

Mortality, Accumulation, and Distribution of Zinc in the Gill System of the Dogfish Following Zinc Treatment

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Besides being an essential element, Zn has been shown to be toxic to aquatic organisms (SKIDMORE 1964, BRYAN 1971, WALDICHUK 1974). Acute lethality of Zn salts to teleosts have been reported (EISLER & HENNEKEY 1977, WONG et al. 1977, HUGHES & FLOS 1978), but few data regarding Zn toxicity to elasmobranchs can be found (CRESPÓ et al. 1979, FLOS et al. 1979). To evaluate the effect of Zn on elasmobranchs and to compare the data with those on teleosts this paper studies Zn toxicity and the accumulation of the metal in the gills of the dogfish *Scyliorhinus canicula* exposed to 180 and 80 ppm of zinc.

MATERIALS AND METHODS

Experiments were conducted on 78 dogfish which were kept for at least one week before experimentation in an open circulation tank (natural seawater pH 7.7; salinity 35.8-37.2; temperature 13-16°C). Specimens were placed in individual tanks. Zn solutions were prepared by adding ZnSO_4 to seawater up to the concentrations of 400, 300, 200, 150, 100, 75 and 50 ppm. Six fish were exposed to each concentration, 50% males, 50% females in each group since differences in Zn content and Zn accumulation due to sex have been described in the dogfish (CRESPÓ & BALASCH 1979a, CRESPÓ et al. 1979). Gills were taken out from the specimen, carefully washed in de-ionized water and filaments were separated from each cartilaginous arch. Filaments and arches were analyzed for Zn by atomic absorption spectrophotometry after digestion in concentrated HNO_3 and dilution with de-ionized water. Left and right samples were analyzed together as no significant differences in Zn content were found in previous experiments between both samples (Student t-test).

RESULTS

Evaluation of the Zn toxicity. Fig. 1 presents mortality curves according to the US standard method (SPRAGUE 1969). Lethal concentrations as well as median survival time (fig. 2) are determined. It appears that LC 50 24 h and LC 50 48 h for the dogfish are 180 and 80 ppm, respectively.

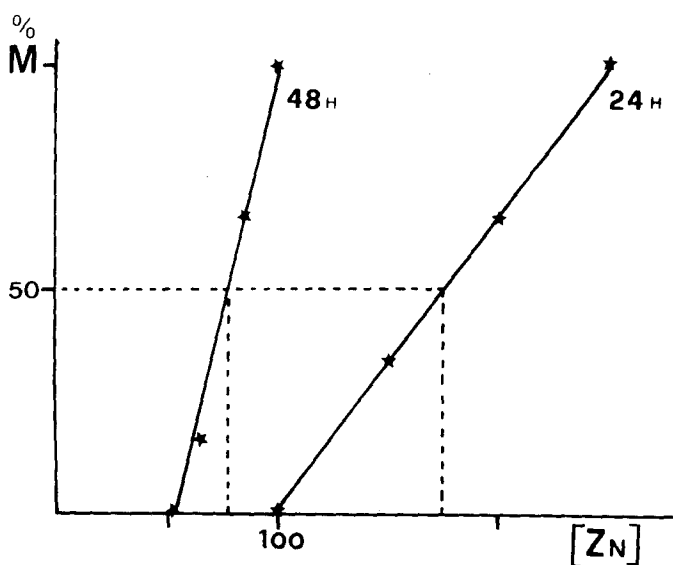


Fig.1 Mortality curves for the dogfish exposed 24 and 48 h to different concentrations of SO_4Zn (ppm Zn)
M%=mortality

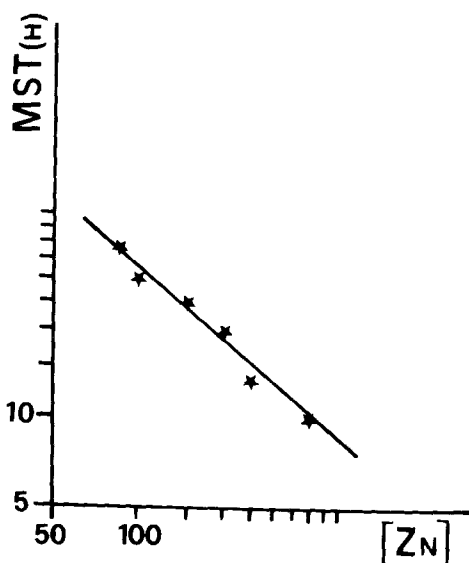


Fig.2 Median Survival Time in h (MST) of the dogfish exposed to different concentrations of SO_4Zn (ppm Zn)

TABLE 1. Zn content($\mu\text{g/g}$ dry weight) in the gill system of the dogfish following Zn treatment. Mean \pm standard deviation. According to Student t-test: ■ $t=4.18 > 3.35$ ($P=0.01$); ▲ $t=3.83 > 3.35$ ($P=0.01$); ★ $t=3.29 > 2.36$ ($P=0.05$); ○ $t=3.14 > 2.36$ ($P=0.05$)

untreat- ed fish	FILAMENTS					ARCHES						
	95 ± 108	84 ± 16	90 ± 16	78 ± 20	82 ± 26	83 ± 21 ▲★	61 ± 16	71 ± 38	62 ± 30	67 ± 38	92 ± 103	65 ± 21 ■
175 ppm 14 h	2700 ± 1500	2800 ± 1600	2800 ± 1600	3300 ± 2000	2600 ± 1700	2800 ± 1700 ▲○	100 ± 34	200 ± 190	210 ± 170	190 ± 90	200 ± 250	170 ± 61 ■
80 ppm 14 h	750 ± 500	630 ± 370	500 ± 350	420 ± 420	400 ± 370	520 ± 330 ○★	90 ± 19	170 ± 50	170 ± 73	130 ± 116	270 ± 220	140 ± 23
80 ppm 24 h	1200 ± 700	810 ± 310	520 ± 240	530 ± 240	420 ± 200	660 ± 240	100 ± 24	110 ± 41	100 ± 41	140 ± 80	180 ± 100	110 ± 21
treat- ment	1	2	3	4	5	ALL	1	2	3	4	5	ALL

Zn accumulation and distribution in the gill system following Zn treatment. Table 1 shows Zn content in the untreated fish (12 animals), and Zn treated groups: 175 ppm-14 h; 80 ppm-14 h; 80 ppm-24 h; (6 animals per group). No fish died during treatment; thus passive uptake of Zn after death (EISLER & GARDNER 1973) was avoided. Zn accumulated in the gill system, accumulating in the filaments more than in the arches. Zn content in the filaments depends on Zn concentration in seawater rather than on exposure time since no significant differences appear between 14 h and 24 h exposure to 80 ppm groups.

On carrying out the multivariate analysis of the variance (SEAL 1964): 3 levels (0 ppm-0 h; 80 ppm-14 h; and 80 ppm-24 h) and 5 variables (Zn concentrations in the five pairs of filaments); the factorial structure of the variables (CUADRAS 1974 a,b; CRESPO et al. 1979) revealed that the filaments carried by the second arch were those which absorbed the greatest amount of zinc per unit of dry weight.

DISCUSSION

On comparing our data on the dogfish with those on teleosts (JONES 1938, DOUDOROFF & KATZ 1953, SKIDMORE 1964, WONG et al. 1977, HUGHES & FLOS 1978) it is apparent that the dogfish is much more resistant to Zn pollution than species studied by these authors (*Gasterosteus aculeatus*; *Salmo gairdneri*; *Ctenopharyngodon idellus*; *Cyprinus carpio*). However all these teleosts are freshwater species. EISLER & HENNEKEY (1977) studied Zn toxicity to an estuarine fish *Fundulus heteroclitus*; the LC described (LC 50 24 h) is 125 ppm Zn. The fact that the LC 50 24 h for the dogfish is higher than the one for the mummichog can be due to the lower salinity and higher temperature of estuarine seawater rather than to the fact of being different species. Nevertheless no conclusion can be given until more elasmobranch species are studied and Zn toxicity to freshwater elasmobranchs is evaluated.

From our results it is apparent that the dogfish accumulates Zn in both gill filaments and gill arches after treatment with lethal concentrations of Zn. However Zn content of the gill system is not a good index to evaluate toxicity since 175 ppm-14 h treated group and 80 ppm-24 h treated group showed both toxicity symptoms and yet Zn concentrations in the gill filaments differed significantly. Moreover 80 ppm-14 h and 24 h treated groups did not differ significantly in their Zn content.

The filaments that showed a greater accumulation per unit of dry weight were those carried by the second arch, i.e. the most developed ones. These results are in agreement with data obtained following subacute treatment (CRESPO et al. 1979) and would suggest a higher specialization of the biggest gills.

Un published data from our laboratory (CRESPO 1979)

show that there is no accumulation of Zn in the organs (liver, spleen, pancreas) after acute treatment while there is an important accumulation following subacute treatment (FLOS et al. 1979). These data would be in agreement with histological observations in teleosts (SKIDMORE & TOVELL 1972, CRANDALL & GOODNIGHT 1963), as well as in elasmobranchs (CRESPO & BALASCH 1979 b).

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