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A study on the mutant pectoral-finless, *pl*, of the medaka, *Oryzias latipes*

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Abstract The pectoral-finless mutant (*pl*) of the medaka has been found and analyzed. The *pl* mutant lacks the pectoral fins. The *pl* gene is autosomal and recessive to the wild-type gene. The *pl* gene does not link to any other genes examined.

Introduction

In 1973, a pair of white medaka (*br*) were found among ordinary orange-red fish of a fish dealer. The gene *r* is linked to the X-chromosome and the white is expressed in the female only (Yamamoto, 1967). Although the male white has been obtained in laboratory through the crossing over that transferred these genes to the Y-chromosome (Yamamoto, 1967), male white medakas have rarely been found in dealers' stocks. Therefore I tried to analyze genetically the male white fish obtained in 1973, and quite unexpectedly a novel mutant which lacks the pectoral fins has been segregated in the progenies derived from the pair of the white. This paper describes the results of the genetic analysis of this mutant *pl* and the morphology of the pectoral-finless medaka. The gene *pl* has been previously listed by Tomita (1992).

Results and Discussion

Morphological observations

The homozygote *pl/pl* has no pectoral fins throughout the life, including the embryonic stage when the fins start to differentiate. Other fins are all normal. The pectoral-finless has no defects in the swimming behavior, presumably due to the functional compensation by other normal fins.

Genetic analyses

The F_1 progenies derived from the male and female of white medakas that were obtained in 1973 were all white and had normal pectoral fins. The F_2 progenies were all white, but were segregated into fish with normal pectoral fins and pectoral-finless fish, in a ratio of 15:1. Crosses between the pectoral-finless white fish thus obtained produced invariably the pectoral-finless white.

When a white female with normal pectoral fins whose genotype was *b/b r/r* [*b*: melanophores colorless. *r*: xanthophores colorless. *b/b r/r* is white, while *b/b R/R* and *b/b R/r* are orange-red (Aida, 1921).] was mated with a pectoral-finless white male (*bplr* that was obtained as an F_2 progeny in the above experiment, the F_1 progenies were all white with normal pectoral fins (*br*, 49 fish). The F_2 progenies were segregated into white fish with normal pectoral fins (*br*: female 30, male 33) and pectoral-finless white (*bplr*: female 14, male 11) in a ratio of 3:1 (χ^2=0.53, p=0.50–0.30) and a sex ratio of 1:1 (χ^2=1.07, p=0.80–0.70). The results indicate that the pectoral finless gene *pl* is autosomal and recessive to the wild type. The genotype of the pectoral-finless white has been deduced to be *b/b pl/pl r/r*. The pectoral-finless white of this genotype was used throughout the following experiments.

I examined the linkage of *pl* to other genes. The genes used are: *Da*: Double anal fins. Anal fin replaces dorsal fin. *df-2*: Deformed membrane fin fold at the larval stage. *dl*: Dilute black melanophores. *gu*: Guanine content in iridocytes decreased. *ha*: Auditory vesicles swollen at the hatching stage. In adults, inner ears deformed. *i*: Albino. *i-3*: Albino but different from the *i* alleles. The *i* and *i-3* alleles are epistatic to the *b* alleles. *ml-3*: Many leucophores (three times in number compared to normal) at the hatching stage. Xanthophores, dilute orange-red. The *Da* gene is an incomplete dominant gene while the others are recessive. All genes are autosomal. In the examination of the F_2 progenies in the following experiments, observations of phenotypes were performed at the time of larval hatching to reduce the investment of labor of rearing fish until adults. Since the gene *r* is not expressed at the time of hatching, the phenotypic expression of the *r* alleles was not recorded. Since the main objective of this study was to examine the linkage of *pl* to other genes, the record of the F_2 segregation ratio

omitting the record of the *r* alleles was sufficient to this purpose.

A brown female with double anal fins and normal pectoral fins (*BDaR*) was mated with a white pectoral-finless male having normal dorsal fin (*bplr*). All of the F_1 progenies were of wild type with respect to the body color and pectoral fins, but their dorsal fin was larger than normal having 7–14 fin rays, due to the heterozygous *Da*. Although the dorsal fin of the *Da* heterozygotes in adults is larger than normal, the fins at the time of the larval hatching is indistinguishable from that of the wild type. For the same reasons stated above for the *r* alleles, the *Da* heterozygotes were included in the wild-type category when the F_2 progeny were examined for phenotype segregation at the time of hatching. Then, the F_2 progenies were segregated into 248 *B* (wild type), 89 *BDa* (black with double anal fins), 89 *Bpl* (pectoral-finless black), 95 *b* (colorless with normal fins), 30 *BDapl* (pectoral-finless black with double anal fins), 35 *bDa* (colorless with double anal fins), 25 *bpl* (pectoral-finless colorless), and 9 *bDapl* (pectoral-finless colorless with double anal fins), in a ratio of 27:9:9:9:3:3:3:1 (χ^2=3.78. p=0.80–0.70). The results indicate that the *pl* gene is not linked to the *b* and *Da* alleles.

A *bplr* female was mated with a brown male having deformed fin fold (*Bdf-2R*). The F_1 progenies were 72 *BR*. The F_2 progenies were 127 *B*, 44 *Bpl*, 34 *Bdf-2*, 38 *b*, 6 *Bdf-2pl*, 12 *bdf-2*, 9 *bpl* and 5 *bdf-2pl*, in a ratio of 27:9:9:9:3:3:3:1 (χ^2=7.48, p=0.50–0.30). The *pl* alleles are not linked to the *df-2* alleles.

A dilute brown female (*BdlR*) was mated with a *bplr* male. The F_1 progenies were 85 *BR*. The F_2 progenies were 149 *B*, 41 *Bdl*, 36 *Bpl*, 75 *b*, 9 *Bdlpl* and 24 *bpl*, in a ratio of 27:9:9:12:3:4 (χ^2= 8.17, p=0.20–0.1). The *pl* alleles are not linked to the *dl* alleles.

A *bplr* female was mated with a transparent orange-red male (*bguR*). The F_1 progenies were 83 *BR*. The F_2 progenies were 94 *b*, 27 *bgu*, 30 *bpl* and 9 *bgupl*, in a ratio of 9:3:3:1 (χ^2=5.43, p= 0.20–0.10). The *gu* alleles are not linked to the *pl* alleles.

A *bplr* female was mated with the orange-red male (*bhaR*) having deformed inner ears. The F_1 progenies were 83 *bR*. The F_2 progenies were 181 *b*, 63 *bpl*, 56 *bha* and 16 *bhapl* in a ratio of 9:3:3:1 (χ^2=2.32, p=0.70–0.50). The *pl* alleles are not linked to the *ha* alleles.

A *bplr* female was mated with an albino male (*biR*). The F_1 progenies were 56 *bR*. The F_2 progenies were 211 *b*, 61 *bpl*, 53 *bi* and 18 *bipl*, in a ratio of 9:3:3:1 (χ^2=4.11, p=0.30–0.20). The *pl* alleles are not linked to *i* alleles.

A *bplr* female was mated with an albino male (*Bi-3R*). The F_1 progenies were 62 *BR*. The F_2 progenies were 109 *B*, 42 *b*, 37 *Bpl*, 41 *i-3*, 10 *bpl* and 13 *i-3pl*, in a ratio of 27:9:9:12:3:4 (χ^2=8.11, p=0.20–0.10). The *pl* alleles are not linked to the *i-3* alleles.

A *bplr* female was mated with a dilute orange-red male having many leucophores (*bml-3R*). The F_1 progeny were 55 *bR*. The F_2 progenies were 48 *b*, 16 *bml-3* and 4 *bml-3pl*, in a ratio of 9:3:3:1 (χ^2=4.05, p=0.30–0.20). The *pl* alleles are not linked to the *ml-3* alleles.

Thus, the gene *pl* does not link to the eight genes tested.

References

Aida, T. (1921) *Genetics*, **6**: 554–576.

Tomita, H. (1975) *Medaka (Killifish)* (T. Yamamoto ed.). Keigaku Publ., Co., Tokyo. pp. 251–272.

Tomita, H. (1922) *Fish Biol. J. MEDAKA*, **4**: 45–47.

Yamamoto, T. (1967) Medaka. In "*Methods in Developmental Biology*" (eds. F.H Wilt and N.K. Wessels), pp. 101–111, Crowell Co., New York.