Bull. Environm. Contam. Toxicol. 27, 376-379 (1981)

Size-Specific Mortality in Fry of Lake Trout (*Salvelinus namaycush*) from Lake Michigan*

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In studies designed to examine the role of PCBs and DDE in the reproductive failure of lake trout (Salvelinus namaycush) in Lake Michigan, BERLIN et al. (1981) observed seemingly faster growth in fish exposed to PCB and DDE than in control fish. Limited length measurements of dead fish indicated that the live fry were slightly larger than fry that died. Size-specific mortality that results in the selective mortality of the smaller fry has been shown to artificially increase growth rates (ALLISON et al. 1964). Because growth rate in salmonids is strongly heritable (KINCAID et al. 1977; BRIDGES 1973; AULSTAD et al. 1972), and because decreasing genetic variability in a fish population is considered to have negative long-term effects on the reproduction of salmonids (MILLER 1957), we tested for the presence and magnitude of size-specific mortality in fry from Lake Michigan lake trout and fry of hatchery origin during exposure to PCBs.

METHODS AND MATERIALS

Two exposure studies conducted simultaneously beginning in January 1978 provided data used for this test (MAC and SEELYE 1981a, b). Lake trout fry hatched from eggs collected from fish in southern Lake Michigan and from eggs of hatchery origin (Jordan River National Fish Hatchery) were exposed for about 50 days after hatching to 50 ng/L PCBs (Aroclor 1254). In addition, fry from both sources were reared in water to which no PCBs were added. Newly hatched fry from the Lake Michigan treatment contained 17.5 µg/g PCBs (Aroclor 1254, based on dry weight of fry), and the hatchery fry contained only 0.4 µg/g (MAC & SEELYE 1981a, b). We used two replicate tanks for each treatment with 650 hatchery or 1000 lake fry per tank. Samples of 25 live fry were removed periodically from each tank for length and weight measurements. Mortality was recorded daily and the total length of each dead fry was measured to the nearest millimeter.

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*Contribution 573 of the Great Lakes Fishery Laboratory, U.S. Fish and Wildlife Service, Ann Arbor, Michigan 48105. This article was written by employees of the United States Government as part of their official duties and therefore cannot be copyrighted.

0007-4861/81/0027-0376 $01.00

Data reduction and analysis included preliminary examination of a curve representing the lengths of live and dead fry versus time for each tank, and subsequent least squares fits of live fish lengths versus time. Resulting regression equations were used to predict the average lengths of fry on days when dead fry were observed and the average predicted lengths were compared (t test) with average observed lengths of dead fry for each exposure.

RESULTS AND DISCUSSION

Regression equations for length of live fry versus time (Table 1) produced correlation coefficients (R^2) ranging from 0.77 to 0.91. Average lengths of dead fry were significantly less ($P \leq 0.005$) than live lengths for all four groups of fry from Lake Michigan lake trout, independent of their exposure to PCBs after hatching (Fig. 1). Conversely, none of the predicted versus observed lengths of fry were significantly different ($P \geq 0.20$) for lake trout fry of hatchery origin. Mortality rates ranged from 4.9 to 6.5% for the hatchery fry and 12.5 to 16.6% for lake fry.

TABLE 1. Regression statistics for growth in length of lake trout fry from Lake Michigan and hatchery origins.

Tank	Slope	Intercept	Standard error of regression	R^2	Treatment
Lake Michigan					
1	0.25	16.8	1.38	0.89	Control
2	0.24	17.1	1.22	0.91	Control
3	0.26	16.9	1.33	0.90	PCB
4	0.25	17.0	1.44	0.88	PCB
Hatchery					
5	0.23	17.0	1.76	0.81	Control
6	0.23	17.0	1.86	0.79	Control
7	0.26	16.4	2.33	0.77	PCB
8	0.27	15.9	2.40	0.77	PCB

Size-specific mortality observed in fry of Lake Michigan origin could be the result of a number of factors, including origin and condition of parents and environmental stresses, including organic contaminants. Size-specific mortality was previously observed in fry of cutthroat trout (Salmo clarki) exposed to DDT by ALLISON et al. (1964). Considering the similarity between the physiochemical properties of PCBs and DDT, the substantial concentration of PCBs in the fry of lake origin may be an important factor in explaining the size-specific mortality observed. The fry of hatchery origin, which contained relatively small amounts of PCBs, did not exhibit size-specific mortality. These results suggest a cause-effect relationship between PCBs and size-specific mortality in lake trout fry. However, we cannot further evaluate

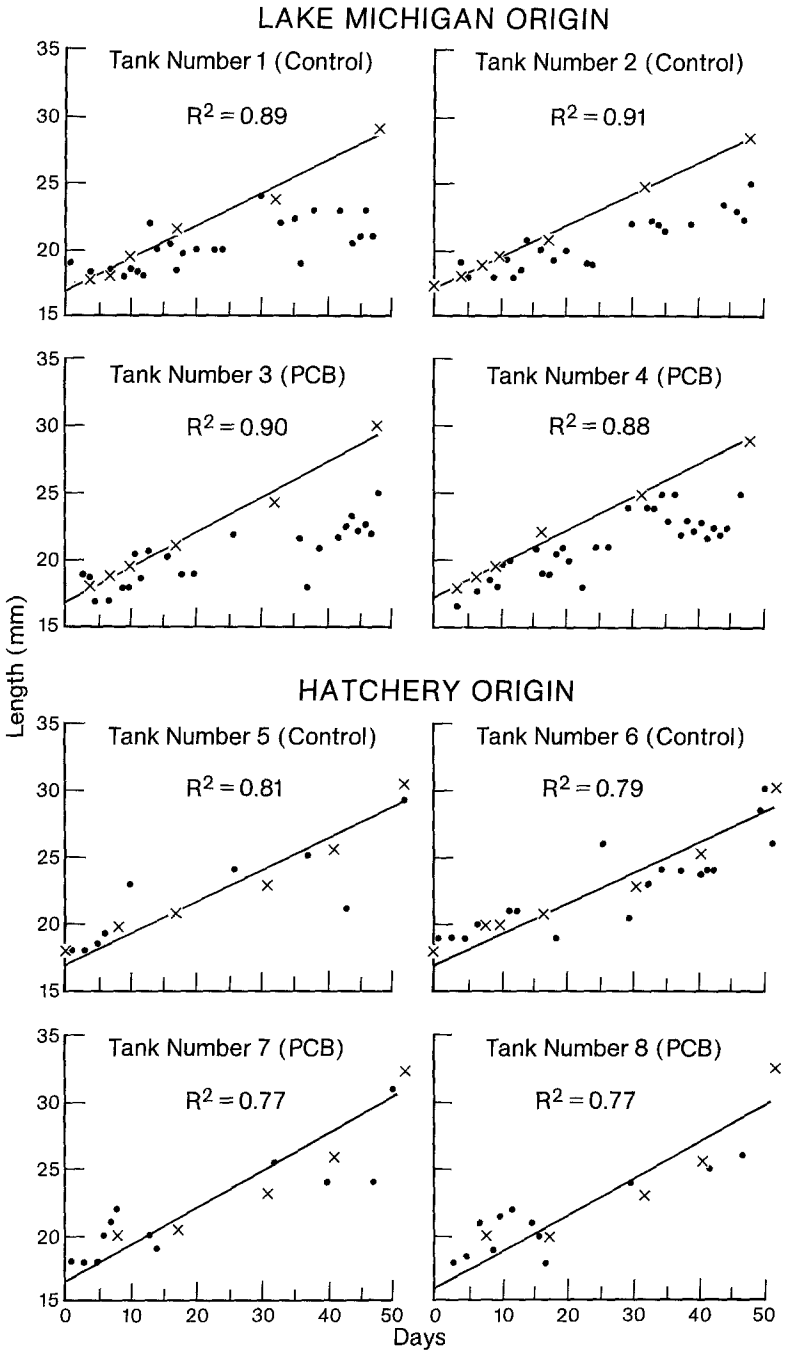

Figure 1. Regression lines and coefficients (R^2) for growth of lake trout fry. Each x represents the mean of 25 fry lengths measured on the various sampling days. Dots represent lengths of dead fry. Fry in tanks 1-4 were of Lake Michigan origin and those in tanks 5-8 were of hatchery origin.

this relationship until fish of the same origin are subjected to testing where chlorinated hydrocarbon content is the only treatment variable.

Higher mortality of the smaller fry might be considered to have a positive effect on long-term fish production in that larger fish are being selected for. Decreasing genetic variability in a fish population, however, can result in lower production. ALLISON et al. (1964) found that when mature cutthroat trout from a population that had undergone size-specific mortality due to DDT exposure were spawned, the resulting fry appeared to be larger and in better condition than fry spawned from control fish. However, the mature trout that produced larger fry had a higher mortality rate after spawning. This higher mortality rate of adults could have serious negative effects on long-term fish production.

The size-specific mortality we observed in fry from Lake Michigan lake trout suggests the need to consider this phenomenon when conducting contaminant effects studies with fry where growth is one of the variables being measured. In addition, if contaminants cause size-specific mortality in natural trout populations, the long term effects should be identified, since a decrease in genetic variability of the population could partly explain the drastic decreases of salmonid populations in the Great Lakes during the time when chlorinated hydrocarbons were increasing in the lakes.

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Accepted June 15, 1981