Pigmentation loss and regeneration in a captive wild-type axolotl, *Ambystoma mexicanum*

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OVER a period of approximately 10 weeks from December 2009 until March 2010 a captive axolotl (*Ambystoma mexicanum*) was observed to change from a wild-type phenotype to a leucistic phenotype. It is common for leucistic axolotls to darken as they mature (Scott, 1995) but a change from wild-type to leucistic is a phenomenon that, to the author's knowledge, has not been documented before.

The animal was purchased by the author from a local pet shop in Bristol, UK, in May 2005 as a juvenile. It was approximately 100 mm TL and had fully developed legs. The gender of the animal was unknown. It was purchased together with a leucistic female of the same size. Since then the pair have been housed together in a 90 cm freshwater tank at room temperature. Both animals are healthy, active, and feed well on a diet of bloodworms with occasional supplementation of waxworms and appropriately sized (gut-loaded) crickets.

Over the winter of 2009 the axolotl began to lose pigmentation across its whole body becoming increasingly pale. Pigmentation loss began uniformly and the animal passed through stages of appearing brown and grey (Fig. 1) until it finally became fully white, with semi-translucent skin and pink gills (Fig 2.). The dorsal surface retained small amounts of pigment giving a pale grey appearance. The pigment of the eyes remained unaffected, as found in leucistic morphs. After the pigmentation loss the animal suffered the complete loss of its left foreleg, past the elbow, and a small section of the tail tip due to an attack by the second specimen (both portions were ingested). The axolotls were then separated with the injured animal being moved to a smaller tank to recover. In the recovery tank, both injured body parts started to regenerate and the areas of new tissue were pigmented. It remains to be seen if the pigmentation will be lost as the tissues mature. Stress caused to this animal due to being the subordinate individual in the aquarium, and the aggressive nature of the female did not seem to be a likely factor in the pigmentation loss. The two specimens have co-habited without any signs of problems for nearly five years, although they have previously had some aggressive interactions. Stress is also associated with loss of appetite and behavioural change, none of which have been observed. Environmental change also seems an unlikely factor contributing to stress as the aquarium set-up and maintenance have been constant since the axolotls were introduced to it, and the second animal has not shown any change. The change appears to be genetic, and is perhaps linked to the maturation of the animal.

Coloration in axolotls is controlled by four genes with all mutations being recessive. These genes produce four mutant phenotypes; albino, leucistic, axanthic and melanistic plus the ancestral wild-type (Frost et al., 2006). Axolotl coloration is produced through three types of pigment containing cells; melanophores, xanthophores and iridophores. In leucistic animals melanophores do not develop correctly but xanthophores and iridophores are still fully formed (Scott, 1995.).

As this animal has retained pigmentation in the eye and exhibits a typical leucistic phenotype it is possible that there is a mutation at the melanophore related loci. Pigment loss may be a result of this individual being heterozygous for the wild-type and possibly having leucistic genes with a loss of function of the dominant allele. It may be possible that the allele is reactivated in the regenerating tissue, or is an unstable mutant of the melanophores, however, this notion is conjecture.

An alternate possibility is that pigment loss may

Figure 1. (Left) and **Figure 2.** (Right). Pigmentation loss in *Ambystoma mexicanum* during winter months of 2009.

be a result of melanophore death. Melanophore death has previously been described in the white leghorn breed of chicken (Jimbow et al., 1974). However, to consider this notion, raises the question of where new melanophores originate in the regenerating blastema. As an amphibian leg regenerates, cells of the stump de-differentiate to form a blastema, which Kragl et al. (2009) showed has restricted potential and normally serves to re-populate the parent cell type. If melanophores have died, then a different cell type must be the source of the new melanophores. It is also possible that the cells do survive but cease to produce melanin. In this case the de-differentiation and re-differentiation processes must re-programme the melanophores to produce pigment.

The ancestry of this animal is not known and so recent hybridisation with the closely related tiger salamander *Ambystoma tigrinum*, is also a possibility.

Pigment loss has been observed with several anecdotal references appearing in the online amphibian forum www.caudata.org. However, none show the extent of change observed in this individual, nor the short time period over which pigment was lost.

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