Bull. Environ. Contam. Toxicol. (1984) 33:127–132

Bulletin of Environmental Contamination and Toxicology

Acute Toxicity of EDB and Aldicarb to Young of Two Estuarine Fish Species

Matthew Landau and John W. Tucker, Jr.

Center for Marine Biotechnology, Harbor Branch Institution, RR 1, Box 196A, Fort Pierce, FL 33450

EDB (ethylene dibromide) and aldicarb [2-methyl-(2-methylthio) propionaldehyde-O-(methylcarbamoyl)oxime] are widely used insecticide/nematicides. Unfortunately, both compounds have been detected in groundwater supplies recently, arousing concern about their future use.

EDB is commonly found in drinking water wells in Florida in concentrations from 0.0001 to 0.01 mg/liter, even in areas that have not been treated with the chemical for 15 years (Florida Department of Health and Rehabilitative Services, Personal Communication). It has been shown to induce mutations in Drosophila (Kale and Baum, 1981) and rats (Wong et al., 1982).

Aldicarb was found in concentrations above 0.007 mg/liter, a recommended upper level, in 13.5% of 8,404 wells examined in Suffolk County, New York, and 16% of these had levels above 0.075 mg/liter (Zaki et al., 1982). Under most conditions, aldicarb is less stable than EDB and is metabolized to its sulfoxide, sulfone, sulfoxide oxime, sulfone oxime, and perhaps several other forms (Richey et al., 1977). It acts primarily as an inhibitor of acetylcholinesterase, resulting in excessive activity of the parasympathetic system, damage to the central nervous system, and overreactivity of voluntary muscles. Gaines (1969) found an LD_{50} for rats of 0.8 mg/kg. The N-nitroso derivative, formed at low pH in the presence of nitrite, has induced tumorigenicity in rats (Quarles et al., 1979), and causes single-strand breaks in human DNA that are not repaired by the normal DNA repairing events in cells (Blevins et al., 1977).

Interestingly, there is a dearth of information concerning the effects of these compounds on aquatic organisms, even though both are likely constituents of runoff from agricultural areas and EDB is an additive in leaded gasoline (Stecher, 1968). We have examined developing embryos and juveniles of common snook, Centropomus undecimalis, and juvenile sheepshead minnows, Cyprinodon variegatus, for acute toxicity responses to these two pesticides.

MATERIALS AND METHODS

Juvenile snook were seined from the Sebastian River (10 °/oo) and juvenile minnows were trapped from an estuarine pond (20 °/oo) at the Center for Marine Biotechnology, both part of the Indian River system in east central Florida. Experimental temperatures were similar to ambient. Juvenile fish were assayed in plastic lined buckets, each containing 8 liters of filtered, diluted, natural seawater (10 °/oo), 6 snook or 5 minnows per bucket. For both tests with snook and for aldicarb tests with minnows, the concentrations used were 0.04, 0.2, 1.0, 5.0 and 10.0 mg/liter. The EDB assay with minnows was conducted with concentrations of 0.1, 1.0, 10.0, 25.0, and 50.0 mg/liter. Pesticides were added to the water in acetone (1.0 ml acetone/8 liters water). Seawater and acetone controls were also run. Experiments were conducted in continuous light for 48 hours at 22.9° to 25.9°C. A death was recorded when opercular ventilation ceased and fish did not respond to gentle prodding. Animals were observed hourly for the first 12 hours and subsequently at longer intervals (2 to 10 hours).

Snook eggs were obtained by stripping ripe females and males and fertilizing the eggs artificially. Bioassays were done in plastic cups and were begun 2 to 3 hours after fertilization. Twenty eggs were used per cup, each containing 100 ml of filtered natural seawater (35 °/oo). The aldicarb was delivered in 0.05 ml of acetone, giving final concentrations of the pesticide equivalent to 0.025, 0.100, 0.250 and 0.500 mg/liter; seawater and acetone controls were also run. All doses were run in triplicate. A light regime of 14h:10h (L:D) was used; the temperature over the 36-hour experimental period ranged from 25.5° to 29.9°C. The organisms were periodically examined and dead embryos and larvae were noted and removed. A death was recorded if the clear egg became opaque, or if the larva halted all motion and began to curl.

Estimates of the LC_{50} values were calculated by the straight-line interpolation method (American Public Health Association, 1976).

RESULTS AND DISCUSSION

Snook and minnow juveniles had similar 48-hour LC_{50} values (Table 1). EDB was less toxic than aldicarb. A peculiar side-effect that EDB had was interference with osmoregulation, resulting in swelling of some of the minnows (Fig. 1).

The results of the snook embryo/larva bioassay (Table 2) yielded a 36-hour LC_{50} of 0.04 mg/liter. All living, healthy eggs hatched before 29 hours. At 0.25 and 0.50 mg/liter, eggs seemed to have a granulated yolk and larvae were more contorted at death.

Table 1. Acute Toxicity Responses by Juvenile Fish

Fish	Mean Weight ± s.d. (g)	Pesticide	48 hour LC_{50} (mg/liter)
Snook	0.23 ± 0.10	Aldicarb	0.10
Minnow	0.60 ± 0.16	Aldicarb	0.10
Snook	0.25 ± 0.10	EDB	6.2
Minnow	0.61 ± 0.14	EDB	4.8

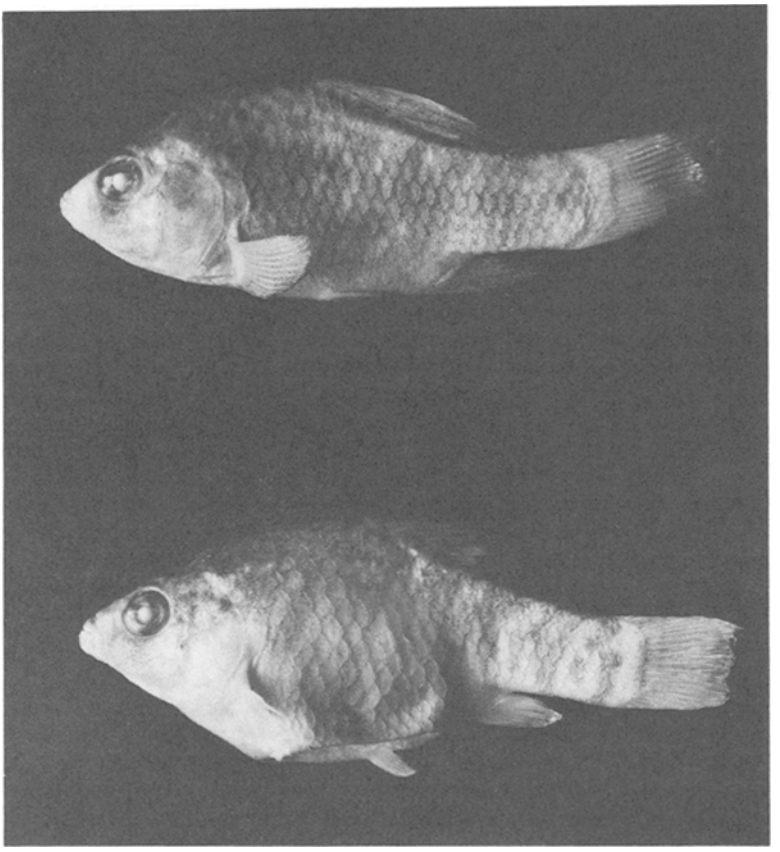

Figure 1. Sheepshead minnow exposed to 10 mg/liter EDB (bottom) compared to unexposed minnow (top). Both fish survived the 48-hour experiment. Note the partially deflated but still swollen abdomen of the lower minnow.

Table 2. Cumulative Acute Toxicity Response of Hatching Snook Eggs Exposed to Aldicarb. Results expressed as percent dead of the total of 60 organisms per treatment.

		HOURS		
	% Dead	0-14	14-25	25-36
Seawater Controls	embryos	1.7	1.7	1.7
	larvae	0	0	20.0
Acetone Controls	embryos	3.3	3.3	8.3
	larvae	0	0	51.7
0.025 mg/liter	embryos	0	0	5
	larvae	0	0	55
0.100 mg/liter	embryos	6.7	16.7	
	larvae	0	83.3	
0.250 mg/liter	embryos	1.7	21.7	
	larvae	0	78.3	
0.500 mg/liter	embryos	0	30	
	larvae	0	70	

Very little is known about the effects of EDB on non-target organisms. Ogino (1978) found that a steady state between goldfish and rearing water was reached in about 5 hours, but EDB was not concentrated in tissue to the extent that other brominated hydrocarbons were. This could indicate that EDB is less likely to be magnified in the food chain. In addition, EDB may be metabolized to its mercapturic acid derivative. However, the intermediates in this reaction, bromoacetaldehyde or S-2-bromoethyl glutathione, may be dangerous themselves (Van Bladeren et al., 1981). Because EDB is a known mutagen, some of its sub-acute effects will probably not be apparent for several generations.

There have been a few studies of the effects of aldicarb on freshwater fish. Konar and Ghosh (1980) claimed that it did not hinder growth and reproduction of *Tilapia mossambica* at concentrations as high as 0.363 ppm. The 48-hour LC_{50} for *Barbus conchonius* was found to be 8.99 mg/liter in hard water and 3.30 mg/liter in soft water (Pant and Kumar, 1981). Pickering and Gilliam (1982) assayed the larvae and juveniles of the fathead minnow *Pimephales promelas*; although 0.07 mg/liter had no effect, 0.156 mg/liter was lethal to fish exposed for 30 days after hatching, and in acute toxicity tests a 96-hour LC_{50} of 1.370 mg/liter was calculated.

While the short term LC_{50} values for EDB reported in the present study are rather high, those for aldicarb were considerably lower than would have been predicted based on reports of its effects on freshwater fish. Snook is an especially valuable gamefish, but Florida stocks have declined in recent years. E.P.A. (1981) determined that Baytex and Malathion were toxic to

snook embryos, and recommended that those insecticides should be used with caution during the snook spawning season. Perhaps many insecticides, possibly including aldicarb, have contributed to reduction of snook populations. Because acetylcholinesterase activity on the surface of the embryos of the fish Oryzias latipes has been detected (Fluck, 1982), we suggest that hatching snook in this study may have been particularly vulnerable to aldicarb. Studies on the extent of aldicarb contamination in estuaries are needed.

Acknowledgements. The authors gratefully acknowledge the assistance of B. Faulkner and C. Sultzman who helped collect the fish, and Dr. J. H. Ryther for his valuable suggestions and support. This is Contribution No. 22 from the Center for Marine Biotechnology.

REFERENCES

American Public Health Association (1976) Standard methods for the examination of water and wastewater. 14th ed. A.P.H.S., Washington, D.C.

Blevins RD, Lijinsky W, Regan JD (1977) Nitrosated methylcarbamate insecticides: effects on the DNA of human cells, Mut Res 44:1-7

E.P.A. (1981) Effects of Baytex and Malathion on early life stages of snook, Centropomus undecimalis. (Mimeo report) E.P.A., Athens, Georgia

Fluck RA (1982) Localization of acetylcholinesterase activity in young embryos of the medaka Oryzias latipes, a teleost. Comp Biochem Physiol 72C:59-64

Gaines, TB (1969) Acute toxicity of pesticides. Toxicol Appl Pharmacol 14:515-534

Kale PG, Baum JW (1981) Sensitivity of Drosophila melanogaster to low concentrations of gaseous mutagens: 3. dose-rate effects. Environ Mut 3:65-70

Konar SK, Ghosh TK (1980) Safe disposal of temik 10G, a carbamate insecticide, to protect fish (Tilapia mossambica), plankton (Cyclops viridis) and worm (Branchiura sowerbyi). Int J Acad Ichthyol 1:13-18

Ogino Y (1978) Intake of brominated hydrocarbons in goldfish. Okayama Igakki Zasshi 90:1451-1456

Pant SC, Kumar S (1981) Toxicity of temik (aldicarb) for a fresh water teleost, Barbus conchonius. Experientia 37:1327-1328

Pickering QH, Gilliam WT (1982) Toxicity of aldicarb and fonofos to the early-life-stage of the fathead minnow. Arch Environ Contam Toxicol 11:699-702

Quarles JM, Sega MW, Schenley CK, Lijinsky W (1979) Transformation of hamster fetal cells by nitrosated pesticides in a transplacental assay. Cancer Res 39:4525-4533

Richey FA Jr, Bartley WJ, Sheets KP (1977) Laboratory studies on the degradation of (the pesticide) aldicarb in soils. J Agric Food Chem 25:47-51

Stecher PG (ed) (1968) The Merck index. 8th ed, Merck & Co, Inc, Rahway, New Jersey

Van Bladeren PJ, Hoogeterp JJ, Breimer DD, Van der Gen A (1981) Influence of disulfiram and other inhibitors of oxidative metabolism on the formation of 2-hydroxyethyl-mercapturic acid from 1,2-dibromoethane by the rat. Biochem Pharmacol 30:2983-2988

Wong LCK, Winston JM, Hong CB, Plotnick H (1982) Carcinogenicity and toxicity of 1,2-dibromoethane in the rat. Toxicol Appl Pharmacol 63:155-165

Zaki MH, Moran D, Harris D (1982) Pesticides in groundwater: the aldicarb story in Suffolk County, New York (USA). Am J Public Health 72:1391-1395

Received January 31, 1984; accepted February 10, 1984