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Quantitative Effects of Pollution on Marine and Anadromous Fish Populations

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NOTE ON SPECIES NAMES

The NMFS Northeast Region's policy on the use of species names in technical publications and reports is to follow the American Fisheries Society's (AFS) lists of scientific and common names for fishes (Robins *et al.* 1991)[a], mollusks (Turgeon *et al.* 1988)[b], and decapod crustaceans (Williams *et al.* 1989)[c], and to follow the American Society of Mammalogists' list of scientific and common names for marine mammals (Wilson and Reeder 1993)[d]. This policy applies to all issues of the *NOAA Technical Memorandum NMFS-F/NEC* and *-F/NER* series.

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[a] Robins, C.R. (chair); Bailey, R.M.; Bond, C.E.; Brooker, J.R.; Lachner, E.A.; Lea, R.N.; Scott, W.B. 1991. Common and scientific names of fishes from the United States and Canada. 5th ed. *Amer. Fish. Soc. Spec. Publ.* 20; 183 p.

[b] Turgeon, D.D. (chair); Bogan, A.E.; Coan, E.V.; Emerson, W.K.; Lyons, W.G.; Pratt, W.L.; Roper, C.F.E.; Scheltema, A.; Thompson, F.G.; Williams, J.D. 1988. Common and scientific names of aquatic invertebrates from the United States and Canada: mollusks. *Amer. Fish. Soc. Spec. Publ.* 16; 277 p.

[c] Williams, A.B. (chair); Abele, L.G.; Felder, D.L.; Hobbs, H.H., Jr.; Manning, R.B.; McLaughlin, P.A.; Pérez Farfante, I. 1989. Common and scientific names of aquatic invertebrates from the United States and Canada: decapod crustaceans. *Amer. Fish. Soc. Spec. Publ.* 17; 77 p.

[d] Wilson, D.E.; Reeder, D.M. 1993. Mammal species of the world: a taxonomic and geographic reference. Washington, DC: Smithsonian Institution Press; 1206 p.

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ABSTRACT

Pollution of estuarine/coastal waters has been viewed as a concomitant of increasing population density and industrialization of the coastal zone. One primary concern has been possible impacts of present and future contaminant loading on abundance of resource species. Information accumulated during the past three decades, especially from experimental studies, indicates a range of lethal and sublethal effects on coastal fish, but has not demonstrated a clear cause-and-effect relationship with population abundance. What has emerged clearly is the extreme difficulty of separating effects of one contaminant or group of contaminants from those of overfishing, or from those of a host of other environmental stresses.

By using a case history approach, the literature on three species of East Coast coastal fish -- Atlantic menhaden (*Brevoortia tyrannus*), striped bass (*Morone saxatilus*), and winter flounder (*Pleuronectes americanus*) -- has been examined for evidence of quantitative effects on populations. General findings with menhaden were that because of extreme variability in recruitment, detection of reductions in abundance because of pollution would be feasible only in the case of catastrophic events (greater than 50-percent year-class reduction). With striped bass, a dominant role of high fishing mortality was proposed, possibly augmented by severe larval mortality in some estuaries if stressful environmental conditions existed concurrently with toxic levels of contaminants. In studies of winter flounder, reduced reproductive success as a consequence of pollution was indicated in several studies, with a range of genetic and cytological abnormalities in developing eggs, and teratogenic effects in larvae.

Studies of fish in other parts of the world suggest that, for species which are subjected to moderate exploitation or which are unexploited, decreases in survival rates due to pollution effects would be too small to detect, whereas in overexploited populations with severely reduced spawning stocks, the added impact of pollution may result in reduced recruitment. However, studies of long-term trends in such heavily fished areas as the North Sea have found no consistent decline of commercial species that could be attributed to deteriorating habitat conditions.

INTRODUCTION

This paper is best introduced by listing a series of hypotheses about effects of pollution on fish and shellfish populations -- hypotheses that have variable data support and may eventually be proved incorrect. They are as follows:

1. **Habitat destruction in estuaries and contaminant disposal in the ocean have had localized adverse effects on resource organisms, but the broad regional effects at the species level have not been determined.** Lack of any early success in assessing effects of pollution on population abundance may be because "the **scale of impact** [emphasis mine] usually is much smaller than the geographical range of the species" (Cross *et al.* 1985).

   Two decades ago, Cole (1972) pointed out a critical guideline for evaluating conclusions reached about marine pollution -- that we must be careful to differentiate large-scale changes due to pollution from purely local, almost parochial, situations, usually in a few square kilometers of estuarine/coastal waters.

   Cross *et al.* (1985) provided cogent additional admonitions: "Improvement in our capacity to predict the effects of pollution on fisheries requires an understanding of the environmental factors that control variability in fish populations and the effect of multiple stresses on these stocks over their entire geographical range. Increased predictive capability can be gained most cost-effectively through closer integration of the disciplines of population dynamics and toxicology."

2. **Significant negative impacts of pollution on commercial fish stocks have not been demonstrated, even for areas such as the North Sea,** where statistical information is reasonably adequate (Cole 1975, 1983; Hempel 1978; Lee 1978; Cushing 1979; McIntyre 1982). Lee, for example, stated "There is no evidence either way as to whether or not contamination of North Sea waters by metals, pesticide/residues, etc. has affected the well being of the fish stocks." This pronouncement is an oversimplification; a more rational viewpoint can be found in other published reports, especially that of Dethlefsen and Tiews (1985): "at present it is impossible to define the role of pollution on fish stocks of the North Sea as a whole. This is largely due to the fact that only drastic changes in marine ecosystems would be detectable and could be interpreted as manmade. Normally chronic and sublethal changes are taking place very slowly and it will be impossible to separate natural fluctuations from anthropogenically caused. Long-term research over decades might be necessary to clearly distinguish between man-made and natural fluctuations especially in offshore waters." Cushing (1979) also stated the issue clearly: "The major problem of population/community monitoring, bearing in mind the non-specific nature of the response to pollutants, is to distinguish pollution induced changes from those due to other causes."

3. **Marine resource populations are subject to large natural fluctuations whose causes are incompletely understood.** Turning again to the North Sea, where resource and environmental data are probably better than anywhere else in the world, Hempel's 1978 detailed report on recent

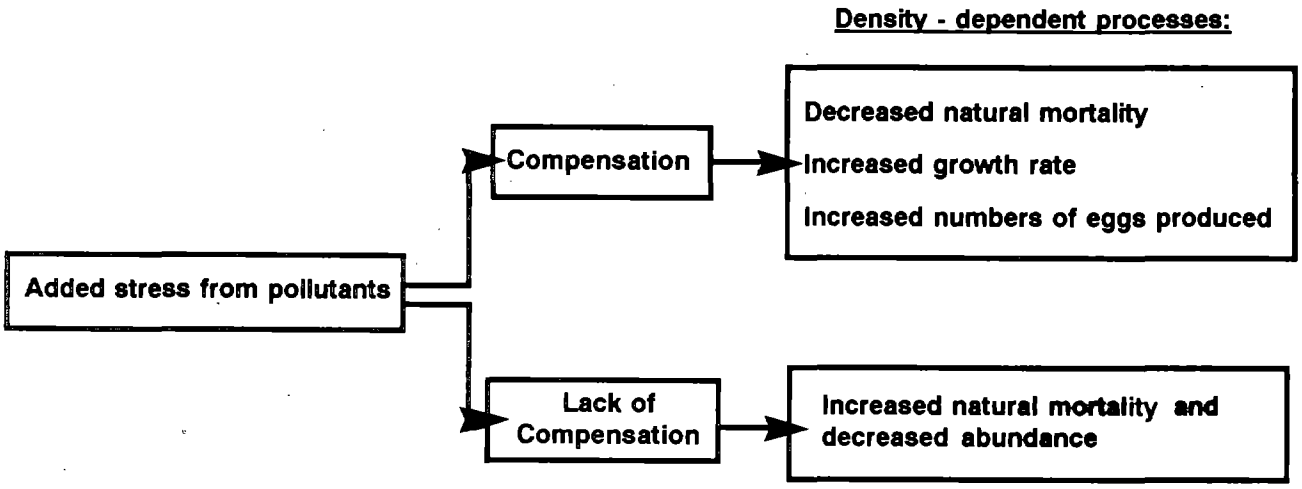

Figure 1. Population responses to pollution stress. Note, in addition, that the gains from compensation are added (in an algebraic sense) to the losses from the added pollution stress. The net result could indeed be decreased **M**, but it could instead be constant **M** (if compensation precisely offsets the contaminant stress) or reduced **M** (if compensation is incomplete).

changes in fisheries and fish stocks concluded with this remarkably nebulous conclusion: "It seems that direct and indirect effects of changes in the fisheries as well as climatic changes in their consequences for the biotic environment caused the recent changes in the fish stocks of the North Sea. It is not possible to quantify the effects of man-made and natural factors separately because of the complexity of interactions between the various fish stocks and the stages of their early life history."

There are really two important points to be made about this and the preceding hypothesis: (1) fish populations are subject to great variability, and partitioning this variability into its components (one of which is pollution induced) is difficult; and (2) population levels (or for that matter recruitment levels) are **themselves** estimated only imprecisely, so that our ability to detect secular changes in population size or recruitment is quite poor (Peterman and Bradford 1987; Peterman 1990). This imprecision is a major reason for difficulty in detecting contaminant effects on populations. So the problem, then, is not only to **partition** the causes of variability, but also to **measure the variability itself** with any precision.

4. **Because of the inability to distinguish population changes due to pollution, such changes might become catastrophic before they are noted.** Effects of pollutants on reproduction, recruitment, behavior, and survival may be particularly critical. Survival can be reduced by parental (inherited) and dietary contaminants, such as polychlorinated biphenyls (PCBs) (Westin *et al.* 1985). Sperm viability and egg fertilizability might be reduced. Larval behavior can be affected, reducing food capture and predator avoidance. Pollution may result in immunosuppression and increased susceptibility to disease in early life stages. The reproductive rate -- usually considered in the literature on environmental toxicology to be a more sensitive parameter than survival (Kooijman and Metz 1984) -- can be severely affected. According to those authors, toxic chemicals may reduce reproductive rates by: (1) causing diversion of maintenance energy to metabolic deactivation or tissue repair; (2) directly impairing protein synthesis, hence growth; (3) affecting digestion and assimilation; or (4) interfering with gonad maturation and gamete production.

These physiological responses of individuals under chemical stress can contribute to reproductive failure of the population as a whole -- although other factors may be involved, as described by Waldichuk (1979): "(1) the fish may be unable to reach its spawning grounds because of unfavourable ecological conditions, or its own weak physical state, and goes unspawned; (2) the eggs may never be released by the female owing to some unsuitable physiological condition; (3) the eggs and larvae may die because of their unhealthy state or poor conditions on the spawning grounds; (4) the eggs and larvae may be poisoned by a substance bioaccumulated in the gonads of the parent, *e.g.* DDT; or (5) the eggs and larvae may be poisoned by toxic substances in the environment."

5. **Offshore fish stocks are not immune to damage from pollutants,** since year-class abundance is determined early in the life history, which for many marine species is spent in coastal/estuarine waters. The reality of this statement is demonstrated in the next section on pollution effects on menhaden stocks.

6. **Many major fish stocks are already overexploited and may be more vulnerable to additional stresses (such as pollution) than unexploited populations.** A fish population that is heavily fished may have limited compensatory reserve and thus may be particularly sensitive to pollution-induced mortality (Cross *et al.* 1985; von Westernhagen *et al.* 1988) (Figure 1). A critical question for each stressed

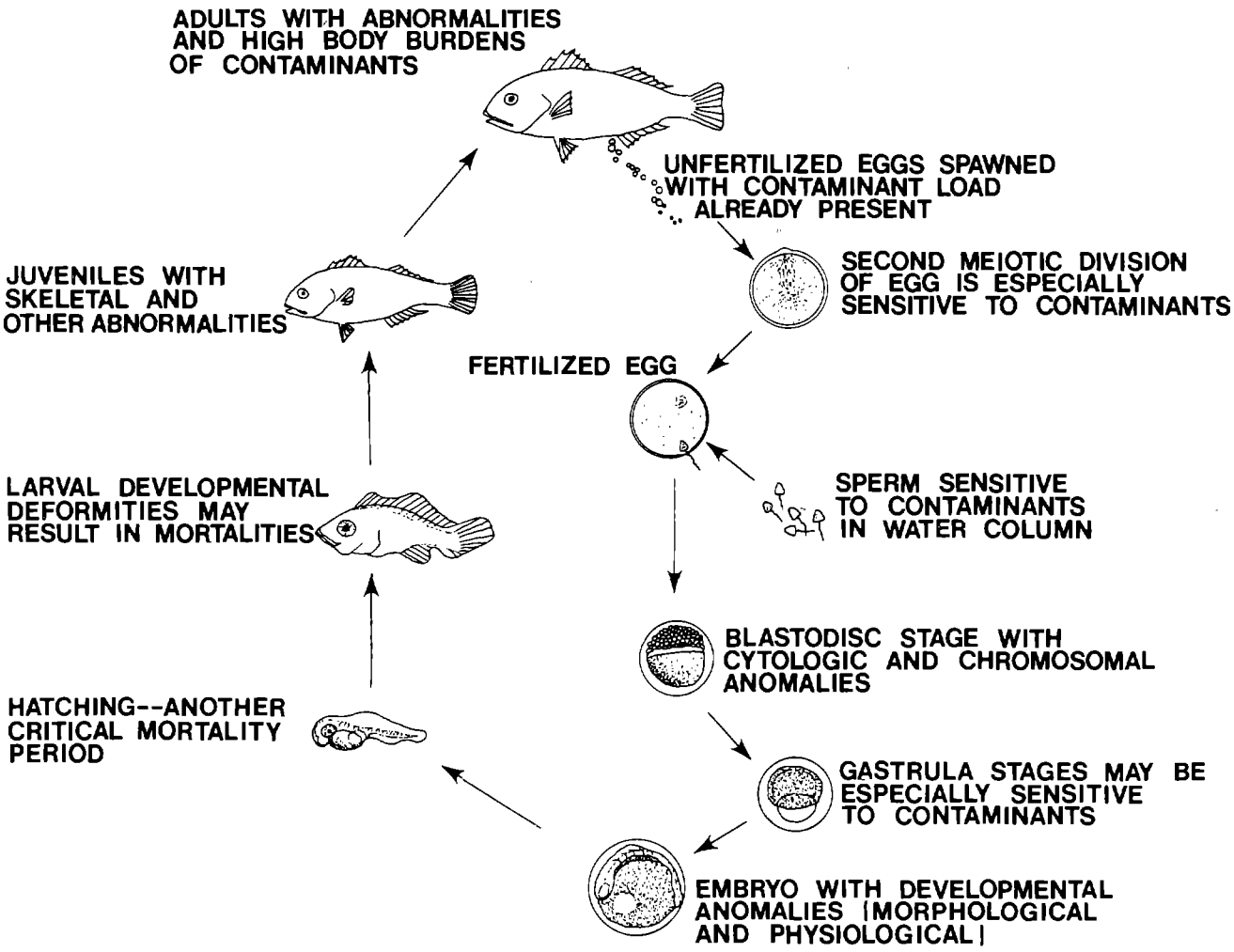

Figure 2. Points in the life cycle when fish are especially sensitive to pollutants.

population concerns the compensatory response -- especially the amount of compensatory reserve that is left in an exploited population.

The point about effects of exploitation was made forcefully by Cushing (1979). Defining the term "impact" as "the loss of eggs, larvae, or juveniles to the recruitment of fish stocks," he stated that: "Variability of recruitment in marine and anadromous fish stocks is high -- one to two orders of magnitude -- and an impact due to pollution would be difficult to detect. However, the effect of an impact depends on the degree of exploitation, so if stocks are low and heavily exploited, no impact can be tolerated until the stock has been returned to the desired objective, the maximum sustainable yield or any lesser quantity that might be optimal."

This point is consistent with a related statement about environmental effects by Jones (1982): "in general, recruitment [annual addition to a fishable stock] is not directly dependent on the size of the spawning stock, but **appears to be dependent on other factors, frequently environmental in origin** [emphasis mine], that affect the fish during the early larval and juvenile stages. Fluctuations in stock size are therefore very largely due to events that occur during the early life stages and are generally less dependent on events that influence the size of the adult stock."

Coastal/estuarine pollution can affect any life stage of fish (Figure 2), but it is during their first year of life -- and more specifically during their first few months of life -- that fish can be particularly sensitive to toxic contaminants (Rosenthal and Alderdice 1976). Factors affecting pre-recruit mortality are therefore of great significance in determining long-term population stability. Death may occur at any point in the life cycle; when translated into population terms, mortality may be chronic or catastrophic, as shown in Figure 3. Pollution impacts may occur at several points in this process.

Life history patterns of fish are important in determining the extent of pollution effects. Potential critical aspects include: (1) location of spawning (freshwater, estuarine, coastal); (2) location of egg deposition (pelagic, demersal); (3) depth preference of hatched larvae in water column -- surface film to bottom; (4) location of nursery area for postlarvae and juveniles; (5) feeding behavior and diets of all life stages; and (6) extent of migration into and out of polluted zones, and duration of occupation of those zones.

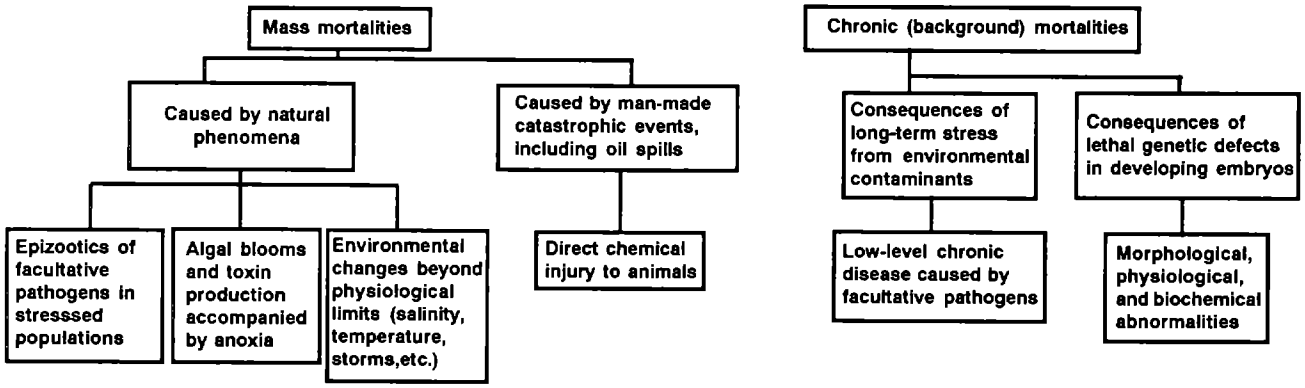

Figure 3. Causes of mortality in marine fish.

These life history factors are, of course, modulated by the nature, extent, and intensity of pollution in every part of the habitat occupied by the species at any developmental stage, from embryo to adult.

CASE HISTORIES OF POLLUTION IMPACT STUDIES

Probably the most critical problem in assessing pollution effects on fish stocks is that of **separating natural and fishing mortality from pollution-induced mortality**. The nature of the problem was explored thoroughly by Jones (1982), whose illustration of the complexity of population responses is presented as Figures 4a and 4b.

The extent of continuing frustration about inability to distinguish causation can be detected in extreme statements such as those of Segar and Stamman (1986): "Biological monitoring programs that cannot separate pollution-induced change from natural change should be terminated and those regulatory requirements which require such programs changed." A reasonable rebuttal to such subjective conclusions might be that "We should intensify research leading to better delimitation of pollution-induced from natural changes in marine populations, and in the interim should adopt a 'precautionary principle' developed recently by Germany (Dethlefsen and Tiews 1985; Dethlefsen 1986) and accepted at the Second International Conference on the Protection of the North Sea in 1987." The principle "**requires action to reduce pollution even in the absence of soundly established scientific proof for cause and effect relationships** [emphasis mine]."

Cause-and-effect relationships -- demonstrations of population impacts of pollutants -- have been subjects of searches by many investigators, especially during the past two decades. Quantitative studies have focused on species of economic importance that may be at risk from coastal/estuarine pollution. Three of these species -- Atlantic menhaden (*Brevoortia tyrannus*), striped bass (*Morone saxatilus*), and winter flounder (*Pleuronectes americanus*) -- will be examined as case histories in the following sections.

ATLANTIC MENHADEN

Estimations of mortality rates in fish stocks from fishing and from natural causes can be made through use of a variety of models developed for assessment and management purposes. Subject to some degree of imprecision, such models may be adapted to assessing effects of pollution by integrating pollution mortality terms into them (Ricker 1954; Schaaf 1979; Cross *et al.* 1985). The modified models can provide estimates of varying pollution mortality on fish stocks at various levels of exploitation (Vaughan, Kanciruk, and Breck 1982; Vaughan, Yoshiyama, Breck, and DeAngelis 1982; Kanciruk *et al.* 1982). Cross *et al.* (1985) described a density-independent variation of a Leslie matrix model (Leslie 1945, 1948; O'Neill *et al.* 1981) that was used to demonstrate population responses of Atlantic menhaden, a relatively short-lived species, to simulated pollution events. They pointed out that all sources of mortality -- natural, fishing, and pollution -- at all life stages of a species throughout its range, must be quantified for effective prediction of pollution effects. A diagram of their model is shown as Figure 5. By using the extensive menhaden data base of the National Marine Fisheries Service's (NMFS's) Beaufort (North Carolina) Laboratory, and imposing both one-time pollution-related catastrophic mortalities and chronic mortalities on one age class, the response of the entire population for the next 30 yr was simulated.

For a simulated catastrophic event (an oil well blowout in coastal waters), and assuming a one-time, 50-percent reduction in survival of 0-age-group menhaden, Cross *et al.* (1985) estimated that the total biomass would be reduced by about 12 percent over 30 yr. Because stocks are heavily exploited and thus may have little compensatory reserve, the simulation indicated a permanent reduction in stock size of nine percent as a consequence of the event. For a simulation of chronic effects, Cross *et al.* assumed slow but continuous decline in estuarine water quality and substantial increase in ocean dumping. The simulation predicted a 40-percent decrease in total population biomass in 30 yr.

In a subsequent paper (Schaaf *et al.* 1987), estimates of acute and chronic pollution effects derived from simulation modeling were made for Atlantic menhaden as well as seven

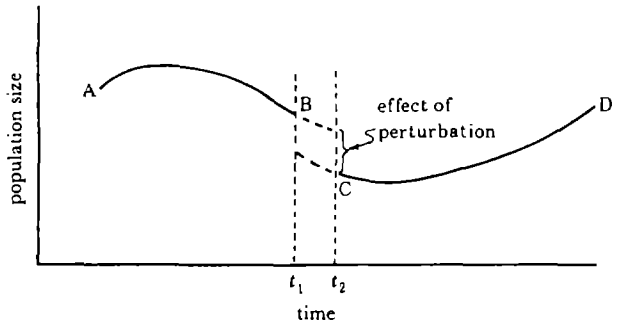

Figure 4a. Simplified response in which a population stabilized at level AB is perturbed from $time_1$ to $time_2$ and becomes stabilized at a new level CD. Responses are shown with and without random fluctuations in population size superimposed on average levels. (From Jones 1982, Figure 3. Copyright The Royal Society. Reprinted with permission.)

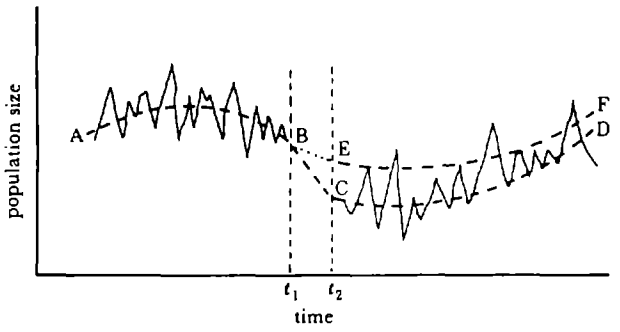

Figure 4b. Simplified response of a population subject to long-term cyclical variation and short-term random variation (AB) that is perturbed from $time_1$ to $time_2$. The result of the perturbation causes a change in population size (CD) to be different than had the perturbation not occurred (EF). (From Jones 1982, Figure 4. Copyright The Royal Society. Reprinted with permission.)

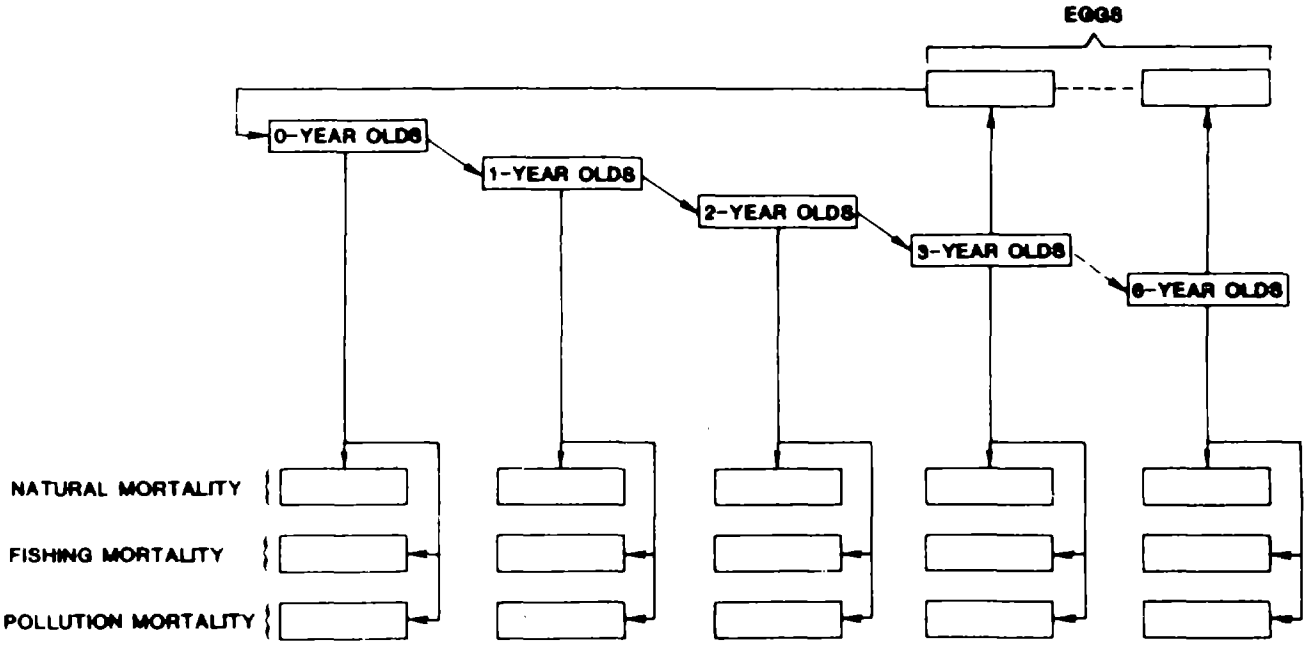

Figure 5. Schematic diagram of Leslie matrix model adapted for menhaden. (From Cross *et al.* 1985. Copyright ASTM. Reprinted with permission.)

other species: spotted seatrout (*Cynoscion nebulosus*), weakfish (*C. regalis*), Atlantic croaker (*Micropogonias undulatus*), striped bass, summer flounder (*Paralichthys dentatus*), bluefish (*Pomatomus saltatrix*), and king mackerel (*Scomberomorus cavalla*). The study, using Leslie matrix modeling, was designed not only to examine population effects, but also to compare the relative vulnerability of several marine fish stocks to pollution. Findings of Schaaf *et al.* included these:

1. "The modeled stocks responded to a simulated catastrophic event (a one-time 50-percent reduction in first year survival) by taking, on average, 10 years to equilibrate at 88 percent of preimpact abundance.

2. Severe problems in the simulations were in estimating the inherent compensatory capacity of the stock, and in random density-independent variability in first-year survival.

3. Species that are impacted the most by coastal/estuarine pollution seem to stabilize most rapidly following acute stress.

4. Stocks most susceptible to acute stress are even more susceptible to chronic stress for at least up to 20 yr.

5. Estimates of first-year survival are critical to any attempts at simulation modeling.

6. Estimates of a species' susceptibility to pollution stress (in terms of stabilization time after impact) can be made from life history data, including age-specific survival and fecundity rates."

As Schaaf *et al.* pointed out, simulations of this kind can provide estimates of the magnitude and duration of pollution impacts that will be useful to resource managers.

Exploring the possible effect of a single human-induced catastrophic event (such as an oil spill) on stock abundance in future years, Vaughan *et al.* (1986) examined the effect of mass mortalities of young menhaden that occurred in 1984, to test the ability to detect reductions in populations following acute pollution events, because of variability in young-of-the-year (YOY) survival. They concluded that: "a catastrophic loss to the Atlantic menhaden 1984 year class (*e.g.*, greater than a 50-percent loss in abundance of the 1984 menhaden year class from the entire Atlantic coast) would have to occur to be detectable at reasonable levels of statistical power (*e.g.*, greater than 70-percent chance of detection), but more subtle reductions (*e.g.*, less than a 25-percent loss to the Atlantic menhaden 1984 year class from the entire Atlantic coast) would undoubtedly go undetected (e.g., chance of detection less than 12 percent). **Such difficulties in detecting reductions are typical of most fish stocks having comparable or larger inherent variability in recruitment or landings** [emphasis mine]." They stated also that "The occurrence of any confounding source of mortality will make it more difficult to detect a reduction due specifically to the cause of mortality under investigation."

These studies with menhaden by Vaughan *et al.* (1986) were preceded by similar attempts to analyze the ability to detect reduction in year-class strength of white perch (*Morone americana*) in the Hudson River (Van Winkle 1977; Van Winkle *et al.* 1981; Vaughan and Van Winkle 1982). Their general conclusions with the white perch analysis were that at least 20 yr of data collection would be required to detect an actual 50-percent reduction in mean year-class strength; and that annual fluctuations can mask major reductions in mean year-class strength.

The simulation models developed for predicting impacts of pollution and other human-induced habitat changes can be designed to do two things: (1) simulate the annual effect on recruitment of YOY fish into the adult population (YOY model), and (2) simulate the long-term effect of reduced recruitment on population size (life cycle model). The YOY models predict either the number of YOY surviving to age 1, or the percent reduction in the YOY. These outputs from YOY models are then used in conjunction with a Leslie matrix model (life cycle model) to predict long-term yields or adult population reduction (Swartzman *et al.* 1977).

An important consideration in any modeling is the determination of density-dependent and density-independent sources of mortality. If the probability of death of an individual decreases as the density of individuals in the population decreases, mortality is termed density dependent or compensatory. The extent of compensatory mortality incorporated in the model can account for large variations in predicted population reductions. The manner in which compensatory mortality acts was hypothesized by Swartzman *et al.* (1977) as follows: "For densities of [YOY] fish below an age-specific, critical density, survival is density independent; above this critical density, survival is density dependent in that it decreases as density increases."

Development and application of simulation models to predict effects of pollution on fish stocks are of course only one approach in the battery of approaches that can be and are being applied to sorting out the causes of mortality in resource species. Other sources of information are data from: (1) fisheries landings and fisheries-independent surveys, useful in estimating stock size, recruitment, and mortality; (2) measurements of physical/chemical changes in the oceans; (3) chemical analyses of contaminant levels in habitats and fish tissues; (4) measurements of physiological responses and biochemical transformations of contaminants by marine organisms; (5) experimental exposures to single or multiple contaminants; and (6) field exposures of resource species in polluted zones.

The effort required for an integrated long-term program that includes all these elements would appear to be overwhelming, but problems of the complexity described can be addressed with modern computer power and a major commitment of research resources.

STRIPED BASS

The striped bass has had a long history of extensive fluctuations in catches (and presumably abundance); its center

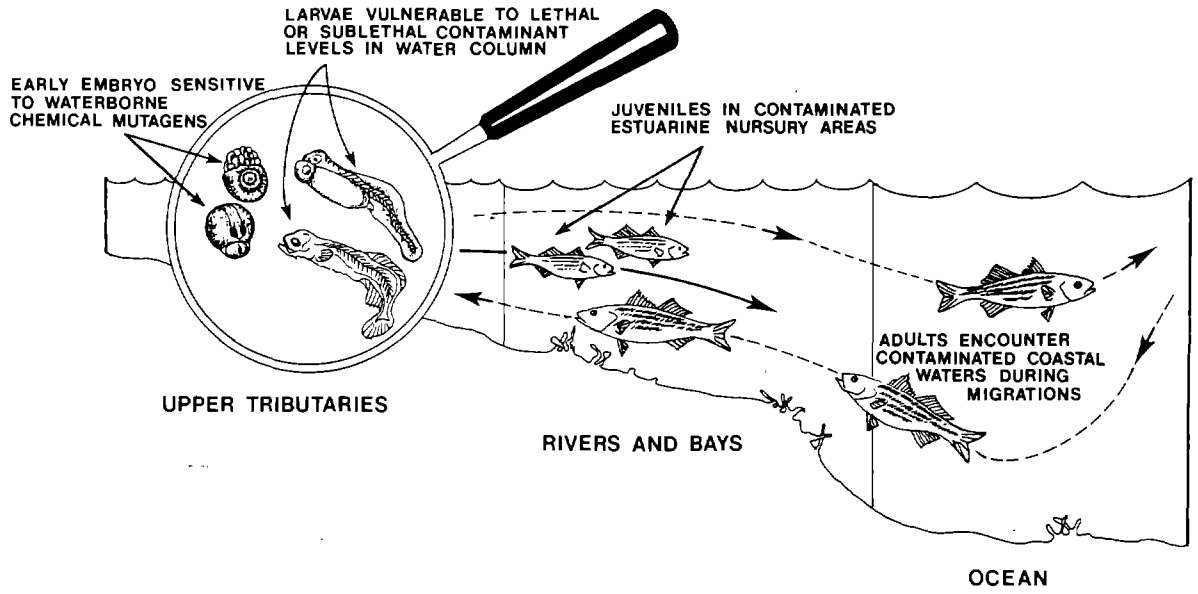

Figure 6. Life cycle of the striped bass, *Morone saxatilis*, with potential pollutant impact points.

of abundance is Chesapeake Bay, with lesser centers in the Hudson and Delaware estuaries (Mansueti 1962; Saila and Lorda 1977).

During their life cycle, striped bass occupy a variety of aquatic habitats, from the lower freshwater zones of spawning rivers to the open sea (Figure 6). For most of their lives, but especially during the first year, they are in habitats exposed to an array of chemical contaminants, as well as to the highly variable estuarine and riverine environments. It is generally accepted that survival during the first 60 days of life determines the size of a year class (Figure 7). It is also well accepted that certain early life stages are particularly vulnerable to environmental toxicants.

Two aspects of human interference with the natural order of things (other than overfishing) have been responsible for extensive quantitative studies of striped bass on the Atlantic coast of United States: (1) possible population impacts of power plant siting and operation on the Hudson River, and (2) the possible role of toxic contaminants in reducing abundance of Chesapeake Bay stocks. The Hudson River studies peaked in the mid-1970s; the Chesapeake Bay studies culminated in extensive documentation in the late 1970s and early 1980s. Investigators were particularly interested in quantitative information linking human activities with effects on striped bass survival. Population aspects of the Hudson River studies were well summarized in a conference volume (Van Winkle 1977) that includes papers by Saila and Lorda, Leggett, Horst, and others. An excellent series of papers by Goodyear (1978-85), Goodyear *et al.* (1985), Boreman and Austin (1985), and others provide analyses based on information from the Chesapeake Bay investigations.

The recent decline in striped bass stocks in the Chesapeake Bay, beginning in 1973 and continuing to 1985, was attributed to various causes, especially overfishing and reduced survival of larvae because of chemical pollution. A major research effort in the late 1970s and early 1980s confirmed that fishing effort had increased since the mid-1960s, and might have affected recruitment (Merriner 1976; Goodyear 1978, 1980, 1984a,b; Levin and Goodyear 1980; Florence 1980; Mehrle *et al.* 1984; Boreman and Austin 1985; Goodyear *et al.* 1985). Evidence was also found of elevated levels of PCBs and other contaminants in tissues of YOY and in their habitats (Mehrle *et al.* 1982; Pizza and O'Connor 1983), and experimental exposure of yolk-sac larvae in the laboratory to environmental levels (but artificial

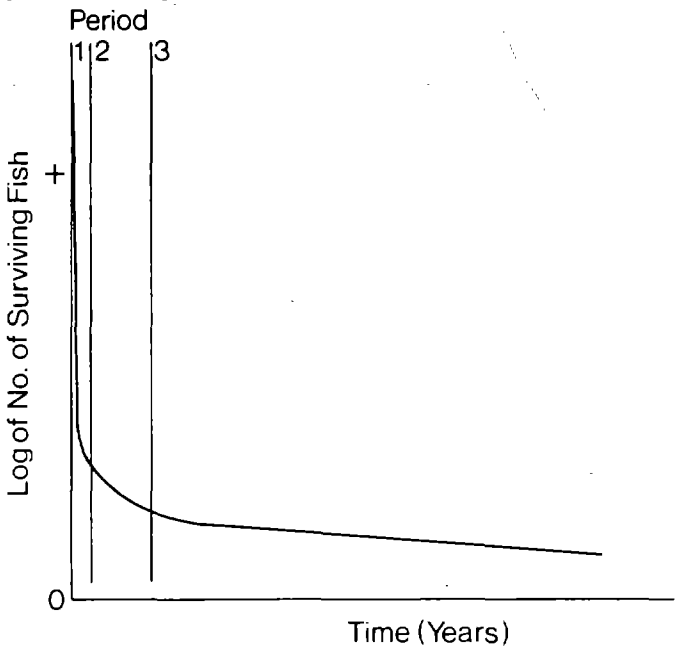

Figure 7. Hypothetical survival curve of one year class of fish. The division of the survival curve into three periods is discussed in the text in relation to the prospect of epidemic diseases contributing to mortality in each period: period 1, eggs and larval fish; period 2, pre-recruit fish; and period 3, fished stocks. (From Munro *et al.* 1983. Copyright ICES. Reprinted with permission.)

mixtures) of selected contaminants resulted in increased mortality (Hall *et al.* 1984, 1987). Yolk-sac larvae exposed in *in situ* chambers to Nanticoke River water (a tributary of Chesapeake Bay) also died differentially; the authors (Hall *et al.* 1985) suspected that high aluminum and low pH levels could be implicated. However, a later experimental study of yolk-sac larvae by Hall *et al.* (1987), also using *in situ* chambers placed in their natural habitat in the upper Chesapeake Bay (Chesapeake and Delaware Canal), did not indicate acute harmful effects although sublethal gill abnormalities were seen in yearlings.

In addition to possible effects of toxic chemicals on larvae, effects on reproduction were also investigated as a possible cause of the population decline. Concentrations of PCBs up to 26 ppm were reported in adult striped bass from the Hudson River, and elevated concentrations of PCBs, DDT, and dieldrin were found in striped bass eggs (Mehrle *et al.* 1982). The organochlorine residues were associated with failure of cleavage in fertilized eggs, but correlations were not significant, according to the authors. In an earlier study of striped bass in California, eggs were found to contain 5-10 ppm DDT, but reproductive depression was not demonstrated (Hunt and Linn 1969).

In other studies, Westin *et al.* (1985) reported that survival of larvae from eggs spawned by contaminated female striped bass was inversely related to concentrations of chlorinated hydrocarbons (hexachlorobenzene, DDT, PCBs, and chlordane) in the eggs, and that parental sources of these contaminants had greater effects on survival than did dietary sources. However, in an earlier study (Westin *et al.* 1983), eggs with PCB content from 1.1 to 8.1 μg/g (ppm) wet weight did not differ from controls in survival and growth after yolk absorption.

In still other studies, tissue contaminants were found to produce abnormalities that might affect stock abundance. Mehrle *et al.* (1982) found that YOY striped bass from the Hudson River had high tissue PCB levels and vertebrae that were fragile and ruptured under minimal force. The authors referred to laboratory studies indicating that contaminants such as PCBs, cadmium, and lead could weaken vertebral structure and contribute to mortality.

Goodyear (1985b) attempted, with simulation models, to examine the relative influence of two factors -- increased fishing mortality and contaminant toxicity -- in producing the observed decline. His principal findings were these:

1. "At low levels of density-dependent mortality, an increase in fishing mortality, or an equivalent decrease in early life-stage survival caused by the toxic effect of a contaminant[,] would cause similar declines in the stock."

2. "At high levels of density-dependent mortality, the effects on the yield are similar if the contaminant-induced mortality precedes the density-dependent mortality. However, if the contaminant-induced mortality occurs after the period of density-dependent mortality, the decline in yield will be more severe than that caused by an equivalent increase in fishing mortality."

However, despite the impressive amount of data and analyses available from Goodyear's and other studies, he concluded that "the actual level of any excess mortality that is imposed on the striped bass population from toxic substances is unknown, and it will probably remain so for some time." But he then went on in a more optimistic vein, stating that reduced fishing mortality could result in a 20- to 30-fold increase in population fecundity which could "offset even rather severe losses due to contaminant toxicity, and could halt or reverse the decline in stock."

It is very interesting, and no doubt significant, that Goodyear's (1985b) conclusions were supported by the behavior of the fishery and the population in the late 1980s. Beginning in 1985, the states of Maryland and Virginia imposed a moratorium on striped bass fishing that remained in effect until 1990. A dramatic increase in spawning stocks occurred (an estimated fivefold increase in spawning females in 1989 as compared to 1984), as well as a high average YOY index. Based on available evidence for the resurgence of striped bass stocks, the Atlantic States Marine Fisheries Commission adopted a conservative management plan for limited harvest and a quota system beginning in 1990.

What can be concluded from results of this research on Chesapeake Bay striped bass, followed by implementation of management measures, and the resurgence of the population in the late 1980s? It begins now (1994) to appear that high fishing mortality was the principal culprit in the decline in stocks since 1973. (It might be noted that to some quantitative biologists it has appeared that way all along, especially because fishing mortality rates on immature striped bass were very high.) It also appears that high larval mortality in some spawning tributaries may have been contributory, if stressful environmental conditions (pH, hardness) occurred coincident with toxic levels of specific contaminants (as, for example, aluminum did in the studies of Hall *et al.* (1985) in the Nanticoke River). These conclusions support the concept that a heavily exploited population may have limited compensatory reserve and may be particularly sensitive to pollution (Cross *et al.* 1985). In the present case, drastic reduction in fishing mortality during 1985-90 because of the moratorium allowed population expansion, even if other factors such as low larval survival due to pollution had contributed to the previous decline.

WINTER FLOUNDER

Over the past two decades, the winter flounder has been the subject of many studies that have emphasized pollution effects on early life history stages (Figure 8). The average female produces about 600,000 eggs, of which an estimated 10-16 percent hatch. Only an estimated 18 individuals per 100,000 hatched larvae survive to age 1 (Saila 1962; Berry *et al.* 1965). The species is estuarine dependent, both for nursery areas and for overwintering sites for adults. Many estuaries are polluted, especially the sediments, and much of the life history of the winter flounder is spent in close association with the contami-

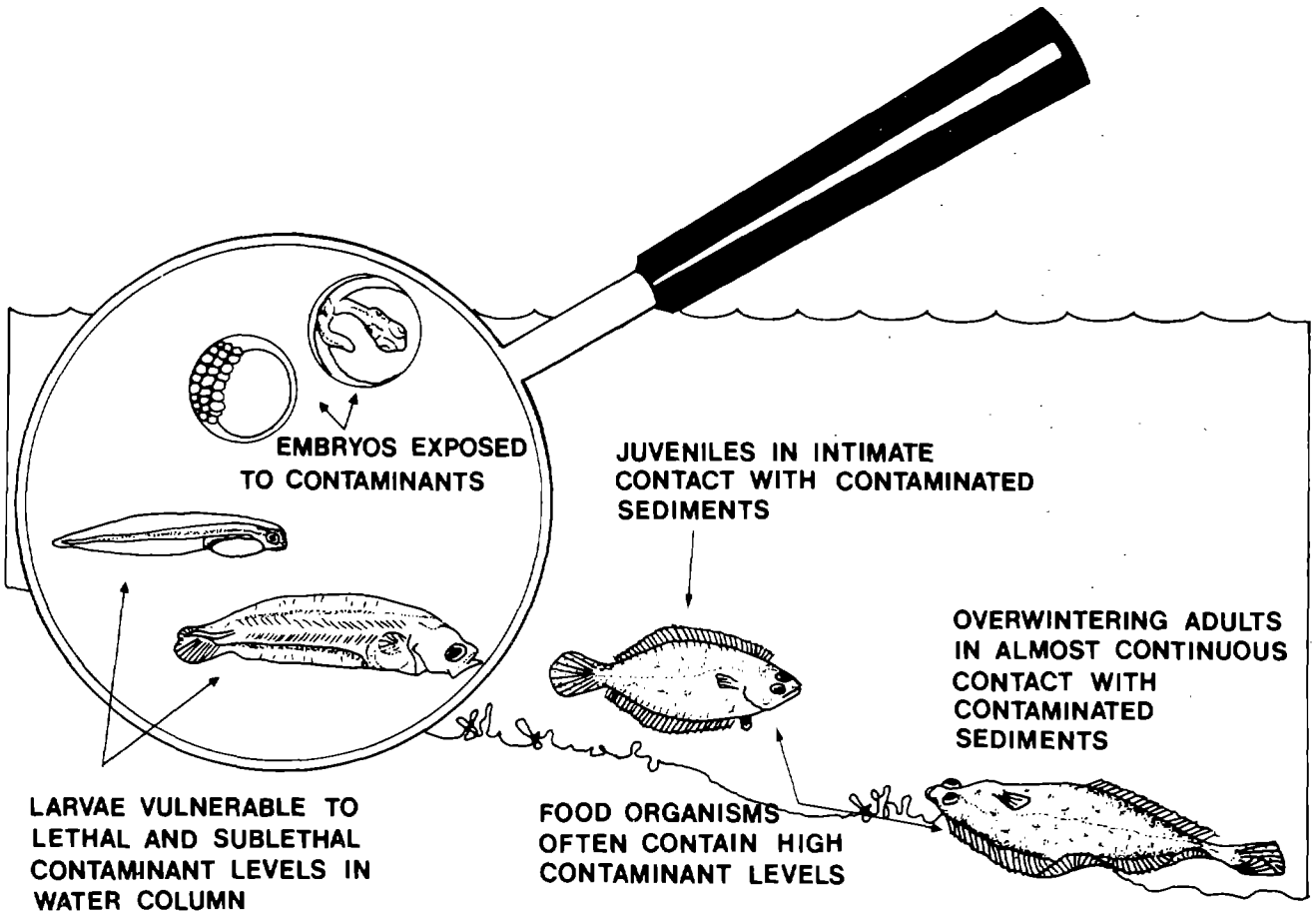

Figure 8. Life history stages of demersal fish that are vulnerable to pollution.

nated bottom sediments in those estuaries. Adults lie partially buried in bottom sediments; spawning occurs near the bottom; eggs sink to the bottom, where they aggregate in clusters; and larvae, after hatching, alternately swim upward and then sink to the bottom (Bigelow and Schroeder 1953; Hughes *et al.* 1986). Fin erosion (a good pollution indicator) is a commonly seen condition in adults from polluted habitats (Ziskowski and Murchelano 1975; O'Connor *et al.* 1987), and tissue levels of pollutants in juveniles and adults can be significantly elevated.

Accumulation of chlorinated hydrocarbons in the tissues of adults and transfer to eggs by females have been found in several studies to contribute substantially to larval mortality. Evidence was found that high mortalities of larval winter flounder in a Massachusetts estuary (a tributary of Buzzards Bay) could be related to pesticide pollution (Topp 1967; Smith and Cole 1970). Adult females concentrated DDT, DDE, and heptachlor epoxide in their ovaries as spawning approached, and mortality of post-yolk-sac larvae was estimated to approximate 100 percent. The authors pointed out the similarity of this pattern of reduced hatchability and larval mortality to that reported for several salmonid species, and considered to be the result of DDT contamination (Burdick *et al.* 1964; Allison *et al.* 1964; Johnson and Pecor 1969). The explanation offered by Burdick *et al.* was that DDT was translocated to the maturing eggs, where, bound to yolk fats, it remained inactive biologically until such fats were metabolized by the developing fry; DDT at this time was then released with lethal results.

In addition to mortality of winter flounder larvae, Smith (1973) also reported that juveniles of age 2 and younger, year-round residents of the polluted Massachusetts estuary, contained higher tissue levels of pesticide residues than did the migratory adults, but mortalities were not observed.

In a related experimental study of DDT effects on developing eggs (Smith and Cole 1973), adult female winter flounder were exposed to sublethal concentrations of DDT and dieldrin. Dieldrin exposure did not affect survival to hatching, but DDT exposure of females resulted in abnormal gastrulation and mortality after fertilization, and in severe vertebral deformities in 39 percent of the larvae at hatching. Experimentally induced levels of the insecticides in gonads of spawning females duplicated levels found in feral fish in an earlier study (Smith and Cole 1970).

A more recent study of the effects of polluted habitats on reproductive success of winter flounder was reported by Hughes *et al.* (1986). Examining samples collected along a composite pollution gradient in Long Island Sound, the authors found no significant differences in percent viable hatch from eggs taken from females at each site. In cytogenetic studies, however, increased percentage of mitotic anomalies and reduced mitotic rates characterized embryos from the more polluted sites, corre-

lating well with the pollution gradient. Analysis of PCBs in eggs did not indicate a correlation between contaminant levels and the cytogenetic data. A previous preliminary study by Longwell *et al.* (1983) had also disclosed high percentages of chromosomal anomalies in winter flounder from the western end of Long Island Sound.

Another pollution gradient, this one a composite of stations in Narragansett Bay, Rhode Island, and Buzzards Bay, Massachusetts, was exploited in a study of effects of inherited contaminants on eggs and larvae of resident winter flounder (Black *et al.* 1988). Progeny from flounders captured in Buzzards Bay, an area noted for PCB contamination, contained significantly higher levels of PCBs (averaging 39.6 µg/g dry weight), and hatched larvae were smaller in length and weight than progeny from reference site adults. No information was given on percent viable hatch, but as larvae grew to metamorphosis the length/weight differences disappeared; the compensatory growth of larvae from contaminated parents but grown in clean water was attributed by the authors to biotransformation and detoxification of contaminants via mixed-function oxidase systems of the embryos and larvae.

Petroleum contamination can affect survival of winter flounder larvae. Experimental exposures of mature female winter flounder and their developing eggs and larvae to low concentrations of No. 2 fuel oil (Kühnhold *et al.* 1978) produced results that would be useful in population analyses. Exposure to 100 ppb throughout gonad maturation of parents, and during fertilization and embryogenesis, resulted in a 3- to 9-day delay in hatching, a 19-percent reduction in viable hatch, and a four-percent prevalence of spinal defects in hatched larvae. Larvae produced from gametes contaminated during parental gonadal maturation, but then reared in clean water, had a mortality coefficient of 0.130, considered by the authors to be much higher than the calculated mortality coefficient for untreated laboratory-reared winter flounder larvae of 0.036-0.059 (Laurence 1975, 1977). Growth of larvae hatched from gametes from oil-exposed spawners was also slower (growth coefficient of 0.220-0.284 compared to the controls of 0.334). [It might be noted that the topic of acute and chronic oil pollution in the sea was examined in detail from a fishery perspective by McIntyre (1982). He concluded a detailed review by stating that "no long-term adverse effects on fish stocks can be attributed to oil, but local impacts can be extremely damaging in the short term...."]

The NMFS's Northeast Fisheries Science Center, and especially its Milford (Connecticut) Laboratory, is carrying on an extensive interdisciplinary examination of the effects of pollution on winter flounder populations of the Northeast. A recent 3-year (1986-88) study of winter flounder from selected stations in Long Island Sound and Boston Harbor (Nelson *et al.* 1991) disclosed a low-percent viable hatch, small larvae, and delayed embryogenesis in offspring from fish taken at the most polluted sites (New Haven Harbor and Boston Harbor). Such findings indicate low larval survival and reproductive impairment at the most severely degraded sites. A related study found greatest prevalences of chromosomal abnormalities and mitotic disruptions in developing embryos from spawning adults taken at the most polluted sites (Perry *et al.* 1991).

Attempts to quantify total pollution impacts on flounder populations of Long Island Sound -- the ultimate objective of this intensive NMFS study -- must employ complex multivariate analytical methods. Variables of greatest significance are extent of reproductive suppression in spawning females, annual larval and YOY mortality, areal extent of toxic levels of pollutants, and annual variations in natural mortality exclusive of that caused by pollution.

Results from earlier studies with other estuarine-dependent species have provided additional evidence that high tissue concentration of chlorinated hydrocarbons in spawning adults can result in mortalities in developing eggs and larvae. Reproductive failure of a spotted seatrout population in Texas was attributed to this phenomenon (Butler *et al.* 1972). The seatrout population inhabited an estuary that was contaminated heavily with DDT, where DDT concentration in ovaries reached a peak of 8 ppm prior to spawning, compared to less than 0.5 ppm in seatrout from other less contaminated estuaries. Spawning seemed normal, but eggs failed to develop.

Spies *et al.* (1985) compared the reproductive success of starry flounders (*Platichthys stellatus*) from polluted San Francisco Bay with that of a reference population from an unpolluted site. Total PCB content of eggs correlated inversely with embryological success and hatching success -- supporting the stated hypothesis that chronic contamination of reproductive tissues by relatively low PCB concentrations (less than 200 µg/kg) has a pervasive deleterious effect on reproductive success of starry flounder in San Francisco Bay.

Baltic flounder (*P. flesus*) with elevated levels of PCBs in their ovarian tissues had a significant reduction in viable hatch of larvae (von Westernhagen *et al.* 1981). A threshold level of 120 ng/g (0.12 ppm) PCB (wet weight) in eggs and ovarian tissue was considered to be a contamination point above which reduced survival of developing eggs and larvae of that species could be expected. Levels of other chlorinated hydrocarbons or heavy metals could not be correlated with reductions in viable hatch. In a subsequent study of whiting (*Merlangius merlangus*) in the North Sea, von Westernhagen *et al.* (1989) selected 0.2 ppm PCB in ovarian tissue as a threshold above which impaired reproductive success could be expected.

Effects of PCB and DDE on reproductive success of herring (*Clupea harengus membras*) in the Baltic Sea were investigated by Hansen *et al.* (1985). Findings included: (1) viable hatch being significantly reduced by ovarian PCB concentrations of more than 120 ng/g, and by DDE concentrations of more than 18 ng/g (wet weight); (2) a positive correlation between ovarian residues of PCBs and DDE; (3) a linear relationship between ovarian residue levels of PCBs and DDE and viable hatch; and (4) effects of PCBs and DDE on reproductive success probably being additive. Levels of contaminants that reduced reproductive success in this study were low; the authors suggested that other contaminants, not analyzed, may also be involved.

Thus far in this section, effects of pollution on early life history stages have been emphasized, but effects on adults must not be ignored. Detailed studies of pollution effects on winter flounder began decades ago, with one of the first experimental studies of tissue lesions resulting from exposure to copper (Baker

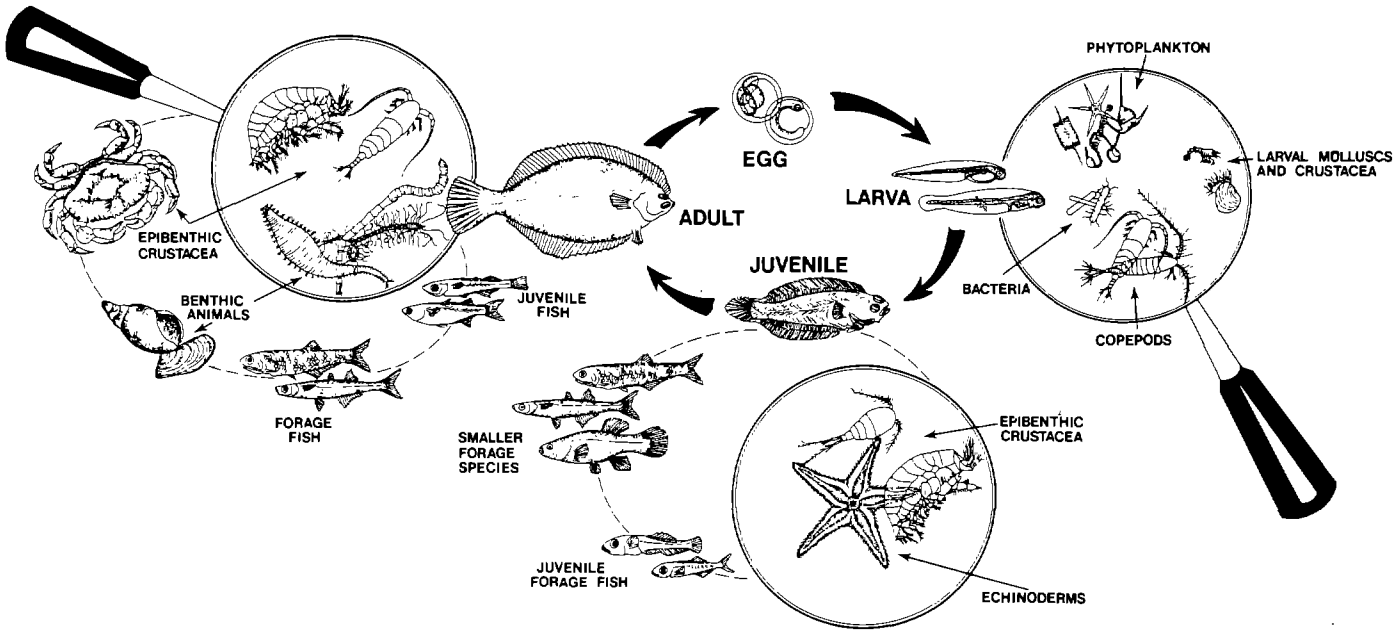

Figure 9. Food chains of winter flounder life history stages.

1969). Principal effects were hemolytic anemia, fatty degeneration of the liver, and renal necrosis -- all potentially lethal. Sublethal physiological effects of two other heavy metals, cadmium and mercury, were reported by Calabrese *et al.* (1975), and the distribution, metabolism, and excretion of DDT and Mirex were examined by Pritchard *et al.* (1973), with the interesting observation that the winter flounder stores its pesticide burden primarily in body muscle.

A recent study of contaminants in winter flounder from a number of polluted sites on the northeast coast of United States (Zdanowicz *et al.* 1986) disclosed that levels of polycyclic aromatic and chlorinated hydrocarbons in stomach contents were higher than those in bottom sediments -- indicating that the compounds were being accumulated by prey organisms (Figure 9). Relating these findings to biological effects, the authors described several kinds of necrotic and degenerative liver and kidney lesions that were relatively more prevalent in fish from severely polluted sites. Zdanowicz *et al.* (1986) did not make inferences about possible pollution-induced mortality, but the correlation of certain sublethal pathological conditions with contaminated habitats and elevated contaminant levels in tissues seemed evident.

An experimental study, exposing adult winter flounders to oil-contaminated sediments for 4-5 mo, resulted in mortalities in summer months, possibly because the oil was acting as a nonspecific stress at a time when temperatures approached an incipient lethal level (Fletcher *et al.* 1981).

The association of progressively severe liver pathology and several types of hepatic neoplasms with badly degraded estuarine/coastal waters is becoming evermore apparent from recent reports. Among them, winter flounder from several degraded areas on the U.S. East Coast (New Haven Harbor, upper Narragansett Bay, Boston Harbor) had prevalences of 3.4-7.5 percent for tumors classified as hepatocarcinomas or cholangiocarcinomas (Murchelano and Wolke 1985) (Figure 10). What seems to be emerging from these studies of tumors in winter flounder and other species in different polluted estuaries, is a sequence of histopathological changes in livers, beginning with fatty deposits and pre-neoplastic changes in liver parenchyma cells. The progression of pathological changes also seems roughly correlated with the extent of estuarine degradation and the length of residence of fish in the estuary. In studies of West Coast flatfish species, positive correlations were obtained between neoplasm prevalence in bottom-dwelling fish and levels of "certain individual groups of sediment-associated chemicals" (aromatic hydrocarbons, chlorinated hydrocarbons, and heavy metals) (Malins *et al.* 1984, 1988). Other studies of East Coast winter flounder have identified an oncogene, derived from tumorous liver and possibly indicating a specific interaction of flounder DNA with polycyclic aromatic hydrocarbons (PAHs) (McMahon *et al.* 1988).

A persistent question, still unresolved fully, is whether the liver tumors kill the fish, or if they regress when the fish moves from the heavily polluted habitat. If the tumors do not regress, the survival of individual fish, as described by Murchelano (1988), "depends on many factors, including the extent of liver damage, the degree of toxicity resulting from decreased hepatic function, the impairment of other essential organs, whether metastasis occurs, and the degree of behavioral modification (the ability to capture prey and avoid predators may be compromised). Population effects are possible if large numbers of fish with hepatic carcinoma die..."

Limited recent information is available about possible reduction in immune responses and hence reduced survival potential of winter flounder as a consequence of exposure to pollutants. In one study, a 4-mo exposure to oiled sediments resulted in statistically significant reduction in liver macrophage aggregates, believed to be important components of the cellular

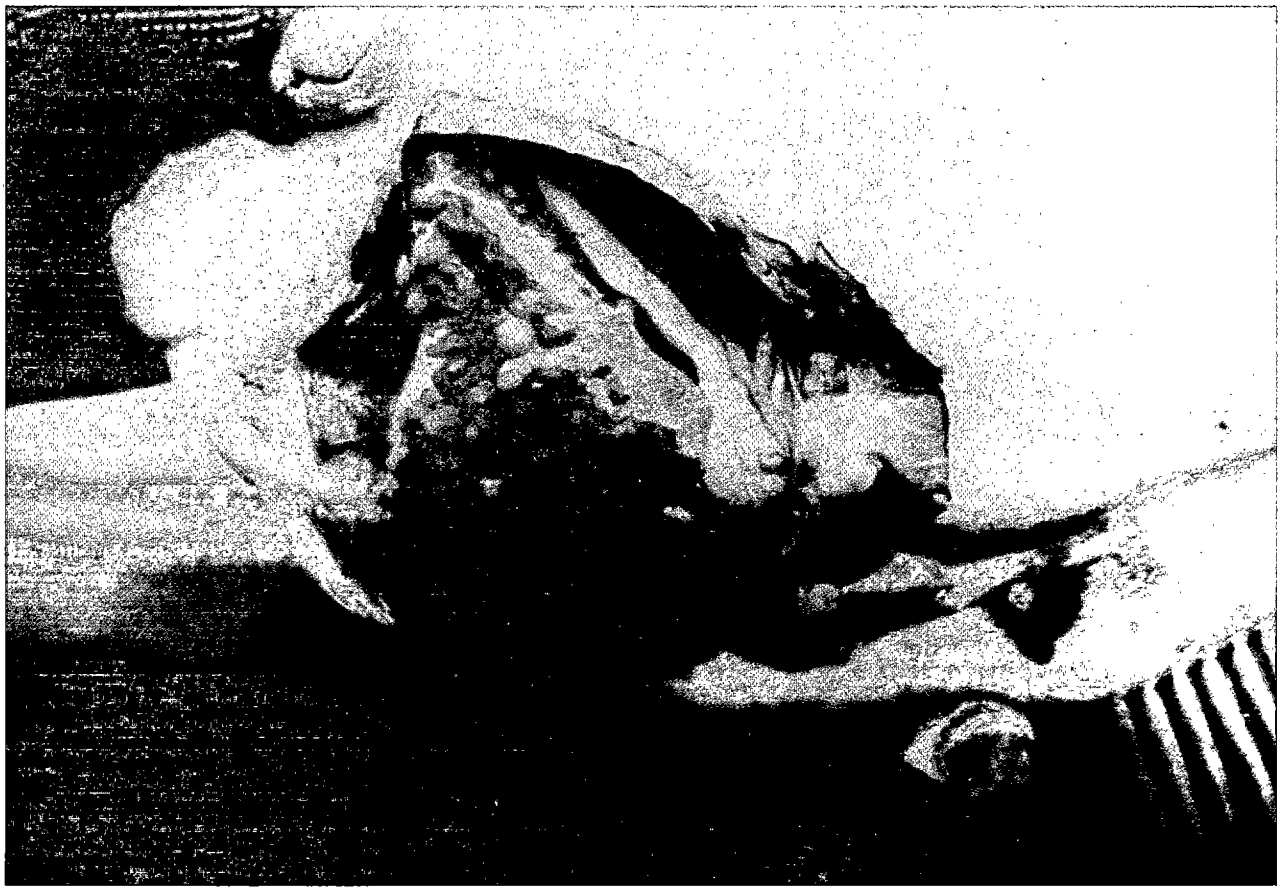

Figure 10. Gross lesions in liver of winter flounder, *Pleuronectes americanus*. (Photograph courtesy of Dr. R. A. Murchelano.)

immune system of fish, and possibly primitive analogues of the mammalian lymph nodes (Payne and Fancey 1989). However, in an earlier study (Wolke, George, *et al.* 1985; Wolke, Murchelano, *et al.* 1985), the numbers of aggregates in fish from polluted areas were not greater than those from reference sites, although their size was. In still other studies with different fish species, aggregates were found to be more numerous and larger in samples from polluted areas than in those from unpolluted sites (Peters *et al.* 1987). It seems plausible, as Payne and Fancey pointed out, that at low chronic levels of pollution, the cellular defense system may function effectively, whereas at higher, more toxic levels of pollution, phagocytosis may be impaired, leading to a decrease in melanomacrophage aggregates.

One final study of pollution effects on adult winter flounder -- this one demonstrating synergism between chronic oil pollution and protozoan (trypanosome) parasitization -- was reported by Khan (1987). Infected fish exposed for 6 wk to oil-contaminated sediments had higher mortality rates than uninfected fish; intensity of infection was higher in oil-exposed fish than in untreated controls; and retardation of gonad development was more pronounced in the oil-treated, parasitized fish than in the other experimental fish.

So here then, with winter flounder, we have a species that has been examined extensively, from pathological, immunological, physiological, and biochemical perspectives, in an effort to understand pollution effects. Probably the most significant findings, from a population point of view, are the demonstrated negative impacts on larval survival due to exposure to chlorinated hydrocarbons -- acting either on prespawning females or on eggs and larvae. Next in order of significance might be the severe morphological changes -- liver tumors and fin erosion in particular -- that have been seen in samples from badly degraded habitats such as the New York Bight apex, western Long Island Sound, and Boston Harbor. Not to be ignored, however, is the observation by Haedrich and Haedrich (1974) that chronically polluted environments seem to have little influence on winter flounder populations.

The one missing ingredient in this scrutiny of pollution effects on the species is a serious attempt to **quantify** the observations made, and **to provide numerical estimates of the extent of population reduction that may result from exposure to pollutants.** Some narrowly focused efforts have been made, such as Smith and Cole's (1973) study of DDT effects on larval mortality, but we are left with little published information that can be extrapolated to the entire population of winter flounder on the northeast coast of United States.

At present, an initial effort is being made by staff members of the NEFSC to develop and test a model to assess pollution

effects on winter flounder population abundance in Long Island Sound (Thurberg, personal communication)[1]. The sheer mass of relevant information available -- biological and environmental -- should enhance the likelihood of success for such a project.

In advance of the availability of any model, it might be instructive to discuss the kinds of information that would be most useful as a data base in developing it. Some elements are: (1) annual landings by geographic subdivisions, with short- and long-term trends and fluctuations; (2) population estimates from trawling surveys, including age structure, age-specific fecundity, and average annual age-specific survival; (3) annual larval abundance and distribution estimates from plankton surveys; (4) data from routine long-term monitoring of physical/chemical variables over the entire range of the species, but emphasizing spawning/nursery areas; (5) descriptions and detailed maps of the levels of principal pollutants (PAHs, PCBs, heavy metals) in sediments and the water column in important estuarine/coastal habitats of winter flounder; (6) a history of the nature and degree of pollution in each major estuary important in the life history of winter flounder; (7) additional descriptions of the effects of pollutants on developing eggs and larvae, with projections of population impacts at various pollutant levels; and (8) additional descriptions of the effects of pollutants on juvenile and adult fish, with estimates of population impacts at various pollutant levels.

Needed, then, is information on: (1)population size, age structure, and age-specific fecundity; (2) natural mortality rates; (3) fishing mortality rates; and (4) pollution-induced mortality, especially in early life history stages. Simulation models can then be constructed to predict population effects of: (1) a one-time acute pollution event (oil spill, other); (2) long-term increasing levels of pollution; and (3) long-term decreasing levels of pollution.

DISCUSSION

The case histories of quantitative pollution effects on populations of Atlantic menhaden, striped bass, and winter flounder presented here are, of course, only examples. Some information is available for certain other species. If this paper were to be written from a European perspective, the long-term research of German workers (Rosenthal, Dethlefsen, von Westernhagen, and their colleagues), examining pollutant effects on larval survival and development, would certainly be emphasized. This series of studies began in 1967 (Kinne and Rosenthal 1967) and continues to the present time (Rosenthal *et al.* 1986; Dethlefsen *et al.* 1987; Dethlefsen 1988; von Westernhagen 1988). Among the many significant findings reported in this series of papers are these:

1. Exposure of maturing females to low concentrations of contaminants -- especially those which are bioaccumulated -- can affect gonad tissue, with effects expressed in the next generation.
2. Life cycle stages most vulnerable to contaminants are maturing females, early embryos, early-hatched larvae, and larvae at transition from yolk-sac to feeding.
3. A wide range of morphological, behavioral, and physiological abnormalities in larvae result from exposure to contaminants, in rough proportion to the environmental level of the particular contaminant.
4. Common morphological abnormalities include malformed lower jaw, eye deformities, anomalies in the vertebral column, and reduced size at hatching.
5. Common physiological abnormalities include reduced heart rate, reduced swimming ability, disturbance in equilibrium, and reduced feeding.
6. Early developmental stages showed the highest malformation rates.

In a recent study of pelagic fish embryos from the North Sea, malformations were found with high frequency in several species -- dab (*Limanda limanda*), Baltic flounder, whiting, Atlantic cod (*Gadus morhua*), and plaice (*Pleuronectes platessa*) -- taken in regions with known high contamination levels (Dethlefsen *et al.* 1986).

In other studies, malformations of embryos of Baltic herring (*Clupea harengus membras*) were found to be abundant and in great variety following exposure to crude oil and chemical dispersants (Linden 1976).

Abnormalities induced by pollutants are, of course, only part of the spectrum of pollution effects; it is also important to examine those **mortalities** that may result from exposure of prespawning females to contaminated habitats. Some evidence exists. Chlorinated hydrocarbons can contribute substantially to larval mortality by accumulation in tissues and by transfer to eggs from the parent females. One specific example of larval mortality which seems related to increased environmental contamination -- mortalities of larval winter flounder in a Massachusetts estuary related to pesticide pollution (Smith 1973) -- was discussed in this paper's case history section. Adult females concentrated DDT, DDE, and heptachlor epoxide in their ovaries as spawning approached, and mortality of post-yolk-sac larvae approached 100 percent.

Another earlier study, referred to previously, provided evidence that high tissue concentrations of chlorinated hydrocarbons in spawning adults can cause mortalities of developing eggs. Reproductive failure of a spotted seatrout population in Texas was attributed to this phenomenon (Butler *et al.* 1972). The seatrout population inhabited an estuary that was contaminated heavily with DDT, and DDT concentration in the ovaries reached a peak of 8 ppm prior to spawning, compared to less than 0.5 ppm in seatrout from other less contaminated estuaries. Spawning seemed normal, but the eggs failed to develop.

Several more recent studies have indicated that high levels of PCBs in the gonads of spawning fish (Baltic flounder, Baltic

[1] Dr. F.P. Thurberg, NMFS, 212 Rogers Ave., Milford, CT 06460.

herring, striped bass, and whiting in the North Sea) can result in low (less than 50 percent) viable hatch of eggs, due to abnormal development and high embryo mortality (von Westernhagen *et al.* 1981, 1988; Hansen *et al.* 1985; Westin *et al.* 1985; Cameron *et al.* 1986). On the California coast, white croaker (*Genyonemus lineatus*) inhabiting contaminated waters near Los Angeles had high body burdens of chlorinated hydrocarbons, greater early oocyte degeneration, lower fecundity, and lower fertilization rates than fish from reference areas (Cross and Hose 1988). Other effects of environmental pollutants on fecundity have been reported. As an example, a study of ovarian development in plaice captured on the French coast in 1979 and 1980, near the site of the Amoco Cadiz oil spill in 1978, revealed reduced concentrations of developing follicles, and no mature follicles in any season (Stott *et al.* 1983).

Effects of contaminants also include hormonal imbalances, which may affect ovulation or spawning (Struhsaker 1977; Wedemeyer *et al.* 1984). Additionally, hatchability of eggs and viability of larvae may be impaired, either by high tissue contaminant levels in the parent females, or by toxic levels of contaminants in the spawning environment (Ernst and Neff 1977; Smith and Cameron 1979; Hansen *et al.* 1985; Johnson *et al.* 1988).

Of course, the basic question that must be asked is **"Can defective embryonic development and high embryo mortality due to pollution affect recruitment?"** Some observations relative to this question have been proposed by von Westernhagen *et al.* (1988). According to their reasoning, total mortality during the embryonic stage of development of marine fish has been estimated to be high -- 95-99 percent for species such as cod and plaice in the Baltic Sea, for example. At this mortality level, decreases in survival rates due to embryo abnormalities, at observed levels from 22 to 33 percent, would be too small to detect in unexploited populations, but in **overexploited populations** in which spawning stocks have been reduced severely, the added impact of abnormal embryonic development and high embryo mortality could result in reduced recruitment. The authors also pointed out that, for the North Sea, the highest prevalences of embryonic malformations occurred in highly polluted areas (off the mouths of the Rhine and Elbe Rivers, and in the vicinity of the dumping zone for titanium dioxide wastes).

From the perspectives of the research scientist and the resource manager, three rather clear responsibilities exist when considering pollution effects on fisheries: one is to demonstrate that estuarine/coastal pollution is really affecting fish and shellfish **stocks**, the second is to define the local or regional stocks (especially shellfish) that **are** affected, and the third is to propose management measures to mitigate damage if it exists.

These responsibilities can be satisfied in four stages which are easy to state but difficult to accomplish: (1) isolate and quantify pollution effects on resource species -- as distinct from effects of natural environmental variations; (2) conduct critical examinations of pollution effects at levels of the individual, the local population, and the species; (3) encourage the identification and quantification of sensitive early-warning indicators of environmental degradation; and (4) attempt to reduce pollutant inputs where damage to living resources has been or can be demonstrated. Item 4 is particularly difficult to achieve; it points out the need for aquatic scientists to interact with those responsible for managing and regulating terrestrial sources of pollution.

Case histories of quantitative effects of pollution on populations of Atlantic menhaden, striped bass, and winter flounder, as presented in this paper, illustrate a series of generalizations useful in stock management:

1. In assessing pollution impacts, the **entire range** of the species should be considered, and precise information about levels of all contaminants throughout that range should be available. Furthermore, migratory characteristics must be considered, since some species may move rapidly into or out of areas of severe pollution.

2. Particular attention must be paid to controlling pollution levels in spawning/nursery areas, since most pollution-associated mortality will occur during the first year of life. It is generally accepted by fish population biologists that survival during the first 60 days of life determines the size of the year class of many estuarine-dependent species, including striped bass (Westin and Rogers 1978); factors affecting early survival (such as pollution) should be of concern and be subjects of management action.

3. "Very little impact [from pollution] should be tolerated at low stock size, because it would prevent recovery to a...maximum sustained yield" (Cushing 1979).

4. Simulation models can supply meaningful information, but the degree of reliability of such models depends on the extent of the data base employed, and very few have worked at all.

5. Marine/anadromous fish populations are characterized by aperiodic dominant year classes which may form the basis for a fishery for many years. (An example would be the 1970 striped bass year class in Chesapeake Bay.) Existence of these dominant year classes, and examination of factors responsible for their production, can lead to important insights about environmental influences on abundance.

Previous sections of this paper examined case histories that indicated points in the life cycles of fish and shellfish where pollutant stresses can exert lethal and sublethal influences. From experimental studies, it seems obvious that with all these potential impact points throughout life cycles, populations in contaminated waters should dwindle and disappear; yet from experience on the North American East Coast, this has not happened (at least not yet). Only in severely degraded local waters, which are a small part of the total range of most fish and shellfish species, have there been localized disappearances; even in those areas, other species which might be expected to be affected are still present and are in some instances abundant. This is true particularly of a number of coastal/estuarine-dependent fish species, many of which spend much of their life

cycles in waters that are to some extent contaminated. It may also be true for migratory species that can move into or through degraded zones, although any possible impacts of transient exposures to contaminants are more difficult to recognize, and much more difficult to quantify.

A statistical analysis of the fishery resources of the United States (Wise 1974) discussed catches of all important marine fish and shellfish, on a species by species basis, for the preceding 20 yr (1950-70). Included were the major Atlantic species usually considered estuarine-dependent or estuarine and nearshore inhabitants, species which might be expected to demonstrate some effects of estuarine and coastal pollution. With few exceptions, according to that publication, catches of those species were relatively steady or showed some increases in the 20-year period. One of the summarizing statements in the report is of particular note: "In general there are no good fishing effort data available for those estuarine species whose catches have remained constant, so it is possible that maintenance of catch levels is due to constantly increasing fishing effort. **Nonetheless, the evidence from catch records of a substantial number of exploited estuarine species in United States waters indicates that pollution and damage to estuaries have not yet shown any measurable overall effect on the part of the marine resource which might be expected to show the first effects** [emphasis mine]." Admittedly, Wise's report covered only 1950-70, but for at least some of the species considered, the situation has not changed dramatically since that time (with the possible exception of striped bass -- but even here, overfishing seems to have been the principal cause of the recent (1973-86) decline in abundance).

Two major recent attempts have been made to assess pollution impacts on fish stocks off portions of the U.S. coasts -- one by Prager and MacCall (1993) examining data from the Southern California Bight, and the other by Summers *et al.* (1986) using historical data for a number of fish species from five estuaries of the northeastern states. Using two modeling methods -- a biomass-based model and a recruitment model -- Prager and MacCall looked for significant effects of climatic conditions and contaminant loadings on spawning success of three Pacific species -- northern anchovy (*Engraulis mordax*), Pacific sardine (*Sardinops sagax*), and chub mackerel (*Scomber japonicus*). By using the models, no climate or contaminant influences on spawning success of northern anchovies were detected, but spawning success of chub mackerel seemed to be strongly influenced by climatic variability, and (with the recruitment model) spawning success of Pacific sardines was strongly negatively correlated with contaminant loadings -- being consistent with the hypothesis that the stock, which had been overfished and had collapsed in the 1950s, was stressed beyond its limits by poor larval survival due to ambient contaminant concentrations. The study of fish populations of five Atlantic Coast estuaries by Summers *et al.* (1986), using a biostatistical modeling approach based on catch statistics, disclosed consistent patterns of pollution effects among similar species across different estuaries.

Another analysis -- this one of recent changes in abundance of North Sea fish stocks -- failed to find an effect of pollution. Tiews (1989) examined abundance trends of 25 species for 35 yr (1954-88) from trawl catches on the German North Sea coast. He found that some species had declined, some had increased, and others had fluctuated irregularly, but that no consistent long-term decline of commercial species could be attributed to deteriorating habitat conditions. Tiews (1983) had earlier expressed suspicion that, at least for some species, declines might be due to pollution effects, but his latest analysis concluded with the comment that "this study shows that the majority of species studied seems to be able to tolerate the present status of environmental deterioration of the ecosystem. Furthermore, the study does not indicate any fundamental impairment of the fishery biological situation of the area during the last 7 years in comparison to the preceding period from 1954 to 1981. Six species have even substantially increased in abundance." Tiews cautioned, however, that 35 yr may be too short a period to reveal natural population fluctuations, so that a very conservative interpretation of the data is necessary.

Goodyear (1984b) has described the dimensions of the problem of interpreting population responses to direct (lethal) and indirect (sublethal) levels of contaminants very succinctly:

1. For cases where contaminants are directly lethal, two types of information are required: (a) "the timing and extent of the excess mortality," and (b) "the nature, timing, and intensity of the density-dependent processes that regulate the size of the population." This, as Goodyear pointed out, is "a key, largely unsolved problem in fishery research and management in general."

2. For cases where indirect (sublethal) levels of contaminants exist, "The interpretation of the population response to the indirect effects of the contaminants on the species of interest requires quantification of the response in terms of a change in survival probability or a change in reproductive rates, and knowledge of the density-dependent processes that control population size." Goodyear then pointed out that "none of the required information is particularly amenable to measurement"!

Experimental demonstrations of the negative effects of chemical contaminants on survival and well-being of marine fish and shellfish are abundant, but the variability that results from the influences of natural factors is so great and so incompletely understood that experimental findings cannot usually be applied directly to assessments of exploited populations -- despite repeated attempts to do so. Some extrapolations from single-contaminant experimental studies can be useful, however. Föyn and Serigstad (1989) described calculations of year-class reductions in cod, herring, and saithe populations of the Norwegian coast that might result from a major oil spill. Detailed field studies of seasonal larval herring distribution and concurrent laboratory studies of effects of oil on larvae, when presented in a worst-case scenario, enabled calculation of what turned out to be a low percentage of potential reduction in recruitment. Field experimental studies, in which fish were exposed *in situ* to contaminated waters, have also produced meaningful results

when combined with adequate chemical analyses of environmental and tissue samples.

The appearance of new reports of sublethal effects of contaminants on marine animals has been aptly described (Lewis 1980) as of "avalanche" proportions. In a thought-provoking discussion of options in environmental management and the deployment of future research efforts concerned with population effects of pollution, he made the point that "The more subtle threats of chronic pollution well away from the hot-spots, and which because of their subtlety have always been seen as potentially the most dangerous, do not appear to have developed and produced effects on the scale that was feared a decade ago." Lewis then asked difficult questions: "If after all the recent, intensive effort there is still difficulty in finding chronic effects upon communities does this not suggest that such effects are negligible? That while the initial concern over chronic effects was justified is it not now time to acknowledge that it is only acute pollution that matters?" Most of us in pollution research would detect a faint odor of heresy in this superficially logical line of thought, probably rejecting it out-of-hand, since it tends to overestimate human ability to sort out effects of natural versus man-induced population changes, and it tends toward too easy acceptance of the significance of acute pollution events in reducing overall abundance of marine populations. However, we would have to admit that our arguments would not have a strong base in adequately demonstrated population impacts. We might also admit that, just as with certain terrestrial species, a point may be reached where contaminants in the marine environment could reach a level, or operate over sufficient time, to greatly affect population abundance.

Assessment of quantitative effects of pollution and other human-induced environmental perturbations on fish populations depends on an understanding of the stock-recruitment relationships of fish populations of concern, and on knowledge of the density-dependent and density-independent factors influencing those relationships (Leggett 1977). Of particular concern is mortality imposed by toxic levels of pollutants on eggs, larvae, and juveniles of economically important fish species. However, models predicting effects of human intrusions (other than fishing) must be used conservatively, especially when impacts on early life stages are included. Leggett (1977) has voiced the concern with precision: "the potential for error in numerical predictions of the effect of proposed levels of increased mortality on pre-recruit stages is large, while the biologically acceptable range of error is small," and further that "Until a better understanding is achieved of the interacting roles of density-dependent and density-independent factors in regulating population size and stability, and until a much better data base is available for the majority of the stocks subject to mortality from industrial activity, precise numerical predictions of the impact of this incremental mortality on adult stocks should be interpreted with great caution."

By assuming the correctness of the generally held principle that recruitment variability is a consequence of events that occur in egg, larval, and postlarval stages, it is logical to focus pollution studies more directly on reproductive success of populations at risk. This has been done in a number of studies, but one disturbing conclusion that can be reached after examining some of the accumulated literature is that **investigators rarely if ever carry their findings to the point of actually estimating quantitative effects on fish populations and species.** Let's consider a few examples. Von Westernhagen *et al.* (1981), von Westernhagen, Sperling, *et al.* (1987), and von Westernhagen, Dethlefsen, *et al.* (1987) examined contaminant content of ovaries of Baltic flounder, and established a threshold level of 0.12 ppm PCB beyond which reproductive impairment would occur. These authors found that 8.5 percent of the sample exceeded that level (with a range of 0.05-3.17 ppm), and they reported that viable hatch was lower than 15 percent if gonad PCB levels exceeded 0.25 ppm. They did **not** take the final quantitative steps. The Baltic flounder catches are known; the extent of PCB contamination in the Baltic is known -- why not make a rough estimate from these data of the possible impact of PCB contamination on recruitment of Baltic flounder? As another example, Cross and Hose (1988) found reproductive impairment and high levels of ovarian DDT and PCB in white croaker from a contaminated site near Los Angeles. Again, white croaker catch statistics are available, and environmental levels of chlorinated hydrocarbons on the California coast have been examined extensively -- why not take another step, armed with the data, and provide a rough estimate of possible impact on recruitment?

Only once, to my knowledge, has even the penultimate step -- of estimating effects of pollution on recruitment -- been taken. This was the report by von Westernhagen *et al.* (1988) on developmental defects in pelagic fish embryos from the western Baltic. Plankton net catches of cod, plaice, and flounder eggs and larvae were examined for mortality and abnormalities. Prevalences of defective embryos and abnormal larvae in various locations in the western Baltic were determined, and effects on survival of larvae of two year classes (1983 and 1984) were estimated. Decreases (18-44 percent) were considered too small in terms of biological significance to cause a detectable impact on recruitment, although the authors pointed out that even a small impact on larval production could, in the case of overexploited Baltic stocks, lead eventually to reduced recruitment. It is puzzling, considering the availability of excellent fishery statistics and environmental information for the Baltic, some of it extending back for more than a century, why the ultimate step -- of estimating total possible population effects of existing pollution levels -- was not taken, since it is only at this stage that pollution information would become meaningful to resource managers.

Part of the problem may be that professionals interested in problems created by coastal/estuarine pollution do not communicate effectively with population dynamics specialists, so full exploitation of available data does not happen. Only rarely, such as in the Mid-Atlantic striped bass program discussed earlier, when simulations based on good resource and environmental data sets are attempted, is some measure of integration achieved. The assessment scientist is often loath to use the minimalist approach necessary with the environmental scientist's data.

To do an effective job of quantifying pollution effects on fish stocks, a large interdisciplinary research and monitoring program is required. The principal ingredients are shown in

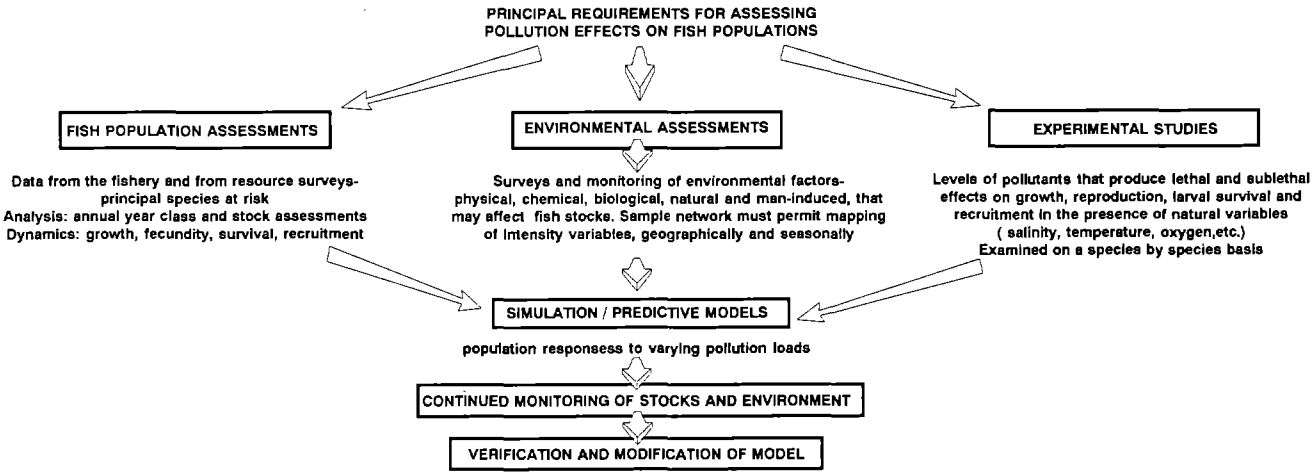

Figure 11. Principal requirements for assessing the effects of pollution on fish stocks.

Figure 11. A triumvirate of population assessments, environmental assessments, and experimental studies constitutes the basic information source for the required modeling effort. Such a program must be long-term as well as geographically broad. Until now, only the Mid-Atlantic striped bass program has approached the level of research commitment required, and even this major effort must face an annual struggle for adequate funding, in spite of its considerable importance and high levels of lobbying for the program.

CONCLUSIONS

More than a decade ago, in a major symposium titled "Protection of Life in the Sea," held at the Biologische Anstalt Helgoland, a series of conclusions was proposed about pollution effects on fisheries -- conclusions that seem still relevant today:

Pollution effects on fisheries have received some scrutiny in recent decades, and information is accumulating, but is still insufficient to be very useful in resource management decisions -- except as they involve local areas. Evidence exists for localized effects of pollutant stress on fisheries, but ***as yet there is little specific evidence for widespread damage to major fisheries resource populations resulting from coastal/estuarine pollution.*** *This may well be because we are unable to separate clearly the effects of pollutant stress from effects of the many other forms of environmental stresses to which marine populations are subject. Other factors, such as shifts in geographic distribution of fish populations, changes in productive ecosystems, or overfishing, may cause pronounced changes in fisheries -- changes which could obscure any effects of localized habitat degradation.* ***It seems, with the evidence presently available, that factors other than pollution are overriding in determining fish abundance, but we lack sufficient quantitative data to make positive statements about cause and effect relationships of abundance and pollution.***

It may be, of course, that estuarine/coastal pollution is exerting some overall influence on certain resource species, but that this influence may be masked by increased fishing effort, or by favorable changes in other environmental factors which create a positive effect on abundance, outweighing any negative effects of pollutants. Many experimental studies, particularly more recent ones concerned with long-term exposure of fish and shellfish to low levels of contaminants, suggest that some long-term effects on abundance should be felt, but our statistics, our monitoring, and our population assessments are not yet adequate to detect them.

Effective long-term monitoring of stocks and environment must be the basis of any attempt to isolate and identify pollution effects. ***A continuous integrated effort in stock assessment, environmental assessment, and experimental studies will be required to understand the role of all environmental stresses -- natural and man-induced -- in determining abundance of resource populations.*** *However, we do not yet have the principal pieces of the puzzle in place, so in the absence of full understanding of the phenomena involved, management decisions affecting estuarine/coastal pollution must be made on the basis of "best available scientific information," just as decisions about allowable resource exploitation are made. In both types of decision processes, a conservative action provides a lower risk of damage and loss than does a more extreme action. Conservatism can be especially significant when decisions are made that might permit pollution to continue or increase, since long-term effects of existing levels on abundance of resource populations are largely undetermined. In addition to advocating conservatism, we must persist in attempts to quantify the effects of pollution, and to determine the precise pathways through which fishery resources are affected* (Sindermann 1980).

Freely admitting to a conservative mind-set (and a mild infatuation with my own prose), there is little about these conclusions that I would change today -- more than a decade after their original publication. Research in the intervening years has

added **substantially** but only **incrementally** and not **conceptually**, to our understanding of pollution effects on resource species; some of that information has been summarized in the case histories just presented. The major problem -- being able to distinguish adequately the effects of pollution from all the other influences on marine fish population abundance -- still confronts us, even though the boundaries of our knowledge have expanded. Recent advances include the creation and implementation of new pollution monitoring and assessment programs, developments in simulation modeling, annual lengthening of critical resource and environmental data sets, and findings from field and laboratory experimental studies.

Some pessimists feel that we will never have adequate data to distinguish clearly the quantitative effects of pollution on fish stocks; I think the accumulation of analyses and insights relevant to the problem forecasts a brighter future than that.

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