THYROID HORMONES IN FISH EXPOSED TO PCDD/F AND TCDD, FROM THE YANGTZE RIVER REGION, CHINA .

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Introduction

Thyroid hormones play a major role in fish metabolism, growth, development and metamorphosis[1,2]. It is therefore of interest to study the effect of persistent organic environmental contaminants on larval and adult thyroid function in fish. Our study examines the effect of dioxins on thyroid hormones in adult silver carp (*Hypopthalmicthyes molitrix)* from the Ya-Er Lake of the Yangtze river basin, and in the early larval development of the Chinese rare minnow (*Gobiocypris rarus*), cultured and treated in the laboratory at the Institute of Hydrobiology, Wuhan, China .

The field study site, the Ya-Er Lake, in the Eastern region of Wuhan, Hubei province, sited in the median lower reaches of the Yangtze river, China is shallow and eutrophic. The lake received a discharge effluent containing a range of persistent chlorinated organic pollutants, from a large

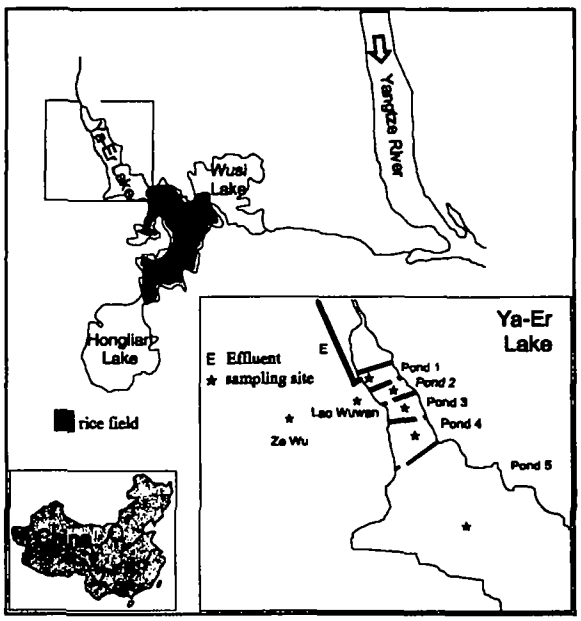

Fig. 1: The field study site

chemical factory situated on its bank, from 1962-1987. In 1978 part of Ya-Er Lake was divided into a series of 5 oxidation ponds for the remediation of wastewater.

Methods and Materials

One-year old male silver carp were sampled from a series of ponds with a gradient of contamination by polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/F) in the Ya-Er Lake. Dissected fish livers were frozen at -20°C, thyroid hormones extracted using barbital buffer and a methanol/chloroform separation matrix and measured using an I^{125} labelled radioimmunoassay[1].

In the first laboratory experiment, eggs of the Chinese rare minnow *G. rarus* cultured at the Institute of Hydrobiology, Wuhan, were exposed continuously to 0 (control), 0.1, 0.5, and 5.0 pg/ml TCDD (2,3,7,8-Tetrachlorinated dibenzo-p- dioxin) from within 24 hours of fertilization for 15 days and thyroid hormones measured. In the second experiment *G. rarus* eggs were exposed to 0 (control),and 0.1, pg/ml TCDD and sampled for assaying thyroid hormones every second day for 18 days. Hatched fry were fed daily with *Artemia salina* and 1/3rd of the test solution in each container was replaced twice a day before hatching and once a day after hatching. Water temperature was 15±1°C.

Water samples from each pond were analysed for PCDD/F on a high resolution mass spectrometer (Finnigan MAT 95S) coupled with an HP GC 5890 for chromatographic separation.

Results and Discussion

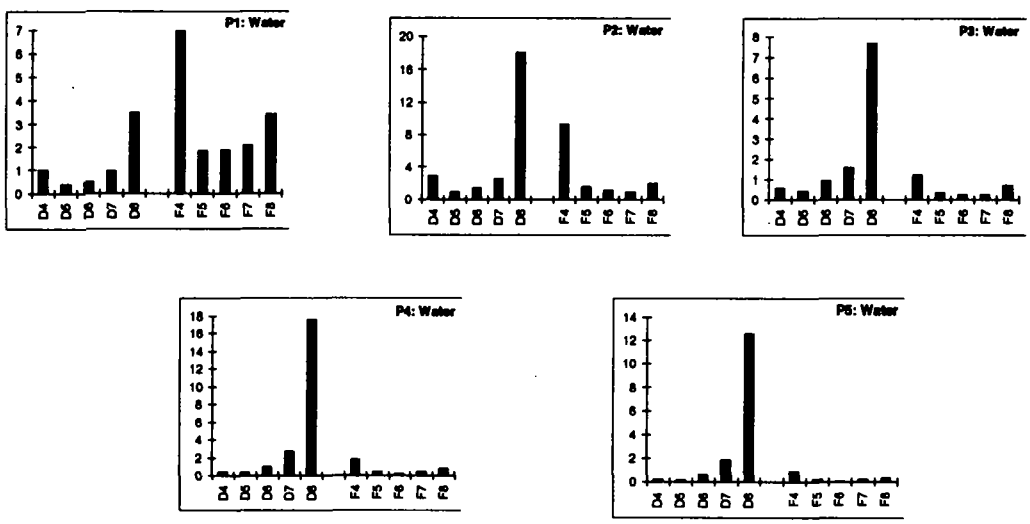

Fig. 2: PCDD/F homologue profiles in water (pg/l) of Ya-Er Lake from five ponds; Pi: Pond i (i=1-5) respectively.

The water in Pond 1 is clearly the most contaminated. Although the single homologue concentrations are lower in the water of Pond 1 the total TEQ is higher and the quality of water contamination changes from Ponds 1 to 5. The homologue pattern in Ponds 4 and 5 reflect the former discharge from the production of pentachlorophenol, while the profile in Pond 1 reflects the subsequent discharge from chloralkali electrolysis.

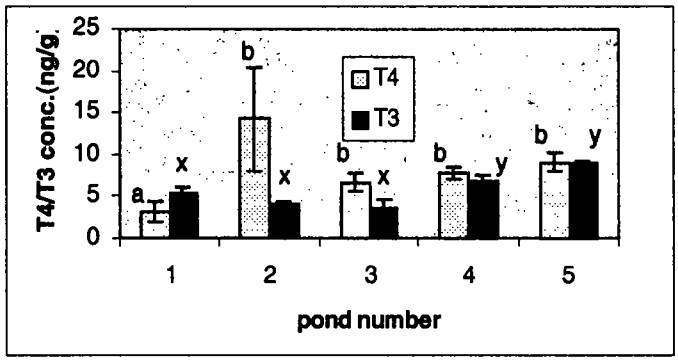

Fig. 3: Thyroxine (T4) and Triiodothyronine (T3) concentrations in yearling *H. molitrix* from ponds 1 to 5 in Ya-Er Lake (mean ± SE , n=6). Means with the same superscript in the same series are not significantly different from each other.T4 concentrations were significantly lower in the liver of silver carp sampled from pond 1 compared with those sampled from ponds 2 to 5. T3 concentrations in liver of fish from ponds 1,2 and 3 were not significantly different from each other but were lower than in fish from ponds 4 and 5. Results indicate that the high level of contamination by persistent organic pollutants of the water and sediments in Pond 1 may have resulted in depressed thyroid activity. In addition the results suggest that deiodinase activity maybe reduced in fish from ponds 1 to 3, since the liver is a major site for the deiodination of T4 to form $T3^3$. T3 is the active form of thyroid hormone and lower T3 levels could result in deleterious effects on the metabolic activities of the exposed fish.

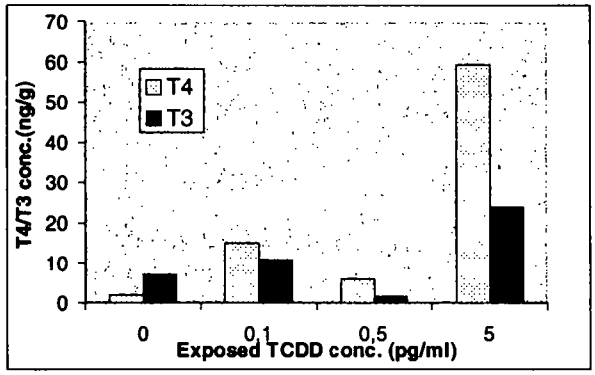

Fig 4:T4 and T3 concentrations (ng/g wet weight) in the whole body of 15 day old larvae of *G. rarus* exposed continuously to 0 (Control), 0.1, 0.5 and 5 pg/ml TCDD from day 1 of development.

Exposure to 0.1 to 5 pg/ml TCDD resulted in increased T4 concentrations in 15 day old *G. rarus* at the end of experiment 1.(Fig 4). However again results indicated that the conversion of T4 to T3 was not as efficient as in control larvae. Whether the increase in T4 concentration in larvae

after exposure to 5pg/ml TCDD is a stress response remains to be investigated. The onset of T4 production was delayed in *G. rarus* larvae treated with 0.1 pg/ml TCDD in experiment 2, while as in the first experiment the treatment seemed to stimulate T4 production on day 13 (Figs. 5 and 6). Again results indicated reduced efficacy of conversion of T4 to T3 in treated larvae.

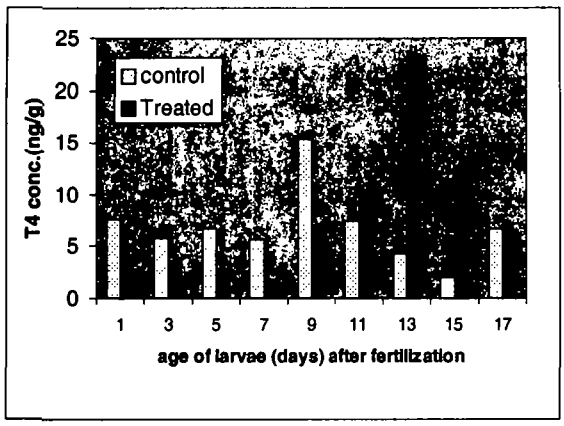

Fig 5:T4 concentration (ng/g wet weight) in the whole body of control and treated larvae of *G. rarus*. Treated larvae were exposed continuously to 0.1 pg/ml TCDD from day 1 of development.

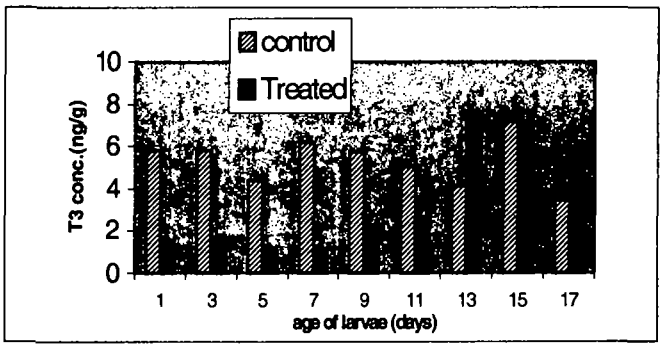

Fig 6:T3 concentration (ng/g wet weight) in the whole body of control and treated larvae of *G. rarus*. Treated larvae were exposed continuously to 0.1 pg/ml TCDD from day 1 of development.

References

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