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Toxicity of malathion to nile tilapia, *Oreochromis niloticus* and modulation by other environmental contaminants

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Abstract

Deliberate or accidental contamination of ponds by widely utilised organophophorous (OP) insecticides such as malathion is a potential problem for aquaculture in tropical countries. The aim of the study was to investigate potential synergistic or protective effects of common environmental pollutants on malathion toxicity in the Nile tilapia (*Oreochromis niloticus*) and by correlation of acute toxicity (LC_{50}) studies with biochemical parameters, identify potential enzyme systems involved in malathion toxicity. Tilapia were very sensitive to malathion (96h LC_{50} 2ppm) and in vitro data indicated that malaoxon, formed by oxidation of malathion, was the effective toxicant. Exposure of fish to an environmentally relevant dose of the insecticide synergist and CYP inhibitor, piperonyl butoxide (PBO) markedly reduced both the sublethal and the acute toxicity of malathion by 2-fold. Correlation of toxicity data with inducer effects and biochemical analyses failed to provide any evidence for CYP1-, CYP2B- or CYP3A-mediated malathion activation or detoxication in this species, thus the effect of PBO could not be attributed to inhibition of these enzymes. Whilst interspecies comparisons implicate hepatic θ class GST and non-specific carboxylesterase in malathion detoxication there was no evidence for alterations in malathion toxicity to tilapia by inducers of these enzymes. Treatment of fish with concentrations of a prototypical polyaromatic hydrocarbon, or cadmium, exceeding those producing effects in field situations, did not alter malathion toxicity indicating a lack of interaction of other common classes of environmental pollutants with OP toxicity.

Keywords

Tilapia;

Malathion toxicity;

Insecticide toxicity;

Acetylcholinesterase;

Carboxylesterase;

Cytochrome P-450;

Glutathione *S*-transferase;
Enzyme induction

Abbreviations

AChE, acetylcholinesterase;

CDNB, chlorodinitrobenzene;

CE, *p*-nitrophenylacetate carboxylesterase;

CYP, cytochrome P-450;

EROD, 7-ethoxyresorufin *O*-deethylase;

GST, glutathione *S*-transferase;

MCE, malathion carboxylesterase;

PAH, polyaromatic hydrocarbon;

PB, phenobarbital;

PBO, piperonylbutoxide;

OP, organophosphorus;

tSOx, *trans* stilbene oxide;
3MC, 3-methylcholanthrene