

## Using Nested Models and Laboratory Data for Predicting Population Effects of Contaminants on Fish: A Step Toward a Bottom-Up Approach for Establishing Causality in Field Studies

K A. Rose,<sup>1\*</sup> C.A. Murphy,<sup>1</sup> S.L. Diamond,<sup>2</sup> L.A. Fuiman,<sup>3</sup> and P. Thomas<sup>3</sup>

<sup>1</sup>Coastal Fisheries Institute and Department of Oceanography and Coastal Sciences, Louisiana State University, Baton Rouge, LA, USA 70803. <sup>2</sup>Department of Biology, Texas Tech University, Lubbock, TX, USA 79409. <sup>3</sup>Department of Marine Science, University of Texas at Austin, Marine Science Institute, Port Aransas, TX, USA 78373

### ABSTRACT

Predicting the effects of contaminants on fish populations is difficult due to their complex life history and high interannual variation in their population abundances. We present an approach that extrapolates laboratory data on contaminant effects, including behavioral effects, to the population level by using a series of nested statistical and simulation models. The approach is illustrated using PCB effects on Atlantic croaker. Laboratory experiments were performed that estimated PCB effects on fecundity, egg mortality, and the swimming speed and predator evasion behavior of larvae. A statistical model converted impaired predator evasion to reduced probability of escaping a predatory fish. An individual-based model then converted the output of the statistical model into changes in larval stage duration and survival, which were used to change elements of the matrix model. A matrix projection model simulated population dynamics for 100 years for baseline conditions and for two hypothetical PCB exposure scenarios. PCB effects were imposed in the model by reducing the fecundity of exposed adults, increasing egg mortality, and increasing the larval stage duration and mortality rate. Predicted population effects of PCBs were small relative to the interannual variation. Our analysis is a step toward understanding population responses to stressors and for ultimately establishing causality in field situations.

**Key Words:** causation, endocrine disruptors, population dynamics, models, Atlantic croaker, PCBs.

---

\* Corresponding author: Tel(voice): 225-578-6346, Tel(fax): 225-578-6513; karose@lsu.edu

## INTRODUCTION

Establishing cause and effect is a critical aspect of assessing the risks of stressors on fish. Ecological risk analysis involves many assumptions, uncertainties, and decisions (Suter 1993). Understanding the underlying causes of a population decline or a shift in community composition provides a basis for intelligent formulation and implementation of a risk assessment (Stahl and Clark 1998). Information on cause and effect reduces the uncertainties of a risk analysis by providing a sound basis for data interpretation and model formulation.

Establishing causality in field situations where fish are exposed to contaminants has mostly involved correlative analysis or evidence of exposure at the individual level. A notable exception was the extensive laboratory toxicity and field studies that documented the decline of lake trout in Lake Ontario during the 1950s due to contaminants with dioxin (TCDD)-like activity (Ankley and Geisy 1998). In the vast majority of studies, however, the effects of contaminants on wildlife, and especially fish, have been difficult to separate from the effects of natural factors (*e.g.*, temperature, food availability) and other anthropogenic stressors (Sinderman 1996; Giesy and Snyder 1998; Rose 2000). We simply do not have a sufficient understanding of how the many factors affecting population dynamics interact to permit us to perform quantitative analyses that isolate the effects of contaminants. Thus, analyses of contaminant effects generally resort to correlations between long-term field data and water quality variables (*e.g.*, Rose and Summers 1992; Chesney *et al.* 2000), or to measurements of exposure on individuals (*e.g.*, Holdway *et al.* 1995; Karels *et al.* 2001). Biomarkers are an excellent example of the use of individual measurements for inferring contaminant exposure (Adams 1990; McCarthy and Shugart 1990); however, inferring ecological effects from biomarkers has proven to be more difficult than inferring exposure (Chambers *et al.* 2002; McCarty *et al.* 2002).

Fish are especially challenging for contaminant cause and effect studies because they exhibit wide variation in population abundance and have complex life histories. Fish tend to be long lived and effects on young fish may not have population implications until years later when these individuals mature. Fish populations are notorious for exhibiting wide interannual fluctuations in abundance (Rothschild 1986; Fogarty *et al.* 1991), often driven by variation in environmental (natural) factors affecting early life stage growth and survival (Shephard *et al.* 1984; Laevastu 1993). Fish also have complex life histories with life stages that utilize different habitats. Inhabiting multiple habitats complicates monitoring and bioassessment, and makes determination of routes of exposure and identification of key bottlenecks in the population life cycle difficult. Fish populations have declined worldwide in recent decades (Garcia and Newton 1997). While overfishing has played a major role, there are also concerns about the role played by deteriorating water quality and habitat loss (Jackson *et al.* 2001). Yet, widespread demonstration of contaminant effects causing declines in fish populations remains elusive (Sindermann 1996; Rose 2000).

Endocrine disrupting chemicals (EDCs) further add to the difficulties in establishing cause and effect relationships between contaminants and fish populations. EDCs are exogenous chemical substances that interfere with the endocrine systems responsible for the maintenance of homeostasis and for the regulation of reproduc-

## Models and Data for Predicting Population Effects

tive and developmental processes. There has been much recent interest in EDCs (Colborn and Thayer 2000; Kolpin *et al.* 2002). Numerous biomarkers have been measured that demonstrate abnormalities of endocrine function in fish and wildlife when exposed to industrial and domestic wastes (Holdway *et al.* 1995; Rolland *et al.* 1997; Kime 1998). Although many of these biomarkers show clear effects at the individual level, many are difficult to directly relate to ecologically relevant endpoints (*e.g.*, mortality), and it is even more difficult to determine their effects on the population within the context of typically highly variable field situations. The ecological implications of EDCs on fish population dynamics remain unclear (Arcand-Hoy and Benson 1998). Our ability to extrapolate mortality effects of EDCs to the population level is technically feasible using population models, although population modeling can be contentious because of disputes over the appropriate assumptions in models and what constitutes model validation (Barnthouse *et al.* 1984; Rykiel 1996; Schnute and Richards 2001). Little progress has been made in quantitatively extrapolating sublethal effects, such as impaired responses to predators (Faulk *et al.* 1999), to the population level. We can measure behavioral effects of contaminants on individuals in the laboratory (von Westernhagen 1988; Little *et al.* 1993), but whether these effects are of ecological significance in field situations remains generally unknown. To fully investigate the cause and effects of EDCs and other contaminants on fish populations, we need to establish a quantitative link between behavioral and physiological effects and population responses (see Johnson and Collier 2002).

In this study, we apply a series of linked statistical and simulation models, coupled with laboratory and field data, to determine the likely magnitude of responses of Atlantic croaker (*Micropogonias undulatus*) to PCB exposure. PCBs are an EDC that is widely distributed in coastal waters, and whose effects on fish reproduction and development have been studied extensively (Monosson 2000). We include the effects of PCBs on egg mortality, fecundity, and the sublethal behavioral responses of fish larvae to their predators. This approach is a bottom up approach to causality, that starts with laboratory experiments on mortality, fecundity, and behavioral effects, which are then carried through a series of models, culminating in 100 year simulations of population dynamics without and with PCB effects. Model simulations provide information on the magnitude of the contaminant effect expected in the field situation. Three models are used (statistical, individual-based, and matrix projection), where the laboratory data on behavioral effects are inputs to the statistical model, the outputs of the statistical model are then used as inputs to the individual-based model, and finally the outputs of the individual-based model are used as inputs to the matrix projection model. The matrix projection model then simulates population dynamics for 100 years. Our analysis using a widely distributed, long-lived coastal fish species such as croaker, and including lethal and behavioral effects of a common EDC such as PCBs, demonstrates the general feasibility of predicting EDC effects on fish population dynamics.

This paper is organized as follows: We first provide a brief background on the life history of Atlantic croaker. We then present a summary of the results of the laboratory experiments on PCB effects on egg mortality, fecundity, and behavior of larvae. This is followed by the description and results of each of the three models. The method and results are presented together to emphasize the sequential nature

of the approach. Analyses reported in this paper are preliminary and should not be interpreted as predictions of PCB effects on the actual mid-Atlantic Bight croaker population. Our primary reason for using croaker was to use an important marine species and associated data to ensure realism of the models. However, the data and models are preliminary, and the PCB exposure scenarios examined are hypothetical. Our goal is to show how we can combine laboratory experiments and modeling to extrapolate lethal and other effects of EDCs to the population level. This information can then be used to help establish causal relationships between specific stressors and population responses.

### ATLANTIC CROAKER LIFE CYCLE

Atlantic croaker is a good species to demonstrate our modeling approach for extrapolating toxicity to the population level. Croaker are widely distributed and common along the east coast of the United States, typify a common fish life history strategy of using estuaries as nursery areas, and are well studied in the laboratory (Thomas and Khan 1997; Khan and Thomas 2000) and in the field (Diamond *et al.* 1999, 2000). Our modeling focuses on croaker in the mid-Atlantic Bight and in the estuarine waters of North Carolina and Virginia. The life cycle of croaker in the mid-Atlantic Bight has been described elsewhere (Able and Fahay 1998; Diamond *et al.* 1999, 2000); we summarize the life cycle here. Adults live in the ocean, migrating south during the fall and winter, and returning in the spring. Atlantic croaker are highly fecund batch spawners (each female distributes her annual production of eggs across several spawning events). Spawning occurs July through March. There appear to be two major spawning modes per year (roughly August-October and January-March) based on the abundance of larvae in North Carolina and Virginia estuaries (Sandra Diamond, unpublished data). Eggs are spawned offshore and hatch into yolk-sac larvae in about 2 days. Yolk-sac larvae initiate exogenous feeding after about 4 to 5 days, and become ocean larvae. Ocean larvae feed on zooplankton (Govoni *et al.* 1986) and are transported inshore by water circulation (Miller *et al.* 1984). After 30 to 60 days, the ocean larvae arrive at the estuary at a length of about 12 mm (Warlen 1982; Nixon and Jones 1997), when they become estuarine larvae. Estuarine larvae transit through the estuaries to reach the primary nursery areas (shallow, brackish creeks and marshes), where they become early juveniles at about 20 mm in length. After reaching 40 to 60 mm, early juveniles become late juveniles and move into secondary nursery areas (deeper sounds, bays, and channels). Most late juveniles migrate to the ocean to join the adults during the fall of their first year, along with those adults that spent their summer in the estuary. About 50% of Atlantic croaker mature at the end of their first year, and 100% of age-2 and older croaker are mature. Historically, adults lived to about 15 years (Hales and Reitz 1992), but a maximum age of 8 to 12 years is more common now due to environmental conditions and fishing pressure.

### MODELING PCB EFFECTS ON CROAKER: METHODS AND RESULTS

#### Overview of Approach

The linkages among the three models (statistical, individual-based, and matrix projection), and between the laboratory and field data and the models, are shown

## Models and Data for Predicting Population Effects

schematically in Figure 1. The results of three PCB laboratory experiments were used to determine the adjustments to the baseline values assumed for fecundity, egg survival, and predator evasion ability of croaker larvae. The reduction in fecundity due to PCB exposure was used directly to adjust the fecundity parameters of the matrix projection model. The reduction in egg survival due to PCB exposure, with some minor calculations, was used to adjust the parameters (denoted G and P) associated with egg stage survival in the matrix model. The reduction in predator evasion ability of the larvae was processed through the statistical and individual-based models in order to obtain the appropriate adjustments to the ocean larva stage parameters of the matrix projection model. Field data were used to configure the matrix model and to ensure matrix model predictions were realistic.

### Laboratory Studies

The results from the low-dose treatments of three PCB experiments were used in this paper (Peter Thomas, unpublished data; McCarthy *et al.* in press). All three experiments used female croaker that were exposed to PCB via their diet. Females were obtained from a relatively pristine estuarine area near Port Aransas, Texas, and either used as controls (no PCBs) or exposed to a low dose (0.4 mg PCB/kg fish/day) for 2 weeks. The low-dose treatment resulted in realistic (albeit high) PCB levels in fish compared to PCBs measured in field monitoring studies of fish. Body burdens of PCBs in the low-dose exposed females were compared to field values of PCBs in croaker and in other species (Figure 2). Field measurements varied in their units and in their tissue of measurement. In order to compare among our laboratory values and the field values, we converted all values to ng PCB per g dry weight (dw) of liver (ppb) using a conversion of 20% dry weight to wet weight and a ratio of concentrations of liver to ovary to muscle of 1 to 2.5 to 0.5. Thus, 1.0  $\mu\text{g}$  of PCB measured in the liver is assumed equivalent to 2.5  $\mu\text{g}$  in the ovary and to 0.5  $\mu\text{g}$  in the muscle. The average PCB concentration in low-dose-treated females was 3.3  $\mu\text{g/g}$  dw of ovary (equivalent to 8250 ng/g dw of liver). This concentration was higher than PCBs measured in croaker and near the upper end of PCBs measured in all fish sampled by the NOAA Status and Trends Program (see Turgeon *et al.* 1992 for methods), but less than the very high concentrations found in highly PCB-contaminated systems such as striped bass in the Hudson River (Zlokovitz and Secor 1997) and largemouth bass in Hartwell Reservoir (Adams and Greeley 1991).

The first laboratory experiment measured the fecundity (eggs/g of ovary) of 17 individual control and 13 individual low-dose exposed females (Figure 3a). Fecundity was measured by counting oocytes in subsamples of ovarian tissue. Average fecundity was reduced by 13%, from 3300 eggs/g in controls to 2879 eggs/g in the low-dose PCB-exposure (Peter Thomas, unpublished data).

The second laboratory experiment measured egg survival (percent) to 24 h post-fertilization (Figure 3b). Two females and two males were placed into tanks and injected with luteinizing hormone releasing hormone analogue (LHRHa) to induce spawning. Eggs were collected from each of five tanks for control and five tanks for low-dose exposed females and males, and monitored for survival. Egg survival after 24 h averaged 43% for the PCB-treated females vs. 87% for control females, or a 51% decrease in egg survival due to PCBs (Peter Thomas, unpublished data).

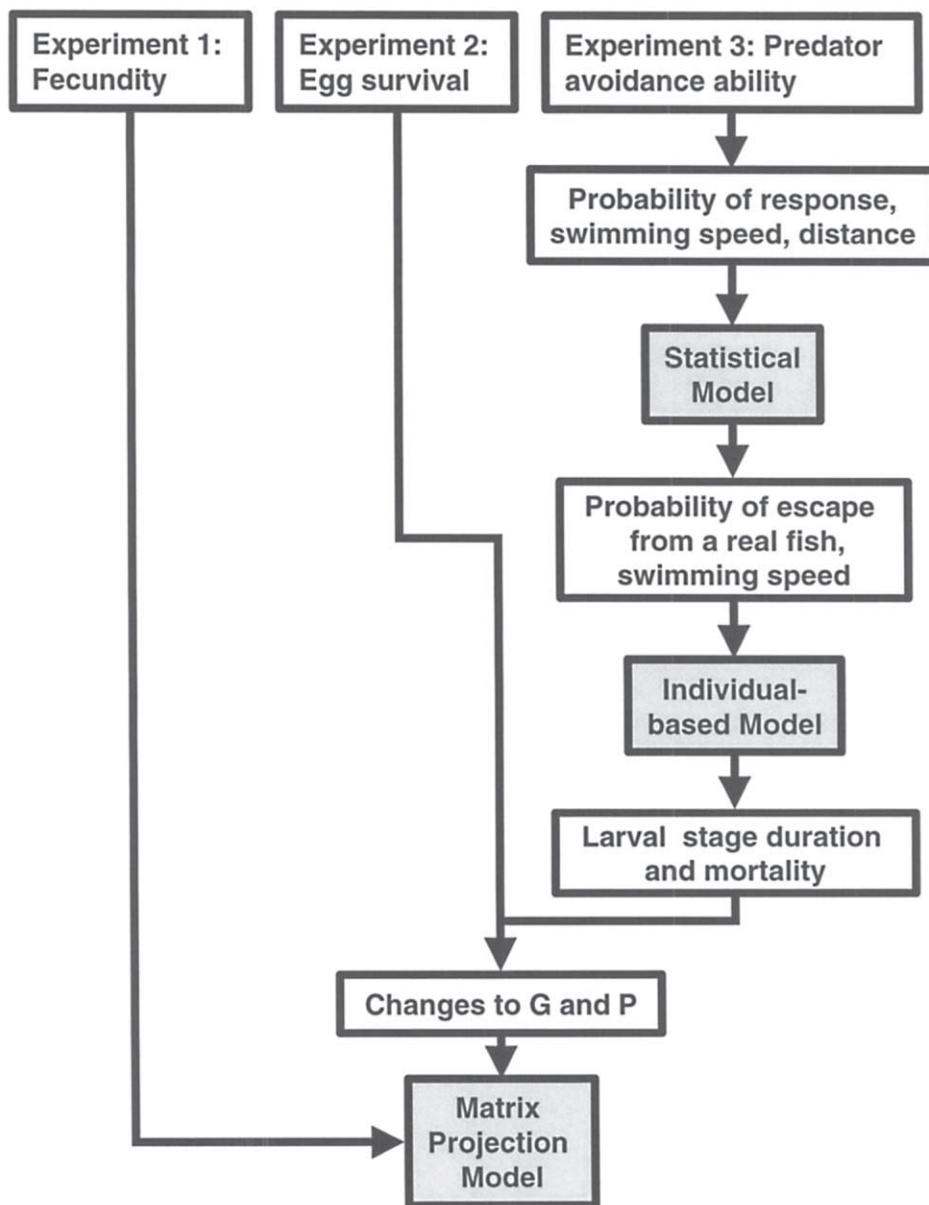
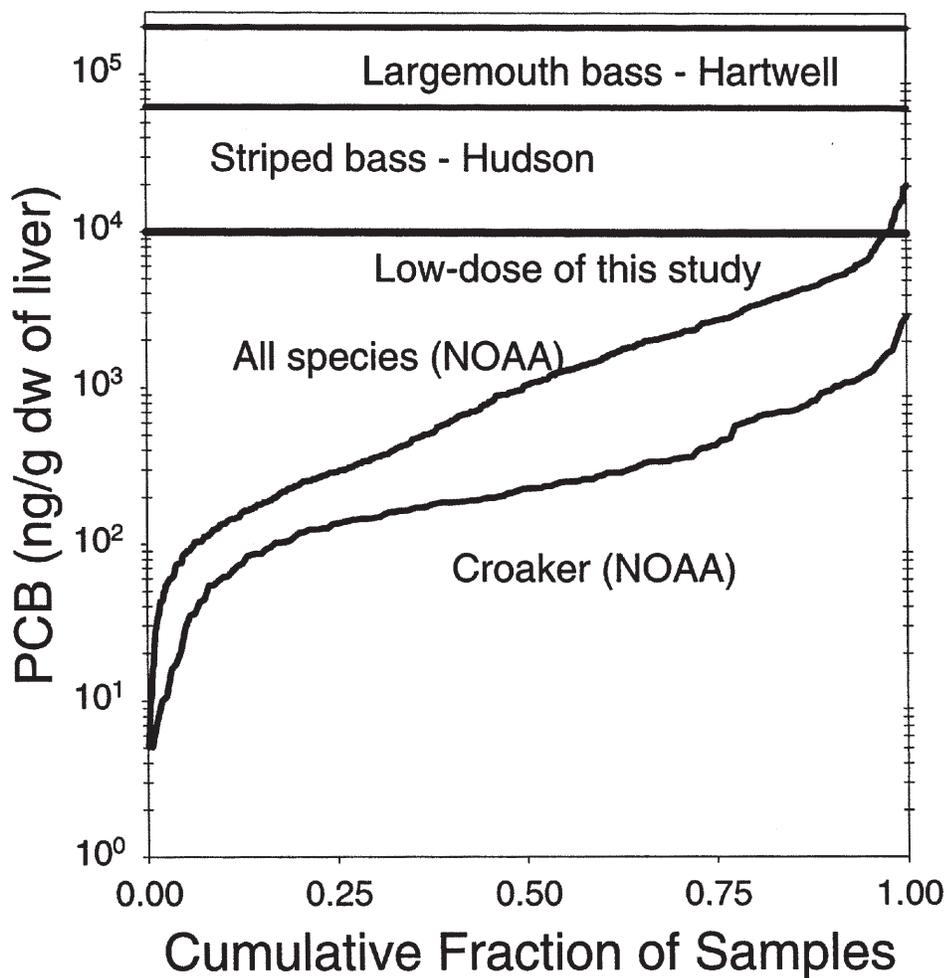
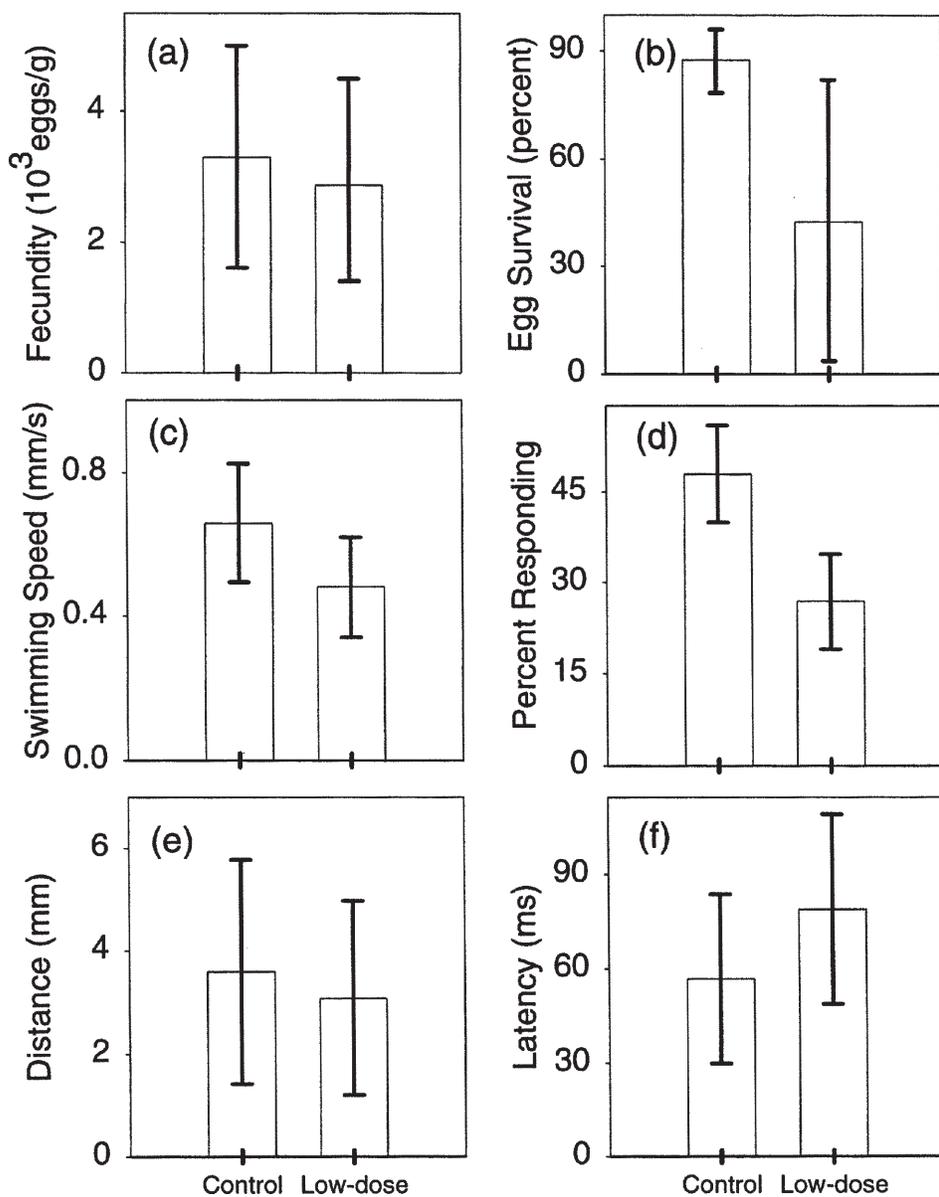


Figure 1. Schematic representation of the relationship among the three models and the three laboratory experiments for extrapolating toxicity results to the population level. G and P are parameters of the matrix projection model.



**Figure 2.** Cumulative distributions and average values of PCB concentrations in field-caught fish compared to the average PCB concentration in female croaker of the low-dose treatment of the laboratory experiments. Data are shown for all fish species and for croaker only measured in the NOAA Status and Trends Program (<http://ccmaserver.nos.noaa.gov>), and for two highly PCB-contaminated locations (striped bass in the Hudson River; largemouth bass in Hartwell Reservoir). The NOAA data were for ten fish species located on the east, west, and Gulf coasts of the US; measurements were made on individuals whose length would indicate they were sexually mature. The mean PCB concentrations of female striped bass were from “hot” areas reported in Table 1 of Zlokovitz and Secor (1997). Adams and Greeley (1991) reported an average PCB concentration of 20 ppm in the muscle of largemouth bass sampled in the polluted area of Hartwell Reservoir.



**Figure 3.** Results from the control and low-dose treatment (mean  $\pm$  SD) of the three laboratory experiments of PCB effects on croaker. (a) fecundity, (b) egg survival, (c) swimming speed, (d) percent responding, (e) distance of response, (f) latency of response. (a) is from Experiment 1; (b) is from Experiment 2; and (c) - (f) are from Experiment 3.

## Models and Data for Predicting Population Effects

The third laboratory experiment involved quantifying the effects of PCBs on the predator evasion behavior of croaker larvae. Larvae at hatching (.2.5 mm in length) were obtained from females exposed to the low-dose PCBs at the same time as females were exposed for the first and second experiments. Individual larvae were then videotaped as they responded to a vibrational stimulus applied to the container (McCarthy *et al.* in press; see Faulk *et al.* 1999 and Fuiman *et al.* 1999 for general methods). The vibrational stimulus was designed to elicit a startle response, similar to that used to evade an attacking predator. The responses of each larva to the vibrational stimulus were measured for five trials at 5, 9, and 13 d after hatching. The videotape was used to determine if the larva responded to the stimulus, and if the larva responded then the videotape was used to determine the time before responding (latency, millisecond) and the distance (mm) and duration (sec) of the response. The five trials were collapsed into single values of percent responding (out of 5), and the average latency, distance, and duration of responses. Percent responding is a measure of the responsiveness of the larva. In addition, the routine swimming speed of individual larva was estimated from videotaping 10 randomly selected larvae for a randomly selected 30-sec period in a 3-min tape. Routine swimming speed reflects the rate of encounter between a larva and its predators and prey. We used the values at 13 d after hatching for 61 control larvae and for 35 larvae from PCB-exposed females.

PCBs affected the predator evasion behavior of the croaker larvae. PCB-exposed larvae had slower average routine swimming speeds (Figure 3c) and a lower percent responding (Figure 3d) than control larvae. When larvae responded, PCB-exposed larvae exhibited responses that were of slightly shorter average distance (Figure 3e) and they took longer (higher latency) to respond (Figure 3f) than control larvae (McCarthy *et al.* in press provide a detailed analysis).

### Statistical Model: From Larval Behavior to Predator Escape Methods

A previous study measured the responses of individual red drum (*Sciaenops ocellatus*) larvae to artificial predator stimuli, as well as their responses to a real predatory fish (Fuiman and Cowan in press). A regression tree was fitted to the data from this previous study. The regression tree related the explanatory variables of routine swimming speed and responses of the red drum larvae to an acoustic stimulus to the response variable of the probability that a larva would escape an attack from the real predatory fish (Lee Fuiman unpublished data). The fitted regression tree was based on control larvae, as no contaminants were used in the study. We assumed that the results of the acoustic stimulus and predatory fish experiment with red drum larvae could also be used to estimate the probability of croaker larvae escaping a real predator based on croaker responses to a vibrational stimulus. The regression tree enabled us to compare how changes in swimming speed and responses to the vibrational stimulus due to PCB exposure in croaker larvae would translate into changes in the probability of croaker larvae escaping a real predatory fish.

Regression trees are a relatively new statistical technique that offer advantages over classical linear regression. Regression trees do not require that the response variable be related to the explanatory variables with the same functional form over

the entire range of the data. Regression trees successively split the response variable (probability of escaping a real predator attack) into branches based on the values of explanatory variables (*e.g.*, swimming speed, distance of response). Each explanatory variable, and each possible value of each explanatory variable, are evaluated and the variable and split value that provides the greatest improvement in fit (measured by reduction in mean square error between predicted and observed) is selected. This process is repeated on the observations in each branch until subsequent splitting would result in a branch with too few observations. Because the resulting regression tree is overspecified, a pruning algorithm is applied to the regression tree to remove some of the branching that reduced the mean square error, but did not reduce the mean square error enough to justify the added complexity of the tree (Breiman *et al.* 1998).

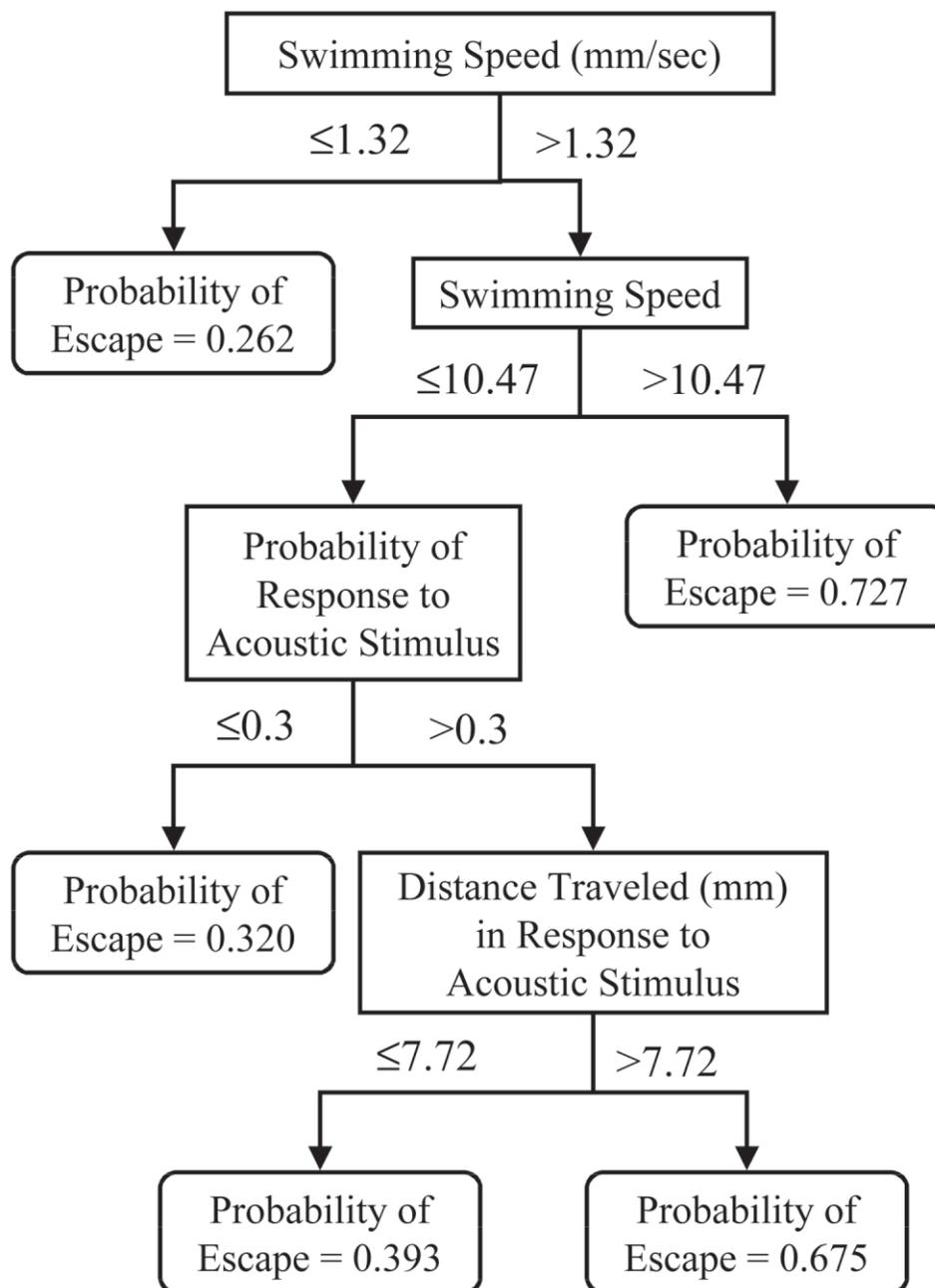
The regression tree estimated from the previous study that used red drum larvae exposed to both the acoustic stimulus and the real predatory fish is shown in Figure 4. Probability of escaping an attack from a real fish predator generally increased in larvae that had faster swimming speeds, greater responsiveness to the acoustic stimulus, and responses of longer distances. To account for differences between the previous study that used red drum larvae and the PCB experiment used here that used croaker larvae, we adjusted the swimming speed and average distance of the responses measured on the control croaker larvae. The swimming speed and distance of responses of the control larvae were adjusted so that the mean values of the control croaker larvae equaled the mean values of the red drum larvae. The same adjustments were then applied to the values of swimming speed and distance of responses measured in the PCB-exposed croaker larvae. The adjusted swimming speed, adjusted distance of response, and the probability of responding (responsiveness) were used in the regression tree to predict the probability of escaping a real predatory fish for each control croaker larva and for each PCB-exposed croaker larva.

## Results

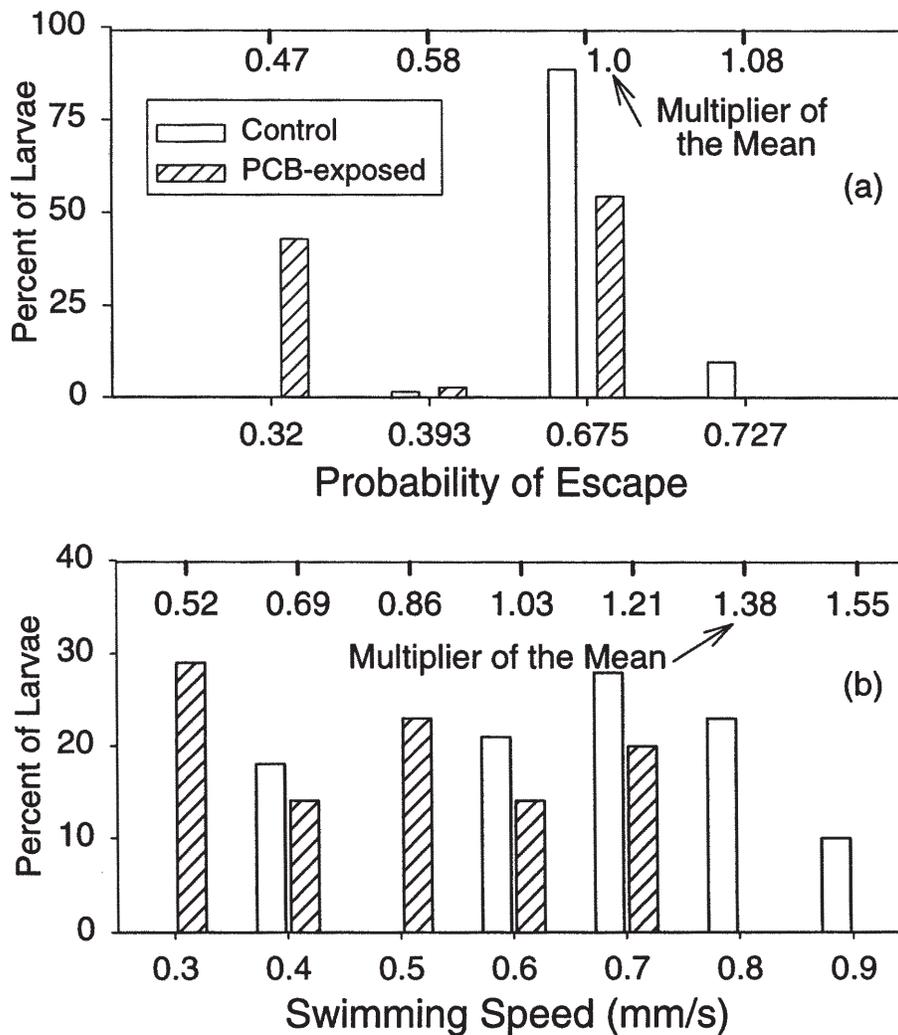
Croaker larvae from PCB-exposed females had generally slower swimming speeds and lower predicted probabilities of escaping a real predatory fish than control larvae (Figure 5). Bar charts of the percentages of larvae with different values of probability of escape (Figure 5a) and swimming speed (Figure 5b) were shifted toward the left (smaller values) for the PCB-treated larvae. Note that none of the control or PCB-treated larvae resulted in the lowest predicted values of probability of escape of 0.262. The multipliers of the mean values of the control larvae were inputted into the individual-based model to convert differences in swimming speeds and probabilities of escape between control and PCB exposed larvae into differences in larval cohort duration and survival.

## Individual-Based Model: From Predator Escape to Larval Stage Duration and Survival Methods

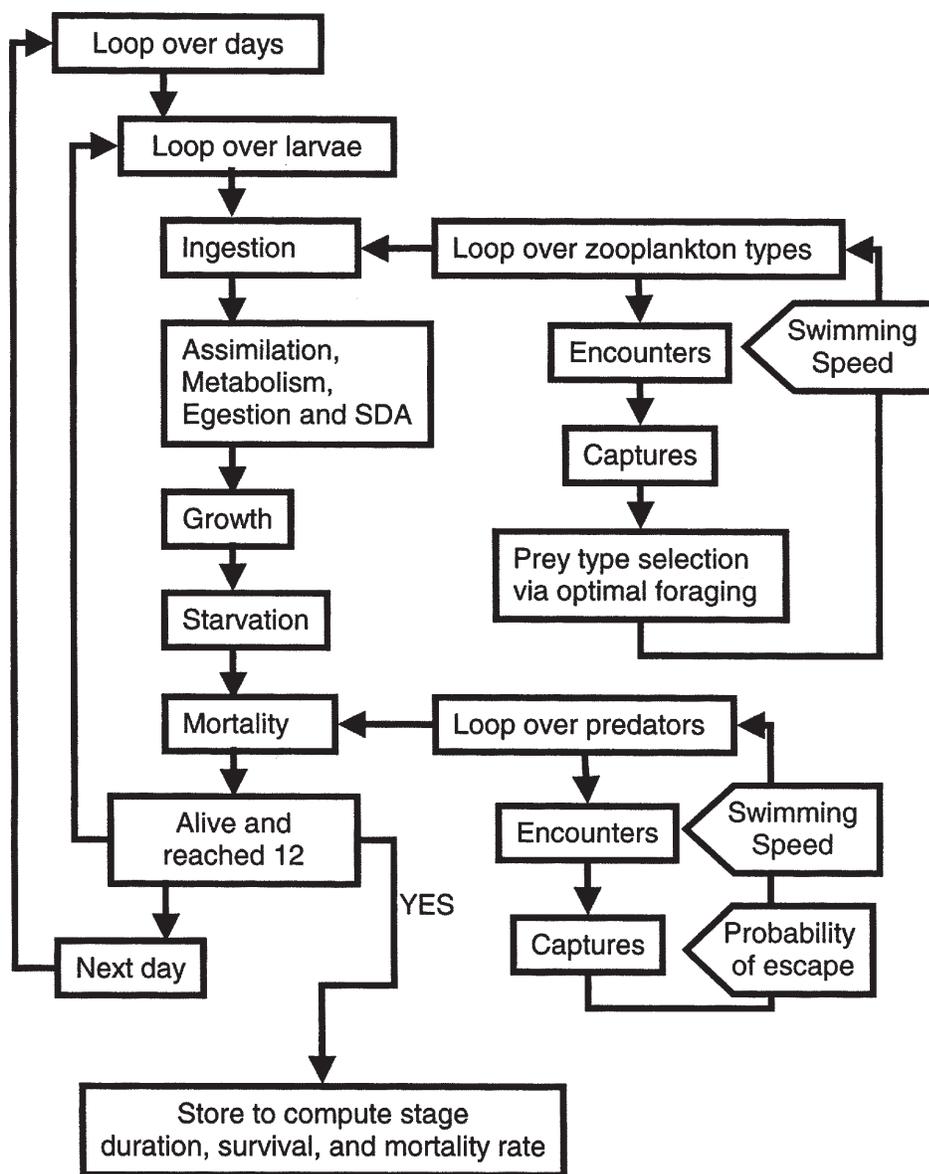
An individual-based model was configured that tracked the daily growth and mortality of individual fish larvae from 3 mm to 12 mm in a well-mixed 2000-m<sup>3</sup> volume (Figure 6). The model was roughly configured to simulate the ocean larval



**Figure 4.** Regression tree relating probability of escaping a real predatory fish attack to the swimming speed, probability of response to an acoustic stimulus, and the distance of response to the acoustic stimulus. The regression tree was based on a previous study involving red drum larvae and the data collection methods are described in Fuiman and Cowan (in press).



**Figure 5.** Bar chart showing the percent of control and PCB-exposed larvae measured at different values of (a) probability of escaping a real predatory fish attack and (b) swimming speed. The values of probability of escaping a real predator were the predictions from the regression tree, after adjusting the values for control croaker larvae to match those for the red drum larvae originally used to estimate the regression tree. Note the probability of escape and swimming speed are also shown as multipliers of the mean value of control larvae along the top axis of each plot.



**Figure 6.** Schematic diagram of the individual-based model that simulated the daily growth and mortality of ocean larvae from 3 to 12 mm. Growth was based on bioenergetics principles, with ingestion dependent on encounter rates of larvae with three zooplankton prey types. Mortality was based on the encounter and capture of larvae by individual sea nettles, ctenophores, and predatory fish. PCBs influenced larval swimming speed, which affected the encounter rates of larvae with zooplankton and with predators, and PCBs influenced the probability of escape of larvae from their fish predators. SDA=specific dynamic action.

stage of croaker in order to convert PCB effects on swimming speed and predator evasion to larval stage survival and duration. All larvae started at 3.0 mm, and growth and mortality were evaluated daily for each larva until it either died or reached 12 mm. Growth of each larva was simulated using bioenergetics principles, with consumption based on encounters and captures with each of three zooplankton prey types. Mortality was determined by simulating the encounter and captures of the larva by specified individual gelatinous zooplankton and predatory fish. Swimming speed determined the encounter rates of the larva with zooplankton and predators; probability of escaping a predator attack affected the capture success of the predatory fish. The individual-based model takes as inputs the differences between swimming speeds and probability of escape between control and PCB-treated larvae from the regression tree (experiment 3), and converts these differences into changes in larval stage duration, survival, and mortality rate for use in the matrix model.

The growth component of the individual-based model was a modified version of the growth model of Letcher *et al.* (1996). Letcher *et al.* (1996) configured their bioenergetics model for generic marine larvae at a water temperature of 15°C. This is reasonably close to the temperatures experienced by larvae spawned in the mid-Atlantic Bight during the fall and winter (Stegman *et al.* 1999). On each day, the assimilated ingestion, metabolism, and egestion plus specific dynamic action components of the growth equation were computed and used to determine the change in weight of each individual larva:

$$\Delta W = W_{t+1} - W_t = A * I - M - E \quad (1)$$

where  $W$  = weight of the larva ( $\mu\text{g dw}$ ),  $A$  = assimilation efficiency,  $I$  = ingestion ( $\mu\text{g/day}$ ),  $M$  = metabolism ( $\mu\text{g/day}$ ),  $E$  = egestion plus specific dynamic action ( $\mu\text{g/day}$ ). The length of the larva was then updated if weight gain was positive and the larva was at least at the average weight expected for its length. Larvae that were less than one-half their expected weight based on their length were considered to have starved to death. Metabolism depended on larval weight and included both routine and active metabolism. Egestion and specific dynamic action were combined and assumed to be 30% of assimilated ingestion. Assimilation efficiency increased with larval weight from about 60% at 11.3  $\mu\text{g dw}$  (3 mm) to a maximum of 80% by 2316  $\mu\text{g dw}$  (12 mm). Ingestion was computed based on encounter rate and capture success of the larva with each of three zooplankton prey types (nauplii, copepodites, adult copepods), with an optimal foraging algorithm determining which of the prey types were actually ingested. Encounter rate depended on the swimming speed of the larvae and their reaction distance to prey of different lengths.

We adjusted the zooplankton densities and capture probabilities of the larvae used by Letcher *et al.* (1996) for an estuarine environment to get realistic growth rates of larvae in an ocean environment. Croaker larvae consume relatively large zooplankton compared to most marine larvae (Govoni *et al.* 1986). We therefore included the nauplii, copepodites, and adult copepods groups but eliminated the smallest zooplankton prey type (rotifers) used by Letcher *et al.* (1996), and fixed the larval capture success probabilities at higher values than those used by Letcher *et al.* (1996).

## Models and Data for Predicting Population Effects

The mortality component of the individual-based model was based on larval encounters and capture by sea nettles, ctenophores, and juvenile fish. Information on the lengths, swimming speeds, encounter radii, and capture success of each of the predator types needed for computing mortality of the larvae were obtained from Cowan *et al.* (1996). Whether each larva was eaten by a predator was evaluated for each day of the simulation. On each day, the mean number of encounters between a larva and each of the predators was computed. Mean daily encounter rate depended on the encounter radius and the distance swum (swimming speed times time active) of both the larvae (prey) and each predator. Predators were assumed active for 24 hours of the day, and the same swimming speed relationship and 13 hours of activity for larvae as was used in the larval growth calculations was also used for the mortality calculations. The realized number of encounters between each predator and a larva was then generated as a random deviate from a Poisson distribution. The number of successful encounters was a random deviate from a binomial distribution, with the probability of success equal to the capture success and the number of trials equal to the realized number of encounters. This process of evaluating encounters and capture success was repeated for each of the predators. If any of the individual predators resulted in at least one successful encounter and capture in a day, then the larva was classified as dead and was removed from the simulation.

The densities of the three predators used in Cowan *et al.* (1996) were reduced to obtain realistic mortality rates of ocean larvae. For simulations reported here, 300 individual ctenophores (mean length of 45 mm), 100 individual sea nettles (mean length of 75 mm), and 20 individual juvenile fish (mean length of 35 mm) were used. These ctenophore and sea nettle densities were 10-fold lower, and the fish density was 5-fold lower than those used by Cowan *et al.* (1996) for the relatively predator-rich Chesapeake Bay.

Two simulations were performed corresponding to baseline (no PCBs) conditions and to PCB-exposed larvae conditions. Each simulation started with 10,000 3.0-mm larvae. For baseline conditions, 10,000 larvae were generated with the values of the swimming speed and probability of escape multipliers in the same proportions as estimated for the control larvae (Figure 5). PCB effects were simulated by repeating the simulation but with 10,000 larvae that had multipliers of swimming speed and probability of escape in the same proportions as estimated for the PCB-treated larvae (Figure 5). Thus, each larva in a simulation had a swimming speed multiplier and a probability of escape multiplier. The swimming speed multiplier was used to adjust the larval swimming speed, which affected their encounters with zooplankton prey and their encounters with each individual predator. The multiplier of the probability of escape was used to alter the capture success of predatory fish. The capture success of the ctenophore and sea nettle predators were not altered because they are tactile predators and the regression tree was estimated for a visual predatory fish. Larval stage duration, survival, and mortality rate were predicted for the baseline and PCB-exposed simulations. The average of three replicate simulations for the baseline and for PCB simulations are reported.

## Results

Larvae from PCB-exposed females were predicted to have a longer stage duration, a higher mortality rate, and a lower stage survival than control larvae. Average larval stage duration and mortality rate was 36.3 days and 0.103/day for PCB-exposed larvae and 30.0 days and 0.064/day for control larvae. The combination of stage duration and mortality rate resulted in lower stage survival for PCB-exposed larvae versus control larvae (2.4% versus 14.6%). The changes in stage duration and stage survival between control and PCB-exposed larvae were used to adjust the values of elements associated with the ocean larval stage in the matrix projection model.

### Matrix Projection Model: From Fecundity, Egg Mortality, and Larval Stage Duration and Survival to Population Response

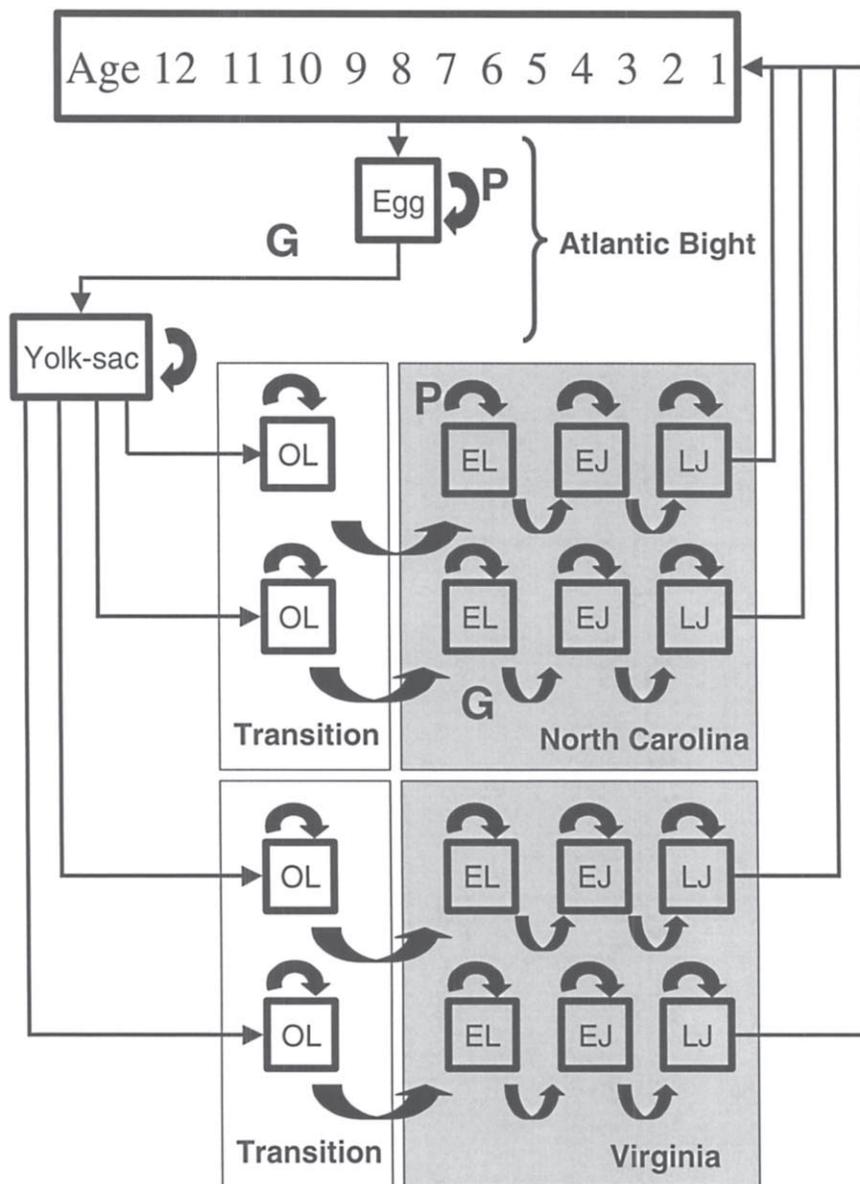
#### Model description

A matrix projection model was configured that simulated croaker population dynamics for 100 years. Matrix projection models have been used previously to simulate stressor effects on fish population dynamics (Goodyear 1985; Schaff *et al.* 1987; Barnthouse *et al.* 1990; O'Connor 2001). Most of these previous applications used the classical Leslie age-based matrix model that follows the numbers of individuals in each annual age class from year to year.

A variation on the classical age-based matrix projection model was used in this study. Egg to age-1 (young-of-the-year) individuals were represented as a series of successive life stages, and two spatial regions and two separate spawning events in each year were simulated (Figure 7). The life stages represented were egg, yolk-sac larva, ocean larva, estuarine larva, early juvenile, and late juvenile. These life stages comprise the first year of life. The two regions simulated (we call North Carolina and Virginia) correspond to two inshore nursery areas utilized by larvae and juveniles spawned by adult croaker in the mid-Atlantic Bight.

Field data showed two distinct influxes of estuarine larvae, with peaks usually in August and January in Virginia and usually in September and February in North Carolina. A model year began on June 1. Total annual egg production was computed from fecundity and numbers of mature females in each age class on June 1, and this production was divided as 70% into cohort 1 and 30% into cohort 2. The number of eggs in each cohort was then split 60% to North Carolina and 40% to Virginia, and the eggs of each cohort were added daily to the model during July through October for cohort 1 and during December through March for cohort 2.

Representing stages (rather than ages) for the young of the year requires estimation of the off diagonal matrix element (as with the classic age-based matrix) as well as the diagonal element. The off-diagonal element ( $G$ ) is the probability of surviving a timestep and progressing to the next life stage and the diagonal element ( $P$ ) is the probability of surviving a timestep and remaining in the same life stage. Estimates of  $G$  and  $P$  for each life stage were obtained from life table information for Atlantic croaker (Diamond *et al.* 1999, 2000), specifically the probability of surviving per timestep (from mortality rate) and the duration of the life stage (see Equations 6.97, 6.98, and 6.103 in Caswell 2001). Because the life stages involve a wide range of durations (days for eggs to months for juveniles), life stage dynamics were simulated



**Figure 7.** Flow diagram of the matrix projection model, which simulates the life stages and spatial aspects of Atlantic croaker in the mid-Atlantic Bight. Adults, eggs, and yolk-sac larvae occur in the Atlantic Bight; ocean larvae transit from the Bight to the estuaries, which is referred to as the transition area; estuarine larvae, early juveniles, and late juveniles occur in the North Carolina and Virginia estuarine nursery areas. The following time steps are used for the life stages: annual for adults; daily for eggs and yolk-sac larvae; 15-day for ocean larvae and estuarine larvae; monthly for early juveniles and late juveniles. OL=ocean larvae; EL=estuarine larvae; EJ=early juveniles, LJ=late juveniles.

using different time steps (daily: eggs and yolk sac larvae; 15 day: ocean larvae and estuarine larvae; monthly: early juveniles and late juveniles; annual: age-1 and older). In order to make interpretation of PCB effects easier, the early and late juvenile mortality rates were adjusted so that total population number in the baseline simulation was stable over the 100 years. The values for eggs and yolk-sac larvae for the ocean environment, and for the remaining young-of-the-year life stages for cohort 1 in North Carolina, are shown in Table 1.

For adults, annual survival fractions, fraction mature, and fecundity were specified for ages 1 to 12. Annual survival fraction of 0.5 was a combination of an assumed natural mortality rate of 0.2/year plus a fishing mortality rate of 0.5/year (instantaneous mortality rate of 0.7/year equals 50% survival per year). Fraction mature by age was 0.5 for age-1, and 1.0 for age-2 and older. Fecundity (eggs/female) increased from a minimum of 46,279 at age-1 to a maximum of 1,089,661 at age-12 (Morse 1980).

In the matrix model, egg production was randomly varied around the model-predicted value from year to year to represent variation from natural sources, and late juvenile survival was specified as density dependent. Twenty years of monthly sampling of young of the year croaker were analyzed for about 60 stations in Virginia estuaries (Virginia Institute of Marine Science, unpublished data) and in North Carolina estuaries (NC Division of Marine Fisheries, unpublished data) to quantify interannual variation in life stage abundances and to identify any density-dependent mortality. Survival and growth rates of croaker vary as result of interannual differences in environmental conditions, such as temperature, prey, predators, and water circulation patterns. The many factors affecting all life stages were crudely simulated by multiplying the model-predicted total egg production each year by a random deviate from a lognormal distribution with a mean of one and standard deviation of 0.7. We compared predicted coefficients of variation (CV, standard deviation/mean x 100) of interannual abundances of early and late juveniles with those from the long-term field data.

The long-term monitoring data also showed that the loss rates of late juveniles (mortality plus those that successfully developed into the next life stage) were negatively related to their average or peak abundance in both North Carolina and

**Table 1. Stage-specific mortality rates, durations, and resulting G and P values for young-of-the-year stages used in the matrix projection model of Atlantic croaker.**

Stage	Timestep	Mortality rate (1/day)	Duration (days)	G	P
Egg	daily	0.49	2	0.229	0.378
Yolk-sac larva	daily	0.14	4	0.172	0.693
Ocean larva	15-day	0.15	35.5	0.004	0.100
Estuarine larva	15-day	0.021	31.7	0.286	0.444
Early juvenile	monthly	0.0147	168.8	0.033	0.611
Late juvenile	monthly	0.0376	118	0.008	0.316

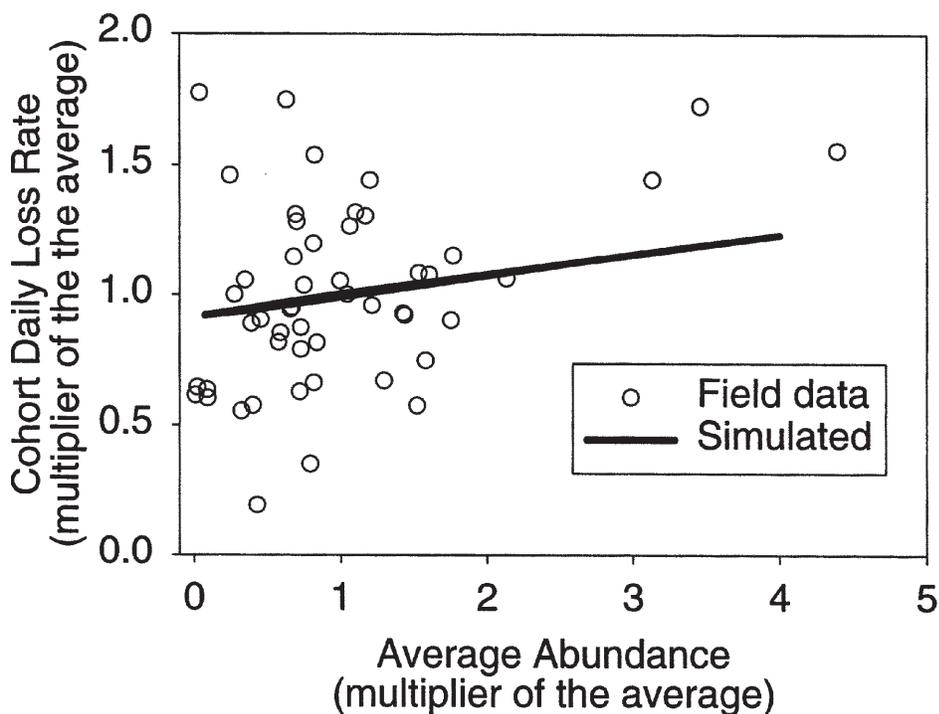
Values for ocean larvae, estuarine larvae, early juvenile, and late juvenile are shown for the first spawning cohort for North Carolina; different values are used for the second spawning cohort for North Carolina and for the two cohorts for Virginia.

## Models and Data for Predicting Population Effects

Virginia estuaries. A relationship between a multiplier of mortality rate (Y) and the abundance of late juveniles (X) was estimated:

$$Y = 0.72 + \frac{1.49}{1 + e^{2.64 - 1.45X}} \quad (2)$$

where X is the number of late juveniles divided by a long-term average abundance. Equation 2 was used each month of the simulation to determine the mortality rate of late juveniles for that month, which was then used to determine the G and P values for that month. Equation 2 was determined so that when a similar range of abundances as was observed in the field data were simulated for seven months as a late juvenile cohort, we obtained a relationship between cohort loss rate and abundance similar to that observed in the field data (Figure 8).



**Figure 8.** Late juvenile cohort loss rate versus average cohort abundance from long-term field data and from the simulation of the density-dependent mortality equation applied to a range of initial cohort abundances. Average abundance on the x-axis and loss rate on the y-axis for the field data and for model simulations are each normalized by dividing by their respective mean values so all values vary around a value of one. The field data estimates are from analysis of field data from North Carolina and Virginia estuaries for both spawning cohorts.

### Baseline Simulations

Simulated croaker population abundances were stable and exhibited realistic interannual variation over the 100 years (Figure 9a). Predicted CVs of early and late juvenile abundances were similar to the CVs observed in the long-term monitoring data. The observed CVs were determined by computing the summed sampling catch of each life stage produced for each year, and then computing the CV of these annual summed abundances. A similar CV was computed from the model predictions; total number entering each life stage by cohort and area were determined for each year and then a CV was computed over the 100 annual values. Predicted CV of early juveniles averaged 88% for both spawning cohorts in both nursery areas vs. the observed values of 82% in Virginia and 72% in North Carolina. Predicted CV of late juveniles averaged from 60 to 80% vs. the observed values of 74% in Virginia and 54% in North Carolina.

### PCB Exposure Scenarios

For both hypothetical PCB exposure scenarios, we assumed that exposure to PCBs occurred during development in the North Carolina nursery area, and effects manifested themselves when these individuals subsequently spawned. Exposure assumed to occur in North Carolina estuaries is purely for illustrative purposes. The first scenario assumed that most of the PCBs were eliminated in the first spawning because eggs are lipid rich and PCBs are highly lipophilic. PCB effects were therefore imposed only on offspring from a female's first year of spawning. The second scenario assumed that the PCB effects occurred through a female's lifetime and so effects were imposed on all offspring year after year.

To simulate the effects of PCBs, fecundity of affected females was reduced by 13% (experiment 1) and P and G values were adjusted for eggs and ocean larvae spawned by these affected females. Egg stage duration was 2 d. Assuming that most of the mortality of eggs due to PCB exposure would occur in the first 24 h, the 51% reduction in 24-h egg survival measures in experiment 2 was assumed to apply to entire egg stage. Therefore, egg survival assumed under baseline conditions was simply reduced by 51%. Keeping the same egg stage duration of 2 d as used in baseline conditions, a new PCB-exposed mortality rate for eggs of 0.855/d was computed. The baseline duration and the new PCB-exposed mortality rate resulted in lowered values of P for eggs under PCB conditions (0.30 vs. 0.38 for baseline) and lowered values of G for eggs (0.13 vs. 0.23 for baseline). The P and G values of all ocean larvae from North Carolina females were adjusted for PCB exposure. Because offspring from North Carolina females went to both Virginia and North Carolina nursery areas, PCB-exposed offspring were kept track of separately in each nursery area. PCB-adjusted values of P and G for ocean larvae were determined using the same procedure as was done for eggs, except the baseline stage duration (in addition to mortality) of ocean larvae was also adjusted for PCB exposure. Based on the results of the individual-based model, survival in each cohort and each area was reduced to 16% of its baseline value, and duration was increased by 21%. We then computed new mortality rates and new durations, which resulted in new values of P and G for ocean larvae in each cohort and in each area. We illustrate this with the first spawning cohort in North Carolina shown in Table 1. The duration of 35.5 d

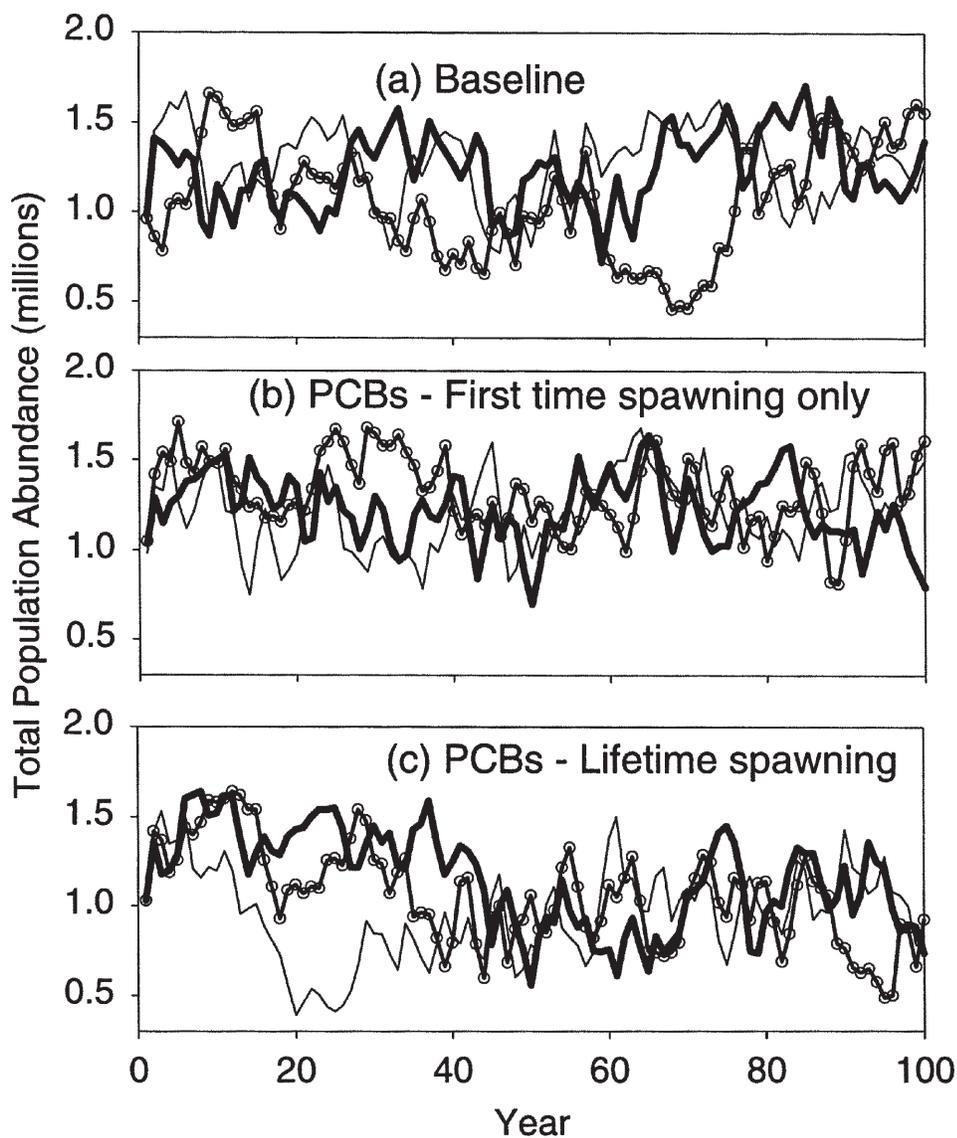


Figure 9. Matrix model simulations of croaker population abundance for 100 years under (a) baseline conditions, (b) PCB exposure with effects on first time spawning only, and (c) PCB exposure with effects on lifetime spawning. Three replicate simulations that differ in their random number sequences are shown for each condition.

and larval stage survival of 0.0049 under baseline conditions were adjusted based on individual-based model results to a stage duration of 42.9 d and a stage survival of 0.00078 under PCB exposure. This increase in stage duration and decrease in stage survival due to PCB exposure resulted in the value of P for ocean larvae decreasing from 0.11 under baseline to 0.08 under PCB exposure and the value G for ocean larvae decreasing from 0.004 under baseline to 0.00072 under PCB exposure. As with baseline conditions, we performed three replicate simulations for each of the two PCB exposure scenarios.

### Matrix Model Simulation Results

Predicted effects of PCBs were negligible under the first time spawning scenario (Figure 9b), and small but consistent under the lifetime spawning scenario (Figure 9c). Predicted average total population abundance across all years and the three replicate simulations was actually slightly higher under the first time spawning PCB scenario (1.25 million) compared to baseline (1.20 million), but this result was well within interannual variability and was due to an unusual sequence of low population abundance years (due to chance) in one of the baseline simulations that was not repeated in the PCB simulations. Overall average total abundance under the lifetime spawning PCBs scenario (1.06 million) was about 10% lower than baseline (1.20 million). Predicted CVs of early and late stage juveniles were unaffected by PCB exposure. Predicted CVs were similar between both PCB scenarios and between the PCB scenarios and the baseline simulations.

## DISCUSSION

This study has demonstrated an approach for predicting the population level effects of contaminants on fish. The approach begins with laboratory toxicity data, and uses a series of nested statistical and simulation models to extrapolate the laboratory results to long-term population dynamics. Laboratory data allow for control of experimental conditions and thus clearer determination of cause and effect (*e.g.*, dose-response relationship). The disadvantages of laboratory data are that some endpoints are difficult to relate to ecologically relevant effects, and the unrealistic environment of the laboratory makes extrapolation to field effects difficult. The modeling approach utilized in this study provides a method for bridging the gap between the laboratory and field. Furthermore, the croaker and PCBs example used a specific series of statistical and individual-based models that permitted extrapolation of sublethal behavioral effects to the population level. Our collection of laboratory methods and modeling tools permitted the temporal scaling from behavioral responses measured in hundredths of seconds to responses of a population over hundreds of years.

Our analyses offer an alternative to the typical approach for establishing cause and effect in field studies. Establishing cause and effect is usually based on extensive laboratory and field measurements on individuals (*e.g.*, see other papers in this February 2002 issue of *Human and Ecological Risk Assessment*). Indicators of stress on individuals are measured and compared among time periods or among different locations. Indicators of increased stress can then be associated with a known source of a stressor or related to contaminant levels measured in the individuals. This

## Models and Data for Predicting Population Effects

commonly used approach is very useful for establishing that contaminant exposure is present, but it is more difficult to use these data to show that the exposure is causing population level effects. In this study, a very different approach was taken to establish causality. Laboratory results are used to predict the likely magnitude of the population-level effects expected in the field. This expected response is then compared to variability in population abundances and dynamics from other natural and anthropogenic sources. In the case study, PCBs were predicted to have a negligible to small population effect on croaker relative to natural variation. These small effects were predicted despite the PCBs simulations incorporating simultaneous reductions in fecundity and increased egg mortality, and incorporating seemingly large reductions in ocean larval survival (*e.g.*,  $G$  from 0.004 to 0.00072 for cohort 1 for North Carolina).

The predictions of small population level effects of PCBs reported here should, however, be viewed with caution. On one hand, the PCB exposure scenario involved only a portion of the possible fish that could be exposed. PCB exposure was restricted to adults that originated in North Carolina estuaries. On the other hand, all individuals in North Carolina were assumed exposed for every year of the 100 years, and the exposure level corresponded to the low-dose treatment in the laboratory experiments. Adult croaker from the low-dose treatment had body burdens that were high relative to those observed in field-caught croaker. Also, many other aspects of PCB effects and croaker population dynamics were ignored. For example, PCB effects on the prey and predators of croaker, and on the ability of croaker to capture their prey were ignored. If the appropriate information was available, these omissions could be added to our modeling approach. Furthermore, the individual-based and matrix models require additional testing before croaker larval stage growth and survival and population dynamics can be simulated with confidence. Further refinement of the models, laboratory data, and exposure scenarios would enable more realistic predictions of PCB effects on croaker population dynamics.

Most fish populations are subject to multiple natural factors and stressors, and yet cumulative population level effects of multiple stressors are rarely considered in studies. The combined effects of multiple stressors can be more than the sum of their individual effects (Power 1997). Situations may occur where one stressor results in no-effect on population abundance, but renders the population more sensitive to another stressor. Cumulative effects of multiple stressors could be examined with our modeling approach. Simulations could be performed without and with PCB effects, and with and without a second stress (*e.g.*, increased harvest). The degree to which the presence of the second stress affected the population response to PCBs would be quantified, and results related to population resilience and resistance (Ives 1995; Grimm and Wissel 1997). Predicting population responses to multiple stressors remains an important but difficult issue.

This study incorporated new and retooled quantitative methods and only addressed a few of the many possible applications of our approach. Regression trees offer a viable alternative to classical regression, when prediction is more important than hypothesis testing and when the dependence of the response variable on explanatory variables is not consistent over the entire range of data. Individual-based modeling is continuing to gain popularity (DeAngelis and Gross 1992; Judson 1994), and is well suited for bridging the gap between laboratory results, especially

behavioral effects, and population dynamics models (Rose *et al.* 1999). Matrix projection models have a long history in ecology (Tuljapurkar and Caswell 1997; Caswell 2001), and in this study some relatively simple extensions were made to the classical model to allow for more realistic simulation of the complex life history of croaker and for realistic simulation of PCB exposure scenarios. Two hypothetical PCB exposure scenarios were presented to demonstrate our laboratory-to-population response approach. Further elaboration could involve attempting to simulate the historical population trajectory using actual environmental conditions. A variety of different assumptions about the contributions of environmental variables and PCBs to population responses could be simulated. The statistical likelihood of each trajectory based on its fit to field data would be compared (Hilborn and Mangel 1997) to determine the most likely contribution of PCBs to the observed population dynamics.

PCB effects on croaker were used to illustrate our approach of scaling laboratory results to the population level. A real fish population was purposely simulated in order that field data could be used to realistically evaluate predicted effects of PCBs vs. natural variation. Detailed analysis of the laboratory results and further refinement of the individual-based and matrix models are underway. These results are preliminary, but we think they convincingly demonstrate the utility and promise of coupling laboratory data with nested models to predict population responses to contaminants. Such analyses would be extremely useful for helping to identify causal mechanisms between contaminant exposure and population-level effects.

#### ACKNOWLEDGMENTS

This research was funded by a grant from the EPA STAR (Science to Achieve Results) Program. We thank Ian McCarthy, Maria Alvarez, Izhar Khan, and Larisa Ford for their work on the laboratory experiments.

#### REFERENCES

- Able KW and Fahay MP. 1998. *The First Year in the Life of Estuarine Fishes in the Middle Atlantic Bight*. Rutgers University Press, New Brunswick, NJ, USA
- Adams SM. 1990. Biological Indicators of Stress in Fish. *Am Fish Soc Symp* 8, Bethesda, MD, USA
- Adams SM and Greeley MS. 1991. Assessment and Evaluation of the Ecological Health of Fish Populations Exposed to PCBs in Hartwell Reservoir. DOE No. 1610-D011-A1. Final Report to the Department of Energy. Washington, DC, USA
- Ankley GT and Giesy JP. 1998. Endocrine disruptors in wildlife: a weight-of-evidence perspective. In: Kendall R, Dickerson R, Giesy J, *et al.* (eds), *Principles and Processes for Evaluating Endocrine Disruption in Wildlife*, pp 349-367. SETAC Press, Pensacola, FL, USA
- Arcand-Hoy LD and Benson WH. 1998. Fish reproduction: an ecologically relevant indicator of endocrine disruption. *Environ Toxicol Chem* 17:49-57
- Barnthouse LW, Boreman J, Christensen SW, *et al.* 1984. Population biology in the courtroom: the Hudson River controversy. *BioScience* 34:14-9
- Barnthouse LW, Suter GW, and Rosen AE. 1990. Risks of toxic contaminants to exploited fish populations: influence of life history, data uncertainty and exploitation intensity. *Environ Toxicol Chem* 9:297-311

## Models and Data for Predicting Population Effects

- Breiman L, Friedman JH, Olshen RA, *et al.* 1998. Classification and Regression Trees. Chapman and Hall/CRC, Boca Raton, FL, USA
- Caswell H. 2001. Matrix Population Models: Construction, Analysis and Interpretation, 2<sup>nd</sup> ed. Sinauer Associates, Sunderland, MA, USA
- Chambers JE, Boone JS, Carr RL, *et al.* 2002. Biomarkers as predictors in health and ecological risk assessment. *Hum Ecol Risk Assess* 8:165-76
- Chesney EJ, Baltz DM, and Thomas RG. 2000. Louisiana estuarine and coastal fisheries and habitats: perspectives from a fish's eye view. *Ecol Appl* 10:350-66
- Colborn T and Thayer K. 2000. Aquatic ecosystems: harbingers of endocrine disruption. *Ecol Appl* 10:949-57
- Cowan JH, Houde ED, and Rose KA. 1996. Size-dependent vulnerability of marine fish larvae to predation: an individual-based numerical experiment. *ICES J Mar Sci* 53:23-37
- DeAngelis DL and Gross LJ. 1992. Individual-Based Models and Approaches in Ecology. Chapman and Hall, NY, NY, USA
- Diamond SL, Crowder LB, and Cowell LG. 1999. Catch and bycatch: the qualitative effects of fisheries on population vital rates of Atlantic croaker. *Trans Am Fish Soc* 128:1085-105
- Diamond SL, Cowell LG, and Crowder LB. 2000. Population effects of shrimp trawl bycatch on Atlantic croaker. *Can J Fish Aquat Sci* 57:2010-21
- Faulk CK, Fuiman LA, and Thomas P. 1999. Parental exposure to *ortho*, *para*-dichlorodiphenyltrichloroethane impairs survival skills of Atlantic croaker (*Micropogonias undulatus*) larvae. *Environ Toxicol Chem* 18:254-62
- Fogarty MJ, Sissenwine MP, and Cohen EB. 1991. Recruitment variability and the dynamics of exploited marine populations. *TREE* 6:241-6
- Fuiman LA and Cowan JH. Behavior and recruitment success in fish larvae: Repeatability and covariation of survival skills. *Ecology (in press)*
- Fuiman LA, Smith ME, and Malley VN. 1999. Ontogeny of routine swimming speed and startle responses in red drum, with a comparison of response to acoustic and visual stimuli. *J Fish Biol* 55 (Suppl A):215-26
- Garcia S and Newton S. 1997. Current situation, trends, and prospects in world capture fisheries. In: Pikitch EK, Huppert DD, and Sissenwine MP (eds), *Global Trends: Fisheries Management*, pp 3-27. American Fisheries Society Symposium 20, Bethesda, MD, USA
- Giesy JP and Snyder EM. 1998. Xenobiotic modulation of endocrine function in fishes. In: Kendall R, Dickerson R, Giesy J, and Suk W (eds), *Principles and Processes for Evaluating Endocrine Disruption in Wildlife*, pp 155-237. SETAC Press, Pensacola, FL, USA
- Goodyear CP. 1985. Toxics materials, fishing, and environmental variation: simulated effects on striped bass populations. *Trans Am Fish Soc* 114:107-13
- Govoni JJ, Ortner PB, Al-Yamani F, *et al.* 1986. Selective feeding of spot, *Leiostomus xanthurus*, and Atlantic croaker, *Micropogonias undulatus*, larvae in the northern Gulf of Mexico. *Mar Ecol Prog Ser* 28:175-83
- Grimm V and Wissel C. 1997. Babel, or the ecological stability discussions: an inventory and analysis of terminology and a guide for avoiding confusion. *Oecologia* 109:323-34
- Hales LS and Reitz EJ. 1992. Historical changes in age and growth of Atlantic croaker, *Micropogonias undulatus* (Perciformes: Sciaenidae). *J Archaeol Sci* 19:73-99
- Hilborn R and Mangel M. 1997. *The Ecological Detective: Confronting Models with Data*. Princeton University Press, Princeton, NJ, USA
- Holdway DA, Brennan SE, and Ahokas JT. 1995. Short review of selected fish biomarkers of xenobiotic exposure with an example using fish hepatic mixed-function oxidase. *Austr J Ecol* 20:34-44
- Ives AR. 1995. Measuring resilience in stochastic-systems. *Ecol Monogr* 65:217-33
- Jackson JBC, Kirby MX, Berger WH, *et al.* 2001. Historical overfishing and recent collapse of coastal ecosystems. *Science* 293:629-38

- Johnson LL and Collier TK. 2002. Assessing contaminant-induced stress across levels of biological organization. In: Adams SM (ed), *Biological Indicators of Aquatic Ecosystem Stress*, pp 533-63. American Fisheries Society, Bethesda, MD, USA
- Judson OP. 1994. The rise of the individual-based model in ecology. *TREE* 9:9-14
- Karels A, Markkula E, and Oikari A. 2001. Reproductive, biochemical, physiological, and population responses in perch (*Perca fluviatilis* L.) and roach (*Rutilus rutilus* L.) downstream of two elemental chlorine-free pulp and paper mills. *Environ Toxicol Chem* 20:1517-27
- Khan IA and Thomas P. 2000. Lead and Aroclor 1254 disrupt reproductive neuroendocrine function in Atlantic croaker. *Mar Environ Res* 50:119-23
- Kime DE. 1998. *Endocrine Disruption in Fish*. Kluwer Academic, Boston, MA, USA
- Kolpin DW, Furlong ET, Meyer MT, *et al.* 2002. Pharmaceuticals, hormones, and other organic wastewater contaminants in U.S. streams, 1999-2000: a national reconnaissance. *Environ Sci Tech* 36:1202-11
- Laevastu T. 1993. *Marine Climate, Weather and Fisheries*. Halsted Press, NY, NY, USA
- Letcher BH, Rice JA, Crowder LB, *et al.* 1996. Variability in survival of larval fishes: disentangling components of variance with a generalized individual-based model. *Can J Fish Aquat Sc.* 53:787-801
- Little EE, Fairchild JF, and DeLonay AJ. 1993. Behavioral methods for assessing impacts of contaminants on early life stage fishes. *Am Fish Soc Symp* 14:67-76
- McCarthy ID, Fuiman LA, and Alvarez MC. Aroclor 1254 affects growth and survival skills of Atlantic croaker (*Micropogonias undulatus*) larvae. *Mar Ecol Prog Ser (in press)*
- McCarthy JF and Shugart LR. 1990. *Biomarkers of Environmental Contamination*. Lewis Publishers, Chelsea, MI, USA
- McCarty LS, Power M, Munkittrick KR. 2002. Bioindicators versus biomarkers in ecological risk assessment. *Hum Ecol Risk Assess* 8:159-64
- Miller JM, Reed JP, and Pietrafesa LJ. 1984. Patterns, mechanisms and approaches to the study of migrations of estuarine-dependent fish larvae and juveniles. In: McCleave JD, Arnold GP, Dodson JJ, and Neill WH (eds), *Mechanisms of Migration in Fishes*, pp 209-25. Plenum Press, NY, NY, USA
- Monosson E. 2000. Reproductive and developmental effects of PCBs in fish: a synthesis of laboratory and field studies. *Rev Toxicol* 3:25-75
- Morse WW. 1980. Maturity, spawning, and fecundity of Atlantic croaker, *Micropogonias undulatus*, occurring north of Cape Hatteras, North Carolina. *Fish Bull* 78:190-5
- Nixon SW and Jones CM. 1997. Age and growth of larval and juvenile Atlantic croaker, *Micropogonias undulatus*, from the Middle Atlantic Bight and estuarine waters of Virginia. *Fish Bull* 95:773-84
- O'Connor TP. 2001. Comparing episodic, chronic, fishing, and non-fishing impacts on resource populations. *Mar Poll Bull* 42:532-5
- Power M. 1997. Assessing the effects of environmental stressors on fish populations. *Aquat Toxicol* 39:151-69
- Rolland RM, Gilbertson M, and Peterson RE (eds). 1997. *Chemically Induced Alterations in Functional Development and Reproduction of Fishes*. SETAC Press, Pensacola, FL, USA
- Rose KA. 2000. Why are quantitative relationships between environmental quality and fish populations so elusive? *Ecol Appl* 10:367-85
- Rose KA and Summers JK. 1992. Relationships among long-term fish abundances, hydrographic variables, and gross pollution indicators in Northeastern US estuaries. *Fish Oceanog* 1:281-93
- Rose KA, Brewer LW, Barnthouse LW, *et al.* 1999. Ecological responses of oviparous vertebrates to contaminant effects on reproduction and development. In: Di Giulio RT and

## Models and Data for Predicting Population Effects

- Tillitt DE (eds), Reproductive and Developmental Effects of Contaminants in Oviparous Vertebrates, pp 225-281. SETAC Press, Pensacola, FL, USA
- Rothschild BJ.1986. Dynamics of Marine Fish Populations. Harvard University Press, Cambridge, MA, USA
- Rykiel EJ. 1996. Testing ecological models: the meaning of validation. *Ecol Model* 106:229-44
- Schaaf WE, Peter DS, Vaughan DS, *et al.* 1987. Fish population responses to chronic and acute pollution: the influence of life history strategies. *Estuaries* 10:267-75
- Schnute JT and Richards LJ. 2001. Use and abuse of fishery models. *Can J Fish Aquat Sci* 58: 10-7
- Shepard JG, Pope JG, and Couzens RD. 1984. Variations in fish stocks and hypotheses concerning their links with climate. *Rapports et Proces-Verbaux des Reunions, Conseil International pour l'Exploration de la Mer* 185:255-67
- Sindermann C.J. 1996. Ocean Pollution: Effects on Living Resources and Humans. CRC Press, Boca Raton, FL, USA
- Stahl RG and Clark JR 1998. Uncertainties in the risk assessment of endocrine-modulating substances in wildlife. In: Kendall R, Dickerson R, Giesy J, *et al.* (eds), Principles and Processes for Evaluating Endocrine Disruption in Wildlife, pp 431-48. SETAC Press, Pensacola, FL, USA
- Stegmann PM, Quinlan JA, Werner FE, *et al.* 1999. Atlantic menhaden recruitment to a southern estuary: defining potential spawning regions. *Fish Oceanog* 8:(Suppl 2):111-23
- Suter GW.1993. Ecological Risk Assessment. Lewis Publishers, Chelsea, MI, USA
- Thomas P and Khan IA. 1997. Mechanisms of chemical interference with reproductive endocrine function in sciaenid fishes. In: Rolland RM, Gilberston M, and Peterson RE (eds), Chemically Induced Alterations in Functional Development and Reproduction of Fishes. pp 29-51. SETAC Press, Pensacola, FL, USA
- Tuljapurkar S and Caswell H. 1997. Structured-Population Models in Marine, Terrestrial and Freshwater Systems. Chapman and Hall, NY, NY, USA
- Turgeon DD, Bricker SB, and O'Connor TP. 1992. National status and trends program: chemical and biological monitoring of U.S. coastal waters. *Ecol Indict* 1:425-57
- von Westernhagen H. 1988. Sublethal effects of pollutants on fish eggs and larvae. In: Hoar WA and Randall DJ (eds), *Fish Physiology, Volume XI, The Physiology of Developing Fish, Part A: Eggs and Larvae*, pp 253-346. Academic Press, NY, NY, USA
- Warlen SM.1982. Age and growth of larvae and spawning time of Atlantic croaker in North Carolina. *Proc Ann Conf SE Assoc Fish Wildl Agencies* 34:204-14
- Zlokovitz ER and Secor DH. 1997. Effect of habitat use on PCB body burden in Hudson River striped bass (*Morone saxatilis*). *Can J Fish Aquat Sci* 56(Suppl 1):86-93