Physiologic Responses of Striped Bass (*Morone saxatilis*) Embryos and Larvae to Low, Sublethal Concentrations of the Aromatic Hydrocarbon Benzene

MAXWELL B. ELDRIDGE, PETE BENVILLE and JEANNETTE A. WHIPPLE
Tiburon Laboratory
Southwest Fisheries Center
National Marine Fisheries Service
Tiburon, California 94920

RESUMEN

El "striped bass" es un pez de importancia deportiva y comercial y que pasa toda su vida en estuarios y aguas costeras. Dado que es un predador final en la cadena alimenticia y emplea los estuarios para desove y desarrollo de la cría, es un buen indicador de las condiciones saludables del biota estuarino. Recientemente, encontramos hembras cargadas en el Delta estuario de la Bahía de San Francisco, en los Estados Unidos, que estaban contaminados con una variedad de contaminantes, los que incluía la benzina hidrocarbónica aromática, derivada del petróleo. Por lo tanto, los experimentos de laboratorio fueron ensayos para probar los efectos que baja concentración, concentraciones subletales (promedio 159 a 248 ppb) de C^{14} rotulados benzina, podían tener en el "striped bass" desde la fecundación a la metamórfosis.

Todas las fases vitales (o evolutivas) absorbienron benzina rápidamente, casi siempre en cuestión de horas, y concentraban los hidrocarburos a niveles mayores que las concentraciones acuosas presentes. Las larvas más desarrolladas, en su alimentación, concentraban la mayor parte de los hidrocarburos, demostrando que la benzina se acumula en los tejidos, tanto por la vía alimenticia como la acuática.

La benzina provocaba desoves anticipados y niveles más altos de sobrevivencia, pero estos beneficios aparentes eran anulados por la mortandad latente después de la contaminación. Larvas prealimentadas y expuestas a la benzina sobrevivían, y no se encontró diferencias en larvas más desarrolladas. Larvas avanzadas y hambrientas sufrieron mayor esfuerzo, muriendo más rápidamente que las no expuestas.

El metabolismo de los embriones y larvas expuestas fué consistentemente más intenso que en los controles, pero ello no se reflejaba en las comparaciones de crecimiento. Al contrario, las larvas expuestas crecian a un ritmo mas rapido que los controles después del período de exposición.

Estos resultados se analizan, dado que se relacionan a vías y efectos conocidos de otros hidrocarburos derivados del petróleo. Se presentan las consecuencias de estos efectos según se manifiestan a nivel de población.

INTRODUCTION

Lay and scientific communities are becoming increasingly more aware and outspoken about the ubiquity of toxic substance (White, 1980; Magnuson, 1980). A large portion of the toxicants found in the aquatic environment originate from petroleum. In fact, most of the Environmental Protection Agency's list of priority pollutants contain compounds which are derived from or are mixed with petrochemicals. Monocyclic aromatic hydrocarbons are generally considered one of the most toxic of the petrochemicals and benzene is the simplest and most basic structure common to all aromatic hydrocarbons. Benzene is found in crude oil and refined products. It is relatively water soluble and has a log partition coefficient of 2.80, meaning it tends to concentrate in fatty tissues.

A small but increasing data base of field measurements show concentrations of benzene in open ocean waters range approximately 0.01 to 5.00 ppb (nll^{-1}) (Whipple et al., 1980a). Nearshore and estuarine waters contain benzene at 1 to 100 ppb and up to 200 ppb in fresh waters. These levels represent residual levels from chronic inputs, whereas petrochemical spills cause higher concentrations. For example, water samples collected around the crude oil plume of the recent IXTOC wellhead spill contained 60 to 100 ppb of benzene. Concentrations decreased with distance from the plume to open ocean residual levels of approximately 1 ppb (Payne et al., 1980).

Studies conducted at our laboratory on the commercially and ecologically important striped bass (*Morone saxatilis*) showed fish tissues (mainly liver and ovary) contained relatively high concentrations of benzene (Whipple, 1979; 1980; Whipple et al., 1980b; Jung and Bowes, 1980). The amounts found suggest the adult fish are being exposed to low level chronic concentrations in the wild. These conclusions are based on our own experience in the laboratory with exposures of adults to benzene.

Striped bass is an anadromous fish found in coastal waters of East, West and Gulf states of the United States. It is a terminal predator in the food chain and spawns large pelagic eggs. Larvae characteristically use shallow, often industralized, estuaries as nursery grounds. Juveniles and adults range into marine environments and sometimes migrate long distances. Unfortunately populations in the northeastern and western United States are experiencing unprecedented declines. Egg viability and survival of early life stages also seem affected (personal communication, Robert Stevens, U.S. Fish and Wildlife Service, Washington, D.C.; Donald H. Stevens, California Department of Fish and Game, Stockton, California).

Pollution is one of the causal factors most implicated in the population declines. Because benzene was present in adult fish it appears likely younger stages might also be exposed to the chemical. We therefore designed a series of experiments (a) to determine amounts and rates the early life stages take up when exposed to low level, sublethal concentrations, and (b) to measure as comprehensively as possible how these accumulated hydrocarbons affect the physiology of these organisms.

MATERIALS AND METHODS

Early life stages of striped bass are easily divided on a physiological and developmental basis into three stages: embryo, from fertilization to hatching (Day-0 to Day-2); prefeeding larva, from hatching to first, exogenous feeding (D-2 to D-7); and feeding (and starved) larva, from the time when feeding normally begins to metamorphosis (D-7 to D-29). The different aspects of this study are organized around these three life phases with benzene exposure occurring within each phase and measurements taken during and after the completion of the particular life stage. The purpose of this strategy was to determine how individuals within a given life stage respond to benzene and to see if latent effects occurred after exposure.

Sexually mature electrofished adults from the Sacramento River, California, were injected with gonadotropins and artificially spawned. Eggs were incubated in flow-through McDonald jars. Newly hatched prefeeding larvae were transferred to semispherical 8-L acrylic plastic rearing containers held in water tables for temperature stability. Animals were reared from hatching to metamorphosis in

Table 1. Experimental conditions of benzene exposure to striped bass and larvae

Life Stage	Type of Exposure	Exposure Duration (days)	Sample Type	Sampling Frequency (time after initial exposure)
Embryo	Continuous slow	0-2	water	0, 3, 6, 12, 24, 33, 42 h
			tissue	1, 3, 6, 12, 24, 36 h
Prefeeding Larvae	Semi-static	2-7	water	0, 1, 3, 6, 12, 24 h; daily thereafter.
			tissue	1, 3, 6, 12, 24 h; daily thereafter.
Feeding & Starved Larvae	Semi-static	8-24	water	0, 3, 6, 12, 24 h; daily thereafter.
			tissue	1, 3, 6, 12, 24 h; daily thereafter.

these containers. Initial stocking densities depended on the type of study being run — approximately 150 larvae in all but survival tests (those had 20 larvae per container). We attempted to duplicate natural water quality conditions as much as possible. Temperatures were held at $18.0 \pm 0.5°C$ and oxygen contents were at or near saturation. Photoperiod and light qualities were controlled and close to natural. Salinities were zero from fertilization to D-4, 1.0 ppt from D-5 to D-13, and 3 ppt from D-14 to D-29. Containers were cleaned and new water and live food (newly hatched *Artemia salina* nauplii in densities of 5.0 nauplii ml^{-1}) added daily.

Animals were exposed to C^{14}-benzene in continuous flow and semi-static systems depending on the type of culture system (Table 1). Target benzene concentrations were 100 to 200 ppb throughout the experiment. Water and tissue samples were analyzed for benzene by gas chromatography (Hewlett Packard 5840) and standard radiometry (tissue digestion and counting on a Packard Tri Carb scintillation spectrometer).

Immediate mortality was measured during each exposure period and delayed mortality after exposure. Each group was monitored to D-29 or complete mortality. Embryonic mortality was determined from direct counts of dead eggs which had floated out of McDonald jars. Larval mortality was estimated by visual counts in test containers.

Growth was determined from subsamples of embryos and larvae that were measured for standard lengths (via ocular micrometer) and dissected apart to separate oil globule, yolk and assimilated tissue. Tissue samples were dried at 60°C for 24 h and weighed on a Cahn electrobalance.

Bioenergetic variables in this study and the methods used to measure them are similar to those of Laurence (1977) and Eldridge et al. (1977). The caloric contents of

Table 2. Summary of sampling frequencies and sizes in the study of the effects of benzene on striped bass embryos and larvae

Life Stage	Age (days)	C^{14} Uptake sf*	Survival sf*	Survival replicate size	Growth sf*	Growth sample size (n)	Bioenergetics sf*	Bioenergetics sample size (n)
Egg	0-2	varied (see Table 1)	every 12 h	2	every 12 h	20-30	every 12 h	30-50
Prefeeding Larvae	2-7	1st 24 h & daily	daily	4	daily	10-20	daily	10-20
Feeding & Starved Larvae	8-29	1st 24 h & daily	daily	4	alternate days	10-20	alternate days	10-20

*sf = sampling frequency

endogenous energy sources were obtained by careful dissection and weighing of egg and larval components, then multiplying these weights against previously determined caloric values (Eldridge et al., 1980b). Yolk and tissue contents were measured directly. Oil globule contents were estimated by measuring globule volumes and comparing them against the initial globule volume in the egg prior to fertilization. Exogenous food intake was determined from counting stomach contents of larvae sampled 1 h after food introduction and entering these counts into the formula: FR = NH/D, where FR = ***daily food ration,*** ***N*** = average number of nauplii in stomachs, H = hours of active feeding, and D = digestion time. Other variables in the equation were previously determined and reported in Eldridge et al. (1980b). Routine metabolism was measured through oxygen consumption in a Gilson differential respirometer.

Sampling frequencies and sample sizes for different aspects of this study are summarized in Tables 1 and 2. Statistical summarizations, analyses and curve fittings were performed with the assistance of a CDC 7600 computer located at the University of California, Berkeley.

RESULTS

Exposure Concentrations

Actual aqueous benzene concentrations in test containers were higher than planned during embryonic prefeeding larva stages (Table 3) and the continuous exposure system proved more stable than the semi-static systems with its repeated daily dosages. Mean exposure concentrations ranged from 159 to 415 nll^{-1}. Prefeeding larvae were exposed to higher initial benzene concentrations than either embryos or feeding larvae (Table 3). Monitoring of benzene in the first 24 h after benzene introduction shows embryos in the continuous flow system had nearly

Table 3. Summary of benzene concentrations in culture containers of striped bass eggs and larvae

Life Stage	Exposure Period	Type of Exposure	Exposure concentrations ($nl\ l^{-1}$, ppb) $\bar{x}$	S.D.	Range
Egg	D-0 to hatching (D-2)	Continuous	248	11	222-260
Prefeeding Larvae	D-2 to D-7	Semi-static	415	199	221-721
Feeding	D-8 to D-24	Semi-static			
	Fed Larvae	Semi-static	159	37	127-247
	Starved Larvae	Semi-static	159	19	127-187

constant benzene exposure (Table 4). Benzene is containers of later stages went through daily fluxes in which benzene was introduced with new water changes. Figure 1 illustrates how benzene decreased in a hyberbolic fashion in prefeeding larval containers and in exponential and linear declines in older stages.

Uptake, Accumulation and Depuration of Benzene in Tissues

In all life stages uptake and depuration of hydrocarbons were rapid, always within a matter of hours. All stages accumulated these hydrocarbons to levels greater than exposure concentrations and the longer the exposure, as in the older larvae, the greater the accumulation.

Table 4 presents results of embryonic uptake. Within an hour of benzene introduction embryos had accumulated over double the water concentration and this trend continued until newly hatched larvae had almost 18 times the average water benzene concentration. Depuration occurred quickly (Table 4). Embryonic tissues had only 57 plg^{-1} (ppb) within 12 h after the end of exposure.

The daily fluctuations of aqueous benzene in test containers of prefeeding larvae were reflected in tissue uptake (Table 4). Tissues reached equilibrium within 3 h after initial exposure then declined for the next 21 h, when the next dose of benzene was applied. Thus tissue benzene declined each day as aqueous benzene decreased. But larvae evidently were not able to completely eliminate benzene from tissues. Tissues sampled at the beginning of each day, before culture water was changed and new benzene added, had benzene in ever increasing amounts (Table 4).

Beginning with D-7 larvae were able to feed on live *Artemia salina* nauplii. This provided another pathway for benzene to enter larval tissues. Larvae deprived of food provided a way to estimate the contribution benzene-laden food made to tissue contamination. In the first 24-h larvae, as in earlier stages, rapidly accumulated benzene (Table 4). Equilibrium was reached within 3 h, the same as prefeeding larvae. But probably because of higher metabolic rates less benzene remained in tissues at the end of 24 h. For the remainder of the exposure period (D-9 to D-24) starved and fed larvae steadily accumulated amounts of benzene in their tissues (Fig. 2). This accumulation was best described by an exponential function in starved larvae and by a power function in fed larvae. After D-9 food appeared responsible

Table 4. Benzene concentrations in culture water and tissues of striped bass embryos, prefeeding and feeding larvae

Life Stage	Hours after initial exposure	Hours after end of exposure	Mean benzene concentrations water (nl l^{-1}, ppb)	tissue (pl g^{-1}, ppb)
Embryo	0		250	—
	1		251	561
	3		257	1340
	6		250	1292
	12		239	2006
	24		256	411
	33		253	4512
	42		232	—
		1		991
		3		354
		6		237
		12		57
Prefeeding Larva	1		(see Figure 1)	2456
	3			2705
	6			2632
	12			1291
	24			1277
	72			2090
	120			3878
	144			4674
		1		991
		3		354
		6		237
		12		57
Feeding Larva Fed —	1		(see Figure 1)	1033
	3			2353
	6			603
	12			991
	24			175
Starved —	1		(see Figure 1)	1174
	3			868
	6			945
	12			338
	24			175

for higher accumulations of benzene.

In separate measurements of benzene uptake over a 24-h period by *Artemia* nauplii, we found nauplii also progressively took up benzene (Table 5). Their maximum accumulation occurred 6 h after initial exposure (4.5 x the initial

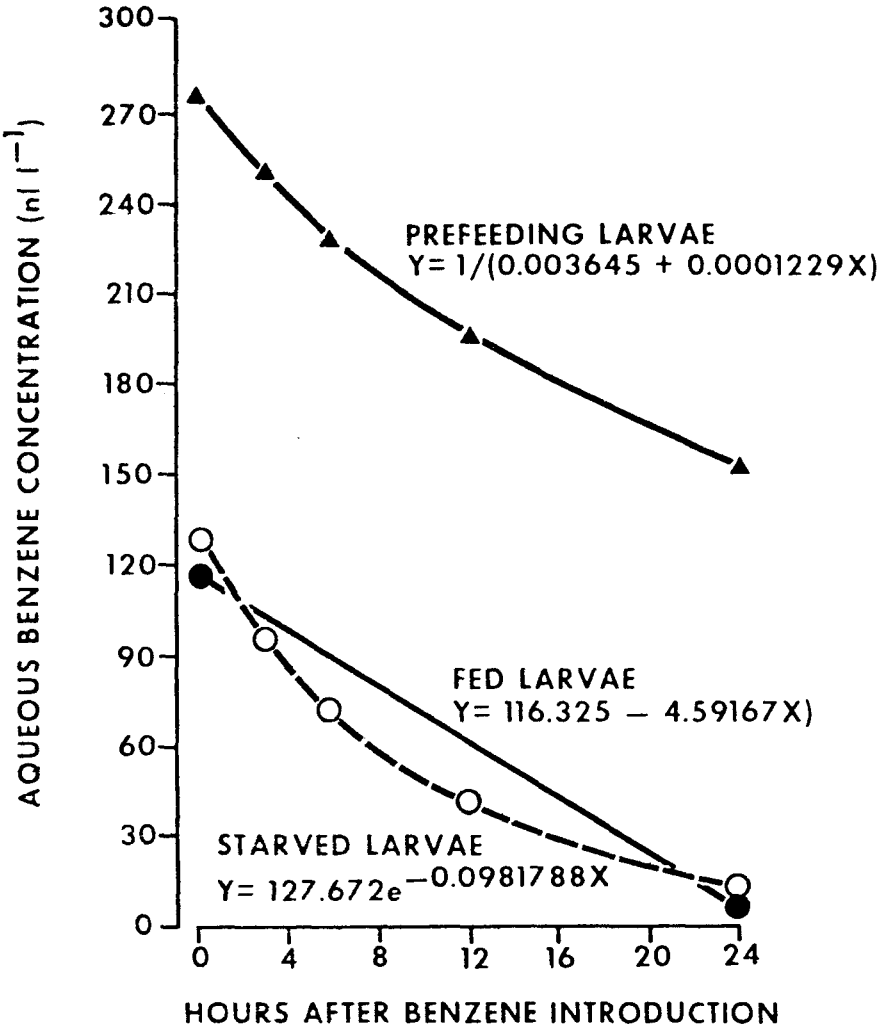

Figure 1. Benzene concentrations in test waters of prefeeding, feeding and starved larval containers during the first 24 h after initial exposure.

exposure concentration). Previous studies on striped bass larval feeding behavior indicate most of the daily food intake occurs within 10 h after food introduction (Eldridge et al., 1980b). Thus fish larvae fed on nauplii when they were most contaminated.

Concentration Factors

Differences in the maximum amounts of benzene accumulated in tissues of various life stages are presented in Table 6. Older, feeding larvae took up much more benzene, and thus achieved higher concentration factors than either prefeeding larvae or embryos. While embryos and prefeeding larvae had approximately the

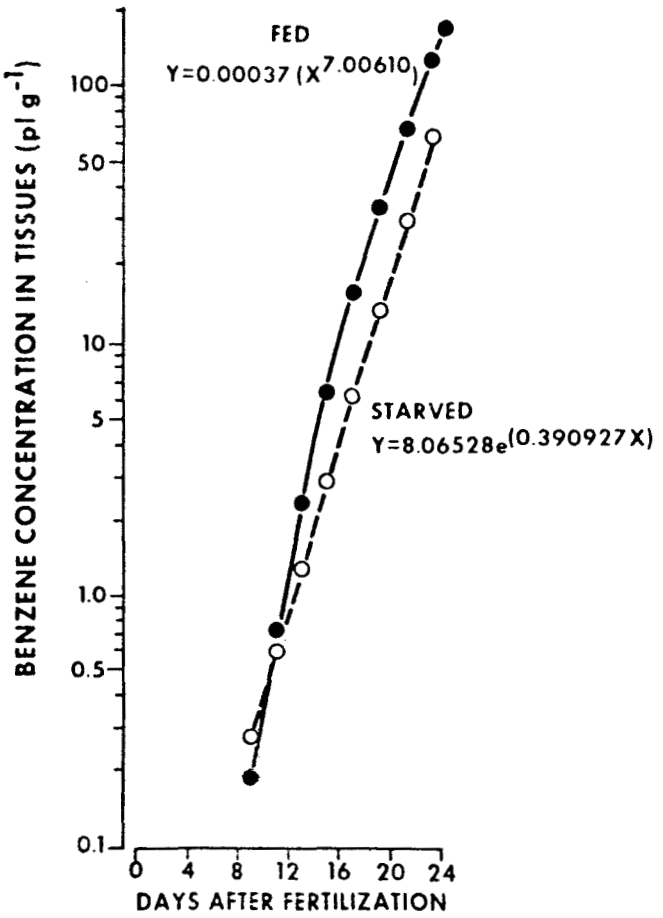

Figure 2. Benzene concentrations in the tissues of feeding and starved larvae from 7 to 29 days after fertilization.

same maximum tissue concentrations, feeding larvae never stopped accumulating benzene.

Survival

Embryos exposed to benzene had lower mortalities to hatching than unexposed (Table 7). Forty-six percent of exposed embryos successfully hatched as compared to 38% of controls. Benzene also caused earlier hatching; total hatching of exposed embryos was completed 5 h before that of controls. Despite better survival to hatching, larvae previously exposed in the embryo stage showed mortality rates higher than unexposed (Table 7) and fewer larvae made successful transition to active feeding.

All prefeeding larvae suffered from high mortality from D-4 to D-6. For the entire 5-day stage, exposed larvae died at rates twice as high as unexposed (Table 7). Delayed mortalities in both treatments were low. The few remaining exposed larvae

Table 5. Concentrations of C^{14}-benzene in culture water and tissues of *Artemia salina* nauplii over a 24-h period

Hours after Initial Exposure	Water ($nl\ l^{-1}$)	Tissue (nlg^{-1})
0	206	0*
1	194	320
3	172	312
6	150	918
12	109	557
24	59	702

*Below levels of detectability with our analytical method.

Table 6. Maximum accumulations and concentration factors of benzene in tissues of different life stages of striped bass

Life Stage	Means aqueous concentrations	Maximum tissue concentrations (plg^{-1})	Concentration factor
Embryo	138	4512	32.7
Prefeeding Larvae	415	4674	11.3
Feeding Larvae	159	226155	1422.4
Starved Larvae	159	73663	463.3

did have more larvae survive to D-29 than controls.

Essentially no effects from benzene exposure were seen in the survival of fed larvae, either during or after exposure (Table 7). Starved larvae, however, died in higher numbers in exposed treatments and near total mortality occurred by the end of the exposure period (D-24).

Growth

We found no significant differences in the sizes of newly hatched larvae from exposed and unexposed eggs. Standard length of exposed averaged 3.6 mm versus 3.5 mm for controls. Assimilated tissues amounted to 34 μg in exposed and 33 μg in controls. Growth of larvae from exposed eggs did achieve larger sizes than unexposed during the period D-2 to D-27. Daily instantaneous growth coefficients ($G_w = \ln (SL_1 - SL_3) / T$, where SL_i = initial size at beginning of test period, SL_e = size at end of test period, T = number of days in test period) for exposed were 0.072 and for control, 0.060.

Table 7. Daily instantaneous mortality* coefficients of striped bass embryos prefeeding larvae and feeding larvae under different benzene exposure conditions

Life Stage	Experimental Condition	Age (days)†	Benzene exposed	Unexposed
Egg	during exposure	0-2	.384	.485
	after exposure	3-29	.111	.085
Prefeeding Larvae	during exposure	2-7	.599	.241
	after exposure	8-29	.028	.050
Feeding Larvae	during exposure	8-23	.024	.029
	after exposure	24-29	-0-	-0-
Starved Larvae	during exposure	8-23	.200	.126
	after exposure	24-29	N/A‡	.081

*Daily instantaneous mortality = $\ln (N_{iT} - N_e)$, where N_i = initial number of embryos or larvae, N_e = number of animals at end of test period, and T = days in test period.

†Days after fertilization

‡Only 1 animal left for this experimental period.

Larvae growth in the short 5-day exposure period of the prefeeding larva stage was similar to that of the embryonic period — no significant differences due to benzene exposure. G_w values by standard lengths were the same (0.118) for both treatments. Larval dry weights were also indistinguishable (77 μg for exposed and 80 μg for controls). Later growth to D-27 was faster in exposed (G_w = 0.073) than controls (G_w = 0.060).

The effects of benzene on growth of larvae after D-8 was readily apparent (Fig. 3), not so much in their standard lengths as in tissue dry weights. After what appeared as a brief adjustment period from D-7 to about D-15, exposed larvae became longer and heavier. By D-29 controls had achieved the same lengths as exposed larvae but they remained lighter in weight.

Bioenergetics

Endogenous energy sources of striped bass eggs are comprised of yolk and oil (Eldridge et al., 1980a). Through standard calorimetric methods we determined the average caloric content of the eggs at the time of fertilization to be 2.231 calories per egg. Original yolk energy content was 0.537 calories and oil energy content 1.694 calories.

In the 2-day embryonic test period, benzene caused almost identical energy consumption of yolk and oil calories. Exposed embryos used 36% of its yolk energy and 26% of its oil energy. Control embryos used slightly less yolk, 31%, and 27% of its oil. After hatching larvae from exposed eggs did not differ from controls in their utilization of yolk and oil energies. All yolks were consumed by D-7. Oil globule

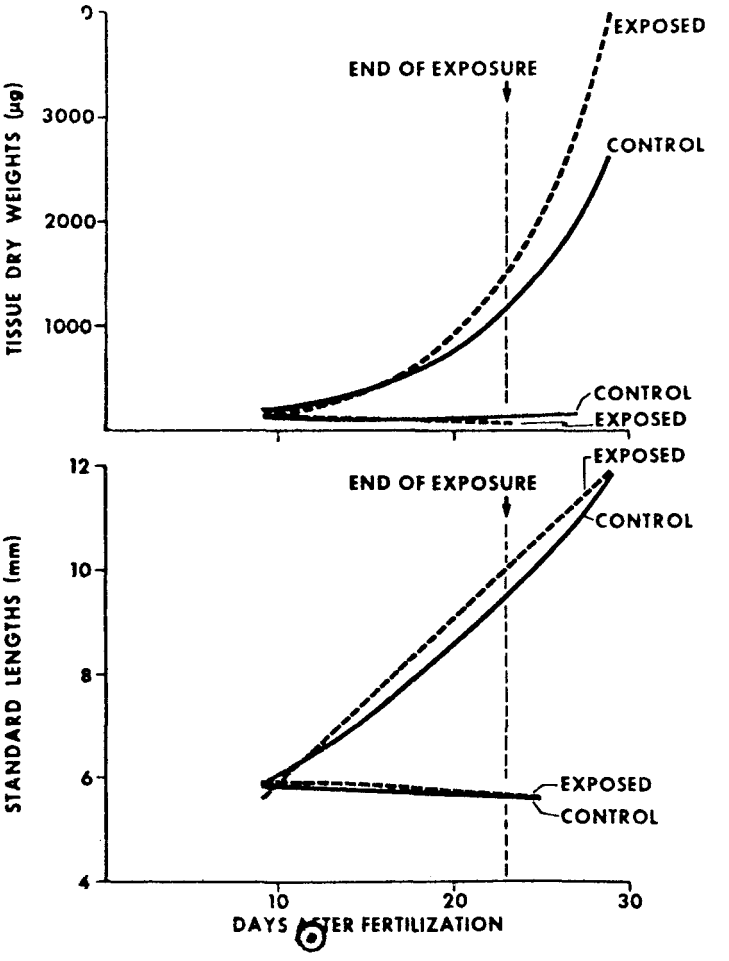

Figure 3. Tissue dry weights and standard lengths of striped bass larvae from 7 to 29 days after fertilization.

energy was used slowly and for a longer period in larvae from exposed eggs. Traces of oil were measured in these larvae to D-21 and only to D-17 in control larvae.

Endogenous energy consumption in prefeeding larvae also had no major differences due to benzene. Yolk from hatching to consumption was used at hyperbolic rates by both treatments. During exposure oil consumption was slightly faster in controls but larvae reared to metamorphosis actually retained their oil globules longer (to D-23 as opposed to D-21). Best fit equations for oil consumption were Y = 1.304-0.056 X for controls and Y = 1.516-.068 X for exposed, where Y = oil calories and X = age in days after fertilization.

Endogenous energy for older, feeding larvae came solely from the oil globule. No significant differences were seen in the consumption of this energy in either starved or fed animals (Fig. 4). Though not statistically different, starved larvae exposed to

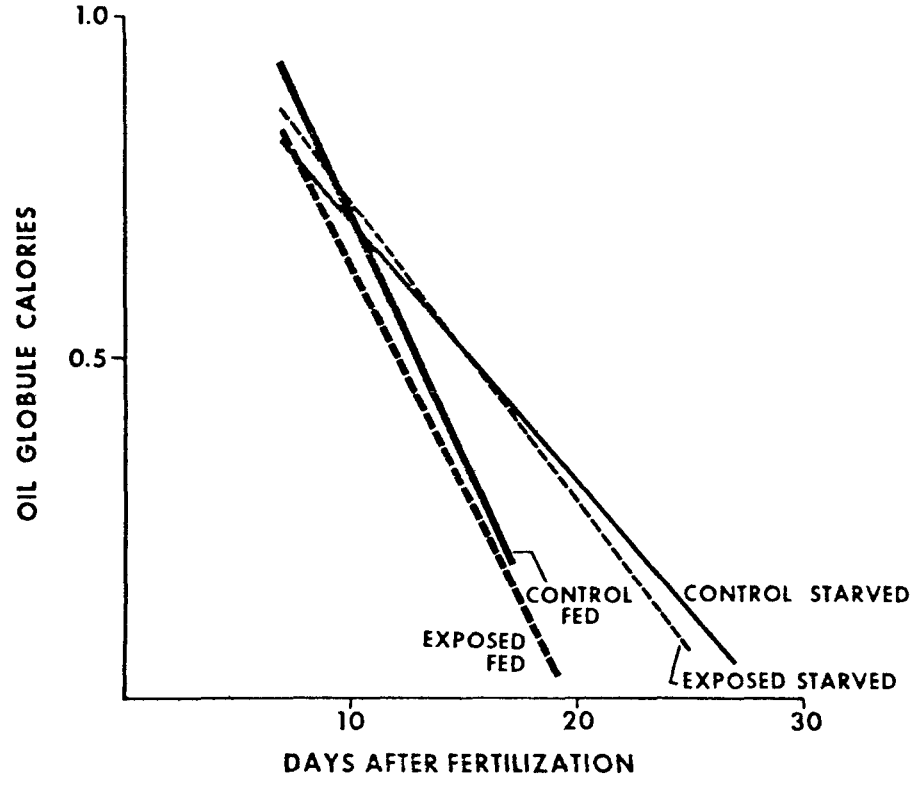

Figure 4. Oil globule calories in striped bass larvae 7 to 29 days after fertilization.

benzene had a faster use rate than controls ('b' value for exposed was -0.044 for exposed and -0.039 for controls). Fed larvae have previously been found to use their oil energies at faster rates than starved animals (Eldridge et al., 1980b). Controls were found to have slightly faster use rates (b = -0.074) and exhausted their oil 2 days before exposed animals (b = -0.067).

Exogenous energy input was estimated for feeding larvae by counting larval stomach contents and using a formula for daily food ration (D.F.R. = (stomach contenst) x (hours of active feeding)/digestive time). Values for feeding time and digestion were predetermined (Eldridge et al., 1980b). Exposure to benzene seemed to reduce the numbers of larvae successfully feeding up to about D-17 (Table 8). After D-17, unexposed were less or about the same. The daily food rations of controls exceeded those of exposed; 7 of 10 ration estimates showed controls with larger rations.

Oxygen consumption was markedly elevated by benzene (Fig. 5). On a weight specific basis benzene increased metabolism in all life stages. Figure 5 also shows the

Table 8. Feeding incidence (percent of sampled specimens with identifiable food in stomach) and estimated daily ration (given in numbers of *Artemia salina* nauplii and equivalent calories)

Age (Days after fertilization)	Percent with food in stomach		Daily food ration			
	Control	Exposed	Control		Exposed	
			Number of Nauplii	Calories	Number of Nauplii	Calories
9	90	40	49.5	0.476	15.2	0.146
11	70	70	34.2	0.329	29.0	0.279
13	100	100	68.8	0.661	63.3	0.609
15	70	50	59.4	0.571	32.7	0.315
17	90	70	57.9	0.556	57.9	0.556
19	30	70	12.1	0.116	14.2	0.137
21	50	60	20.6	0.198	19.1	0.183
23	100	100	178.5	1.715	112.1	1.077
25	100	100	240.6	2.312	267.9	2.574
27	100	50	170.9	1.642	32.1	0.309

larger sizes attained by exposed larvae.

DISCUSSION

Developments in analytical capabilities have allowed scientists to detect extremely low concentrations of aromatic hydrocarbons, including benzene, in waters where previously they were believed to have volatilized. Results of our study demonstrated that planktonic striped bass eggs and larvae take up benzene at low sublethal concentrations and accumulate it to levels many times that of the surrounding water. Early life stages of other fish species have also been found to accumulate benzene and related monoaromatic hydrocarbons. They did not, however, concentrate them to such high degrees and they differed in the life stages which took up the most hydrocarbons and the times required to reach equilibrium. In a study by Eldridge et al. (1977) Pacific herring eggs and larvae all reached equilibrium with benzene within 6-12 h. Eggs accumulated the most benzene, 10.9 times the initial concentration, yolksac larvae, 6.9 times, and feeding larvae, 3.9 times. The reverse pattern was found by Korn and Rice (1980). Coho salmon alevins preceded fry, which exceeded eggs in the uptake and accumulation of toluene. No stage achieved a concentration factor higher than about 20. Salmon eggs required 10 days to reach equilibrium, alevins around 36 h, and fry only 3 h. Striped bass eggs, in continuous exposure, never reached an equilibrium state by hatching. Later larval stages did likewise. We believe striped bass continued to accumulate benzene

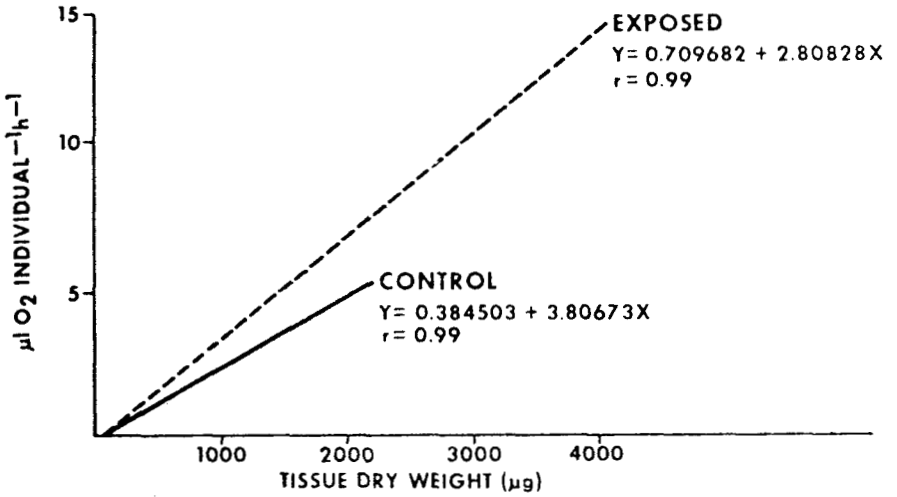

Figure 5. Oxygen consumption of striped bass embryos and larvae from fertilization to 29 days after fertilization.

because of its extraordinary high amount of oil, located mainly in the oil globule. Fifty two percent of the embryo's total dry weight consists of this oil (Eldridge et al., 1980a). Older larval stages may relocate these lipid stores in other areas of their bodies as oil globules are consumed.

Greater amounts of benzene in tissues of feeding larvae as compared to starved larvae and the uptake of benzene measured in *Artemia* nauplii lead us to conclude that ingested contaminated food served as a pathway for benzene into larval tissues. Herring larvae feeding on live rotifers showed similar results (Eldridge et al., 1977; Echeverria, 1980). Nonetheless benzene in water still appears to be the principal source of the hydrocarbon.

Among the effects due to exposures to low level sublethal concentrations of benzene (Table 9) direct mortality was not readily apparent. Delayed mortalities occurred in larvae from exposed eggs, exposed prefeeding larvae had slightly higher mortalities, and feeding larvae did not seem affected. The older starved larvae, however, suffered most from the added stress of benzene. Their behavior was different from unexposed, starved larvae in that they were much less active and they died much sooner than their unexposed counterparts. This suggests that larvae feeding on less than maximum food rations have a potential for higher mortalities.

Growth of feeding larvae, measured in tissue weight, was definitely advanced by benzene exposure. Similar findings were found by Eldridge et al., (1977) in Pacific herring exposed to low benzene concentrations. The proposed explanation for the herring results seems also appropriate with striped bass. That is, benzene in low chronic doses stimulates adaptive physiological responses which are often measured in what might be interpreted as beneficial mode. The hypothesis is termed "sufficient challenge" (Smyth, 1967).

Among the energetic variables measured, the consistently higher metabolism of exposed animals is most noteworthy. Brocksen and Bailey (1973) found that low concentrations of benzene increased oxygen consumption in striped bass juveniles.

Table 9. Summary of multi-disciplinary study on the effects of low level concentrations of benzene on early lifestyles of striped bass

Life Stage	Uptake accumulation and depuration	Mortality	Growth	Bioenergetics		
				Endogenous Energy	Exogenous Energy	Metabolism
Embryo	Rapid and continuous uptake; 33x concentration factor; rapid release.	Benzene enhanced survival to hatching but offset by delayed mortality.	No effects during exposure; larvae from exposed egg larger.	No distinct differences.	———	Benzene caused increase.
Prefeeding larvae	Rapid uptake; gradual accumulation; lowest concentration (llx) factor.	Benzene increased mortality during exposure.	No effects during or after exposure	Slightly faster oil utilization in controls.	———	Benzene caused increase.
Feeding larvae	Rapid uptake and rapid loss; continuous accumulation; benzene entered tissues via food; very high concentration factor in fed larvae (1422x).	No effects on fed larvae; starved larvae suffered high mortalities from benzene.	More tissue assimilation due to benzene.	Benzene slightly enhanced oil consumption in fed larvae.	Benzene reduced feeding incidence and ration amounts.	Benzene caused increase.

Evidently low level chronic concentrations of benzene elicit resistent stress responses. In the general adaptation syndrome (G.A.S.) caused by stress, animals first have an alarm reaction followed by a stage of resistance. If the stress is highly concentrated or prolonged, a stage of exhaustion then takes over (Selye, 1950). We hypothesize that the high metabolic rates and possibly the increased growth caused by low benzene concentrations are due to the animals being in the second stage of the G.A.S., that of resistance. Exposed striped bass embryos and larvae accelerate development (i.e., early hatching, faster anabolism) since it is to the animal's advantage to grow and differentiate so as to increase its resistance capabilities (such as increased mobility, detoxification capability). The sufficient challenge effect is then interpreted as a resistance reaction which, over the short term, more than meets the needs to counter the stress and temporarily benefits the animal. Prolonged exposure could eliminate these benefits if the animal becomes exhausted. To fully explore this possibility it will be necessary to expose early life stages through and beyond metamorphosis.

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