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Geographical Distribution of Electrophoretically Detected Protein Variation in Australian Commercial Fishes. I. Jack Mackerel, *Trachurus declivis* Jenyns

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Abstract

Eight polymorphic loci were detected in a survey for electrophoretically detectable protein variation, carried out using liver samples from the Australian jack mackerel, *T. declivis*. The distribution of gene and genotype frequencies in sample sets from different areas shows that distinct subpopulations of the species occur in Western Australia and in New Zealand and that two or more geographically overlapping but genetically distinct subpopulations occur in the waters around south-eastern Australia.

Introduction

The declaration of the 200-mile Australian fisheries zone makes Australia responsible for the proper management of the living resources within this area and, as a result, there is a clear need for studies on the distribution, population structure and basic biology of the exploitable fish species of the zone (Anon. 1952). To this end, research aimed at identifying, by genetic means, the subpopulation structure of several species has been undertaken and the results are reported in this and following papers.

A 'subpopulation' may be defined as a 'self-sustaining genetic unit of population' (Anon. 1976) or as 'a reproductive community of individuals who share a common gene pool' (Dobzhansky 1950). For convenience in this series of papers, the exploitable group of fish found in a particular area will be termed a 'stock' (Anon. 1976). Thus, 'stock' refers to the fish available to a particular fishery, whereas 'subpopulation' describes a biological unit of population. Clearly, different stocks may be based on the same subpopulation, on different subpopulations, or may include members of two or more subpopulations, and different management regimes would be needed in each case.

Genetic techniques can be used to identify the type of structuring present in a population and this has been done in the past for a number of species (e.g. de Ligny 1968; Jamieson 1970; Fujino 1976). Two distinct sets of information are present in genetic data. Firstly, the relative frequencies of alleles can be determined and these may or may not change significantly between sample sets or stocks, giving information about the relationship between the groups sampled when interpreted in the light of other information about the species. Secondly, the distribution of phenotypes within a sample set can be compared with that predicted for a randomly mating population and the result interpreted in terms of the genetic structure of the sampled groups. In the past, most studies using genetic techniques to identify subpopulations in stocks of fish have concentrated on the information present in the allele-frequency data and have largely ignored the implications of abnormal phenotype distributions within sample sets in the interpretation of gene-frequency data.

The various species of horse mackerel, scad and jack mackerel of the genus *Trachurus* found around the world constitute a significant resource, with about $1 \cdot 6 \times 10^6$ t being taken each year. In Australia, the catch of *Trachurus declivis* Jenyns, the Australian species, is small but the potential for a larger catch is present (Maxwell 1979; Webb and Grant 1979) and so it was chosen as one of the species to be studied in this program. Using electrophoretic techniques to find inherited variation in specific enzymes and then by interpreting the distribution of this variation in a series of sample sets taken from the same and different areas, an attempt was made to identify the number and distribution of subpopulations of the species in the various stocks around southern Australia.

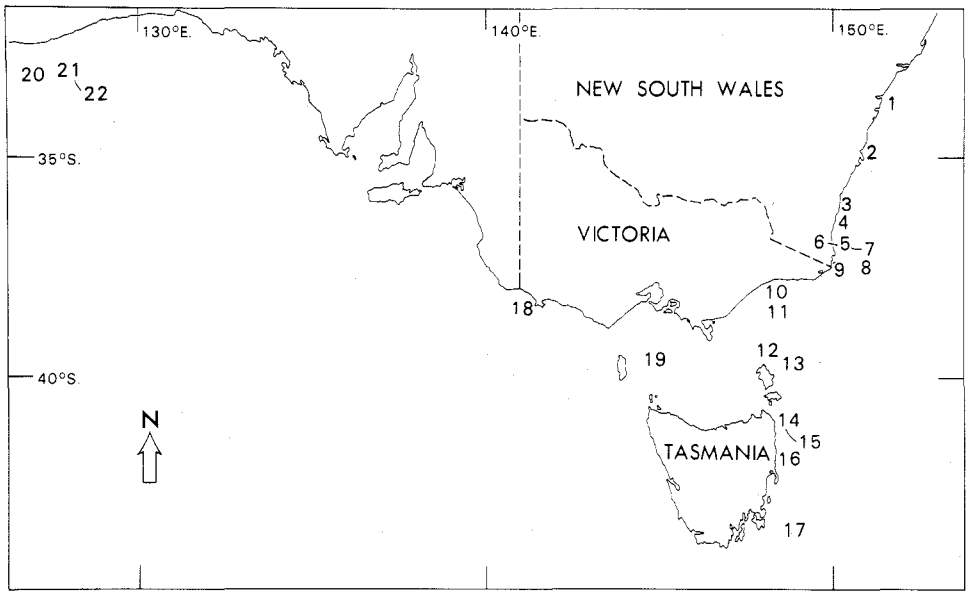

Fig. 1. Distribution of sampling locations around the Australian coast. Numbers identify the sample sets in Table 2.

Methods

Twenty-two sample sets were collected from areas around southern Australia (Fig. 1). The intention was to take replicate sample sets from each area on at least two occasions; however, this proved impossible in Western Australia, which was sampled on only one occasion. Each sample consisted of a piece of liver taken from a freshly caught mackerel and snap frozen in liquid nitrogen. The length, sex and breeding condition of each specimen were noted. When the samples arrived in Canberra, they were taken out of the liquid nitrogen and stored at -20°C until analysis. The sampling locations, dates and sample sizes are given in Table 1 and Fig. 1.

A survey for electrophoretically detectable protein variation was carried out on cellulose acetate medium (Cellogel: Chemitron, Milan) using the methods described by Richardson (1983). The running conditions used for each of the polymorphisms found are given in Table 2. All the available material was then typed for the polymorphic loci found.

Statistical analysis of the collected data was carried out using the computer program 'POPGEN' (Rossiter and Richardson, unpublished data), which carries out a nested analysis of the data using up to three run-specified station or individual variables. The program calculates for the lowest level of the hierarchy (1) the allele numbers and frequencies (with confidence limits), (2) the phenotype frequencies and the expected frequencies under Hardy–Weinberg equilibrium expectations, and (3) Smith's *H*-value for each locus. Smith's *H*-statistic (Smith 1970) is an unbiased estimate of half the difference between the expected and observed proportion of heterozygotes. Consequently, it is zero at equilibrium, positive when there are

Table 1. Location and date of sampling, gene frequencies, Smith's *H*-values and sample sizes for each station and stock studied for the enzymes shown

Rare *Gpi* 1, 2 and 5 alleles have been combined for analysis. The frequency of the rare allele 1 can be calculated by difference from the other frequencies. $*P<0\cdot05$; $**P<0\cdot01$; $***P<0\cdot005$; n.s., not significant

Stn No.	Date	Glucosephosphate isomerase Allele frequency 2	3	4	5	$10^3 \times H$	$2N$	Carboxylesterase Allele frequency 2	3	4	$10^3 \times H$	$2N$
A. Central New South Wales												
1	7.xii.76	0·06	0·52	0·42	0·01	−25	156	0·06	0·91	0	18	158
B. Southern New South Wales												
2	11.xi.76	0·02	0·50	0·46	0·02	−48	100	0·04	0·93	0·03	36	100
3	29.viii.76	0·02	0·55	0·43	0	70	60	0·20	0·77	0	−52	60
4	29.viii.76			—						—		
5	29.viii.76	0·03	0·53	0·40	0·04	1	136	0·09	0·89	0	−11	138
6	1.ix.79	0·04	0·57	0·40	0	48*	372	0·15	0·82	0	49*	368
7	15.ix.79	0·04	0·48	0·48	0	−3	114	0·11	0·87	0	−15	102
8	28.viii.76	0·02	0·59	0·40	0	−30	58	0·10	0·90	0	25	60
9	28.viii.76	0·02	0·67	0·29	0·02	−35	140	0·13	0·84	0·02	17	140
Σ		0·03	0·56	0·40	0·01	11	980	0·12	0·85	0·01	20*	968
Homogeneity test of stations 2–9		$\chi^2_{12}=18\cdot6$, n.s.						$\chi^2_6=15\cdot6$*				
C. Eastern Victoria												
10	5.ii.78	0·05	0·45	0·44	0·06	44	82	0·10	0·87	0·03	−15	78
11	27.viii.76	0·04	0·61	0·33	0·01	−47	140	0·14	0·84	0	62*	140
Σ		0·04	0·55	0·37	0·03	2	222	0·13	0·85	0·01	35	218
Homogeneity test of stations 10–11		$\chi^2_2=6\cdot1$*						$\chi^2_1=0\cdot3$, n.s.				
Homogeneity test of stations 1–11		$\chi^2_{18}=31\cdot6$*						$\chi^2_9=19\cdot8$*				
Homogeneity test of stocks A–C		$\chi^2_4=6\cdot9$, n.s.						$\chi^2_2=3\cdot3$, n.s.				
D. North-eastern Tasmania												
12	22.xi.76	0·04	0·58	0·37	0·01	−20	294	0·14	0·83	0·01	14	294
13	27.xi.76	0·03	0·59	0·36	0·03	−16	118	0·09	0·89	0·01	23	120
14	26.viii.76	0·03	0·58	0·38	0	−3	60	0·20	0·78	0	91*	60
15	25.viii.76	0·01	0·55	0·41	0·02	28	140	0·14	0·83	0	15	140
16	12.i.78	0·07	0·58	0·33	0·02	4	304	0·14	0·84	0·01	35*	328
Σ		0·04	0·58	0·37	0·02	−3	916	0·14	0·84	0·01	27*	942
Homogeneity test of stations 12–16		$\chi^2_8=8\cdot2$, n.s.						$\chi^2_4=4\cdot0$, n.s.				
E. South-eastern Tasmania												
17	14.i.78	0·03	0·60	0·37	0·01	−10	222	0·08	0·89	0·01	8	218
Homogeneity test of stations 1–17		$\chi^2_{30}=44\cdot3$*						$\chi^2_{15}=28\cdot5$*				
Homogeneity test of stocks A–E		$\chi^2_{10}=13\cdot5$, n.s.						$\chi^2_4=8\cdot0$, n.s.				
F. Western Bass Strait												
18	10.iii.77	0·08	0·50	0·40	0·03	−88	38	0·13	0·85	0·02	10	68
19	14.i.77	0·06	0·57	0·36	0·02	5	178	0·13	0·86	0·01	15	176
Σ		0·06	0·56	0·37	0·02	−11	216	0·13	0·86	0·01	14	244
Homogeneity test of stations 18–19		$\chi^2_2=0\cdot9$, n.s.						$\chi^2_1=0$, n.s.				
Homogeneity test of stocks A–F		$\chi^2_{10}=13\cdot5$, n.s.						$\chi^2_3=8\cdot0$, n.s.				
G. Southern Western Australia												
20	8.iii.77	0·07	0·53	0·37	0·03	−10	30	0·28	0·69	0	82	36
21	7.iii.77	0·13	0·67	0·13	0·04	68	24	0·23	0·77	0	32	26
22	6.iii.77	0·27	0·44	0·28	0·01	−7	78	0·14	0·86	0	7	78
Σ		0·20	0·50	0·27	0·02	−6	132	0·19	0·80	0	31	140
Homogeneity test of stations 20–22		$\chi^2_4=8\cdot2$, n.s.						$\chi^2_2=3\cdot5$, n.s.				
Homogeneity test of stations 1–22		$\chi^2_{40}=126\cdot2$***						$\chi^2_{20}=35\cdot0$*				
Homogeneity test of stocks F and G		$\chi^2_2=16\cdot0$***						$\chi^2_1=1\cdot7$, n.s.				
Homogeneity test of stocks A–G		$\chi^2_{12}=78\cdot4$***						$\chi^2_6=10\cdot4$, n.s.				

too many homozygotes, and negative when there are too many heterozygotes. Smith's H-value has an advantage over the usual χ^2 test of observed and expected distributions of genotypes in that estimates of H from a series of sample sets may be combined to give an estimate of $\bar{H}$ with much narrower confidence limits (due to the larger sample size) without increasing the Wahlund effect. The program then gives summed or recalculated values for all of the described variables for higher levels in the nested hierarchy, as well as testing for homogeneity of gene numbers between data sets at each level. A test for the non-random association of phenotypes between loci is also carried out.

The sampling stations used in this study were grouped together into geographical areas for analysis before the data were analysed. The areas chosen were selected for biological or management purposes, e.g. State boundaries, extremes of range, port of origin of fishing fleets and the common belief amongst fishermen that fish of several species show differences in behaviour on the northern and southern sides of eastern Bass Strait.

Results

The eight loci that were found to be polymorphic and the number of alleles at each locus are given in Table 2. The alleles are numbered in order of increasing mobility.

Table 2. Summary of the polymorphic loci found, the number of alleles observed and the running conditions used

Staining systems are described in Richardson (1983). TEB, 15 mM Tris, 5 mM EDTA, pH adjusted to 8·2 using boric acid; TEM, 50 mM Tris, 5 mM EDTA, pH adjusted to 7·8 with maleic acid; TM, 100 mM Tris, pH adjusted to 7·8 with maleic acid

Locus	No. of alleles	Running conditions		
		System	Voltage (V)	Time (h)
Glucosephosphate isomerase (*Gpi*; EC 5.3.1.9)	5	TEB	250	1·5
Carboxylesterase (*Est*; EC 3.1.1.1)	4	TEB	200	2·5
Isocitrate dehydrogenase (NADP+) (*Icd*; EC 1.1.1.42)	4	TEM	180	2
Aconitate hydratase (*Acon*: EC 4.2.1.3)	4	TEM	180	2
L-Iditol dehydrogenase (*Sord*; EC 1.1.1.14)	4	TM	250	2
Adenosine deaminase (*Ada*; EC 3.5.4.4)	5	TM	250	2
Peptidase A (*Pep A*; EC 3.4.11?)	4	TEB	250	2
Peptidase B (*Pep B*; EC 3.4.11?)	5	TEB	250	3

Work using two of these polymorphisms (peptidases A and B) was stopped after it was found that it was not possible to differentiate between all the phenotypes. The location of sampling stations, gene frequencies and Smith's H-values are given for the other loci in Tables 1 and 3, as well as the results of the between-station and between-stock homogeneity tests. Not all the stations have been analysed for all loci as the sample material was lost because of a freezer failure.

The phenotypes of the five alleles at the glucosephosphate isomerase locus (Table 1) are distributed in each area as would be expected if the population was in Hardy–Weinberg equilibrium and, with the exception of stock C, the estimates of gene frequency within each area are homogeneous. However, the range of station gene

Table 3. Gene frequencies, Smith's *H*-value and sample size for each station and stock studied for the enzymes listed
*$P<0.05$; **$P<0.01$; ***$P<0.001$; n.s., not significant

Stn No.	Isocitrate dehydrogenase						Aconitate hydratase						L-Iditol dehydrogenase						Adenosine deaminase						
	Allele frequency				$10^3 \times H$	$2N$	Allele frequency				$10^3 \times H$	$2N$	Allele frequency				$10^3 \times H$	$2N$	Allele frequency					$10^3 \times H$	$2N$
	1	2	3	4			1	2	3	4			1	2	3	4			1	2	3	4	5		
A. Southern New South Wales																									
4	0·28	0·12	0·57	0·03	39	134	0·38	0·46	0·15	0·01	119*	128	0·02	0·98	0	0·01	15*	132	0·46	0·46	0·01	0·07	0·01	11	138
5	0·34	0·07	0·54	0·05	−4	138	0·40	0·49	0·09	0·09	94*	126	0·04	0·96	0	0	−2	138	0·40	0·54	0	0·07	0	40	128
6	0·32	0·09	0·53	0·06	30	350	0·54	0·36	0·07	0·04	112*	278	0·03	0·96	0·01	0	10*	354	0·41	0·52	0·01	0·06	0	43*	366
7	0·27	0·08	0·60	0·06	93*	120	0·38	0·49	0·10	0·03	133*	108	0·05	0·93	0·03	0	50*	112	0·33	0·55	0·02	0·11	0	30	114
Σ	0·31	0·09	0·55	0·05	35*	742	0·45	0·43	0·09	0·03	113*	640	0·03	0·96	0·01	0	15*	736	0·40	0·52	0·01	0·07	0	35*	746
Homogeneity test of stations 4–7					$\chi^2_9=6·6$, n.s.						$\chi^2_9=23·1$*						$\chi^2_3=3·7$, n.s.							$\chi^2_9=9·3$, n.s.	
B. Eastern Victoria																									
10	0·37	0·05	0·55	0·03	103*	74	0·40	0·54	0·07	0	28	76	0·02	0·96	0·01	0	−1	84	0·40	0·50	0·02	0·07	0	96*	82
C. **Tasmania**																									
16	0·31	0·05	0·62	0·03	29	308	0·48	0·44	0·07	0·01	65*	208	0·04	0·96	0·01	0·01	5	308	0·51	0·45	0·01	0·03	0	8	308
17	0·27	0·08	0·64	0·01	23	210	0·31	0·62	0·07	0·01	90*	128	0·04	0·96	0	0·01	26*	218	0·46	0·46	0·02	0·06	0	45	220
Σ	0·29	0·06	0·63	0·03	26	518	0·41	0·51	0·07	0·01	75*	336	0·04	0·96	0·01	0·01	14*	526	0·49	0·45	0·02	0·05	0	23	528
Homogeneity test of stations 4–17					$\chi^2_{18}=25·3$, n.s.						$\chi^2_{18}=48·4$*						$\chi^2_6=3·8$, n.s.							$\chi^2_{18}=25·7$, n.s.	
Homogeneity test of stocks A–C					$\chi^2_6=14·6$*						$\chi^2_6=12·8$*						$\chi^2_2=0·08$, n.s.							$\chi^2_6=11·9$, n.s.	
D. Western Bass Strait																									
18	0·36	0·05	0·55	0·05	−6	62	0·47	0·44	0·08	0·02	32	64	0	1·0000		0	0	70	0·43	0·49	0	0·07	0·01	−11	72
Homogeneity test of stocks A–D					$\chi^2_9=15·9$, n.s.						$\chi^2_9=13·2$, n.s.						$\chi^2_3=3·1$, n.s.							$\chi^2_9=12·0$, n.s.	
E. Western Australia																									
20	0·19	0	0·72	0·10	54	32	0·61	0·33	0·06	0	−51	36	0·03	0·95	0·03	0	−1	38	0·38	0·45	0·08	0·10	0	4	40
21	0·25	0·20	0·40	0·15	−50	20	0·36	0·50	0·14	0	121	28	0·12	0·89	0	0	71	26	0·29	0·57	0	0·11	0·04	41	28
22	0·24	0·06	0·66	0·04	116*	70	0·47	0·39	0·14	0	67	74	0·06	0·92	0·01	0·01	22	72	0·37	0·53	0	0·11	0	−11	76
Σ	0·23	0·07	0·63	0·07	73*	122	0·49	0·40	0·12	0	47	136	0·06	0·92	0·02	0·01	25*	136	0·35	0·51	0·02	0·10	0·01	3	144
Homogeneity test of stations 20–22					$\chi^2_6=12·8$*						$\chi^2_4=4·8$, n.s.						$\chi^2_2=0·8$, n.s.							$\chi^2_4=2·0$, n.s.	
Homogeneity test of stocks A–E					$\chi^2_{12}=21·5$*						$\chi^2_{12}=19·8$, n.s.						$\chi^2_4=8·0$, n.s.							$\chi^2_{12}=19·1$, n.s.	

frequencies on the eastern coast (i.e. stocks A–E combined, Table 1) is too wide to have been sampled from a single distribution. There is a general decrease from north to south in the frequency of allele 4 on the eastern coast but this is not great enough to be statistically significant, and the eastern coast heterogeneity is not due to clinal variation in gene frequency but to interstation variation in gene frequencies. The western Bass Strait gene frequencies are similar to those found on the eastern coast and the overall estimated gene frequencies in Western Australia are very significantly different from those found elsewhere.

Table 4. Allele frequencies for aconitate hydratase, calculated for fish from each available year-class on the eastern coast

As the species breeds in the spring and summer, the data have been calculated annually from July each year. $*P < 0{\cdot}05$

Year of birth	Allele frequency 1	2	3	4	$2N$	$10^3 \times H$
1968–1969	0·47	0·41	0·09	0·03	194	131*
1969–1970	0·43	0·43	0·13	0·02	56	108*
1970–1971	0·36	0·52	0·12	0·01	206	106*
1971–1972	0·36	0·57	0·19	0·02	124	87*
1972–1973	0·41	0·50	0·07	0·02	102	106*
1973–1974	0·49	0·42	0·08	0·02	280	77*
1974–1975	0·51	0·41	0·04	0	90	79*

Overall homogeneity $\chi^2_{18} = 24{\cdot}3$, n.s.
Homogeneity of allele 2 $\chi^2_6 = 13{\cdot}6$*

The frequencies of the four carboxylesterase alleles (Table 1) do not differ between the areas studied, however, there is significant between-station variation in gene frequency in area B. As well, the combined data for stocks B and D are out of Hardy–Weinberg equilibrium, in each case due to an excess of homozygotes.

The four isocitrate dehydrogenase ($NADP^+$) alleles (Table 3) are not distributed at random within stations as the observed distributions of phenotypes of many of the samples show an excess of homozygotes over that expected under Hardy–Weinberg equilibrium conditions; as well, the Tasmanian and the Western Australian data differ in gene frequencies from those from other areas.

The four aconitate hydratase alleles (Table 3) show the most extreme case of divergence from Hardy–Weinberg equilibrium of any of the loci studied, with all but one of the eastern coast schools being out of equilibrium in the direction of excess homozygotes. As well, there is between-school and between-area heterogeneity on the eastern coast. The gene frequencies of the Western Australian and western Bass Strait samples are not significantly different from the eastern coast data.

Both L-iditol dehydrogenase (four alleles) and adenosine deaminase (five alleles) show similar patterns, with some schools and areas out of genetic equilibrium because of an excess of homozygotes but with no evidence of significant between-station or between-area changes in gene frequency (Table 3).

The combined eastern coast data were examined for variation in gene frequency due to other parameters. There was no significant difference in gene frequency between the sexes nor was there an increase in H with age (i.e. length). When the year of birth was calculated from the published growth curves for each fish (Webb and Grant 1979),

analysis showed that there were no significant differences between the gene frequencies of the different year-classes except for one aconitate hydratase allele (Table 4) and in no case did the subdivision reduce appreciably the significant H-values. The lengths of fish in adjacent year-classes overlap in all the older age classes and consequently some between-year variation may have been missed because of misclassification.

Table 5. Summary of combined observed and expected genotype numbers from the eastern coast data

Enzyme		No. of homozygous genotypes				No. of heterozygous genotypes					
		11	22	33	44	12	13	14	23	24	34
Glucosephosphate isomerase	Observed	4	428	201	1	61	32	1	572	23	13
	Expected	2	429	195	0	57	39	14	576	23	14
Carboxylesterase	Observed	4	27	1007	4	2	34	0	230	2	9
	Expected	0	20	976	0	5	38	0	279	3	16
Isocitrate dehydrogenase ($NADP^+$)	Observed	80	25	248	12	17	224	6	32	2	21
	Expected	62	4	224	1	31	236	16	59	4	31
Aconitate hydratase	Observed	150	164	24	4	133	16	8	22	2	3
	Expected	99	112	4	0	211	39	9	41	9	2
L-Iditol dehydrogenase	Observed	3	627	2	0	28	1	4	5	3	0
	Expected	1	618	0	0	38	0	0	9	7	0
Adenosine deaminase	Observed	145	185	3	9	261	4	33	4	27	4
	Expected	128	163	0	3	288	8	36	9	40	1

Discussion

The reason for the excess of homozygotes at seven of the eight loci studied must be considered before any between-area variation can be interpreted. The extent of the homozygote excess can be seen in Table 5 where the observed and expected numbers of each phenotype are given. Several explanations of these data need to be considered.

(1) *Errors in typing.* While this material was being typed, parallel studies using the same loci were being carried out on several other species without such abnormal distributions of phenotypes being found. As well, the only loci where the different phenotypes are not easily distinguishable (i.e. peptidases A and B) were discarded from the analysis. Variation due to secondary bands and breakdown bands was occasionally present but differed markedly in relative intensity from sample to sample and was easily distinguishable from the variation assumed to have a genetic basis (by analogy with similar patterns in other species).

(2) *Selection against the heterozygotes.* It can be seen that the deficiency is normally against all classes of heterozygotes (Table 5). This would mean that severe disruptive selection was occurring at seven of the eight loci studied and this is extremely unlikely.

(3) *Null alleles present.* The presence of alleles at a locus that do not lead to any activity for the resulting enzyme will lead to a situation of apparent excess of phenotypic homozygotes. However, such situations are rare and usually associated with non-essential enzymes, e.g. esterases. It is extremely unlikely that five enzymes of intermediary metabolism would all carry null alleles at relatively high frequencies in one species, as these are fatal in the homozygous condition.

(4) *Assortative mating*. This is a process whereby animals deliberately choose mates with a similar phenotype. It seems unlikely that such behaviour could be ascribed to fish differing only in liver enzymes. It is possible that one or more of the loci studied may be linked to loci controlling visible or behavioural characteristics that, in turn, are subjected to assortative mating but it is extremely unlikely that a randomly selected set of genes that show no non-random between-loci association of phenotypes would all be tightly linked to such loci.

(5) *Inbreeding*. The unusual distribution of phenotypes obtained could be due to the fact that only a limited number of matings produced the animals in each school or to mating between close relatives for several generations. The habits of jack mackerel, which include external fertilization and spawning aggregations involving many thousands of animals, make such a result unlikely. As well, such behaviour would lead to similar H-values at all loci and similarly heterogeneous gene frequencies at each loci, and this was not found.

(6) *Presence of two or more genetically distinct groups in the population*. If the sample sets include members of two or more groups and these have markedly different gene frequencies at a locus, then the combined sample will show an apparent excess of homozygotes (i.e. a Wahlund effect) and this seems the likeliest explanation of the data.

The Wahlund effect could be due to gene-frequency differences between the sexes, age-classes or year-classes within the sample sets. However, subdivision of the eastern coast data by these factors does not reduce H and, except for one allele at the aconitate hydratase locus for year-classes (Table 4), there were no significant differences in gene frequency between such groups. Alternatively, the Wahlund effect could be due to the presence of two or more genetically distinct subpopulations in each area. The size of the H-value found (Table 5) at each locus would change with the size of the difference between gene frequencies of the subpopulations present. If the two groups are in Hardy–Weinberg equilibrium and equally represented in the samples, then an H of $0{\cdot}01$ is equivalent to a difference of $0{\cdot}2$ in gene frequency and an H of $0{\cdot}02$ is equivalent to a difference of $0{\cdot}3$.

The heterogeneity then is most likely due to the presence of several widely distributed allopatric subpopulations of the jack mackerel in Australian waters. The lack of detectable non-random associations between phenotypes at different loci make it impossible to define the genetic characteristics of these subpopulations though it is clear that the divergences are due to differences in gene frequencies, not to fixed differences between the groups. When the gene frequencies of animals from the eastern coast that were in reproductive condition (stages 3–6) in spring are compared with the frequencies calculated for animals in reproductive condition in summer (i.e. testing the possibility of temporal, rather than geographical, isolation of the subpopulations), there are no significant differences between the groups at any locus. Thus, as measured in this fashion, temporal isolation does not supply the basis for maintaining the differences in gene frequency that are implicit in the data. As a consequence, the critical aspects in the breeding structure of the Australian jack mackerel population that maintain the observed structuring are still unknown and their clarification must await further research.

Because of the presence of more than one subpopulation within each stock, management of the species in the face of heavy fishing pressure would be a complex matter. It is not possible at present to draw any final conclusions regarding the relationship between the various stocks studied as differences in gene frequency

between the stocks may simply reflect differences in the proportion of each subpopulation present in each sample set. However, the material from Western Australia differs markedly in frequency at two loci from that from other areas. One of these loci is glucosphosphate isomerase in which no Wahlund effect was detected, and it seems likely that this stock belongs to a distinct subpopulation. There is no evidence that the western Bass Strait stock can be differentiated from those east of Bass Strait. There are differences in gene frequencies between stocks on the eastern coast, but the underlying structuring leaves any interpretation of these differences in doubt. The dangers implicit in using gene-frequency data in a simplistic fashion to identify the presence of different subpopulations in a species can be clearly seen in this study.

Gauldie and Smith (1978) carried out a survey for genetic variation on New Zealand jack mackerel and reported the presence of variation in a carboxylesterase and in glucosephosphate isomerase. Though no direct comparison has been made between New Zealand and Australian samples, the gene frequencies at the glucosephosphate isomerase locus in New Zealand were quite different from those found in Australia (Gauldie and Johnston 1980). As well, isocitrate dehydrogenase, which was polymorphic in Australia, was monomorphic in New Zealand and it seems probable that different subpopulations of the species are exploited in the two countries.

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