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ALCL3 TREATMENTS INDUCED MORPHOLOGICAL AND MICROSCOPICAL ALTERATIONS IN PELOPHYLAX LESSONAE EMBRYOS

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The embryonic epidermis of amphibians is recognized as a sensitive and highly informative biomarker for detecting alterations induced by environmental contaminants, owing to its direct exposure and rapid ability to modulate mucus secretion (Semeraro et al., 2026). Aluminum chloride (AlCl3) is further identified as an acid-reactive toxicant capable of inducing physiological stress, ionoregulatory disruption, and developmental impairment in early aquatic life stages, with effects exacerbated under reduced pH conditions (Gensemer & Playle, 1999). We assessed the morphological and epidermal effects of two AlCl3 concentrations in P. lessonae embryos, comparing individuals retaining the egg-jelly layer with those experimentally deprived of it. Exposure to AlCl3 produced a coherent pattern of physicochemical and developmental alterations indicative of stress and sublethal toxicity. Increasing aluminum concentrations led to a measurable acidification of the medium, which was closely associated with enhanced melanization: both the number of melanin granules and the total melanized area in the embryonic epidermis increased in a dose-dependent manner, particularly in embryos lacking the protective mucous envelope. Developmental outcomes reflected this stress gradient. Mortality remained relatively low across treatments, yet the incidence of deformities rose sharply under intermediate concentrations without mucus, suggesting a threshold-dependent sensitivity. Total body length was reduced under the aluminum treatment, indicating impaired growth and morphogenesis. Across all endpoints, the presence of the egg-jelly layer consistently mitigated acidification, pigment activation, and developmental damage. Overall, these findings support a model in which aluminum toxicity during early amphibian development is mediated primarily through pH-driven physiological stress, with the mucous barrier acting as a significant protective factor.

References

1. Semeraro et al. MRT 2026;89.4:611-623.
2. Gensemer & Playle. CREST 1999;29.4:315-450.