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Assessing the Effects of Pollutant Exposure on
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Narragansett Bay Estuary Program

Current Report

The Narragansett Bay Project

A FRAMEWORK FOR ASSESSING THE EFFECTS OF POLLUTANT EXPOSURE ON ESTUARINE POPULATIONS

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FOREWORD

The United States Congress created the National Estuary Program in 1984, citing its concern for the "health and ecological integrity" of the nation's estuaries and estuarine resources. Narragansett Bay was selected for inclusion in the National Estuary Program in 1984 and designated an "estuary of national significance" in 1988. The Narragansett Bay Project (NBP) was established in 1985. Under the joint sponsorship of the U.S. Environmental Protection Agency and the Rhode Island Department of Environmental Management, the NBP's mandate is to direct a five-year program of research and planning focussed on managing Narragansett Bay and its resources for future generations. The NBP will develop a comprehensive management plan by December, 1990, which will recommend actions to improve and protect the Bay and its natural resources.

The NBP has established the following seven priority issues for Narragansett Bay:

- * management of fisheries
- * nutrients and potential for eutrophication
- * impacts of toxic contaminants
- * health and abundance of living resources
- * health risk to consumers of contaminated seafood
- * land-based impacts on water quality
- * recreational uses

The NBP is taking an ecosystem approach to address these problems and has funded research that will help to improve our understanding of various aspects of these priority problems. The Project is also working to expand and coordinate existing programs among state agencies, governmental institutions, and academic researchers in order to apply research findings to the practical needs of managing the Bay and improving the environmental quality of its watershed.

This report represents the technical results of an investigation performed for the Narragansett Bay Project. The information in this document has been funded wholly or in part by the United States Environmental Protection Agency under an interagency agreement with the National Oceanic and Atmospheric Administration (#DW13931613-02). The results and conclusions contained herein are those of the author(s), and as they do not necessarily represent the views or recommendations of the NBP, no official endorsement should be inferred. Final recommendations for management actions will be based upon the results of this and other investigations.

EXECUTIVE SUMMARY

A logical framework for analyses quantifying the effects of anthropogenic stress on exploited populations was developed by the Northeast Fisheries Center. The framework was developed with a two-fold objective. The primary objective was as a guide for the EPA Estuarine Programs' Living Marine Resources Working Groups in research planning, recognizing that several important baseline studies had to be accomplished before a thorough analysis of the variations in population levels for major species in the estuaries could be conducted. The second was to demonstrate how data obtained through these projects could be use in analyzing the effects of contaminant exposure on populations through the use of modelling techniques.

Two approaches that may be utilized to quantify the effects of pollution on estuarine species are described. The first is a simple life history model framed in terms of elementary demographic parameters - birth rates (egg production), survival rates at each stage, and growth rates. The implications of varying levels of anthropogenic sources of mortality on population biomass and potential yield are considered. The second approach differs fundamentally from the first in that a sequence of catch (or population) estimates is related to measures of environmental stress using time series analysis to empirically identify relationships between contaminant levels or other anthropogenic factors and changes in yield or abundance. These two types of approaches are used as examples to illustrate the function of models in research planning.

Sensitivity analysis of the life history model indicated that modest increases in pollutant-related mortalities during the early stages (egg and larval) may result in dramatic declines in population size and yield while the juvenile and adult stages were more resilient to pollutant related effects.

These results indicate that continued support of scientific research examining the effects of contaminant exposure on life history stages of estuarine dependent species is of critical importance to the understanding of the overall population level effects of pollution. Dosage-response experiments in particular provide some of the most direct input into models such as those described in this report. The development of long time series of both catch statistics and abundance estimates in association with pollutant loadings is of importance in the development and use of time series models. Support of projects collecting and analyzing these data are vital.

INTRODUCTION

The overall scientific objectives of the EPA funded Estuarine Programs are to identify major problems threatening the 'health' of the nations estuaries with respect to both recreational and commercial interests; and to conduct research to define and analyze those problems with the purpose of relating the environmental quality of water and sediments in the estuaries to the status of their living marine resources. General management objectives of the projects are to develop recommendations for improved management of the estuaries and their resources based on the results of the scientific research; to define regulatory structures to implement the recommendations; and to generally inform and involve the public in the formulation and implementation of the management plans. These overall objectives are carried out through the several committees and working groups in each of the estuaries.

An important component of the scientific objectives of the Estuarine Programs is the status of the living marine resources of the estuaries and their reaction to contaminant exposure on a population level. The living marine resources working groups' responsibilities include the determination of 1) the status of the resources, 2) whether or not the condition of these resources is declining or improving, and 3) whether or not contaminants (either in the form of toxicants, hypoxia, etc) are impairing the 'health' of the resources.

A logical framework for an analysis quantifying the effects of anthropogenic stress on exploited populations was developed by the Northeast Fisheries Center. The framework was developed with a two-fold objective. The primary objective was as a guide for the EPA Estuarine Programs' Living Marine Resources Working Groups in research planning, recognizing that several

important baseline studies had to be accomplished before a thorough analysis of the variations in population levels for major species in the estuaries could be conducted. The second was to demonstrate how data obtained through these projects could be use in analyzing the effects of contaminant exposure on populations through the use of modelling techniques. The use of models to examine the population level effects of pollutant exposure is currently limited since much of the information required to parameterize some models is currently not available, however meaningful insights may be obtained through the use of generalized data obtained through the literature and from projects currently funded or planned by the Estuarine Programs.

In this report, we describe two types of models that demonstrate approaches to quantifying the effects of pollution on estuarine species with particular reference to winter flounder. In the first, a simple life history model is described. The model is framed in terms of elementary demographic parameters - birth rates (egg production), survival rates at each stage, and growth rates. The implications of varying levels of anthropogenic sources of mortality on population biomass and potential yield are considered. The second approach differs fundamentally from the first in that a sequence of catch (or population) estimates is related to measures of environmental stress using time series analysis to empirically identify relationships between contaminant levels or other anthropogenic factors and changes in yield or abundance. We therefore attempt to deduce the dynamics of the system by its output (yield or abundance). For an overview of statistical issues involved in the detection of impacts of this type see Stewart-Oaten et al. (1986), Rose et al. (1986), and Summers and Rose (1987).

To determine the effects of contaminant exposure and fishing on biomass and

yields of estuarine fish stocks, it is necessary to understand the interaction between the natural dynamics of populations and pollution levels. The fishable biomass of a population can be characterized as a balance between gains realized through growth and recruitment, and losses from several types of 'competing' mortalities (Figure 1). These mortalities include natural mortality (M), from predation or disease, and anthropogenic mortality, from fishing (F), pollution, and habitat alteration/loss. A goal of stock assessment research is to partition and determine the levels of each of these mortalities and examine the effects of each on population levels, anticipating that different mortalities affect distinct life history stages of the individuals in a population. For instance, predation mortality may primarily affect early life history stages (eggs, larvae and juveniles) because potential predators of smaller individuals are more numerous (e.g. Sissenwine 1984, Bailey 1984, Rothschild 1986). In contrast, fishing mortality is concentrated on adults, (and in some cases, juveniles) of most exploited species. Pollutant-related stresses may affect population levels by: 1) reducing fecundity or the viability of ova, and 2) decreasing the survival of larvae, post-larvae, juveniles, and adults. Secondary effects may include increased vulnerability to disease and predation and reduction in growth rates.

LIFE HISTORY MODEL

A conceptual diagram of a life history model incorporating the effects of pollutant exposure and fishing effort on population biomass and potential yield is given in Figure 2. In this model, a basic four stage life history including egg, larval, juvenile, and adult components is presented.

Transition between the stages is accomplished through growth except when the life cycle is completed; in this case, the transition is made between adult spawning biomass and egg production. Losses at each stage are due to natural causes such as predation, disease, starvation, adverse environmental conditions etc., pollution and/or habitat related causes, and fishing mortality (for recruited size classes). Note that for simplicity, only adults are subject to exploitation in this example. Pollution-related factors may also affect growth rates and there may be synergistic effects between natural and pollution-related sources of mortality. For example, larvae and juveniles in polluted environments may have lower condition factors and be more vulnerable to predation or disease. Clearly, natural or pollution-related factors which increase mortality rates at any point in the life cycle reduce the stock of fish available for harvest. Reduced growth rates may have indirect effects such as increasing the time spent in vulnerable life history stages (e.g. egg and larval stages), thereby increasing overall mortality rates. In addition, since maturity and fecundity are often size dependent, reduced growth rates may result in a reduction in reproductive output.

In the model, we assume that density dependent effects are important only during the larval phase. This assumption is based on generalized information for flatfish species indicating that spatial limitations on the nursery grounds may be an important density-regulating mechanism (see Beverton 1984). Density dependence may also be important at other life history stages, however, the basic model structure may be modified to account for additional density-dependent effects. A brief description of each model component, written in PC-based FORTRAN77, is given below.

EGG PRODUCTION Production of viable eggs by a population is a function of

the number of females surviving to reproductive size, fecundity, and fertilization and hatching success. A sub-model for this component would be:

$$N_0 = \{[G H E] N\} \{ \exp(-M_0 - M_0^{\wedge}) t_0 \}$$

where N_0 is the number of viable eggs which survive to the end of the egg stage (or alternatively, the initial number of larvae), G is percent fertilization success, H is percent hatchability, E is the mean fecundity, N is the number of females surviving to maturity, M_0 is the instantaneous mortality rate during the egg stage due to natural causes, M_0^{\wedge} is the instantaneous rate of egg mortality attributable to pollution, and t_0 is the duration of the egg stage; (all parameters are evaluated at a specified pollutant load). For information on the effects of chronic exposure to toxicants on fecundity and hatchability, see Logan (1986) and Hughes et al. (1986).

Extensions of this stage may include partitioning the number of adults into smaller size classes since fecundity (and possibly other parameters) are size dependent. Total egg production would then be obtained by summing over individual size classes.

LARVAE The basic model describing survivorship during the larval stage assuming a density-dependent effect during this phase is:

$$N_1 = 1/\{[M/(M_1 + M_1^{\wedge})][(\exp(M_1 + M_1^{\wedge}) t_1) - 1] + (1/N_0)[\exp(M_1 + M_1^{\wedge}) t_1]\}$$

here N_1 is the number surviving to the end of the larval phase (or the initial number of juveniles), N_0 is the number of viable, fertilized eggs surviving to

the end of the egg stage (the output from the egg production component), M_1 is the instantaneous rate of natural mortality (predation, starvation, etc.), M_1^{\wedge} is the instantaneous rate of pollution/habitat related mortality, M is the density-dependent mortality rate, and t_1 is the duration of the larval stage. This model describes an asymptotic relationship, appropriate if competition among members of a cohort for space or other critical resources are important. This function is a modification of the Beverton-Holt stock recruitment model with an explicit term for pollution/habitat stress.

JUVENILES The basic structure of the sub-model for survival during the juvenile phase is:

$$N_2 = N_1[\exp(-M_2 - M_2^{\wedge}) t_2]$$

where M_2 and M_2^{\wedge} are instantaneous rates of natural and pollution-related mortality during the juvenile phase and t_2 is the duration of the juvenile phase.

ADULTS The sub-model for survival during the adult stage is:

$$N_3 = N_2[\exp(-M_3 - M_3^{\wedge} - F) t_3]$$

where M_3 and M_3^{\wedge} are instantaneous rates of natural and pollution-related mortality for adults, F is the instantaneous rate of fishing mortality, and t_3 is the duration of this stage. Chronic exposure to toxics may also result in increased incidence of neoplasia and disease (Murchelano et al. 1986, Sass 1986) however, clear links between disease incidence and mortality rates have not been determined. Increased mortality in this stage, whether by fishing or caused by pollutant exposure will have a direct effect on the levels of egg

production and that losses at this stage represent biomass that cannot be harvested.

Because pollution and fishing mortality are measured in the same units in the model, we can describe pollution-related mortality in fishing mortality equivalents. The expected yield in weight is:

$$Y = [F/(M3 + M3^{\wedge} + F)]\{[1 - \exp(-M3 - M3^{\wedge} - F) t3]\} N2 w$$

where w is the mean weight of adults and all other terms are defined as before. The tradeoffs in yield as pollution related mortality and fishing mortality are illustrated in Figure 3. The figure depicts contours of equal yield as F and $M3^{\wedge}$ vary for a given initial input of $N2$ individuals. In this example, yield is highest at low levels of F and at the lowest levels of pollution-related mortality. As $M3^{\wedge}$ increases however, the 'competition' between fishing and pollution-related mortalities becomes more evident. In this case, the best strategy is to exert high levels of fishing mortality before the fish are lost due to pollution-related mortality (assuming of course that the fish are edible). Note also that sublethal effects such as reduction in growth rates can have an effect on yield. It is clear from the above yield equation that any reduction in biological parameters (mean weight-at-age, for example) will result in reduced overall yield.

Projected changes in population size based on this type of model may be expressed in a number of different ways, however, a graphical representation of the model provides an easily interpreted overview (see Figure 4). Each life history stage occupies one quadrant of the figure. Relationships between each of the stages, except that between the larval and juvenile stages, are

assumed to be linear (i.e. density-independent). We assume density dependence during the larval stage as described earlier. The thin line in each quadrant represents the inter-stage relationships for a population inhabiting a relatively non-polluted environment. The projected change in population size can be traced by simply following the sequence of dashed lines in this arbitrary example. Note that after one generation, the population has increased in size from the starting spawning stock size. After one additional generation, however, the population size is approaching an equilibrium level. The asymptotic relationship during the larval phase (quadrant 3) is stabilizing.

If the effect of pollution-related stress occurring at each of the life history stages is now considered, the position of each of the lines shifts downward (represented by the bold lines in the figure), reflecting reduced survival levels in each stage (or reduction in the number of viable eggs in the first quadrant). In this example, the cumulative effects of pollution-related stress are so severe that the population cannot sustain itself. Note that the population trajectory does not stabilize as before but, rather, spirals downward (traced by dotted lines in the figure). Clearly, pollution-related effects may not be this dramatic; significant impacts may not occur at each life history stage. Of course, the magnitude of the effect at any stage will be a function of the pollutant load. The objective of the analysis is to quantify the inter-relationships among life history stages and to estimate the magnitude of the parameters.

SENSITIVITY ANALYSIS

To examine the sensitivity of each of the stages to the simulated effects

of pollutant exposure, each of the pollutant-related mortality parameters were varied while holding all other levels of mortality constant during repeated runs of the model. Initial input parameters to the model were obtained primarily from the literature, and where possible, specific to winter flounder from Southern New England waters and estuaries. Simulations were performed using levels of pollutant-related mortalities equivalent to the removal of 20, 40, 60, and 80% of the individuals surviving the effects of natural mortality in each stage and applying these mortalities continually over 10 generations. Additional simulations were also performed varying the levels of percent successful hatchability and fertilizability with levels ranging between 20 and 100% of the base levels (arbitrarily set at 90 and 80% successful fertilization and hatchability, respectfully). Determination of base values for each of the parameters are described below.

Fecundity Estimates of mean fecundity were calculated utilizing results of studies conducted in Rhode Island waters (Saila 1962) and in Cape Cod Bay (Topp 1968) for individuals averaging about 300-400 mm (approximately age 3). Saila (1962) estimated that winter flounder from Rhode Island waters with an average length of 334 mm produced about 610,000 eggs, while Topp (1968) estimated that fish of about 300-400 mm produced approximately 435,000 eggs. For the purposes of this analysis, a rounded average of 500,000 eggs was used.

Mortality Mortality estimates used in the base runs were obtained from summaries given in Klein-MacPhee (1978) and Percy (1962). For the purposes of these sensitivity analyses, natural mortalities during each of the stages were:

	Mortality Rate (%)
Egg Stage	99.0
Larval Stage	98.7
Juvenile Stage	95.3
Adult Stage	25.6

Fishing mortality was held constant at approximately 18% ($F=0.2$) and was applied during the adult stage only.

Stage Durations The length of time that individuals spent in each of the stages was standardized to the adult stage. The duration of this stage was set equal to the amount of time the initial cohort (200 individuals; sex ratio of 1:1) would have survived given an estimate of natural mortality (M_3) of .30 and subjected to a fishing mortality (F) of .20 (approximate levels obtained from Klein-MacPhee (1978)). The calculated estimate of this stage was approximately 8 years (2920 days). The duration of each of the remaining stages was then scaled to this standard. Estimates of the actual times spent in each stage, obtained from Pearcy (1962) and standardized durations were:

	Months	Days	Scaled Duration
Egg Stage	0 - 0.7	21	.007
Larval Stage	0.7 - 2.4	51	.017
Juvenile Stage	2.4 - 22.4	621	.213
Adults	22.4 ->	2920	1.000

Mean Weight The mean weight of an average adult, used to calculate yield was estimated to be 0.6 kg for the entire duration of the adult stage.

RESULTS

The results of the sensitivity analysis of the simple life history model are shown in Figures 5-6. The figures illustrate the effect of varying each

of the parameters on the total adult population (in numbers) and yield (kg) over 10 generations. In each of the figures, the solid black lines represent the base run. In this simulation, the population increased steadily to an equilibrium level approximately 2.5 times larger than the initial population size and remained at that level. Yield levels in the base run also increased steadily for 2 generations then remained at a constant level of about 37 kg per generation. This simulation corresponds to the thin lines in Figure 4.

Modest increases in pollutant-related mortalities during the early stages (egg and larval) resulted in rather dramatic declines in both population size and yield over the period simulated, while the juvenile and adult stages were more resilient to pollutant related effects in the simulations conducted here (corresponding to the thick lines in Figure 4).

While the effects of contaminant exposure may not have been simulated precisely in the examples, the results indicate that pollution-related mortalities induced on the early, vulnerable life history stages of a population may have rather profound effects on future stock sizes and yields if not controlled.

TIME SERIES MODELS

Given a time series of abundance such as that available for Narragansett Bay from the University of Rhode Island, or catch estimates and measures or indices of pollution/habitat degradation, empirical models to predict the impacts of these factors on yield or abundance may be developed. In this section we briefly describe an approach based on time series analysis methods which directly account for common statistical problems in data sets of this type - autocorrelation and non-stationarity. A full discussion of these

methods is beyond the scope of this report and the reader is referred to Box and Jenkins (1976). Examples of the use of another type of model utilizing categorical regression models in this context are provided by Summers et al. (1985), Rose et al. (1986) and Summers and Rose (1987).

The strategy of model building advocated by Box and Jenkins (1976) is an iterative process of tentative model identification, estimation, and checking of the adequacy of the fitted model. A sequence of models is proposed based on certain diagnostic characteristics, parameters of the model are estimated and the residuals of the model are checked for independence. If the model is not adequate (i.e. if there is still autocorrelation in the residuals), an alternative model is identified and checked for adequacy. The process is continued until a suitable model is found. Nonstationary series (i.e. series which exhibit trends or changes in level) are typically handled by

'differencing' the series (forming a new series $X = Y_t - Y_{t-b}$; where b is the period of differencing). Typically, taking first differences ($Y_t - Y_{t-1}$) is sufficient to make the series stationary. Non-stationarity is a significant problem in many of the time series of relevance here. Other methods of trend removal are possible but these are generally less desirable than the approach outlined above (see Box and Jenkins 1976).

Two general classes of time series models may be useful for impact assessment depending on the nature of the available information: intervention models and transfer function models. A brief description of each is provided below.

Intervention models These models are used to describe the effect of one or more discrete events on an output variable (in this case, yield or abundance). For example, suppose a series of catch estimates over a period of years is

available and during the course of this time series, discharge of a specific pollutant was initiated. Suppose further that this discharge had an immediate effect on yield and/or abundance. The form of the intervention model for this case is:

$$Y_t = w_0 I_t + N_t$$

where w_0 is the intervention term (the magnitude of the pollution effect), I_t is the intervention variable (0 before the start of pollution and 1 thereafter) and N_t is a 'noise' component which may itself be modeled as an autoregressive-integrated-moving-average (ARIMA) process (Box and Jenkins 1976). Note that the intervention variable (I_t) essentially serves as an on-off switch. The 'noise' or error term (N_t) accounts for other factors which are affecting the output variable. The model for this term may be quite complex and include expressions for the past history of the catch series (i.e. autoregressive terms) and/or expressions for the effects of past 'shocks' or perturbations (the moving-average component). If the pollution effect is not immediate, but instead has a gradual impact, the form of the intervention model is:

$$Y_t = [w_0/(1 - dB)]I_t + N_t$$

where the term d is restricted to the interval +1 to -1; this coefficient represents the graduated effect of the intervention. As d approaches either +1 or -1, the effect becomes more gradual. At intermediate values (around 0) the effect is more abrupt. Here, B is the so-called backward shift operator.

It is recognized that it may be difficult or impossible to identify exact starting points for specific pollution effects. In other instances, there may

be an intermittent effect or episodes of relatively brief duration (e.g. an anoxic event); problems of this sort can be modelled using intervention analysis.

Transfer Function Models If there exists a time series of measures of pollution or habitat degradation as well as catch or abundance estimates, a transfer function model relating the series may be developed. The general form of the transfer function is:

$$Y_t - d_1Y_{t-1} - d_2Y_{t-2} \dots - d_mY_{t-m} = w_0X_{t-1-b} - w_1X_{t-2-b} \dots + w_nX_{t-n-b} + N_t$$

where Y_t is the catch or abundance series, X_t is a measure or index of pollution, d_i and w_i are model parameters, and N_t is an error term which may also be modelled as an ARIMA process. The transfer function model approach is useful if we are dealing with the outcome of a process in which both catch/abundance and pollution/habitat degradation has been changing over time. Possible sources of information on pollution/habitat degradation that could be used in development of transfer function models include:

- Trends in Human Population
- Agricultural Land Use
- Dredge Activity
- Dissolved Oxygen
- Municipal Sewage Discharge
- Power Plant Discharge
- Industrial Discharges

RECOMMENDATIONS FOR FURTHER RESEARCH

Continued support of scientific research examining the effects of contaminant exposure to the various life history stages of estuarine dependent species is of critical importance to the understanding of the overall

population level effects of pollution. Dosage-response experiments in particular provide some of the most direct input into models such as described in this report. The development and/or preservation of long time series of both catch statistics and abundance estimates in association with those pollutant loadings determined by the Estuarine Program's to be of significance is important in the development and use of time series models. Support of projects collecting and analyzing these data are vital.

Life History Models The simple life history model described may be expanded in a number of ways. The model may be partitioned into sub-stages, or alternatively, an age-structured rather than a stage-structured model may be specified. In the stage-structured model, growth rates are important only in terms of defining the time spent in each stage. In an age-structured model, we would shift the emphasis to size-at-age and specify size-dependent effects and parameters such as density-dependence in other stages of a species life history. This technique has been successfully applied by Deriso (1980) and Shepherd (1982) to offshore species, however not specifically to pollutant mortalities.

The pollution-related mortality terms may be linked to dosage-response experiments to allow evaluation of the model as a function of a range of pollutant stress levels; i.e. the functional relationship between dosage and mortality levels may be incorporated directly in the model.

The spatial distribution of both the organisms and contaminant levels will, in general, be heterogeneous. Accordingly, as information accrues, it will be desirable to incorporate spatial dynamics into the model, similar to the broadscale approach utilized by Spaulding et al. (1983). This will also provide a linkage with models of physical transport processes and gradients in

pollutant loads. Sedentary species, of course, are most easily linked with the information on spatial distribution of pollutants. Mobile species may be subjected to differing pollutants or pollutant levels at one or more points in the life cycle (or even during a seasonal cycle) and it will be necessary to understand the distribution patterns for each life history stage to evaluate the effects of differing pollutant levels in time and space.

Time Series Analysis Several potential difficulties in the development of intervention or transfer function models must be identified. First, the models require variables which are measured at equally spaced points in time; further, there must be no missing observations. Missing data may, in particular, be problematical in development of time series models. Objective methods of filling missing observations, however, are available (see Summers et al. 1985). Secondly, relatively long time series are generally required to fit multivariate time series models (greater than 30 observations); series of this length may be difficult to obtain for some variables (e.g. population abundance or relative abundance) but not others (e.g. catch). Finally, the development of transfer function models is an extremely involved process for more than one or two input variables and complex multivariate models will be difficult or impossible to develop. Note however, that there is likely to be a close relationship between many potential input variables and that use of only one or two of these will be necessary to develop useful models.

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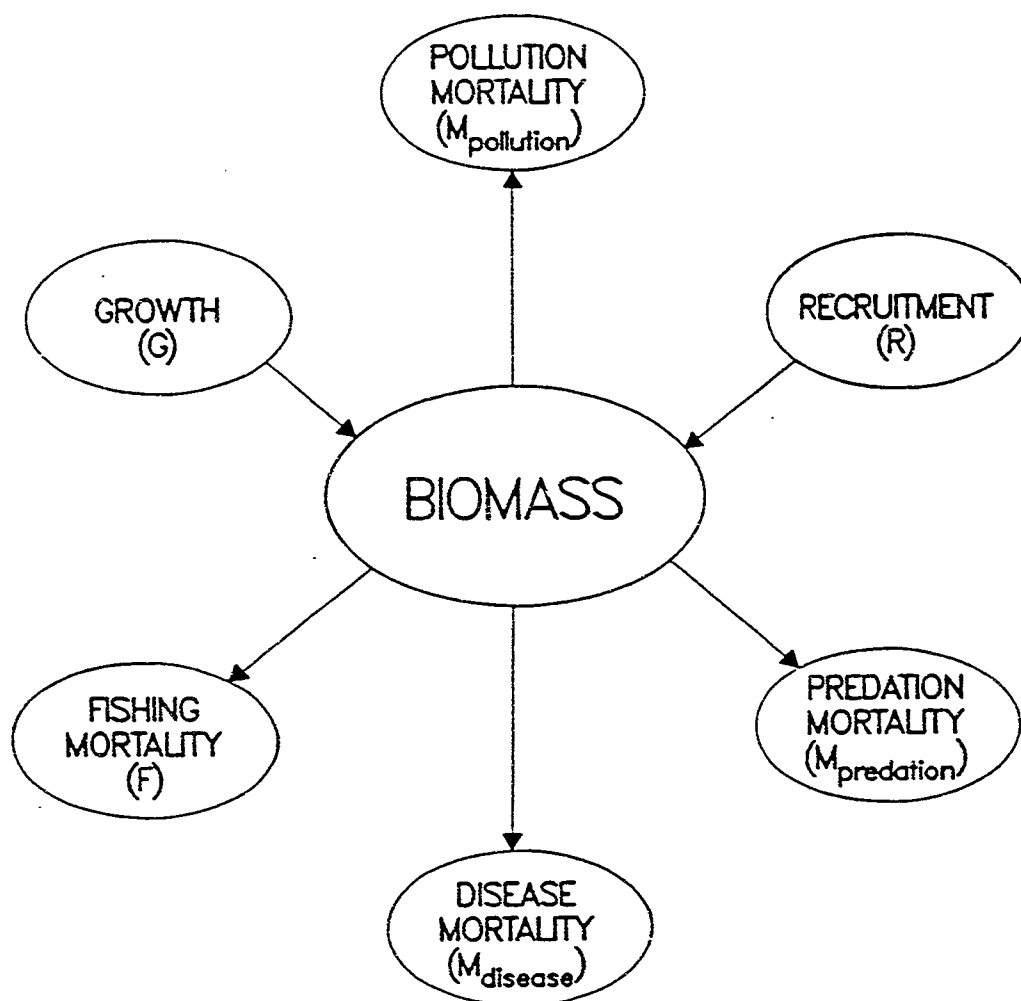
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$$B_{t+1} = B_t + G + R - F - M_{\text{predation}} - M_{\text{disease}} - M_{\text{pollution}}$$

Figure 1. Generalized relationship between forces affecting the biomass of an exploited population.

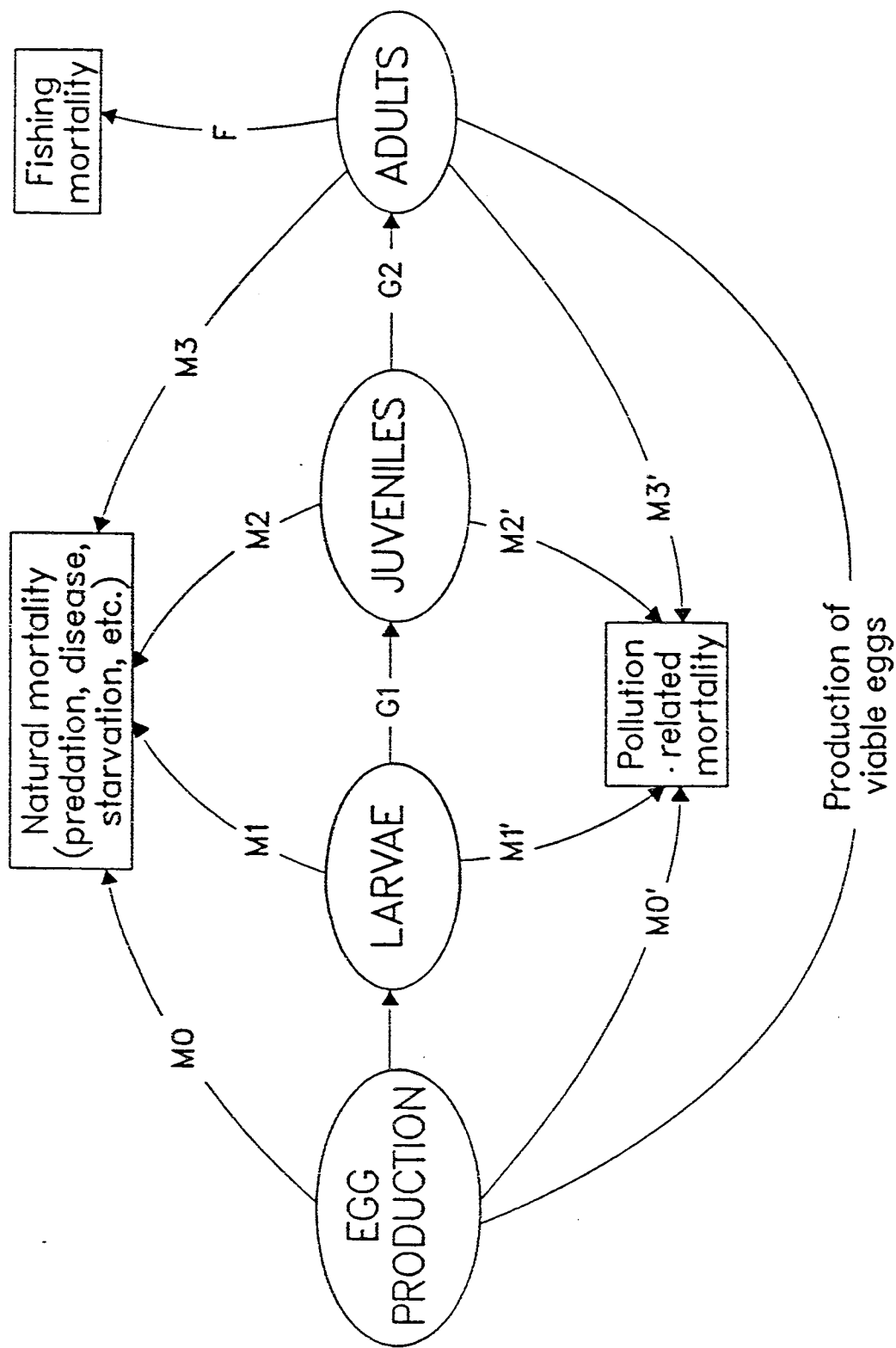


Figure 2. A simple four-stage life history model to assess the effects of natural and anthropogenic sources of mortality on an exploited population.

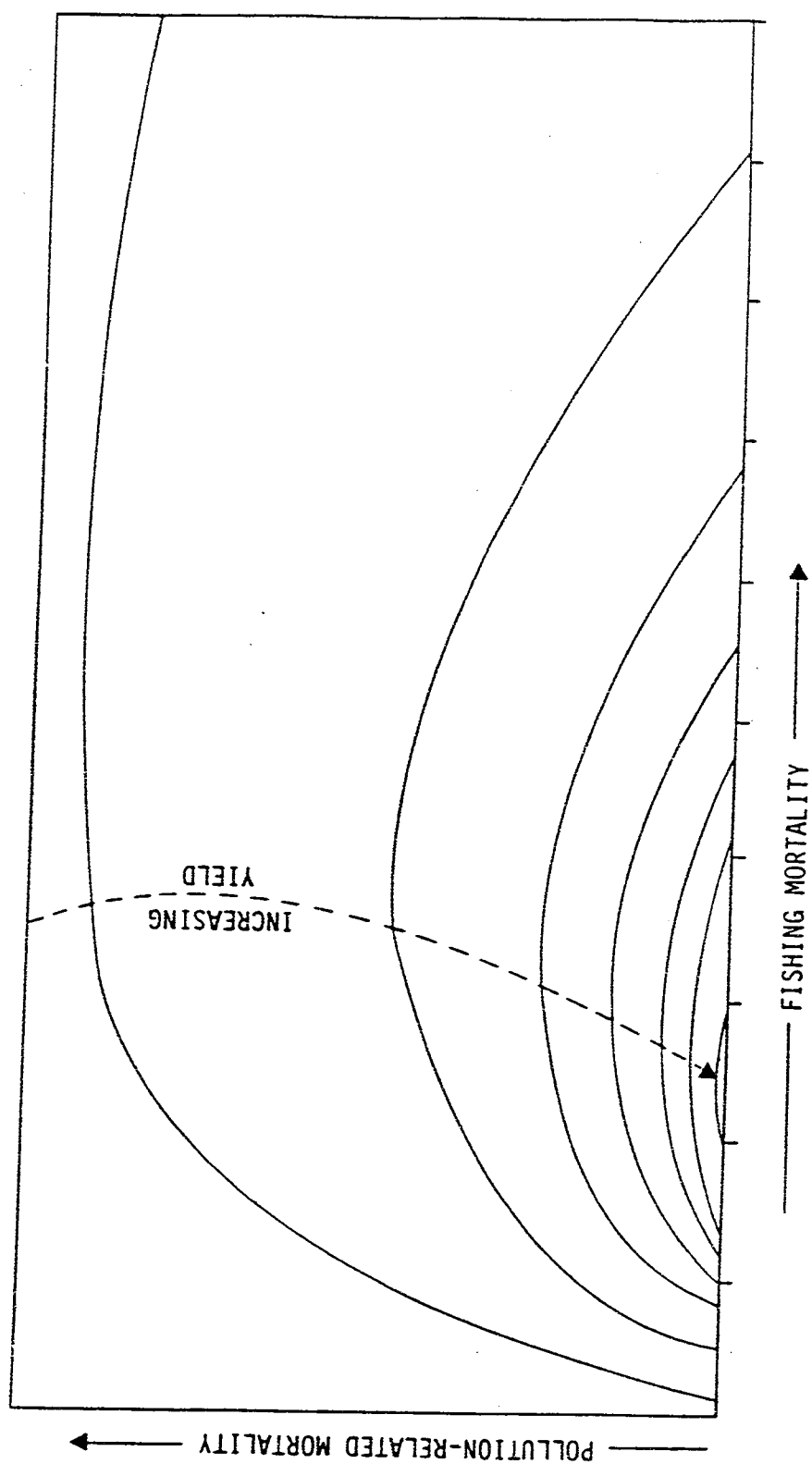


Figure 3. Contours of equal yield at varying levels of fishing and pollution-related mortality.

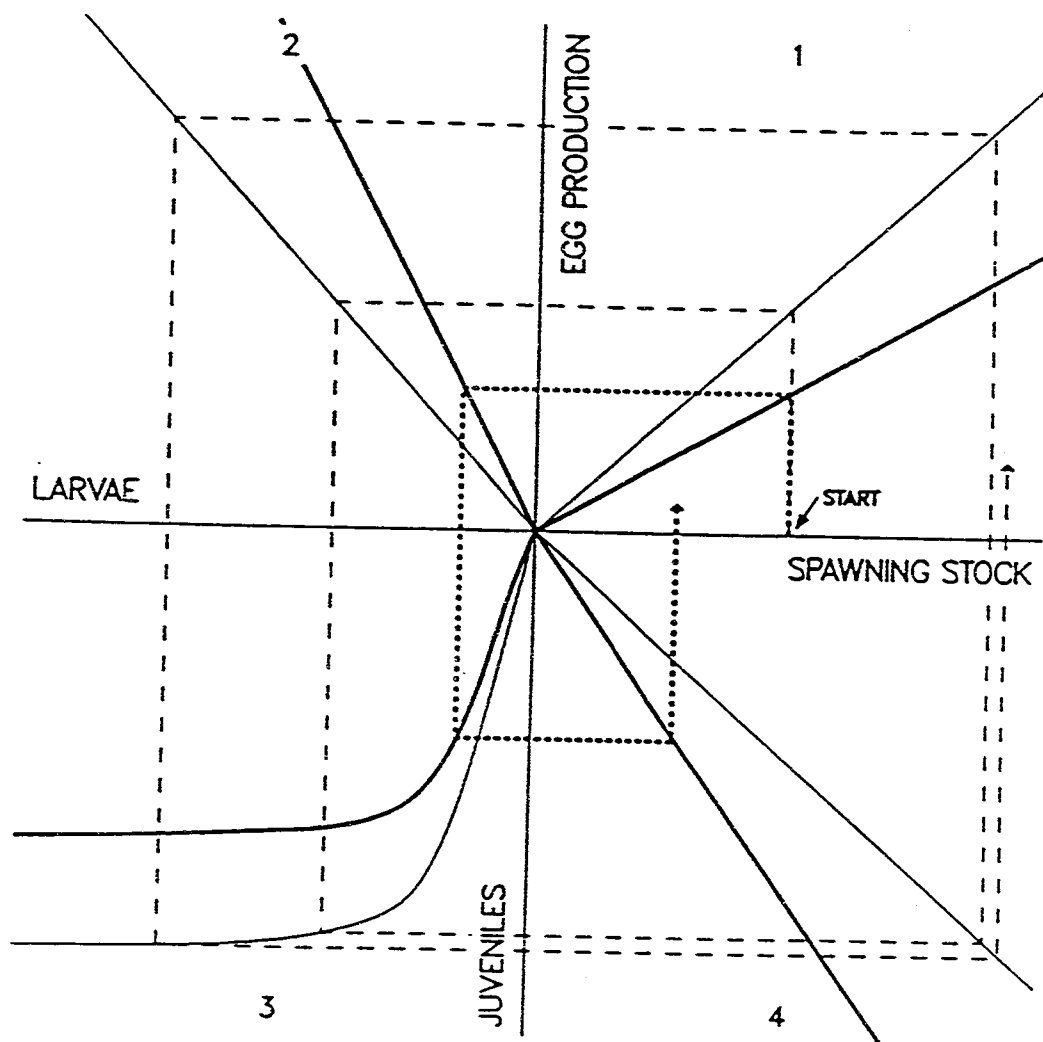


Figure 4. Generalized four-stage life history model indicating the effect of increased pollution-related mortality on population size. (Bold lines represent relationships between stages in a population exposed to pollutants, thin lines represent one in a non-polluted environment.)

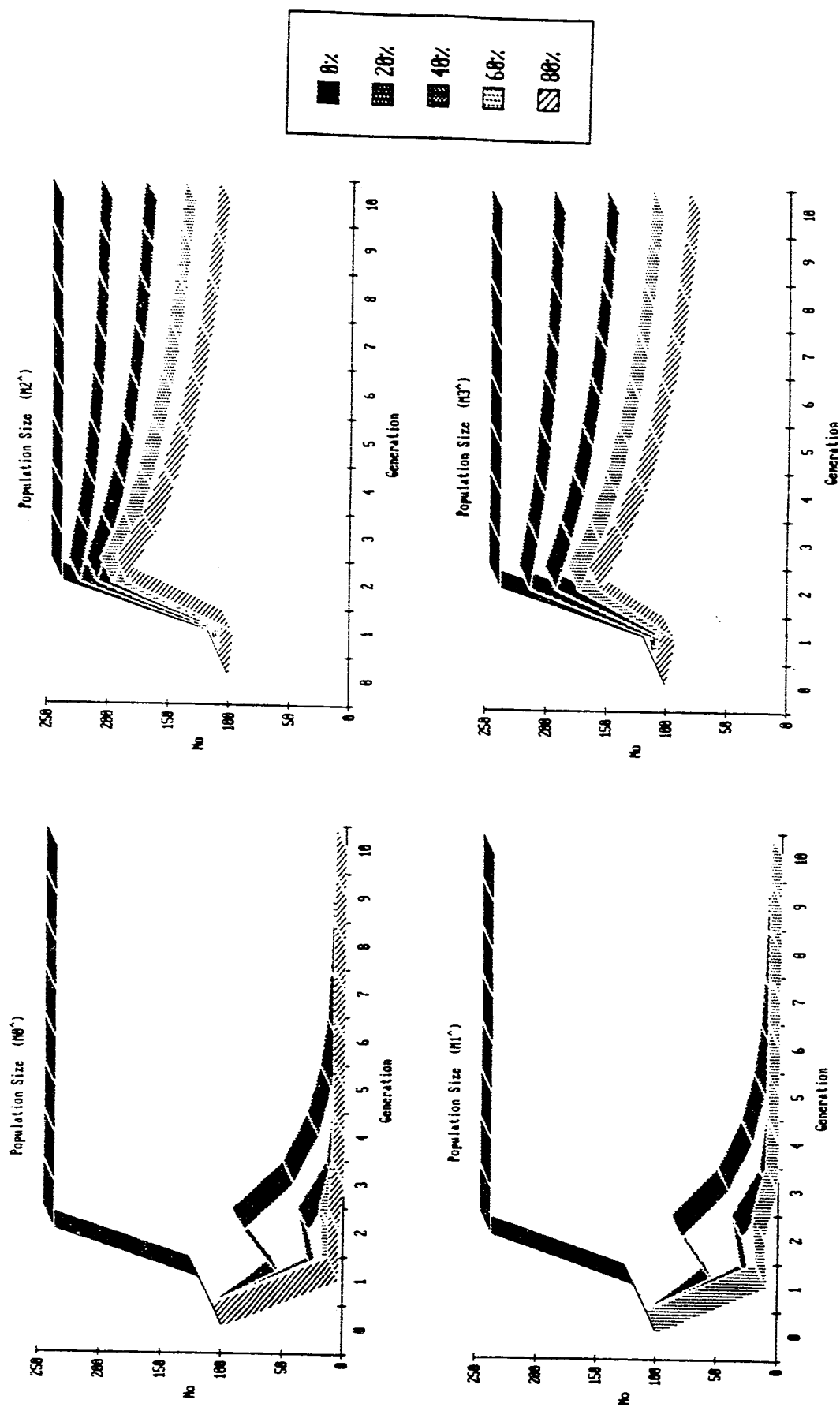


Figure 5. Results of sensitivity analyses indicating the effect of varying pollutant-related mortalities in the egg ($M0^{\wedge}$), larval ($M1^{\wedge}$), juvenile ($M2^{\wedge}$), and adult ($M3^{\wedge}$) stages on adult population size over 10 generations.

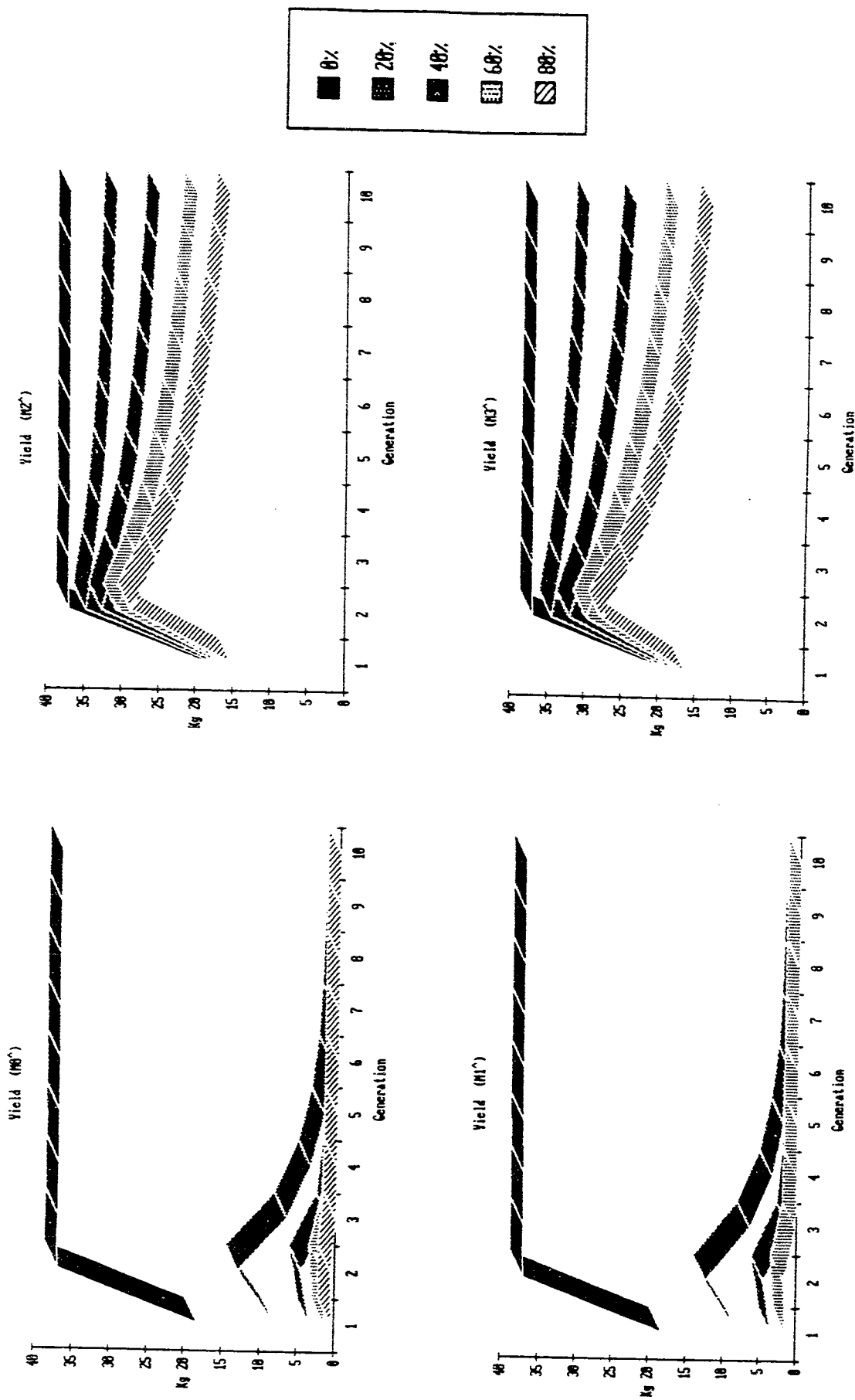


Figure 6. Results of sensitivity analyses indicating the effect of varying pollutant-related mortalities in the egg ($M0^{\wedge}$), larval ($M1^{\wedge}$), juvenile ($M2^{\wedge}$), and adult ($M3^{\wedge}$) stages on yield.