

Ruditapes philippinarum is a clam native to Japan, with a wide distribution in the Indopacific area. This species is presently nearly cosmopolitan, as it has been imported into many countries for its better productivity in comparison with native clams. Introduction into the Adriatic dates back to 1983, when it was first cultivated in the lagoon of Venice. Breeding plants are now present in several brackish basins of the Northern Adriatic.

In order to increase the productivity of hatchery stocks, it is very important:

- 1) to acquire detailed knowledge of the genetic structure of wild and artificial populations,
- 2) to verify to what extent the genetic architecture of the strains can be modified by natural selection, and
- 3) to test whether the process of adaptation is similar in different lagoons, and in marine areas.

Moreover, the fact that *Ruditapes philippinarum* is undergoing a rapid phenomenon of naturalization means that it is a good model to study the evolution of the genetic structure of populations during the spreading phase. Comparison with native species, which are probably poorer competitors, may throw some light on the relations existing between the genetic architecture of a species and its performance as competitor and colonizer.

Genetic variability was estimated by the electrophoretic analysis of allozymes. Heterozygosity may be a relevant parameter, as it has frequently been proven to be related with morphometric traits and growth. Besides, heterozygote deficiencies appear to be a common feature in many bivalve species, but the explanation of these findings is still rather unclear.

Samples from cultivated stocks were collected in two brackish basins in the delta of the river Po: the lagoons of Caleri (samples C1 and C2), and Scardovari (S1 and S2). Sampling was repeated in April and September 1991. Electrophoresis was carried out according to BORSA and THIRIOT-QUIEVREUX (1990), testing eight enzyme systems for a total of 15 loci, only one of which (*Mdh-2*) did not show any variation. The number of animals scored, number of alleles per locus [NA], observed heterozygosity [Ho] and heterozygote deficiencies [(Ho-He)/He] are reported in the table.

As can be seen in the table, the strains have a rather high level of heterozygosity, similar to that found in previous studies (BORSA and THIRIOT-QUIEVREUX, 1990; MATTOCCIA, 1991). The presence of a good degree of genetic heterogeneity may represent a potential advantage for breeding, as it provides raw material to artificial selection.

Allele frequencies of a number of loci differed in the two basins. In the lagoon of Caleri a difference was found with respect to the estimates obtained from naturalized populations (MATTOCCIA, 1991). These findings suggest the possible involvement of selection and adaptation to local ecological conditions.

LOCUS	NA	N. INDIVIDUALS				Ho x 1000				(Ho-He)/He x 100			
		S1	C1	C2	S2	S1	C1	C2	S2	S1	C1	C2	S2
<i>Aat-1</i>	3	100	87	45	80	230	207	156	275	-8	-8	-39	-1
<i>Aat-2</i>	4	73	86	33	55	370	233	182	273	-3	-17	-53	-16
<i>Pgm-1</i>	3	93	88	56	94	419	432	339	468	-23	-17	-37	-16
<i>Pgm-2</i>	3	/	71	/	59	/	169	/	254	/	-13	/	-41
<i>Pqi-1</i>	6	100	89	60	98	570	742	650	653	-9	+7	-10	+6
<i>Pqi-2</i>	3	100	87	/	20	80	207	/	100	-9	+9	/	-55
<i>Glo</i>	3	89	84	61	98	270	440	262	367	+12	-2	-13	+7
<i>Idh-1</i>	4	89	92	45	98	326	446	289	367	-11	0	-26	-12
<i>Idh-2</i>	3	94	93	--	78	96	237	/	141	-31	-6	/	-33
<i>Lap-1</i>	4	92	86	61	96	359	267	180	281	-28	-25	-50	-23
<i>Lap-2</i>	3	--	67	--	76	/	388	/	553	/	-25	/	+39
<i>Mdh-1</i>	3	80	71	61	100	75	56	66	10	+7	+4	+3	0
<i>Mdh-2</i>	1	80	71	61	100	0	0	0	0	/	/	/	/
<i>Sod-1</i>	4	--	52	56	100	/	135	55	70	/	+7	+4	+3
<i>Sod-2</i>	3	80	86	55	100	137	209	291	150	-21	-10	-3	-6

Our data are in accordance with the common finding of a heterozygote deficiency. As the table shows, the defect is the most frequent condition (35 out of 49 cases), although only six revealed a significant difference. Several hypotheses, reviewed by GAFFNEY *et al.* (1990), have been suggested to explain this phenomenon but, at the present state of research, it is not possible to make a choice. It is probably better to conclude that "The causes of heterozygote-deficiency.....remain obscure" (ZOUROS *et al.*, 1988).

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