Mutant genes in the Mexican axolotl (Ambystoma mexicanum)

Thirty-five mutant genes have been described for the axolotl. The mutants have been put into 5 general catagories based upon the affected aspect of development. All the mutations are recessive. All of the mutations, except micro (#12), n^3 (#17) are presently maintained in the I.U. Axolotl colony.

Group I. Maternal effect mutations

1. o — Ova deficient; arrest of all eggs spawned by an o/o female at gastrulation due to an ooplasmic deficiency
2. cl — Abnormal cleavage
3. f — Fluid imbalance; swelling of the blastocoel
4. v — Arrest at blastula stage accompanied by a stratification of cell organelles according to their relative densities.
5. nc — No cleavage; the genetic lesion may involve a defective "seeding center" in tubulin assembly.

Group II. Specific effects on embryonic organs and tissues

6. c — Cardiac non-function
7. an — Transitory anemia.
8. e — Eyeless; gametogenesis stops at diplotene. In the eye induction system the gene affects only the ectodermal component.
9. sp — Spastic; the genetic lesion may involve a defect in the induction of the cerebellum.
10. as — Ascites; the Wolffian duct fails to open into the cloaca.
11. ph — Phocomelia; the growth of the major long bones is delayed, giving the limbs a flipper-like character.
12. micro — Variable degrees of micropthalmia. (This mutation was discovered by Signoret and Le Fesque and is kept in their colony in France.)
13. s — Short toes due to a reduction in the number of phalanges; no formation of the ribs; and incomplete development of the Mullerian duct.
14. st — Stasis of the blood circulation in the liver.

Group III. Nucleolar mutants

15. n^1 — Small nucleolus; no effect upon the amount of rDNA
16. n^2 — Small nucleolus; no reduction in the amount of rDNA
17. n^3 — Small nucleolus (mutation left no descendents)
18. n^4 — Reduced amount of rDNA; no effect upon the size of the nucleolus.
19. n^5 — Small nucleolus

Group IV. Recessive autonomous cell lethal mutations

20. p — Premature death at the stage of simple unbranched gill rudiments (stage 36)
21. q — Quivering lethal; cartilage is deficient and the notochord is flaccid.
22. t — Twisted gills with few filaments.
23. mi — Micropthalmic lethal; eyes are very small and sometimes the effect of the gene is asymmetric.

24. g Gill lethal; cell membranes of g/g show reduced capacity for binding enzymes.
25. r Renal insufficiency. Death is probably the result of a metabolic abnormality.*
26. x Fragile gills; mesonephros fails to develop.*
27. y Limb development arrested at the 2-digit stage.
28. ut Abnormal gills; slow growth and front feet stop development without forming digits.
29. l Small eyes with the pupil almost lacking; fragile gills and the front limb is retarded in growth.
20. h Hand lethal; the first digit of the front forelimb is oriented at right angles to the other digits so that it gives the appearance of a human thumb.
31. b "Brandon lethal"; retarded development of the front limbs.

Group V. Effects on pigmentation

32. d "White" phenotype due to a restriction of the number and the distribution of the melanophores and xanthophores.
33. a "Albino" pehnotype due to a failure to synthesize melanin.
34. m "Melanoid" phenotype due to an increased number and distribution of melanophores; the iridophores are not present and the number of xanthophores is reduced.
35. ax "Axanthic" phenotype; the homozygous mutant animals do produce xanthoblasts but the synthesis of pteridines is blocked.

* Larvae homozygous for both x and r show ~~microphthalmia~~, which may be asymetric in some instances.

A more detailed discussion of each of the mutations, plus a biblography of the published articles will be presented in subsequent issues of the Newsletter. General review articles of the axolotl mutants are:

Briggs, R. (1973) in Genetic Mechanisms of Development (ed. F.H. Ruddle) Academic Press.
Humphrey, R.R. (1975) in Handbook of Genetics (ed. R.C. King) Vol. 4. Plenum Press.
Malackiski, G.M. and A.J. Brothers (1974) Science 184: 1142.

Some of the descriptions and the placement of the mutants into certain groups are based upon preliminary information provided by investigators who are not directly associated with the Axolotl Colony. We wish to thank those involved for the personal communication of preliminary results to Dr. R.R. Humphrey or to A.J. Brothers.

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