

Sensitivity of Colonial Hydroids to heavy metal contaminations

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Comparably to larval bioassays, hydroid sensitivities to contaminants have been shown to be more relevant respect to other test organisms (3, 4). Results of experimental growth data with a single clone of the athecate hydroid *Clavopsella michaeli* (Berrill, 1948) exposed to single heavy metal contaminations (Hg⁺⁺, Cu⁺⁺, Cd⁺⁺) are provided here. The stock colony was collected at Ischia (Gulf of Naples) in May 1988, first record in Mediterranean Sea. One-week laboratory experiments and data analysis were carried out according to Stebbing (4), with few modifications (2).

Before the appearance of morphological abnormalities or colony death (1), chemical stresses cause variations in growth rate depending on concentrations and relative toxicities of pollutants. Exposure to different concentrations of three metal ions (Hg⁺⁺, Cu⁺⁺, Cd⁺⁺) resulted in recognizable, reproducible variations of the hydroid growth rate of *C. michaeli* colonies (Fig.1). Firstly, hormesis appeared: growth significantly increased in the presence of very low concentrations of copper (0.5, 1 µg/l) and cadmium (1 µg/l) ions while there was only a transient stimulation of growth rate after a 24h-exposure to 0.1 and 1.0 µg/l of mercuric ions. Threshold concentrations causing *C. michaeli* growth rate inhibition were consistent with environmental levels of many polluted coastal ecosystems. In fact, significant reduction in growth rate occurred with exposure to about 0.5-1.5 µg/l Hg⁺⁺ (Fig.1a), 2-3 µg/l Cu⁺⁺ (Fig.1b) or 25-30 µg/l Cd⁺⁺ (Fig.1c). Nevertheless, in these cases, resilience was still maintained: K values quickly rose to control levels, after restoration of uncontaminated conditions (3). Full degeneration of colonies, without possibility of recovery, occurred after exposure to 5 µg/l Hg⁺⁺, 10 µg/l Cu⁺⁺, or 100 µg/l Cd⁺⁺. Here, different stages of hydranth degeneration have been observed, comparable to those described by Karbe (1) in *Eirene viridula*.

The process of growth of colonial hydroids appears to be controlled by homeostatic mechanisms which regulate the co-operation of multi-interacting cellular systems during colonial morphogenesis and morphostasis. Such mechanisms counteract the possible inhibitory effect of any external disturbance of low intensity, thus maintaining colonial growth at an optimal, preferred rate. When the counteractive capacity is overloaded, inhibition of growth rate occurs (2, 3, 4). The physiological basis of the control mechanisms of hydroid development still remain unclear. Nevertheless, alterations in hydroid growth rate resulted as early and highly sensitive indexes of environmental stresses (2, 3, 4) and they can constitute a useful tool for the assessment of sublethal episodes of water pollution.

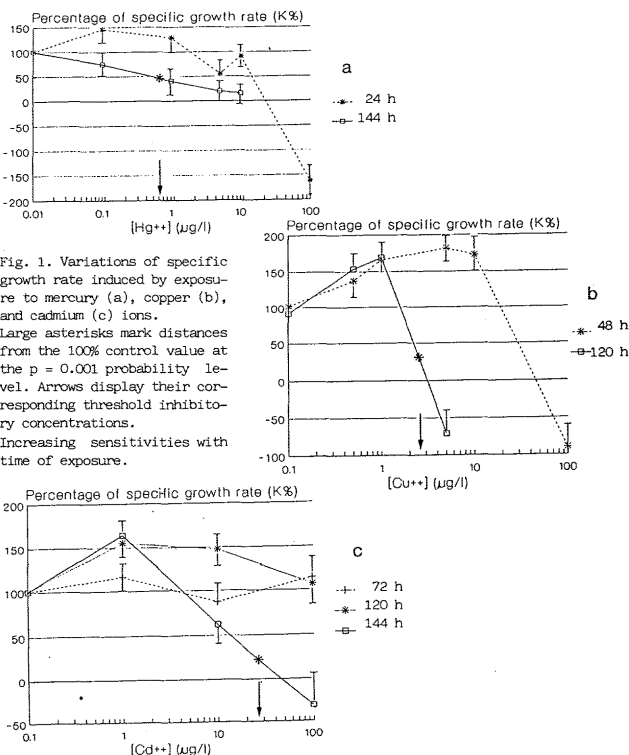


Fig. 1. Variations of specific growth rate induced by exposure to mercury (a), copper (b), and cadmium (c) ions.

Large asterisks mark distances from the 100% control value at the $p = 0.001$ probability level. Arrows display their corresponding threshold inhibitory concentrations.

Increasing sensitivities with time of exposure.

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