Bull. Environ. Contam. Toxicol. 31, 526–529 (1983)

Toxicity of Carbaryl and 1-Naphthol to Goldfish (*Carassius auratus*) and Killifish (*Fundulus heteroclitus*)

Thomas B. Shea and Eugene S. Berry

Department of Biology, Northeastern University, Boston, MA 02115

An important factor in assessing the potential danger of a pesticide to higher organisms is the rate of natural decrease of the compound (HERZEL et al. 1976); the longer a pesticide persists in the environment, the more likely it is to be toxic. In vitro studies have shown that pesticides which persist are taken up by cells to a greater extent than pesticides which do not persist (MURIKAMI & FUKAMI 1978). The pesticide carbaryl readily hydrolizes to 1-naphthol in model ecosystems (KARINEN et al. 1967) and in cell culture medium (SHEA & BERRY 1983). The 1-naphthol has been observed to undergo no further breakdown, thus persisting, in cell culture medium for at least 48 h (SHEA & BERRY 1983) as well as no further breakdown in seawater over 24 h, if not exposed to sunlight (STEWART et al. 1967).

Previous studies have shown 1-naphthol to be more toxic than its parent compound, carbaryl, to several species of mollusks (BUTLER et al. 1968; STEWART et al. 1967) and to several species of fish (STEWART et al. 1967; TILAK et al. 1979). A recent study has shown 1-naphthol to be as toxic as carbaryl to protozoal cultures (WEBER et al. 1982), and an in vitro study has shown 1-naphthol to be at least twice as toxic as carbaryl to goldfish-derived cell cultures (SHEA & BERRY 1983). This report presents an extension of this in vitro work by examining the relative toxicities of carbaryl and 1-naphthol to goldfish (Carassius auratus) and the common killifish (Fundulus heteroclitus).

MATERIALS AND METHODS

Technical grade carbaryl (99.07% pure) and technical grade 1-naphthol (99.7% pure) were obtained as gifts from the Union Carbide Corp. (Wilmington, DE). Compounds were initially dissoloved in 100% ethanol then dispensed at 1, 5, or 10 parts per million (ppm) into glass aquaria containing 15 L of dechlorinated water for goldfish, or half-strength artificial seawater for killifish. The maximum concentration of ethanol in the water was 0.0001%, which was also included in control aquaria, and was harmless to the fish. On separate occaisions, ten fish were introduced into each aquarium 30 minutes after dispersal of the compounds. The aquaria were aerated but not filtered, and food was withheld for the duration of the study (10 days).

RESULTS AND DISCUSSION

1-naphthol was observed to be more toxic than carbaryl to both goldfish and killifish (Figure 1). Goldfish exposed to 1 ppm carbaryl had no observed mortality, as did the control fish. Two goldfish had died by day 10 in 5 ppm carbaryl, and 4 goldfish had died in 10 ppm carbaryl by day 10. In contrast, 10 ppm 1-naphthol resulted in the death of all goldfish within 2 days, 5 ppm 1-naphthol resulted in the death of all goldfish within 5 days, and a total of 3 goldfish died by day 10 in 1 ppm 1-naphthol (Figure 1). No control mortality was observed in goldfish.

Killifish were unaffected by 1 and 5 ppm carbaryl, and 10 ppm carbaryl resulted in the death of a total of 4 killifish by day 10 (Figure 1). Killifish exposed to 10 ppm 1-naphthol all died within 2 days, 5 ppm 1-naphthol resulted in the death of 4 killifish by day 10, and no killifish died in 1 ppm 1-naphthol or in control conditions (Figure 1).

Along with the observed increase in mortality of 1-naphthol over carbaryl, 1-naphthol was observed to induce neurological trauma in both goldfish and killifish. Following 4 h exposure to 10 ppm 1-naphthol, all fish exhibited tremors, erratic swimming, darting, and an increased frequency of opercular beats. Within 24 h all fish exposed to 5 ppm 1-naphthol exhibited the same symptoms, and by day 6 all goldfish exposed to 1 ppm 1-naphthol were exhibiting these symptoms. Killifish were not observed to demonstrate any morbidity in 1 ppm 1-naphthol. All surviving fish, both goldfish and killifish, continued to exhibit neurological symptoms until the termination of the study at day 10. In contrast, there was no observed morbidity or neurological trauma in either goldfish or killifish at any of the concentrations of carbaryl.

As this is a static toxicity test under laboratory conditions, the toxic concentrations of these compounds are somewhat artificial and extrapolation to the environment would not necessarily represent a true assessment of the possible dangers of carbaryl and 1-naphthol. However, the relative toxicities of carbaryl and 1-naphthol can be compared. In goldfish, 1-naphthol is approximately 5 times more toxic than carbaryl on day 10 and in killifish, 1-naphthol is twice as toxic as carbaryl (Figure 1). Furthermore, all surviving fish exposed to 1-naphthol exhibited neurological trauma, whereas no neurological trauma was observed in fish exposed to carbaryl. The observed increase in toxicity and the induction of neurological trauma may be related to *in vitro* studies suggesting 1-naphthol, but not carbaryl, was taken up by goldfish-derived cell cultures (SHEA & BERRY 1983). If the cellular uptake is similar *in vivo*, then the relatively slow rate of hydrolysis of carbarylunder non-alkaline conditions (KARINEN et al. 1967; STEWART et al. 1967) may allow fish, exposed to carbaryl, to metabolize and/or tolerate the gradual increse in 1-naphthol.

Previous studies have shown 1-naphthol to be more toxic than carbaryl to several species of molluscs (*Mytilus endulis*, *Crassostrea gigans*, and *Clinocardium nuttallii*) (BUTLER et al. 1968; STEWART et al. 1967) and several species of fish (*Cymatogaster aggregata*, *Parophrys vetulus*, *Gasterosteus aculeatus*, and *Labeo rohita*) (TILAK et al. 1979; STEWART et al. 1967). In addition

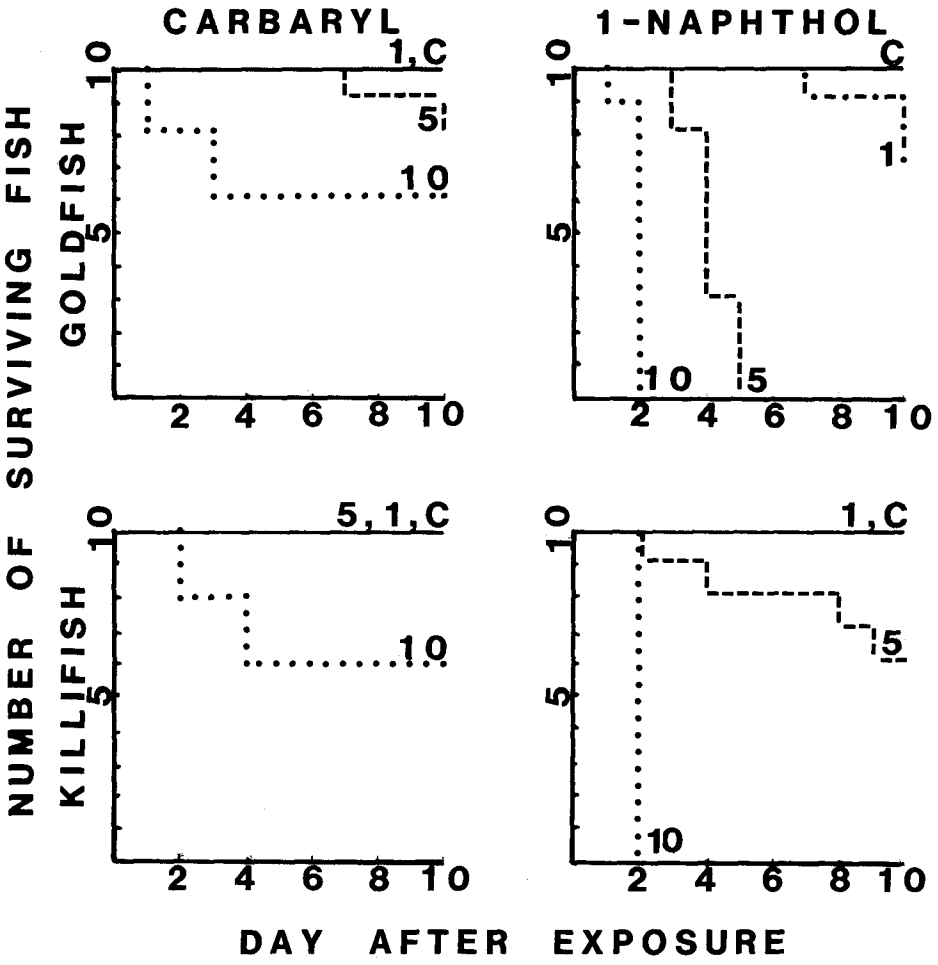

Figure 1: Effect of carbaryl and 1-naphthol on goldfish and killifish. Numbers along curves represent parts per million of each compound. C = control. Note the differences between the number of fish surviving in carbaryl versus those surviving in 1-naphthol.

1-naphthol was shown to be as toxic as carbaryl to protozoal cultures (WEBER et al. 1982). Based on these previous studies, the results presented here, and the report of detection of both carbaryl and 1-naphthol in aquatic systems adjacent to treated areas (OSMAN & BELAL 1980), it may be suggested that 1-naphthol may be responsible for a significant portion of the effects observed as a result of application of carbaryl.

REFERENCES

BUTLER, J.A., R.E. MILLMAN, N.E. STEWART: J. Fish. Res. Board Can. 25, 1621 (1968).

HERZEL, F., G. SCHMIDT, P. SOBHANI: Z. Angew. Zool. 63, 137 (1976).

KARINEN, J.E., J.G. LAMBERTON, N.E. STEWART, L.C. TERRIERE: J. Agric. Food Chem. 15, 148 (1967).

MURIKAMI, M. and J. FUKAMI: Bull. Environ. Contam. Toxicol. 19, 423 (1978).

OSMAN, M.A. and M.A. BELAL: J. Environ. Sci. Health B15, 307 (1980).

SHEA, T.B. and E.S. BERRY: Bull. Environ. Contam. Toxicol. 30, 99 (1983).

STEWART, N.E., R.E. MILLMANN, W.P. BREESE: Trans. Am. Fish Soc. 96, 25 (1967).

TILAK, K.S., D. MOHANARANGA RAO, A. PRIYAMVADA DEVI, A.S. MURITY: Ind. J. Exp. Biol. 18, 75 (1980).

WEBER, F.W., T.B. SHEA, E.S. BERRY: Bull. Environ. Contam. Toxicol. 28, 628 (1982).

Accepted June 21, 1983