POSTER

DEVELOPMENTAL TOXICITY OF LIGNIN AND NATURAL EXTRACTS-BASED NANO(BIO)PESTICIDES IN ZEBRAFISH EMBRYOS

Alessandra Lama, Beatrice Negrini, Pamela Floris, Patrizia Bonfanti, Anita Colombo, Maurizio Gualtieri, Paride Mantecca

Research Centre POLARIS, Dept. of Earth and Environmental Sciences, University of Milano-Bicocca, Italy

The transition toward more sustainable agriculture management practices is a growing priority. In this context, nano(bio)pesticides (nanoBPs) represent a promising alternative to conventional synthetic formulations. NanoBPs consist of a nanoscale carrier (e.g. lignin), combined with active ingredients of biological origin (e.g. plant, fungal extracts). Despite their eco-friendly design, nanoBPs may still interfere with embryonic development and physiological regulation. This study evaluated the potential hazard of novel lignin-based nanoBPs grafted with grapevine by-products, cone flower extracts, fungal extracts, and copper oxide. The physico-chemical properties of nanoBPs were analized by Dynamic Light Scattering, while (eco)toxicity was assessed using the Fish Embryo Toxicity (FET) test (OECD 236/2013). Zebrafish embryos (3 h post-fertilization, hpf) were exposed to freshly prepared nanoBPs over a concentration range of 0.1–100 mg/L until 120 hpf. Lethality and hatching rates were recorded from 72 hpf, and malformations and morphometric parameters were evaluated at 120 hpf. Only lignin–-fungal filtrate nanoBP induced embryo lethality at 100 mg/L, attributable to the fungal extract. Sublethal effects included hypopigmentation and reduced locomotor activity in larvae exposed to the fungal filtrate, as well as impaired swim bladder inflation in those treated with lignin and lignin–cone flower formulations.These effects may result from disruptions of thyroid hormone–mediated processes involved in pigmentation, growth, and swim bladder development. Accordingly, they will be further investigated within an adverse outcome pathway (AOP) framework focusing on thyroid-related sublethal endpoints. In particular, AOPs 155 and 157 link inhibition of deiodinase enzymes as the molecular initiating event to impaired posterior swim bladder inflation, while hypopigmentation will be explored as an additional endpoint, emphasizing tyrosinase activity as a key molecular event in melanogenesis.

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