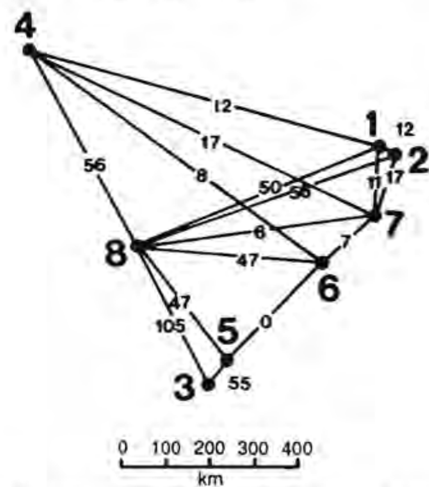

Fig. 4. Diagram showing inter-locality genetic distances in *C. penicillata* in Southern Africa. Locality numbers and locations shown in Fig. 1. Inter-population distances represent Nei's[19] D_N × 1000.

Discussion

Intrapopulation genetic variability

Previous studies have reported relatively low heterozygosities ($\bar{H}$) for carnivores,[8,9,26,47–52] and for large mammals generally[27] (and references therein), although an absence of empirical correlation between body size and genetic variability in mammals was demonstrated by Wooten and Smith.[27] The mean $\bar{H}$ value of 0.034 for *C. penicillata* (calculated from Table 2) is only slightly lower than the mammalian mean of 0.039 (for 128 species) given by Wooten and Smith,[27] and somewhat higher than mean $\bar{H}$ values obtained for other carnivores such as coyotes (0.009)[9] and raccoons (0.028).[8] If the 'fast' esterase and serum loci are excluded, the mean $\bar{H}$ for *C. penicillata* is somewhat lower (0.026: data not shown). On the other hand, if these fast loci had been scored for all specimens (instead of just six specimens: see Material and methods), then a somewhat higher mean $\bar{H}$ value would have been obtained for *C. penicillata*.

Interpopulation variability: F statistics

As a result of the high proportion of loci having 'private alleles' (Table 2), the mean F_{ST} for *Cynictis* populations was considerably higher (0.271 and 0.585 for five- and eight-population analyses, respectively: Table 4 and results) than recorded

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for populations of humans (0.148),[28] house mice (0.12),[29] Scandinavian moose (0.096),[53] coyotes (0.080),[9] prairie dogs (0.103 and 0.068 for *Cynomys ludovicianus*[30] and *C. mexicanus*[31] respectively), and deer mice (0.075),[54] although not as high as values recorded for pocket gophers (0.412 and 0.752 for *Thomomys bottae*[32] and *T. umbrinus*[55] respectively).

This result is unusual as population genetical theory predicts that, in large, continuously distributed and potentially mobile animals occupying fairly homogeneous ranges (as applies to *C. penicillata* in Southern Africa), gene flow would be sufficient to prevent spatial heterogeneity in gene frequencies (i.e. high F_{ST}). The unusual F_{ST} results for *Cynictis* merit further discussion on the biological reality of these data, given the small mean population sample sizes of 5.0 and 3.6 for the five- and eight-population analyses, respectively.

It might be argued that the high proportion of variable loci having 'private alleles' is an artifact of sampling whereby, on average, polymorphic alleles occur at a frequency of only one individual (and therefore a single population) in the entire sample, resulting in an apparent, high mean F_{ST} value for all variable loci. From a probabilistic point of view, though, it seems unlikely that chance effects should result in polymorphic alleles in six out of the eight variable loci occurring only in restricted populations, and that none of these polymorphic alleles should be expressed in the largest population from Bloemfontein ($N = 9$ or 15 depending on the locus sampled). Furthermore, in a simulation model comparing 'true' F_{ST} with estimated values based on different methods and sample sizes, van den Bussche *et al.*[56] demonstrated a general correlation between true and estimated F_{ST} with Nei's[33] method (which was used in this study), at sample sizes varying from five to 50. Finally, in a study of Mexican prairie dogs,[31] involving a sample size (N = 29) similar to that in the present study, the mean F_{ST} was relatively low (0.068) and not relatively high as in our study. Different lines of evidence therefore support the validity of the present gene frequency data for *Cynictis*, although it is important that preliminary hypotheses based on these data are tested in the light of further data. It is also possible that the predominance of slowly evolving loci in this study has contributed to high F_{ST}, and that the picture may have been somewhat different if data on faster evolving loci such as esterases and transferrin had been obtained for all the populations.

Social organization and life-history attributes may have profound effects on the distribution of genotypes among populations (and therefore on mean F_{ST} values). Spatial heterogeneity of alleles may be promoted by small, inbred breeding units (such as 'coteries' of prairie dogs[30]), or by fossorial or sedentary habits and restricted, fragmented distributions (e.g. pocket gophers, whose burrows are limited to isolated patches of friable soil[55]). The yellow mongoose is fossorial, colonial and territorial.[34] According to Walker,[35] the species seldom moves more than 1 km from its warren, and Stuart[36] mentioned that tagged animals were observed feeding up to 1.3 km from the burrows.

Zumpt[61] found that home ranges of yellow mongooses varied from 600 to 3000 metres in radius from a colony, and that juveniles remain in a colony until the end of their first year, when the new litters are born. The occurrence of GPD^a polymorphic alleles in two individuals collected from the same colony, one of these a subadult of about one to two years of age, is consistent with the idea that juveniles remain in their natal colony for at least a year.

Further indirect evidence for low vagility in the yellow mongoose comes from the careful documentation of the occurrence and incidence of rabies outbreaks where *Cynictis* has been shown to be the carrier. Snyman[37] showed that rabies outbreaks caused by *Cynictis* were always confined to very restricted foci, and successive outbreaks could often be traced back to the same spot on the same farm up to 11 years later. Low vagility in *Cynictis* — whether this is due to social structure or possibly to the fragmented distribution of suitable soils for burrowing — may therefore be an important factor in explaining the extraordinarily high level of spatial heterogeneity in gene frequencies observed in this species.

Current behavioural studies on the yellow mongoose (A. Rasa, personal communication) indicate that: (1) the species is spatially and socially stable over periods of years (i.e. vagility is low); (2) the dominant male (whose tenure may encompass several years) conducts the majority of matings; (3) young remain with their parents in the colony and act as 'helpers' over several years; (4) matings occur between neighbouring colonies. These factors promote genetic homogeneity and inbreeding in a restricted area, and consequently genetic heterogeneity over a wider geographic area. In this regard the behavioural results support the genetic data obtained in this study.

The relative strengths of gene flow and gene drift in determining allele frequencies can be estimated by the number of migrating individuals per generation (N_m). Where N_m is greater than one, gene flow is sufficient to prevent differentiation of populations due to drift.[38,54,39] N_m can be calculated from F_{ST}, using the formula

$$N_m = {}^1/_4(1/F_{ST} - 1)$$

or from the conditional average frequency of 'private alleles' $[p(1)]$, by solving for the equation

$$\log_{10}[p(1)] = -0.49 \times \log_{10}(N_m) - 0.95$$

and correcting for the fact that this equation is based on a sample of 10 individuals per population by multiplying by 10 and dividing by the mean sample size for the present study.[39] According to Slatkin and Barton,[39] both methods work equally well under ideal conditions but estimates obtained from F_{ST} are more robust under realistic conditions.

Calculated from F_{ST}, N_m in the yellow mongoose was 0.18 and 0.67 for the eight- and five-population analyses, respectively. N_m, calculated from 'private alleles', was 0.06. Fewer than one individual per generation is therefore exchanged between local populations, suggesting that gene drift, and not gene flow, is the major factor determining the genetic structure of populations of yellow mongooses. However, these analyses were based, on the whole, on very widespread geographic localities, and these conclusions need to be verified on the basis of samples taken over a more restricted geographic area.

Ryman *et al.*[53] demonstrated significant allele frequency differences among local populations in the moose, a large and potentially highly mobile species, and they concluded that 'Even in large mammals local differentiation seems to have been documented in every case for which it has been tested.' The data for the yellow mongoose support the idea that spatial heterogeneity in gene frequencies may be more widespread in large mammals than was previously thought.

In species in which gene flow plays a dominant role in moulding the genetic structure of populations, one would expect a correlation between genetic and geographic distances indicative of an 'isolation by distance' model. The lack of association of genetic and geographic distances in the yellow mongoose (Fig. 4) is consistent with the high F_{ST} value obtained for the species and indicates a model of drift in which gene flow plays a minor role.

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Interpopulation variability: genetic distances

Multivariate craniometric analysis of geographic samples in *C. penicillata*[63] resulted in the proposal of four parapatric subspecies, separated by transition zones of variable steepness. The hypothesis was advanced that some measure of genetic divergence had occurred between these craniometrically defined subspecies. One of the primary aims of the present study was to test this hypothesis, using electrophoretically detectable genetic characters. The present study includes two subspecies, *C. p. bradfieldi* (Windhoek and Riemvasmaak localities) and *C. p. penicillata* (remaining localities).

Genetic identities (I) between populations of *C. penicillata* varied from 0.900 to 1.000 (Table 5) with a mean of 0.968, which is higher than the mean I of 0.96 for 1680 conspecific population pairs, given by Thorpe.[3] Yellow mongoose populations are therefore homogeneous genetically. Phenetic analysis of genetic distances does not indicate any clustering on the basis of morphometrically defined subspecific affiliation (Fig. 3). Disagreement between biochemical and morphological data at the subspecies level appears to be more common[30,40,41,54,57,58] than does agreement.[42,43,55]

Hafner *et al.*[55] have criticized the common assumption among systematists that taxonomic and genetic distances are generally correlated.[3,44,59] They point out that cladogenetic speciation events (often occurring rapidly, e.g. where certain chromosomal changes are implicated) need have no effect on genetic distance *per se*, and that both temporal as well as spatial aspects of gene flow should be considered, as genic mutations are accumulated in a clockwise fashion over time. Thus, high genetic similarities between populations (as in the present study) may represent either a high degree of gene exchange between present populations, or they may represent recency of divergence of isolated or partially isolated populations. A further possibility, which may apply in the case of *C. penicillata*, is that extinction of new mutations in relatively large, outcrossed populations (the mean F_{IS} of 0.039 suggests an overall balance in the proportions of heterozygotes and homozygotes) prevents long-term genotypic divergence between populations (assuming absence of selection), even though spatial heterogeneity of gene frequencies may be pronounced, as discussed above for *Cynictis*.

Table 5 Value of (*A*) Nei's standard genetic distance (below the diagonal) and identity (above the diagonal) and (*B*) Roger's genetic distance (below the diagonal) and similarity (above the diagonal) between eight populations of *C. penicillata* in Southern Africa, based on 22 loci. Locality codes are explained in Fig. 1.

	Localities							
	1	2	3	4	5	6	7	8
A								
1	—	0.989	0.942	0.989	0.997	0.998	0.989	0.951
2	0.012	—	0.936	0.987	0.992	0.992	0.983	0.945
3	0.059	0.066	—	0.936	0.946	0.946	0.949	0.900
4	0.012	0.013	0.066	—	0.992	0.992	0.983	0.945
5	0.003	0.008	0.055	0.008	—	1.000	0.993	0.955
6	0.002	0.008	0.055	0.008	0.000	—	0.993	0.954
7	0.011	0.017	0.053	0.017	0.007	0.007	—	0.946
8	0.050	0.056	0.105	0.056	0.047	0.047	0.056	—
B								
1	—	0.954	0.917	0.953	0.985	0.986	0.951	0.939
2	0.046	—	0.901	0.950	0.969	0.967	0.935	0.923
3	0.083	0.099	—	0.900	0.932	0.930	0.920	0.886
4	0.047	0.050	0.100	—	0.968	0.966	0.934	0.922
5	0.015	0.031	0.068	0.032	—	0.998	0.966	0.955
6	0.014	0.033	0.070	0.034	0.002	—	0.964	0.953
7	0.049	0.065	0.080	0.066	0.034	0.036	—	0.920
8	0.061	0.077	0.114	0.078	0.045	0.047	0.080	—

Four hypotheses may be put forward to explain the discordance between morphological and genetic data in *Cynictis*: 1) morphometric variation may be non-genetic and environmentally induced, as shown by Smith and Patton[43] for pocket gophers (where diet — crops versus natural habitat — determined skull size; this seems unlikely in a carnivorous species such as *Cynictis*); 2) allopatric differentiation in skull size may have occurred recently; 3) morphometric transition zones (i.e. clines) separating phena may have arisen parapatrically due to a selection gradient acting on genes affecting skull size but not on neutral enzyme loci; 4) morphometric (craniometric) subspecies can be distinguished genetically, but the level of resolution at the 22 loci employed in the present study was insufficient to detect existing genetic differences (possibly the use of 'fast' enzymes such as esterases, or the technique of mitochondrial DNA, may yet uncover genetic markers between subspecies and consequently help to determine the extent of gene flow in the transition zone between subspecies).

According to Chesser,[30] the combination of pronounced spatial heterogeneity of gene frequencies and lack of association of genetic and geographic distances (conditions found in yellow mongoose populations: see above) are in agreement with the expectations of a model of differentiation by founder effect (i.e. allopatric speciation[45] or Type 1b allopatric speciation of Bush[13]). On the other hand, computer simulation studies have shown that high F_{ST} (i.e. reduced gene flow) promotes the formation of stepped clines across selection gradients resulting in parapatric differentiation and speciation.[14] This process is accelerated when partial barriers correspond with existing clines. Such a scenario may help to explain why, in the yellow mongoose, morphometric clines are steep (i.e. stepped) when associated with potential barriers such as the Orange river and more gradual when they correspond with an important ecotone marking the limits of Kalahari sands.[63] Furthermore, low vagility, as suggested for the yellow mongoose (see previous discussion), is predictive of parapatric speciation according to Bush.[13]

While the genetic data do not unequivocally support an allopatric rather than a parapatric mode of differentiation, there seems to have been ample opportunity for the allopatric origin of subspecies of *C. penicillata* during the late Pleistocene.[63] Furthermore, a phylogenetic analysis of predicted range expansion in the species suggested that at least the major, north–south transition zone (separating the two phena sampled in the present study) is allopatric in origin. An allopatric mode of morphometric differentiation is therefore taken as being the most parsimonious explanation of the data on *C. penicillata*.

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