GENE ANALYSIS IN THE MEDAKA *(ORYZIAS LATIPES)*

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We have analyzed the genetics of the mutant strains of the medaka, *Oryzias latipes*. Up to 1980, 54 mutant genes were found in wild populations and in domesticated strains. All are spontaneous mutants : 51 recessives, 1 dominant and 2 incomplete dominants. Mutant individual fish were very rare in wild populations (8 cases) and in domesticated strains (3 cases). The remaining mutants (43 genes) were found among the heterozygous offspring resulting from breeding tests. The mutants which are controlled by a single gene were observed in 50 cases, whereas there were only 2 with polymeric characteristics. Some mutants were difficult to be cultured suggesting that the trait may lethal sometimes.

As shown in Table 1, 4 mutant genes were established from the orange-red stock at Yatomi near Nagoya, while 3 other mutant genes were introduced into our stocks from other sources. About 60 mutant genes are now being maintained in our laboratory.

Some interesting characters are polygenic in nature. One is a character which reciprocally changes the body color of wild type to orange-red type. The character in which papillar processes appear on the anterior parts of anal fin rays of adult male is also controlled by more than one gene. Similarly, the absence of pectoral fins may be polygenic types, or may be under the control of a single gene *(pl)*.

Polymorphism in wild population is very rare (2 cases). The *dx-1* gene has approximately 7% gene frequency in populations of medaka from paddy fields in Sugashima, Toba, Mie Prefecture. The *sm* gene was frequently found in fish from paddy fields in Nagakute near Nagoya. However, the gene frequency for this trait was not determined because the population of fish was variable.

Some mutant genes have a pleiotropic effect on the expression of certain characters. For example, the *i* (albino) gene affects not only melanin formation (amelanotic melanophores), but also the state of xanthophores (dilution of carotenoid content) and leucophores (increased number and development). The *dm* (dispersed melanophore) gene acts on both melanophore and leucophore dispersal in the adult fish. The *mm* (mixed melanophore) gene causes the formation of differentiated and undifferentiated (punctate) melanophores and leucophores. The *vc* (variegated chromatophore) gene induces the variegation of melanophores and leucophores.

The interaction of genes in the above mutants may be divided into three types. The first is the independent type in which body color genes are usually not affected by the expression of gene which causes deformities. The state of different chromatophores are controlled by independent genes, except in pleiotropic cases. The *co* and *di* genes control the concentration and the dispersion of xanthophores, respectively. The *cm* gene promotes the concentration of melanophore, while the *dm* gene, the dispersion of melanophores and leucophores. In the double recessive, *di-dm* the dispersal of melanophores, xanthophores and leucophores is promoted, while in the double recessive, *cm-co*, the concentration of xanthophores and melanophores is enhanced. In these cases, the genes act independently on chromatophores. The second is the interactive type. The *dm* mutant gene induces the dispersal of melanophores and leucophores, whereas the *cm* gene, the concentration of melanophores. The double recessive *cm-dm* shows an effect which is intermediate between the *dm* and *cm* types. In the *di* mutant makes xanthophores disperse while in the *co* mutant xanthophores are concentrated. Similarly, the double recessive *co-di* promotes the formation of intermediate

xanthophores (incompletely concentrated) between the *co* and *di* types. The third type is epistasis. The *i* (albino) gene is epistatic for other genes affecting melanin formation. In all gene combinations, the homozygote (*ii*) induces the albino state. The *lf* is epistatic for the coloration of leucophores; i. e., in combination with genes, *lf* causes the absence of leucophores. The *r* gene induces colorless xanthophores in any combination with genes affecting the coloration of xanthophores.

Autosomal linkages have been found in 3 combinations in this fish: (1) *i* (albino) and *ci* (color interference), (2) *co* (concentrated xanthophore) and *dx-2* (diluted xanthophore-2), (3) *di* (dispersed xanthophore) and *wl* (white leucophore). The *r* alleles (colorless xanthophore) and *lf-2* alleles (leucopnore-free-2) are sex-linked.

Table 1. A brief explanation of mutant genes in the medaka.
a: autosomal, s: sex-linked, r: recessive, id: incomplete dominant, d: dominant.

(1) Genes introduced to our laboratory.		
ci	a, r	Decreased number of xanthophores and increased number of developed leucophores. (T. Aida, and T. Takeuchi).
fm	a, r	Decreased number of melanophores throughout life. (K. Takahashi)
fu	a, r	Fused vertebra and short body length (T. Aida).
(2) New genes.		
of	a, r	Delay of fusion of oil drops at early development (Y. Taguchi).
(3) Known genes established from orange-red stocks at Yatomi.		
b	a, r	Colorless melanophores.
B'	a, r	Black variegation with colorless melanophores.
r	s. r	Colorless xanthophores.
wy	a, r	Vertebral column bent wavily.
(4) Genes originally found in our laboratory.		
as	a, r	Some fin rays have partially defective segments.
b^d	a, r	Colorless melanophores at the larval stage which gradually change to black color in adult fish.
b^l	a, r	Dilute black colored melanophores at hatching.
b^p	a, r	Delay of melanin formation at the embryonic stage and dilution of melanin content of the eye.
b^v	a, r	Variegation with dilute black and black melanophores at hatching. ($B > b^v > b^l > b^d > B' > b > b^p$)
cm	a, r	Concentrated melanophores.
co	a, r	Concentrated xanthophores.
co-2	a, r	Phenotype similar to *co* but no linkage with *co*.
Da	a, id	Double anal fins (dorsal fin resembles anal fin).
de	a, r	Decreased melanophore number in adult fish.
df-1	a, r	Deformity of membrane fin at hatching.
df-2	a, r	The same phenotype as *df-1* but no linkage with *df-1*.
di	a, r	Dispersed xanthophores.
dl	a, r	Diluted color in melanophores.
dl-2	a, r	Phenotype similar to *dl* but no linkage with *dl*.
dm	a, r	Dispersed melanophores and leucophores in adult fish.
dm-2	a, r	Phenotype similar to *dm* but no linkage with *dm*.
dx-1	a, r	Diluted orange-red color in xanthophores.
dx-2	a, r	The same phenotype as *dx-1* but no linkage with *dx-1*.
em	a, r	Enlarged median fins (dorsal and anal fins) toward posterior direction.
fl	a, r	Decreased numbers of leucophores at hatching.
fl-2	a, r	Phenotype similar to *fl* but having different alleles.
fs	a, r	Fused interhaemal and interneural spines, and small dorsal and anal fins.
fu-2	a, r	Fused vertebra and short body. All are different alleles.
fu-3	a, r	
fu-4	a, r	
fu-5	a, r	

fu-6 a, r Notochord bent wavily at the embryonic stage; adult with fused vertebra and short body.

gu a, r Reduced deposition of guanine in iridocytes.

i a, r Albino.

i^b a, r Delay of melanin formation at the embryonic stage: $(+ > i^b > i)$

i-3 a, r Albino similar to *i* but no linkage with *i*.

if a, r Partial defect of fin rays.

il-1 a, r Polymeric gene.

il-2 a, r Defect of guanine in iridocytes, except abdomen and the eye.

lf a, r No visible leucophores throughout life.

lf-2 s, r Few leucophores throughout life.

ml-1 a, r Polymeric gene. Increased number of leucophores.

ml-2 a, r About two times leucophores at the embryonic stage.

ml-3 a, r The same phenotype as polymeric genes, *ml-1* and *ml-2*.

mm a, r Mixture of normal and undifferentiated (punctate) melanophores and leucophores.

pc a, r Polycyst in kidney.

pl a, r No pectoral fins throughout life.

r^d s, r Dilute orange-red color of xanthophores. $(R > r^d > r)$

rf a, r Smaller fins.

ro a, r Swims rollingly.

rs a, r Reduced scales.

si a, d Defect of a pair of guanine spots on the brain membrane.

sl a, r Delay of pigmentation of melanophores at the larval stage.

Sm a, r Delay of physiological color changes of melanophores.

Va a, id Black, variegated, with melanophores and leucophores.

vc a, r Variegated chromatophores.

vl-1 a, r Leucophores disappear 2 weeks after hatching, although some leucophores reappear in adult fish.

vl-2 a, r Phenotype similar to *vl-1* but different alleles.

wl a, r White leucophores.