

Recessive lethal mutants of the medaka (*Oryzias latipes*) maintained in the Division of Biology, National Institute of Radiological Sciences

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Abstract Four morphological mutants (*ge*, *nc-1*, *nc-3* and *tb*) of the medaka (*Oryzias latipes*) are described. The mutations were induced by X-rays or ethylnitrosourea (ENU). The mutants are all autosomal and recessive lethal.

Introduction

In the course of a study on the transmission of radiation- or chemical-induced malformations to subsequent generations in the medaka (*Oryzias latipes*), several morphological mutants were found (Hyodo-Taguchi and Ishikawa, 1993; Ishikawa and Hyodo-Taguchi, 1993; Ishikawa *et al.*, 1994). The following is a brief description of four of them.

Mutagenesis

Males of H04C, an inbred strain of the medaka (Hyodo-Taguchi and Sakaizumi, 1993), were treated with X-rays or ethylnitrosourea (ENU) and then pair-mated with untreated females of the same strain. The resultant morphological mutations were recovered in the F₃ progeny. Among the mutants described below, *nc-1* was found among the progeny of an X-ray irradiated male, and the others were recovered among those of ENU-treated males.

Mutants

The mutants are all autosomal and recessive lethal.

(1) *ge* (*gray eyes*)

The *ge* mutant embryo is distinguishable from the wild-type embryo at Iwamatsu stage 28 (Iwamatsu, 1994), when pigmentation in the eyes in *ge* fails to advance. The development of pigmentation in the eyes in *ge* starts at about Iwamatsu stage 32, but the distribution of the pigment shows variegation; pigmented and unpigmented sectors coexist on the same eyeball. The gallbladder becomes dark green or black in the mutant embryo, whereas it is yellowish-green in

the wild-type embryo. The mutant embryo shows only the color abnormalities described above, and no structural abnormalities. After hatching, most of the fry swim exhibiting a rolling behavior. All of the mutant fry die within several days following hatching.

(2) *nc-1* (*non-circulation-1*)

The earliest expression of the *nc-1* mutation is detected at Iwamatsu stage 23, when the body cavity (pericardial region) of the mutant embryo expands more than that of the wild-type embryo. The heart begins to pulsate, as in the wild-type embryo, at Iwamatsu stage 24. However, the blood circulation is never established in the mutant embryo (It is established at Iwamatsu stage 25 in the wild-type embryo.). The heart continues to pulsate for several days, and a dwarfish and malformed embryo is formed. The embryo develops necrosis and finally dies before hatching.

(3) *nc-3* (*non-circulation-3*)

Blood circulation is not established in the *nc-3* mutant embryo. The phenotype of *nc-3* is similar to that of *nc-1*, and the mutant embryo dies before hatching. Complementation tests showed that *nc-3* was a different gene from *nc-1*.

(4) *tb* (*twisted brain*)

The earliest expression of the *tb* mutation is detected at Iwamatsu stage 16, when the head-to-tail length of the embryonic shield (embryonic body) of *tb* is shorter than that of wild-type embryo. At Iwamatsu stage 20, a short and fairly normal embryonic body is formed in the mutant. However, the neural rod in the mutant embryo is twisted in either the right or the left direction in the midbrain region. The heart begins to pulsate in the mutant at about Iwamatsu stage 24, but blood circulation is not established. The heart continues to pulsate for several days, and a dwarfish and malformed embryo is formed. The embryo develops necrosis and dies before hatching.

References

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