THEORETICAL WORK ON THE SIZE-SELECTIVE VULNERABILITY TO PREDATION DURING THE EARLY LIFE HISTORY STAGES OF FISHES

ANNE R. PARADIS

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THEORETICAL WORK ON THE SIZE-SELECTIVE VULNERABILITY TO PREDATION DURING THE EARLY LIFE HISTORY STAGES OF FISHES

by

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in partial fulfilment of the requirements for the degree of

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Abstract

Scientists believed that if we understood the factors governing the high mortality rates during the larval period of fishes, we could predict recruitment levels in coming years. This thesis concentrated in the investigation of size-selective predation mortality of fish larvae. First, I investigated the general patterns of size-selective predation from experimental studies and evaluated the empirical evidence support for the theoretical models. I found that fish larvae measuring 10% the size of predators were most susceptible to predation. This pattern was constant across a variety of experimental conditions and for four different types of predators.

Second, I investigated how encounter and susceptibility to predation generate size-selective mortality of fish larvae and how it was affected by the abundance and size of the predators. I found that encounter and susceptibility were counter-acting functions. The detection of size-selective removal of individuals and thus the balance between these two models was closely related to the cohort's overall mortality. I also found that the predator characteristics were important in determining the characteristics of survivors.

Third, I investigated how individual larval characteristics may influence their survival and how survivors differed under different selection pressures. I demonstrated that the effect of the larval characteristics in determining the number, length and growth

rate of survivors depended on the characteristics of the predator population. This implied that growing faster or being larger does not translate into a universal survival advantage.

Finally, I assessed how the predictions of the individual-based model compared with changes in the length frequency distributions observed in natural populations. I demonstrated that adding predation to the model make better predictions of the changes in the length frequency distribution observed in Conception Bay for some larval fish species. I also demonstrated that this result was highly sensitive to the accuracy of the estimates of growth rates.

This thesis is an important contribution to the theoretical framework of early life history of fishes. It posed serious questions about the effectiveness of current sampling protocols, the use of the statistical analytical tools, and most importantly the approach in the investigation of size-selective mortality of fish larvae.

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I dedicate this thesis to my parents for giving me strength, determination and confidence in myself.

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Part of this thesis has been published with the approval of my supervisory committee and the School of Graduate Studies. My supervisor, Dr. Pierre Pepin has initially designed and written the research proposal of Chapter II. The practical aspects of the research, data analysis and manuscript preparation were my responsabilities. As for the other chapters, I was the lead investigator at all stages of the work.

Chapter II: Paradis, A.R., Pepin, P., and Brown, J.A. 1996. Vulnerability of fish eggs and larvae to predation: review of the influence of the relative size of prey and predator. Can. J. Fish. Aquat. Sci. **53**: 1226-1235.

Chapter III: Paradis, A.R., Pépin, M., and Pepin, P. Disentangling the effects of size-dependent encounter and susceptibility to predation with an individual-based model for fish larvae. Can. J. Fish. Aquat. Sci. *In press*, May 1999.

Chapter IV: Paradis, A.R. and Pepin, P. Investigations of the effect of larval initial size and growth rate with an individual-based model of the size-selective vulnerability to predation of fish larvae. Fish. Bull. *Submitted*, May 1999.

Chapters V and VI: Paradis, A.R. and Pepin, P. Modeling field length distributions of fish larvae with an individual-based simulation model and with field estimates of predator population characteristics. Fish. Oceanogr. *Submitted*, June 1999.

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Chapter I: **General Introduction**

"Recruitment variability remains the single least understood problem in fishery science." Houde (1987).

More than ten years of research later, we are still struggling with this problem. Predicting recruitment levels from year to year has been the ultimate goal of most ichthyoplankton research. Mortality rates during the larval stages are very high and quickly decrease towards the end of the larval period and the start of the juvenile stage. Scientists believed that if we could understand the factors governing the high mortality rates during the larval period, we could predict recruitment levels in the coming years.

The major focus of research during the late 1980's was to determine which one of the causes of mortality were most important in controlling the number of surviving fish larvae. To date, the general consensus is that predation plays a greater role in affecting mortality during the early life stages than any other factor (Bailey and Houde 1989). It also appears that growth during the larval stage plays a critical role in recruitment variability as it affects the time spent during a period of high vulnerability to predation and allows increasing variability in individual larval fish characteristics (such as length, age, weight). More recently, scientists have realized that the high and variable mortality during the larval period prevents reliable and accurate predictions of

future recruitment levels. It may be that gross recruitment levels are set during the larval period but that the fine tuning occurs during the juvenile period.

Growth and mortality rates during the early life history of fishes are extremely high. Fishes may increase in weight 10^5 to 10^7 times from the fertilized egg to juvenile stage (Houde 1987); while numbers are reduced by four to seven orders of magnitude between hatching and recruitment (Cushing 1974). Factors which influence variability in growth rate as well as mortality will have tremendous effect in determining if an individual fish larva will survive to the juvenile stage. Whatever factors come into play in generating growth rate variability, the end result is the same - the generation of size variability among individuals of a cohort of fish larvae (DeAngelis and Huston 1987). This thesis will focus on asking: How does variability in size among individual larvae affect survival to the juvenile stage? The different causes of mortality may affect differently which larvae survive to the juvenile stage and as such it is important to identify the most likely source of mortality. Starvation and disease may affect the weakest individuals of the cohort. It may be that these weak individuals are also the smallest individuals and thus, the argument is that if they were not eaten by predators, they would have died of starvation or disease. Physical processes such as advection, diffusion and migration to unfavorable areas may not be influenced by individual characteristics. It may be a matter of luck as opposed to inherent characteristics that increases or decreases their survival probability. Mortality due to predation may be the

most intense selective pressure faced by fish larvae and has the potential to be greatly influenced by larval characteristics such as size and growth rate.

A prey-predator interaction (vulnerability to predation) is the combination of specific events occurring between two individuals: an encounter between prey and predator and the attack and capture of the prey by the encountered predator. Several factors may affect the different probabilities associated with these events: sizes of prey and predator, behavioral responses of individuals, morphological and ontogenic development of larvae (see Bailey and Houde 1989 or Fuiman and Margurran 1994 for extensive reviews). These factors are among the primary determinants of the selective pressure of predation on a cohort of fish larvae. This thesis will concentrate specifically on the influence of body sizes and growth rates of individual fish larvae, as well as sizes of predators, as factors influencing their vulnerability to predation.

Size-selective predation has long been recognized as a structuring effect in population dynamics as well as community composition. Brooks and Dodson (1965) have demonstrated a shift in prey size and composition given the presence or absence of a major predator in different lakes and proposed the size-efficiency hypothesis. Paine (1969, 1971) demonstrated that the removal of a specific predator may completely reverse the structure of the prey community by changing the abundance and dominance hierarchy of prey species as well as their size. He introduced the concept of keystone

predators (Paine 1971). Considering size-spectra theory that relates biomass to size, Peterson and Wroblewski (1984) predicted a general decline of mortality as a function of size of marine fishes. Since then, predation has been recognized as an important selective pressure. The most important agent influencing predation is the relative size of the prey organisms. Bailey and Houde (1989) in an extensive review of predation of marine fish larvae and the recruitment problem, have proposed specific general patterns of predation mortality as a function of both prey and predator size. They predicted that a general dome-shaped function of predation would be truncated given different functional types of predators. Bailey and Houde (1989) broke up these general patterns of predation into its component parts: encounter and susceptibility, based on concepts introduced by Zaret (1980) and Greene (1986). Zaret (1980) and Greene (1986) defined susceptibility to predation as the product of attack and capture while vulnerability to predation as the product of encounter, attack and capture.

Thesis layout

Chapter II: Empirical model of size-selective predation rates.

In this thesis, I used several tools in investigating size-selective predation mortality of individual fish larvae. Bailey and Houde (1989) have offered conceptual models of encounter rates, larval susceptibilities and vulnerabilities to different predator types. I evaluated the empirical support for these conceptual models. First, I

investigated the general patterns of size-selective predation mortality from published experimental studies. Given that studies investigating the components of predation experimentally are specific to the questions and hypotheses under study and despite variable laboratory conditions, were general patterns of size-selective predation mortality of fish larvae observed?

Chapter III: Disentangling encounter and susceptibility with an individual-based model

Laboratory experiments offer information and relationships governing the predation process. This basic information is the foundation of individual-based simulation models. The power of these tools for exploring consequences of individual interactions at the population level depends on the quality of the information which forms the basis for the model's formulation. Simulation and modelling analyses based on general patterns of encounter and susceptibility (combination of attack and capture) may reveal qualitative results with broad implications (Rice et al. 1997).

I used the general empirical relationships from Chapter II as the susceptibility component in an individual-based model to investigate how these relationships interacted with the mathematical encounter model proposed by Gerritsen and Strickler (1977). Specifically, I investigated how encounter rate and susceptibility to predation generated size-selective mortality of fish larvae. I also investigated how the size-selective mortality of fish larvae generated by these size-specific models was affected by the abundance of predators as well as size structure of the predator population.

Chapter IV: Effects of larval characteristics on size-selective predation with the individual-based model

In this chapter, I investigated how individual characteristics of fish larvae may influence their survival potential and how the characteristics of surviving fish larvae differed under different selection pressures identified in Chapter III. I also compared the results with other individual-based simulation models that used either the same encounter model (Cowan et al. 1996), the same growth model (Rice et al. 1993a), or a similar size-structured predator population (Rice et al. 1993b).

Chapter V: Confronting simulation results with field surveys

Finally, I assessed how the predictions of the individual-based simulation model compared with changes in the length frequency distributions observed in a natural population. Specifically, if information about the predator population dynamics, initial length frequency distributions as well as gross estimations of growth rates was used as the initial condition of the individual-based model, could I explain changes in the length frequency distributions of surviving fish larvae over time? Are the fish larvae surviving in the field the same individuals 'surviving' simulated predation?

Concluding remarks

I started this thesis with the objective to answer a specific question by using different tools and assessing how the answer to this question is affected by the choice of tools. I originally intended to investigate and answer the following question: Which larvae are better able to survive predation? Is it always the biggest larvae or is it that the size of the fish larvae has little effect on their survival and mostly that predation is affected by environmental conditions?

I have come to realize that the answers to these questions are of little importance when we start to investigate the power of our answer or, in other words, the power of our predictions. Are we actually capable of detecting size-selective removal of individuals from field samples? When one or two individuals survive out of a million, does it really matter how big they are and how fast they are growing? What is the actual difference in probability of survival of two individuals of different sizes? How is this difference affected by the precision of our measurement in size of larvae? Ultimately, is this difference in survival probability significant? May we expect to detect such difference from field sample using current statistical analytical tools and sampling procedures?

Chapter II

Vulnerability of fish eggs and larvae to predation: review of the influence of the relative size of prey and predator.

Abstract

I investigated the potential influence of relative body size of early life stages of fishes on their vulnerability to predation by crustaceans, ctenophores, medusae and fishes, and contrasted the patterns with predictions based on different conceptual models. I found that vulnerability of ichthyoplankton to predation by ctenophores and by predatory fishes were dome-shaped. Laboratory estimates of predation rates of these two predator types were negatively influenced by the volume of the container in which the experiments were conducted and by the duration of the experiment. Medusae and crustaceans showed decreasing predation rates with increasing relative size of fish larvae. Laboratory estimates of predation rates of medusae were influenced by container volume, temperature, and duration of the experiment whereas predation rates of crustaceans were influenced by container volume alone. Independent of predator type, the vulnerability of ichthyoplankton to predation was maximal when fish larvae were 10% of the length of the predator.

Introduction

Mortality of larval fish is believed to be mostly due to two overlapping causes: starvation and predation. Mortality owing to starvation is assumed to occur over a short period and at small sizes (Cushing 1974; Hunter 1981, 1984), whereas predation occurs throughout all stages; hence, predation may be a more important source of mortality.

Recent reviews have summarised the factors that influence the mortality of fish eggs and larvae (e.g., Houde 1989; Pepin 1991; Heath 1992; Fuiman and Margurran 1994; Leggett and DeBlois 1994). Although many factors influence predation rates, sizes of both predator and prey have been found to be important (Werner and Gilliam 1984; Kerfoot and Sih 1987; Bailey and Houde 1989; Pepin et al. 1992). Despite the recognition that body size is important, there have been few attempts to provide general empirical evidence of the overall pattern of size-selective vulnerability to predation. A commonly expressed view is that larger and older larvae are less vulnerable to predation, although this paradigm has been criticised (Litvak and Leggett 1992). This concept is based principally on experiments that focused on the probability of capture of larvae (Litvak and Leggett 1992), ignoring the encounter and attack probabilities that play major roles in the predation outcome (Fuiman and Margurran 1994). Bailey and Houde (1989) proposed four vulnerability curves for fish larvae to illustrate the theoretical effect of altering the prey:predator sizes ratio on the basis of Zaret's (1980) and Greene's (1986) characterisation of the search and feeding modes of different functional predator groups. The predictions from Bailey and Houde's (1989) conceptual model were that the

vulnerability of larval fish to all functional predator types follows a dome-shaped function, except for cruising invertebrates for which the relationship was hypothesised to show a continuously decreasing vulnerability with increasing relative size.

To explore generalities in size-selective patterns in vulnerability to predation and to contrast these with Bailey and Houde's (1989) conceptual models, I reviewed laboratory and enclosure studies of predation rates on fish eggs, larvae and juveniles. As with other such approaches (Miller et al. 1988; Houde 1989; Pepin 1991), my aim was to unify observations on common variables. In past reviews, the principal independent variable has been the size of one of the organisms (Pepin and Miller 1993). In the case of predation rates, the ratio of sizes may serve as a better descriptor (Bailey and Houde 1989, Pepin et al. 1992) than either prey or predator size alone. Therefore, I constructed the vulnerability curves in relation to prey:predator size ratios, thus permitting a direct comparison with Bailey and Houde's (1989) predictions. Despite the extent of information dealing with larvae-predator interactions, the concepts dealing with size-dependent processes have been used primarily as interpretative rather than applied tools. This may be due to differences in protocols of experiments that form the basis for the conclusions from previous reviews. To account for differences in experimental design, I investigated the effects of variables that have been shown to significantly effect predation rates, namely container volume (deLafontaine and Leggett 1987; Monteleone and Duguay 1988); duration of experiment (Westernhagen and Rosenthal 1976); and temperature (e.g. Houde 1989; Pepin 1991).

Methods

Data collection and standardisation

Data were compiled from a review of papers reporting results from laboratory and enclosure experiments using fish eggs, larvae, or juveniles as prey. Studies did not have to focus on size-selective relationships to be included in the analysis. Functional response and behavioural studies were also included if authors provided information on the size of their organisms, if standardised predation rates could be calculated and if the experimental protocol was clearly explained. Ideally, functional response and behavioural studies served to provide information on the variability of the selection patterns found in size-selective studies. Variables included in our analysis were predation rate (PR), length of prey (L_f), length of predator (L_p), duration of the experiment (D), temperature (T), and container volume (V).

Data were computed from tables or digitised from figures in articles if the original data were not provided by investigators. I grouped predators into four groups: crustaceans (crabs, shrimps, euphausiids, amphipods, and copepods), ctenophores, fishes, and medusae. I found it impossible to group predators in functional groupings (as in Bailey and Houde 1989) because some predators may exhibit different foraging tactics (e.g., *Alosa pseudoharengus*). Length measurements differed somewhat among predator groups. For crustaceans, length was the crabs' carapace width (van der Veer and Bergman 1987) or length (Ansell and Gibson 1993), shrimp and amphipod total length (van der Veer and Bergman 1987; Ansell and Gibson 1993; Westernhagen and Rosenthal 1976;

Westernhagen et al. 1979), and copepod prososome (Bailey and Yen 1983) or body length (Lillelund and Lasker 1971). The lengths of ctenophores and medusae were given as diameter of the organisms but excluded the expanse of the tentacles. Cowan et al. (1992) reported the size of their ctenophores and medusae as water displacement, i.e. volume of water displaced when the organism was added to a beaker. I transformed this measurement into a diameter by taking the cubic root of the volume. For predatory fishes, size was reported as either total or standard length. If lengths were not included in a study, I substituted the value reported in previous or later work by the same author(s) on the same species. If only a range of values was given, I used the midpoint between the minimum and maximum.

Standardised predation rates (PR) were calculated as:

$$PR = n_e \times n_i^{-1} \times m^{-1} \times D^{-1} \quad (2.1)$$

where n_e is the number of prey eaten, n_i is the initial number of prey offered, m is the number of predators per experimental trial, and D is the duration of the trial in hours.

Statistical analysis

Predation was related to size as follows:

$$\ln(PR) = a + b \times \ln(L_f/L_p) \quad (2.2)$$

where L_f is prey length (mm) and L_p is predator length (mm). The natural logarithm of predation rate ($\ln(PR)$) and size ratio ($\ln(L_f/L_p)$) approached normal distribution. The equation was then modified to include a second order term, $(\ln(L_f/L_p))^2$, if it was

significantly correlated ($\alpha = 0.05$) with the residuals of the basic model such that:

$$\ln(PR) = a + b \times \ln(L_f/L_p) + c \times (\ln(L_f/L_p))^2 \quad (2.3)$$

The second-order term was chosen because Bailey and Houde (1989) and Fuiman and Margurran (1994) suggested that the vulnerability of fish larvae to different predators is dome shaped. The methodological variables ($X = V$, T, or D) were included in order of importance if they were significantly correlated with residuals from equations 2.2 or 2.3 and if they had few missing observations such that:

$$\ln(PR) = a + b \cdot \ln(L_f/L_p) + c \cdot (\ln(L_f/L_p))^2 + d \cdot \ln(X) \quad (2.4)$$

To avoid problems due to a limited number of levels of observations (i.e. clusters of observations at two points resulting in an apparent linear trend), each residual relationship was plotted to assess the generality of the effect of that variable. A variable was included in equations 2.2 or 2.3 only if a continuous and uniform visual trend was apparent. Because I was dealing with multiple regressions, partial F-values were computed to assess if the amount of variation explained by individual variables of the final models was significant ($\alpha = 0.05$). Analyses of covariance and t-tests were performed to test if the model's parameters (intercept, first-order term and second-order term) computed for the different predator taxon were significantly different from each other. Parameters (a, b, c, and d) for any model are presented as the computed estimates with their standard error.

The graphical representations of the data were designed to highlight the size-selective predation model for each predator type. The first panel of each figure shows the standardised predation rates with the underlying effects of experimental conditions (i.e.,

the raw data). The second panel shows the corrected predation rates using the overall average value of each significant experimental variable.

Empirical reviews are limited by the diversity and the quality of data sets from which they are developed (Pepin and Miller 1993). Within a model, data come from a variety of sources that may differ extensively in methodological approaches. Therefore, several of the model's variables may be cross-correlated. To provide the reader with some insight on potential confounding factors, I have reported the significant cross-correlations between the size ratio and all methodological variables.

To determine if different prey life stages suffered different levels of predation, I performed an analysis of variance on the residuals among prey life stages for each predator type.

Results

The data set (Table 2.1) included 72.4% size-selection predation experiments, 44.8% functional response experiments and 13.8% behavioural experiments. Some articles (34.4%) were combinations of these types of studies.

Crustaceans

The model for crustaceans summarised nine studies and 411 observations (Table 2.1, Figure 2.1*a*). Predation rate was negatively related to the prey:predator size ratios (Figure 2.1*b*). Only container volume was negatively correlated with the residuals of the

model (τ= -0.13, P< 0.001, n= 411). The regression of the residuals against the container volume was weak but still significant ($F_{1, 409}$= 18.1, P< 0.001, r^2= 0.04). The final equation is:

$$\ln(PR)_{\text{crustaceans}} = -(5.2\pm0.1) - (0.97\pm0.09) \times \ln(L_f/L_p) - (0.42\pm0.07) \times \ln(V) \qquad (2.5)$$

with ($F_{2, 408}$= 60.3, P< 0.001, r^2= 0.23) (Figure 2.1*b*).

In the data set, prey:predator size ratios were negatively correlated with duration of the experiment (τ= -0.35, P< 0.001, n= 411) and container volume (τ= -0.45, P< 0.001, n= 411), but not with temperature (τ= -0.07, P= 0.06, n= 411). These cross-correlations indicate that relatively small larvae were used in longer experimental trials and bigger container volumes.

Ctenophores

The model summarised three studies and 169 observations with 95% of the data from the study by Monteleone and Duguay (1988) (Table 2.1, Figure 2.2*a*). Predation rates followed a dome-shaped function in relation to prey:predator size ratios (Figure 2.2*b*). The addition of the squared size ratio $(\ln(L_f/L_p))^2$ in the model significantly improved it from r^2= 0.18 to r^2= 0.37 ($F_{1,166}$= 50.1, P< 0.001; Sokal and Rohlf 1981, p. 635). The model's curvature was greatly influenced by the seven data points at the upper end of the range of size ratios but four of them were from Cowan and Houde's (1992) study and the remaining were from Monteleone and Duguay's (1988) study (Table 2.1, Figure 2.2*a*). Because similar observations arose from independent studies, I believe there

is no justification at this point to consider them as outliers. Container volume (τ= -0.46, P< 0.001, n= 169) and duration of the experiment (τ= -0.23, P< 0.001, n= 169) were strongly correlated with the model residuals ($F_{1, 167}$= 159.3, P< 0.001, r^2= 0.49 and $F_{1, 167}$= 18.8, P< 0.001, r^2= 0.10, respectively). The final equation is:

$$\ln(PR)_{\text{ctenophores}} = -(2.6\pm0.2) -(2.1\pm0.2)\times\ln(L_f/L_p) -(0.47\pm0.05)\times(\ln(L_f/L_p))^2 -(0.42\pm0.03)\times\ln(V) -(0.9\pm0.2)\times\ln(D) \quad (2.6)$$

with ($F_{4, 164}$= 134.5, P< 0.001, r^2= 0.77) (Figure 2.2*b*).

Monteleone and Duguay (1988) found that bigger containers yield higher predation rates when initial prey density was kept constant (i.e., positive impact of container size on predation rates). Combining data of different initial prey densities, I found a very strong negative impact of container volume on predation rates.

Fishes

The model for fishes summarised 12 studies with 301 observations (Table 2.1, Figure 2.3*a*). Predation rates followed a dome-shaped function in relation to the prey:predator size ratios (Figure 2.3*b*). Duration of the experiment (τ= -0.56, P< 0.001, n= 301) and container volume (τ= -0.18, P< 0.001, n= 301) were correlated with the residuals of the model ($F_{1, 299}$= 622.9, P< 0.001, r^2= 0.68 and $F_{1, 299}$= 8.4, P= 0.004, r^2= 0.03, respectively). The final relationship is:

$$\ln(PR)_{\text{fish}} = (2.4\pm0.4) -(1.9\pm0.3)\times\ln(L_f/L_p) -(1.08\pm0.04)\times\ln(D) -(0.43\pm0.05)\times(\ln(L_f/L_p))^2 -(0.13\pm0.03)\times\ln(V) \quad (2.7)$$

with $F_{4, 296}$= 409.8, P< 0.001, r^2= 0.85 (Figure 2.3*b*).

The second-order term $(\ln(L_f/L_p))^2$ was a significant parameter only after duration of the experiment was included in the model and it only slightly improved the model from r^2= 0.81 to r^2= 0.84 ($F_{2,297}$= 27.8, P< 0.001; Sokal and Rohlf 1981, p. 635).

The prey:predator size ratios were correlated with duration of the experiment, temperature and container volume (τ= 0.17, P< 0.001, n= 301; τ= -0.30, P< 0.001, n= 219; and τ= -0.28, P< 0.001, n= 301, respectively). These cross-correlations suggest that relatively small prey were used in shorter experimental trials, at higher temperatures and in bigger container volumes.

It seems that the effect of relative size might be obscured by other factors affecting the interaction between larval and predatory fishes. To further investigate the size-selective relationship, I plotted the best fit of the first- or second-order regression estimated for individual studies (Figure 2.3*c*). This allows viewing the individual size-selective patterns without the effect of different methodological approaches and cross-correlated variables. I identified maximum vulnerability to occur when fish larvae are approximately 10% of the length of the predatory fishes (range 3-18%). Below the 10% mark, all studies indicated increasing vulnerability to predation by fishes with increasing relative prey size and above this mark, six of eight studies indicated negative slopes (Figure 2.3*c*).

Medusae

The model for medusae summarised nine studies and 464 observations (Table 2.1, Figure 2.4*a*). Predation rates were negatively related to the prey:predator size ratios (Figure 2.4*b*). Container volume (τ= -0.42, P< 0.001, n= 464), temperature (τ= 0.24, P< 0.001, n= 340) and duration of the experiment (τ= -0.11, P= 0.004, n= 340) were strongly correlated with the residuals of the models ($F_{1, 462}$= 225.7, P< 0.001, r^2= 0.33; $F_{1, 338}$= 21.5, P< 0.001, r^2= 0.06; and $F_{1, 338}$= 28.6, P< 0.001, r^2= 0.08, respectively). The final equation is:

$$\ln(PR)_{medusae} = (1.3\pm1.1) - (1.0\pm0.1)\times\ln(L_f/L_p) - (0.37\pm0.02)\times\ln(V) - (1.5\pm0.4)\times\ln(T) - (1.03\pm0.07)\times\ln(D) \quad (2.8)$$

with $F_{4, 335}$= 347.4, P< 0.001, r^2= 0.81 (Figure 2.4*a*).

Even though the addition of temperature produced a loss of 123 data points because of a failure to report this information in four studies (Table 2.1), I chose to include it in the model because of the large significant impact of this variable on predation rates.

I confirmed the results of deLafontaine and Leggett (1987) that the highest predation rates occurred in the smallest containers. The prey:predator size ratios were positively correlated with temperature (τ= 0.67, P< 0.001, n= 340). This could explain the conflicting results between the positive residual correlation with temperature and the negative effect of temperature on the predation rates of medusae. The prey:predator size ratios were negatively correlated with the duration of the experiment (τ= -0.32, P< 0.001,

n= 464) and the container volume (τ= -0.31, P< 0.001, n= 464). These cross-correlations suggest that relatively small prey sizes were used consistently at lower temperatures, in longer predation trials, and in bigger containers.

Categories of predators

I found that vulnerability to predation by crustaceans and medusae decreases with increasing relative prey length to predator length (Figure 2.5). Vulnerability to predation by ctenophores and fishes followed dome-shaped functions, with peak predation rates at about 10% relative prey length to predator length (Figure 2.5).

The corrected vulnerability of crustaceans and medusae have similar slopes: -0.97±0.09 and -1.0±0.1, respectively, with t= 0.223, P> 0.05. The corrected vulnerability curves of ctenophores and fishes had similar slopes (-2.1±0.2 and -1.9±0.3, respectively (t= 0.55, P> 0.05)) and second-order terms (-0.47±0.05 and -0.43 ±0.05 (t= 0.566, P> 0.05)). This implies that the vulnerability curves of the same order are not significantly different from each other, but the different intercepts indicate that the predator groups do not show the same type of response to the methodological variables. This is demonstrated with the partial F-test (Table 2.2). This test computes the amount of variability explained by the individual variables within multiple regressions. One can easily see that the impact of methodological variable is different for different predator types.

Developmental stages

There was relatively little difference in the vulnerability of different developmental stages to predators although there was a general tendency toward lower predation rates as fish developed from eggs to juveniles (Figure 2.6). Only in the cases of crustaceans feeding on juveniles ($F_{2, 405}$= 17.6, P< 0.001) and fishes feeding on yolk-sac larvae ($F_{3, 293}$= 11.8, P< 0.001) were predation rates significantly lower and higher, respectively, than for other developmental stages.

Discussion

This review of laboratory and enclosure studies of predation on ichthyoplankton revealed two general patterns of vulnerability in relation to the relative size of prey to predator. Vulnerability to ctenophores and predatory fishes followed a dome-shaped relationship typical of raptorial predators and filter-feeding fishes (Bailey and Houde 1989). Vulnerability to medusae and crustaceans decreased as the size of prey relative to that of the predator increased, typical of cruising invertebrate predators (Bailey and Houde 1989). In all instances, vulnerability of ichthyoplankton to predation decreased when prey were 10% or more of the predators' size.

Bailey and Houde (1989) divided crustaceans into two functional predator groups and predicted a simple linear vulnerability curve for cruising invertebrates (e.g., amphipods, shrimps and euphausiids) and a dome-shaped function for raptorial invertebrates (e.g., crabs and copepods). I found no such differentiation among

crustaceans, which instead showed a negative relationship between vulnerability of ichthyoplankton to predation and the prey:predator size ratio. This simple linear relationship is biologically similar to the model for vulnerability of ichthyoplankton to medusae (Figure 2.5). There is a need to assess if crustaceans and medusae are more similar predators than previously expected or if the similarity between both predators is an artefact of this review and affected by the data used. The ranges of relative prey size studied for crustaceans and medusae were not as well spread out in the smaller relative sizes as for fishes and ctenophores. This may be one reason why a dome-shaped relationship was not apparent in the former two groups. Before concluding that larger ichthyoplankton are less vulnerable to predation by medusae and crustaceans, we should try to investigate the predation impact on relatively small prey sizes (less than 10% predator length). The scarcity of data at the small end of the relative size distribution could be due to the limited variability of predator size, which constrains the prey:predator size ratio logistically available to investigators. Conclusions concerning the impact of predation on relatively small ichthyoplankton should be made with caution because some of the information dealing with medusae and crustaceans is unavailable at this time. There is still the possibility that the impact of these two predator types on relatively small ichthyoplankton may have been underestimated in laboratory studies. Because I failed to note any significant difference in the slopes of the size-selective vulnerability curves for both pairs of groups with similar relative size ranges and functional curves, there remains the possibility that the overall vulnerability

of fish eggs and larvae to predators is independent of predator type. However, the overall magnitude of the impact of individual predator types could depend on their search rates and metabolic requirements.

The results pertaining to the vulnerability of young fish to predation by adult fishes are consistent with earlier predictions, which state that vulnerability should follow a dome-shaped function (Bailey and Houde 1989; Fuiman and Margurran 1994). However, much of the variability in the vulnerability curve of fishes is explained by the duration of the experiment. Only after this variable was added to the model did the second-order term for size, $(\ln(L_f / L_p))^2$, significantly influenced the relationship. Despite a high model r^2 (0.85), it is clear that much of our understanding about the vulnerability of ichthyoplankton to predatory fishes may be greatly influenced by the experimental conditions under which studies are conducted. Although equally difficult situations exist in studying predation rates by fish from stomach analysis (Hunter and Kimbrell 1980), there is a need to reconcile laboratory and field studies. Both avenues of research must be developed on spatial and temporal scales that will allow us to understand the factors that influence the potential impact of predatory fishes. The importance of predation as a regulatory factor of larval fish mortality rates was partly based on results from large mesocosms (e.g. Øiestad 1985). Such experimental systems may provide the means by which populations of larval fish can be exposed to predation by pelagic fishes. Changes in abundance could then be monitored over time, in a manner similar to field-based populations studies.

Effect of control variables

I have found that duration of the experiment, container volume and temperature could help reconcile some variation in the vulnerability to predation by crustaceans, ctenophores, fishes, and medusae among different studies.

The duration of the experiment appeared to be a significant variable for ctenophores, fishes, and medusae. Mathematically this is not unexpected, since it is part of the predation rate (calculated per hour), and regression of an independent variable divided by its dependent variable will most likely be significant and negative (Atchley et al. 1976; A.R. Paradis, unpublished data). Lower predation rates were found in longer experiments. In longer experiments, a level of predation may have been obtained that allowed enough variation for statistically significant differences between treatments. Furthermore, longer experiments tended to be performed in larger containers. I suggest that the high number of encounters, attacks, and captures resulting from a short trial conducted in a small container volume are not representative of natural events in the field. Therefore, the problem in the current calculations of predation rate lies in our general approach to the manipulation of time in experimental trials. I propose that predation rates ought to be calculated as the time until a certain proportion of prey are eaten. Instead of manipulating time to get adequate levels of predation, we should use this time as a measurement of predation rate.

Container volume was a significant variable for all predator types. Higher predation rates were found in experiments using smaller container volumes. This could

be due to increased encounter rates in small container volumes (Bailey and Houde 1989; Fuiman and Margurran 1994) or it could be due to the impact of the container on the swimming behaviours of predator or on the escape responses of larval fishes. Cowan and Houde (1992) found that encounter rate increased more rapidly as a function of the relative velocities of medusae and ichthyoplankton than susceptibility decreased as larvae grew. Tang and Boisclair (1993) found that the size of enclosure affected the swimming characteristics (median speed, acceleration rate, and median turning rates) of juvenile rainbow trout (*Oncorhynchus mykiss*). When studying the effect of container volume on predation rates by medusae, deLafontaine and Leggett (1987) demonstrated that either initial density or container volume had to be kept constant to produce consistent results. They demonstrated decreasing instantaneous mortality rate (Z corrected for predator area) with increasing container volume and, when initial prey density was kept constant, increasing predation rate (number of larvae taken per day) with increasing container volume (deLafontaine and Leggett 1987). With various levels of initial prey density, I found a negative impact of container volume on medusa predation rates. I argued previously that the impact of the duration of the experiment on predation rates may be due, in part, to a mathematical artefact. The same argument might apply for initial prey density and container volume because these two variables are similarly linked in the calculation of predation rate. Predation rate is often calculated as the proportion of prey eaten. The use of proportion has been argued to allow comparisons of predation rates between experiments using different

initial prey densities (e.g., Gotceitas and Brown 1993). This could lead to erroneous predictions of predation impact if the comparisons between treatments do not attempt to keep container volume or initial prey density constant. Furthermore, small container volume and (or) high initial prey densities may overestimate the encounter rates and therefore overestimate the predation rates.

The lack of an effect of temperature on predation rates in this study is surprising because of the overwhelming evidence of its impact in a number of other studies. Pepin (1991) demonstrated strong temperature effects on either cumulative or stage-specific larval mortality. Unlike this review, which is based entirely on laboratory data, Pepin's (1991) temperature models were based on field observations of daily and cumulative mortality rates of eggs, yolk-sac larvae, and post-larvae. Lower vulnerability to predation by medusae were found in studies performed at higher experimental temperatures. At these higher temperatures, however, larger prey:predator size ratios were used. I cannot conclude that temperature has an impact on the laboratory estimates of predation rates of fish larvae because of these confounded correlations.

Predation during the early life history of fishes

From hatch to metamorphosis, a larval fish's vulnerability to predators will change continuously depending on their co-occurrence with different functional groups as well as the latters' size distribution. Rapid growth may reduce the impact on larval fish of a specific predator type by reducing the time spent within a vulnerability

window (Houde 1987; Beyer 1989; Pepin 1989; 1991). However, I agree with Leggett and DeBlois (1994), that this is not necessarily beneficial as an individual may pass from the niche of one predator to another. Figure 2.7 illustrates the range of predator sizes used in the experiments summarised in this study and their adjusted predation rates. Invertebrate carnivores should be dominant predators during the early larval stage whereas fishes should be of greater importance during the latter parts of the larval period. Although mortality rates in an ideal uniform ecosystem should decrease with increasing size of larvae (Peterson and Wroblewski 1984), spatial and temporal variations in prey-predator interactions owing to patterns in patchiness, migration, and production are likely to lead to departures from theoretical expectations. The patterns of selective predation suggest many similarities among predator groups. However, to understand the role of predation as a regulator of early life history survival, it appears to be essential that scientists initiate research into factors that influence the timing of encounters between larval fish and potential predators throughout ontogeny.

These results provide an increased understanding of the vulnerability to predation of larval fish, regardless of habitat or predator type, over a wide range of experimental conditions. In aquatic ecosystems, most outcomes of interactions between individuals, species, and populations are determined by their relative body sizes (Werner and Gilliam 1984; Miller et al. 1989). The survival of a given prey can depend on its relative body size. The growth of a predator can depend on the relative size of its prey item. Reflecting this, most models of populations or individual interactions are

based on body size and in computer simulations most individuals are followed according to their growth and therefore their present and past body sizes. It is imperative that we understand the effect of body size on predation interactions because these may underlie the evolutionary and ecological trends governing populations dynamics.

For all predator types of this study, maximum vulnerability was attained when the relative size of the fish larvae was 10% of the predator size. This will have implications for life-history strategies of fish, which will be dependent on the nature of the community in which they release their offspring.

Table 2.1: Continued.

References	stages	L_f	L_p	PR	D	T	V	n
			Medusae					
Arai and Hay 1982	y, l	10.0-11.0	5.3-40[g]	0.08-0.4	24	10	3.6	15
Bailey 1984	y, f, s, l	0.6-21.1	14.3[h]	0.01-0.62	24	9	5	18
Bailey and Batty 1983	f	11.0	5.1-22.1	0.0002-0.8	1 or 4	11.5	5	93
Cowan and Houde 1992	l	2.6-9.4	32-109	0.13-0.67	24	-	3200	22
Cowan et al. 1992	e	2.0[c]	0.7-1.0[c]	0.0006-0.002	1	20.6	2200	23
deLafontaine and Leggett 1987, 1988	y, l	3.7-5.3	18-71	0.04-0.49	36.4-46.4	-	1000-9000	65
Fancett and Jenkins 1988	e, l	1.8-6.5[i]	14-62	0.0001-0.003	-	-	7-25	29
Gamble and Hay 1989	y, s, l	2.0-13.0	13-84	0.003-0.23	24-27	8	5000	192
Purcell et al. 1987	l	8.1-19.0	61.7	0.09-0.63	1-5	-	12.1	7

Note: The developmental stage are e, eggs; y, yolk-sac larvae; f, first-feeding larvae; s, starved larvae; l, larvae; and j, juveniles. The variables are L_f, fish larvae length (mm); L_p, predator length (mm); PR, predation rate calculated as the proportion of prey eaten per predator per hour; D, duration of the experiments (hours); T, temperature of the experiments (°C); V, container volume of the experiments (litres); and n, number of observations from a given study. -, information was missing in the original article and could not be found from other studies. Subscript letters indicate that the data were missing in the original article and derived from: [a] Yen (1983,1985), [b] Kathman et al. (1986), [c]Weight-length or age-length relationships from Lasker et al. (1970) were used to convert data from the original article into a form appropriate for this review, [d] Olney(1983), [e]Volume of water displaced by a ctenophore or a medusa; to transform it into length, we used the cubic root of the given value, [f] Leim and Scott (1966), [g] Arai and Brickman-Voss (1980), [h] Bailey and Batty (1983), [i] Jenkins (1987).

Table 2.1.: Continued.

References	stages	L_f	L_p	PR	D	T	V	n
			Fishes					
Ansell and Gibson 1993	j	42.1-43.1	17-232	0.03-0.9	16	10	350	39
Cowan and Houde 1992	l	6.4	42-50	0.01-0.37	24	-	3 200	4
Folkvord and Hunter 1986	l	6.4-50.8	86-191	0.05-0.33	0.08	22.1	1 350	12
Fuiman 1989	l	8.8-16.9	66-104	0.04-0.37	8	10.5	81.66	21
Fuiman and Gamble 1988	l	8.6-10.1	58-193	0.01-0.96	24	11.8	15 500	32
Fuiman and Gamble 1989	l	10.35	150-190	0.03-0.15	2-8	-	15 800	22
Gamble and Fuiman 1987	y, s	8.4-9.3	68.5	0.02-0.47	4.3-7.7	9.5	5 000	42
Margulies 1990	y, s, l	3.0-14.0	45.5	0.07-1.0	0.25	-	38	45
McGovern and Olney 1988	y	4.9[f]	16.5-92.5	0.19-0.94	1	-	6.5	9
Monteleone and Houde 1992	e, y, l	1.7-13.3	58-65	0.09-0.77	0.25	19	76	27
Pepin et al. 1987	l	1.7-9.0	350	0.005-0.05	0.08	17.0	3 000	28
Pepin et al. 1992	y	5.0-5.8	36-61	0.8-1.0	24	13.2	2 700	20

Table 2.1: Summary of the studies used in this review.

References	stages	L_f	L_p	PR	D	T	V	n
			Crustaceans					
Ansell and Gibson 1993	j	27.8-44.0	49.5-64.8	0.01-0.1	12-16	10	40	8
Bailey 1984	y, f, s, l	3.2-21.1	4.2[a]-22.5[b]	0.01-0.36	24	9	5	33
Bailey and Yen 1983	e, y, s, l	1.3-5.2	4.2[a]	0.02-1.0	24	8	1-2	47
Lillelund and Lasker 1971	y	2.5-4.7[c]	1.8-3.4	0.0007-0.99	20-24	18	3.5	96
Theilacker and Lasker 1971	y	2.5	3.5-10.5	0.04-0.96	22	17	3.5	73
Turner et al. 1985	y, l	1.6-3.9	1.8-4.2	0.0001-0.32	20	22	1	33
van der Veer and Bergman 1987	j	10.0-55.0	23-65	0.0003-0.45	36	18	12.5	41
Westernhagen et al. 1979	l	7.5-16.0	1.1-3.0	0.0003-0.01	5	9	0.5	61
Westernhagen and Rosenthal 1976	y	8.75	1.64	0.0002-0.009	1	9	0.5	19
			Ctenophores					
Cowan and Houde 1992	l	6.4	64-82	0.01-0.05	24	-	3 200	5
Cowan et al. 1992	e	2.0[d]	0.7-1.5[e]	0.0007-0.002	1	18.	2 200	4
Monteleone and Duguay 1988	e, s, l	1.1-3.1	0.5-47.5	0.0002-1.0	4	-	1-200	160

Table 2.2: Partial F of significant variables of the equations empirically derived for four types of predators of ichthyoplankton.

Predator type	df	$\ln(L_f/L_p)$	$(\ln(L_f/L_p))^2$	$\ln(D)$	$\ln(V)$	$\ln(T)$
Crustaceans	1, 408	116	NS	NS	36	NS
Ctenophores	1, 164	110	88	20	196	NS
Fishes	1, 296	40	74	729	19	NS
Medusae	1, 335	100	NS	217	342	14

Note: NS, not significant.

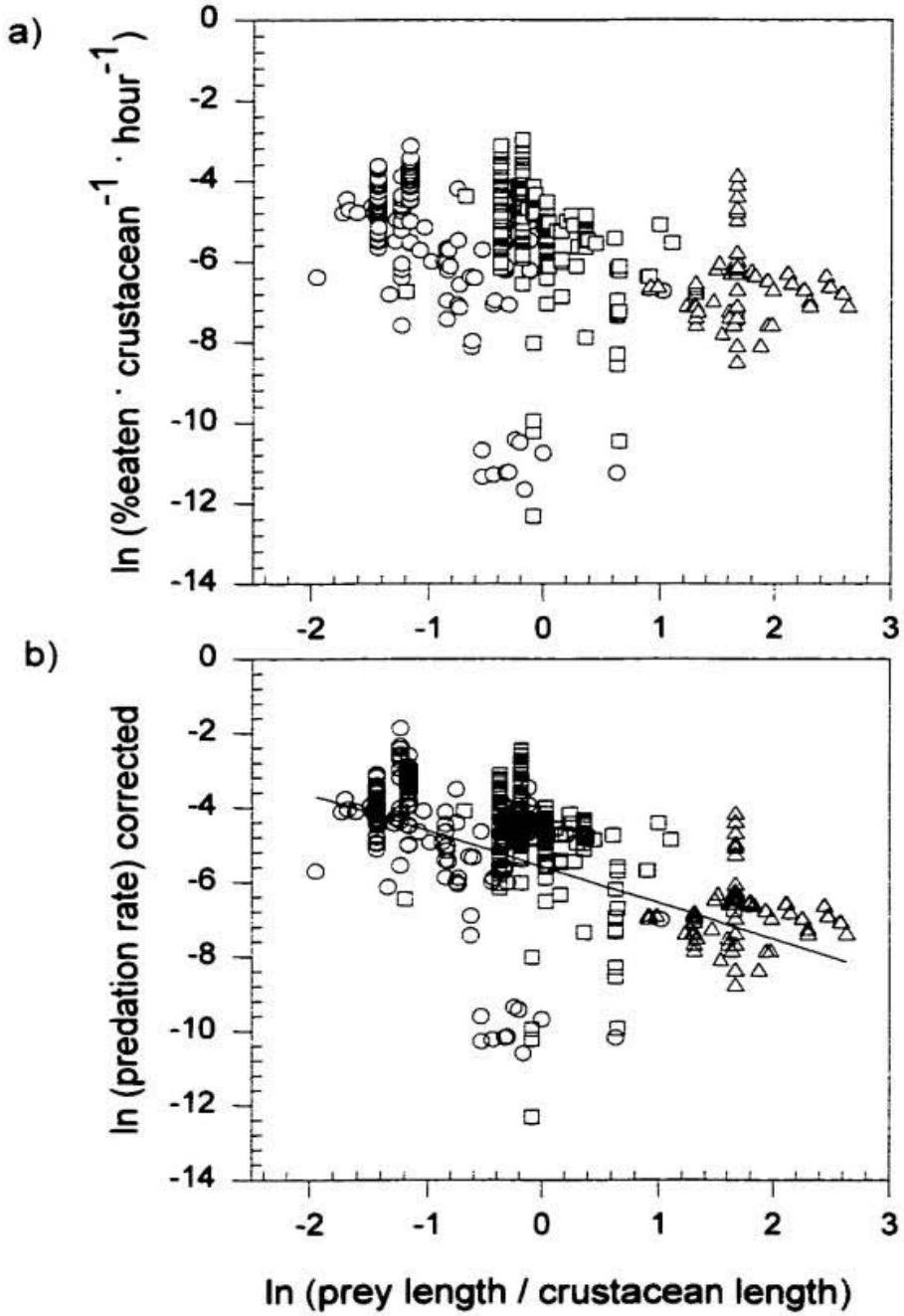

Figure 2.1: Predation rates of crustaceans on ichthyoplankton in relation to the prey:predator size ratios: **(a)** raw data, **(b)** data corrected for the effect of container volume (mean of 2.5 L) in experiments that used shrimps, crabs, and euphausiids (circles), copepods (squares), and amphipods (triangles).

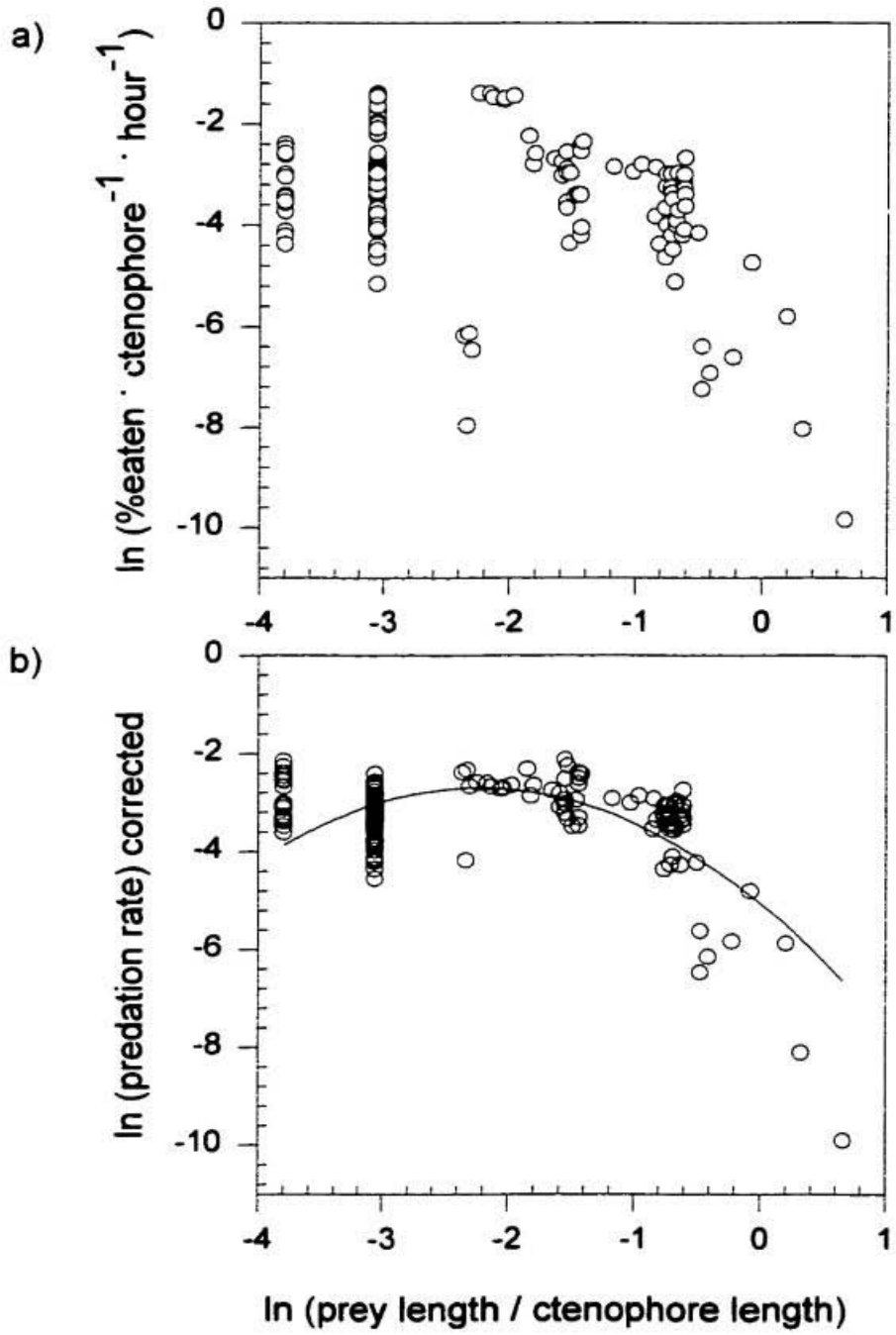

Figure 2.2: Predation rates of ctenophores on ichthyoplankton in relation to the prey:predator size ratios: **(a)** raw data, **(b)** data corrected for the effect of container volume (mean of 17.2 L) and duration of the experiment (mean of 4 h).

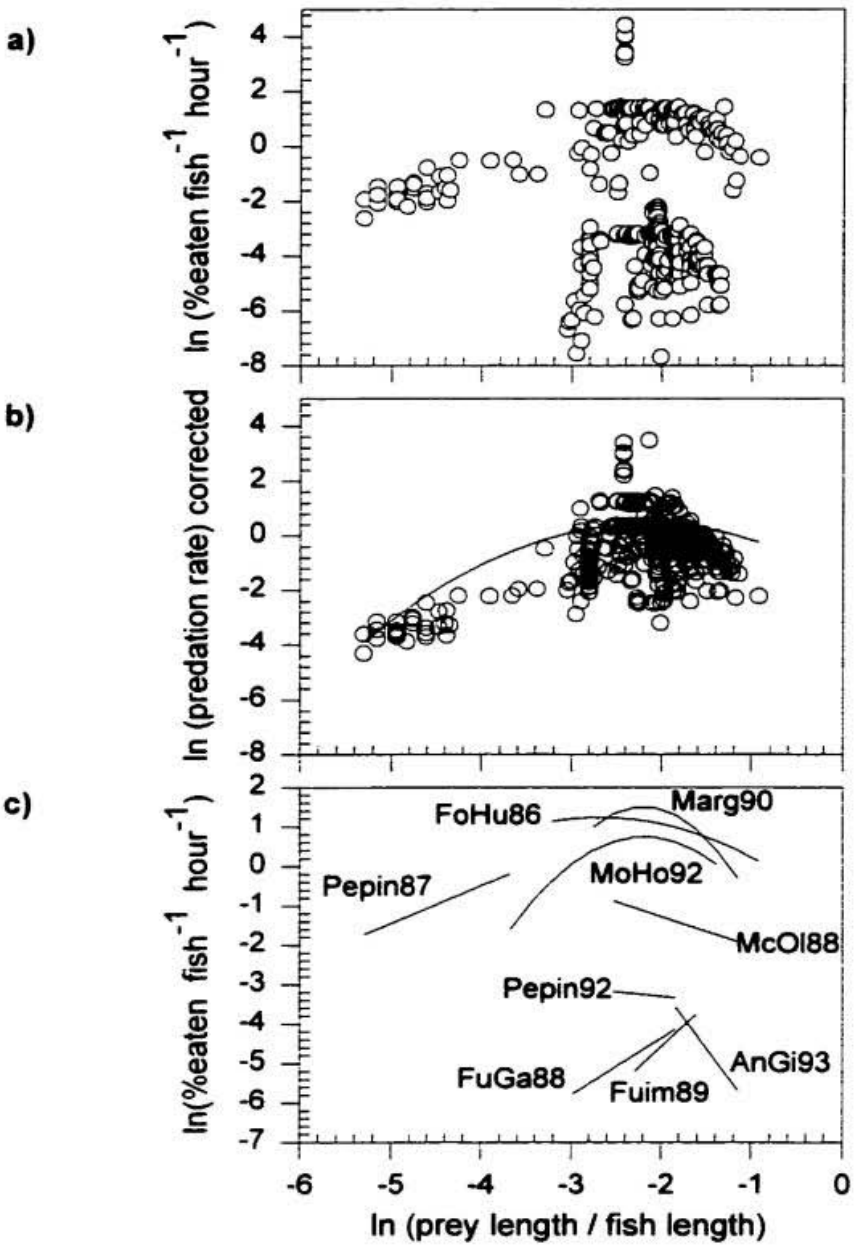

Figure 2.3: Predation rates of predatory fishes on ichthyoplankton in relation to the prey:predator size ratios: **(a)** raw data, **(b)** data corrected for the effect of duration of the experiments (mean of 2 h) and container volume (mean of 690 L), **(c)** each line represents the best of first and second order regression for individual studies that are identified by the first four letters of the first author's name or the first two letters of both authors' names and the year of publication.

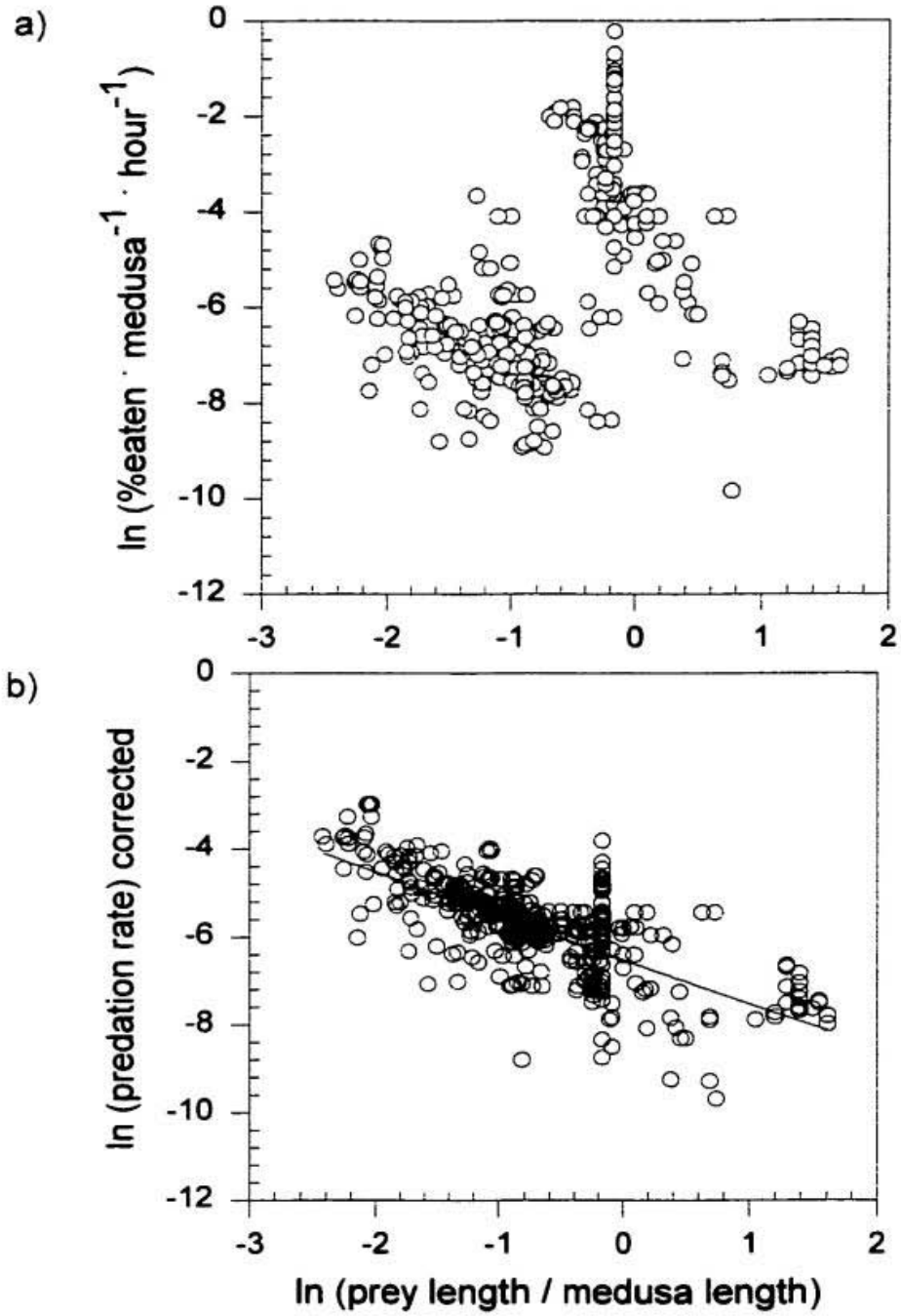

Figure 2.4: Predation rates of medusae on ichthyoplankton in relation to the prey:predator size ratios: **(a)** raw data, **(b)** data corrected for the effect of container volume (mean of 372 L), temperature (mean of 9.6 oC), and duration of the experiment (mean of 9 h).

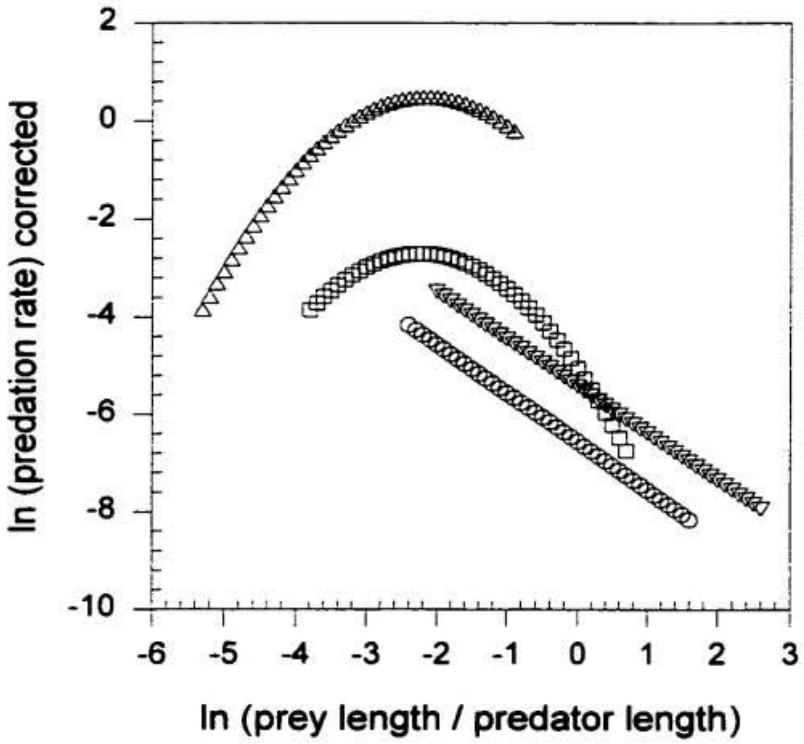

Figure 2.5: Curves describing the vulnerability of fish early life stages to predation by medusae (circles), crustaceans (inverse triangles), ctenophores (squares), and fishes (triangles). The predation rates were corrected with the mean value of the specific experimental conditions affecting predation rates of each predator type. Line were drawn according to the equations of the final model for each predator type and the range of prey:predator size ratios used in the analysis.

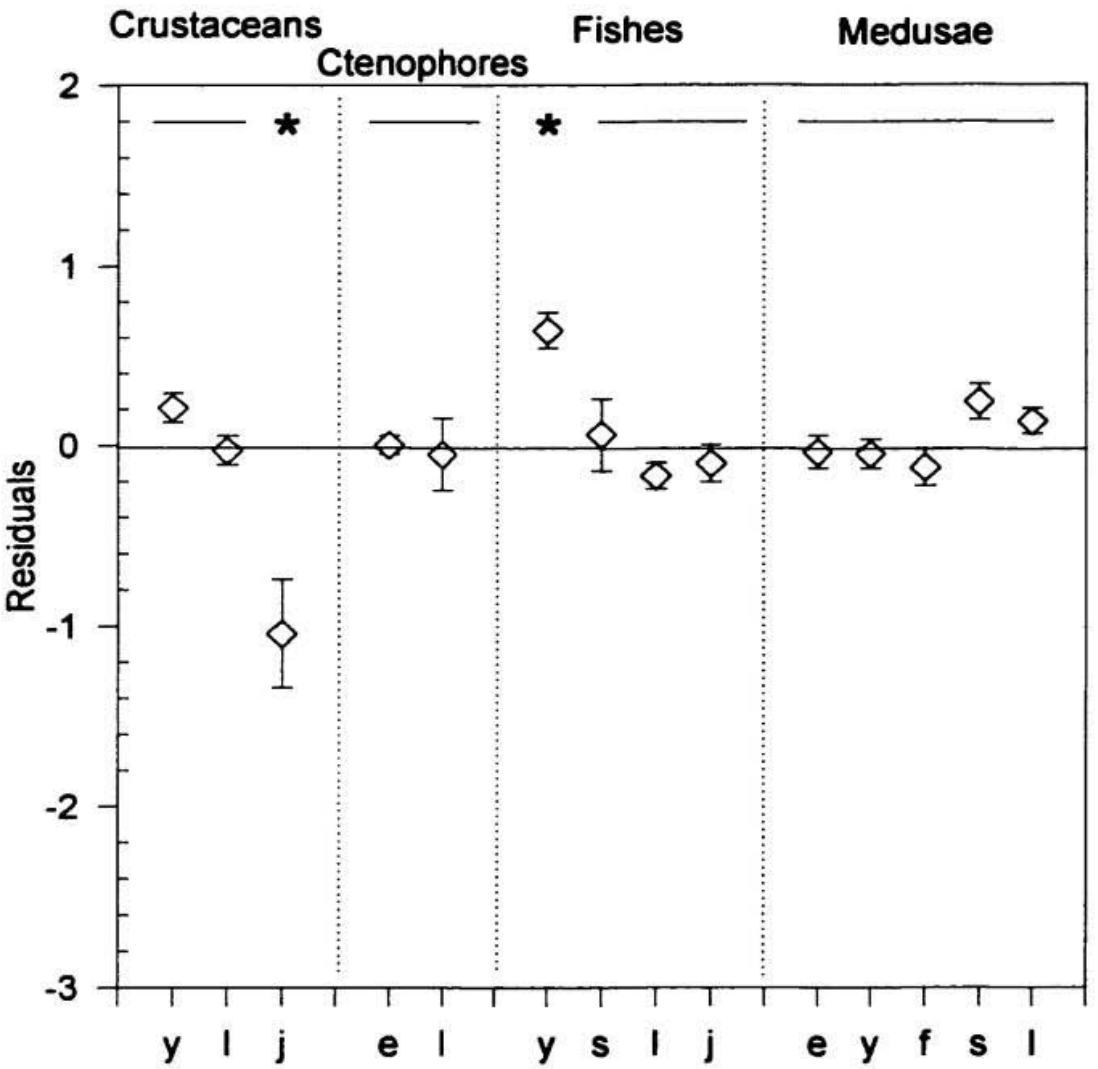

Figure 2.6: Mean residuals of the size-selective predation rates by different predator types plotted against the prey life stage. A statistically different mean is reported with a star (multiple comparisons test) and all nonsignificant means within a predator type are marked by a horizontal line on the x axis. e, eggs; y, yolk-sac larvae; f, first-feeding larvae; s, starved larvae; l, older larvae; j, juveniles. Error bars show ±1 standard error.

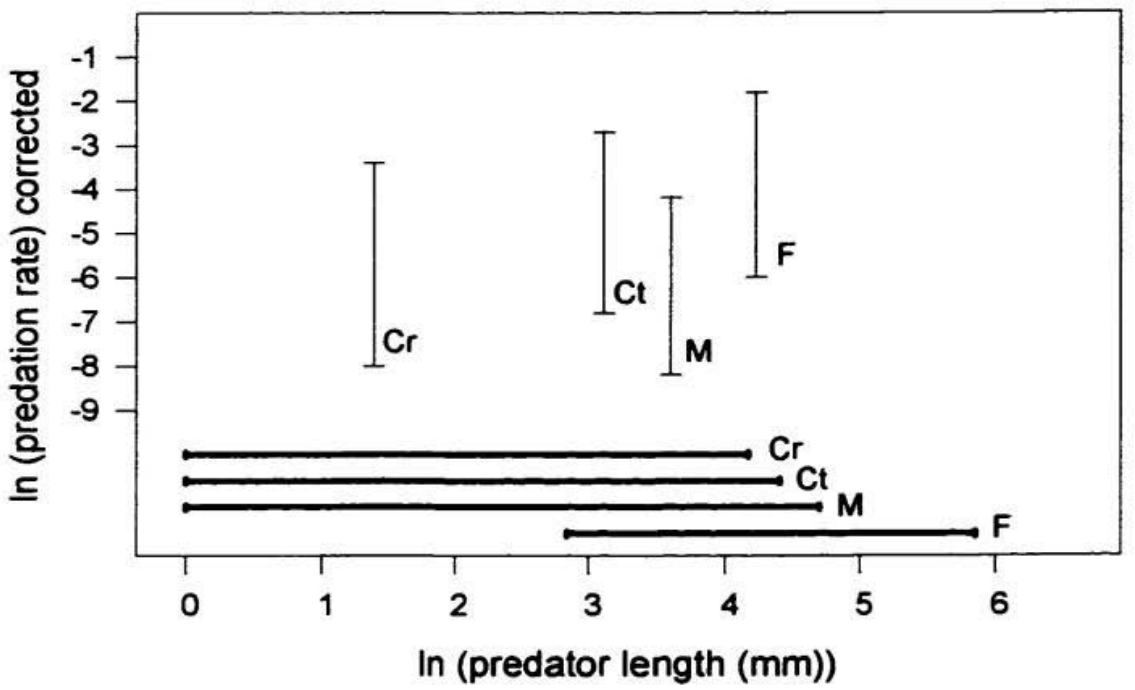

Figure 2.7: Schematic diagram showing the range of lengths of the different predator types used in the experiments summarized in this study (horizontal bars). The vertical error bars indicate the range of predation rates, after correction for experimental conditions, observed in the studies associated with each predator type. The location of the vertical error bars along the x axis represents the median predator length from the studies summarized in this review. Cr, crustaceans; Ct, ctenophores; M, medusae; F, fishes.

Chapter III

Disentangling the effects of size-dependent encounter and susceptibility to predation with an individual-based model for fish larvae.

Abstract

I investigated the effects of size-dependent encounter and susceptibility, the role of variation in the size distribution of predators, and the timing of prey-predator interaction during the larval phase in shaping the length frequency distribution of surviving fish larvae. These analyses based on general empirical size-dependent relationships may have broad implications to understanding larval fish cohorts dynamics. I demonstrated that the formulations of encounter and susceptibility to predation counteract each other, an increased range of predator sizes reduces the evidence for size-selective mortality only slightly, and synchronous spawning and hatching events have the potential to produce strong size-selective mortality of a cohort of fish larvae. The important factors in generating size-selective mortality are either the timing of encounters between fish larvae and their predators or high mortality rates. I demonstrated a direct relationship between the potential of size-selective mortality and the overall mortality rate of the cohort. I suggest that it may be difficult to detect the effect of size-dependent processes in the field. A better understanding of the factors influencing encounter represents a critical element in extrapolating laboratory studies of predation to the field.

Introduction

Predation is a major source of mortality during the early life stages of fishes (Houde 1987; Bailey and Houde 1989). A prey-predator interaction is the sequence of three events: encounter between a prey and a predator, attack by the predator, and capture of the prey (Greene 1986). The sizes of both prey and predator are important factors determining the outcome of predation interactions (e.g., Werner and Gilliam 1984; Bailey and Houde 1989; Chapter II). Analyses based on general patterns of encounter and susceptibility (where the latter is the combination of attack and capture) may reveal qualitative results with broad implications (Rice et al. 1997). In previous individual-based models, the relationships for either attack or capture were derived from single experimental studies or from species-specific observations (e.g., Rice et al. 1993a,b 1997; Letcher et al. 1996; Cowan et al. 1996; 1997).

Previous individual-based models have contributed significantly to our understanding of the mechanisms by which predation mortality influences survival during the early stages of fish. Rice et al. (1993a) found that faster growing cohorts of fish larvae had a higher survival rate than slower growing cohorts, and that the size distributions of survivors were generally larger than expected from considerations of growth rates alone. Rice et al. (1993b) have demonstrated that choosing a different size class of predator can produce the opposite effect of size-selective predation on the size distribution of survivors. Cowan et al. (1996) also found that faster growing cohorts had a higher survival rate than slower growing cohorts of fish larvae when they combined

variations in the parameters of the growth rate distribution as well as predator size structure and foraging strategies. Cowan et al. (1996) further demonstrated that size-selective mortality was apparent only after a significant number of fish larvae died (~70% of the original cohort) and that growth needs time to develop differences in the characteristics of survivors.

Cowan et al.'s (1996) modeled different size classes of predators separately and compared foraging patterns of large and small predators. Rice et al. (1993b) and Letcher et al. (1996) have argued that it is the full range of predator sizes that is an important factor in shaping the size distribution of survivors. Rice et al. (1997) further developed their model by incorporating growth rate and size distributions of predators. Fish larvae and predators grew at differential rates such that fish larvae outgrew the predator's preferred prey size. They found that small changes in these factors had strong effects on the size distribution of surviving fish larvae. Most important was their demonstration of how these effects change over time. Rice et al. (1997) argued that strong selection in one direction late in an interaction may completely erase evidence of strong selection in the opposite direction occurring at earlier stages, leaving the false impression that no size selection was occurring at all.

In this study, I extended previous models to investigate the role of variations in predator size and abundance and the effects of encounter and susceptibility in shaping the length frequency distribution of surviving fish larvae. I also investigated the influence of different timings of spatial overlaps between fish larvae and predatory fishes. I compared

these results for four different predator types: crustaceans, ctenophores, medusae, and pelagic fishes.

Methods

Model description

The model was designed to simulate the growth and predation mortality of fish larvae for 30 days. The initial cohort of fish larvae was composed of 10 000 individuals, where each larva was characterised by a discrete length and growth rate (Table 3.1) drawn at random from a normal distribution.

Fish larvae were subject to predation by four types of predators: crustaceans, ctenophores, medusae and pelagic fishes. The size distribution of each predator was generated from a normal distribution. The minimum and maximum sizes of predators were restricted (Table 3.1) so that the relative size of fish larvae to predator remained within the range of previous laboratory studies (Chapter II). This was possible because predators were not allowed to grow during the simulation run. Predator size referred to the length of crustaceans and pelagic fishes, and the diameter for ctenophores and medusae.

Each individual fish larva was exposed to predation by one of the four predator populations each day for 30 days. I evaluated whether the larva encountered a predator and whether it was captured by the predator. The predation model of this study was a combination of three processes: encounter between fish larvae and predator, susceptibility of fish larvae to the encountered predator, and growth of survivors. Encounter rates were

calculated according to the Gerritsen and Strickler's (1977) equation modified to account for non-zero prey sizes (Bailey and Batty 1983). Susceptibility probabilities were based on the general laboratory-derived predation equations from Chapter II. Growth of fish larvae was based on the constant growth model of Rice et al. (1993a).

The individual-based model presented in this study is similar to Cowan et al.'s (1996) individual-based model. However, I explicitly incorporated the size of predator as an important factor determining the outcome of predation, as suggested by Rice et al. (1993b). The susceptibility probability was a function of both prey and predator sizes and was based on multispecific-empirical relationships for the four predator types (Chapter II) whereas the susceptibility probability of Cowan et al. (1996) was a function of prey size only and was based on laboratory measurements of realised capture probability (from Cowan and Houde 1992).

Encounter model

The encounter rates (E, encounter $\cdot$ d^{-1}) between a fish larva and a predator were based on a modification of the Gerritsen and Strickler (1977) model for randomly moving organisms in a three-dimensional space such that:

$$E = \pi \times D \times C \times A_p \times R_{total}^2 \times \left(\frac{V_f^2 + 3 \times V_f^2}{3 \times V_p}\right) \quad for\ V_p \geq V_f$$
$$E = \pi \times D \times C \times A_p \times R_{total}^2 \times \left(\frac{V_p^2 + 3 \times V_f^2}{3 \times V_f}\right) \quad for\ V_p \leq V_f \qquad (3.1)$$

where V_f and V_p were the swimming speeds of prey and predator respectively ($mm \cdot s^{-1}$), A_p was the size-specific abundance of predators ($predator \cdot m^{-3}$), D was the proportion of daylight hours in a day (13h / 24h), C was a time and volume conversion ($8.64 \times 10^{-5} s \cdot d^{-1} \cdot m^3 \cdot mm^{-3}$). I assumed that fish larvae were motionless at night time (as Cowan et al. 1996) so that encounters could only occur during the day. Including size-specific abundance for predators allowed me to include the possibility that a fish larvae are more likely to encounter certain predators than other ones. Originally, Gerritsen and Strickler (1977) defined an encounter radius independent of the sizes of organisms. The total encounter radius (R_{total}, mm) was the sum of the encounter radii of fish larvae and predator such that the encounter radius of prey or predator was defined as:

$$R = a \times L \tag{3.2}$$

where L was either the length of fish larvae (L_f, mm) or the size of predator (L_p, mm), and a was a parameter (Table 3.2). Equation (3.2) allowed me to relax the original assumption that fish larvae and their predators are dimensionless points in space (Bailey and Batty 1983). The swimming speeds of fish larvae and predator (V) were also length-dependent and were calculated as:

$$V = b + c \times L^d \tag{3.3}$$

where b, c, and d were parameters (Table 3.2).

I allowed several encounters between a prey and a predator by calculating the probability of several encounters ($P(N)$) with a standard Poisson distribution as follows:

$$P(N) = \frac{(E \times t)^N \times e^{-E \times t}}{N!} \quad (3.4)$$

where N is the number of possible encounters and t is one day.

Each day and for each larva, I determined at random the numbers of encounters with a predator (N). In addition, I determined the size of the predator encountered (L_p) with another random number, based on the normal size frequency distribution of the predator population. Then, I used another random number to determine which of N encounter(s) lead to a capture given that I had determined the length of predator encountered.

Susceptibility model

Susceptibilities (S) were derived from the laboratory-corrected predation equations of Chapter II for specific predator types such that:

$$ln(S) = g + h \times ln \frac{L_f}{L_p} + i \times (ln \frac{L_f}{L_p})^2 \quad (3.5)$$

where g, h, and i were parameters (Table 3.2). The parameter g included the laboratory corrections calculated in Chapter II. Under average laboratory conditions, container volumes are small and duration of an experiment is short. Under these conditions, encounter rates between all four types of predators and fish larvae are greater than one, if they are calculated with Gerritsen and Strickler's (1977) equation (see Appendix 1). This implies that the prey and predator under average laboratory conditions are contained

within the same body of water. I assumed that on the larger spatial (thousands of cubic meters of water) and temporal (30 days) scales as in this simulation model, the probability of encounters between fish larvae and their predators is less than one. Therefore, a predation rate calculated under laboratory conditions represented susceptibility under field conditions (see Appendix 1).

Vulnerability curves

In this model, I defined encounter and susceptibility as functions of both prey and predator sizes. The product of these equations is vulnerability. The model predicts that small fish larvae will be most vulnerable to all three invertebrate predators (Figures 3.1, 3.2, 3.3) whereas larger fish larvae will be most vulnerable to predation by pelagic fishes of intermediate sizes (Figure 3.4). Changes in vulnerability of larval fishes to predation by pelagic fishes are mostly governed by variations of predator sizes (Figure 3.4). Furthermore, the peak vulnerability changes given different sizes of predators and occurs for similar length of fish larvae. It is obvious that including predator sizes at all stages of a predation interaction (encounter and susceptibility) may lead to different conclusions about the effects of a predator population on the survival rates and the characteristics of surviving fish larvae.

Growth model

Each larva was assigned a growth rate ($mm \cdot d^{-1}$) drawn at random from a normal distribution (Table 3.1) and its assigned growth rate was maintained throughout the entire simulation. At the end of each day, the lengths of all surviving individuals was updated with the individual's growth rate.

Design of simulation hypotheses

I performed three simulation trials to investigate the three objectives of this study (Table 3.3). First, other simulation studies (Rice et al. 1993a,b; 1997; Letcher et al. 1996; Cowan et al. 1996; 1997) implicitly assumed that the abundance of predators will only affect the overall survival rate of the cohort and will not interfere with the size-selective mortality of individuals. To evaluate this assumption, I chose five abundance levels for each predator (Table 3.4). The abundance of predator was chosen to yield mortality of the order of 10%, 30%, 50%, 70% and 90% total loss from the initial cohort. I was not concerned if these abundance were representative of specific field abundance estimates because the main focus was to evaluate how changes in abundance which yield different mortality levels interact with the size-selective mortality of a cohort of fish larvae. For these simulations, I present the characteristics of the cohort of fish larvae consumed by the predators as well as the length frequency distribution of the growth and survival cohorts (Table 3.3). I also investigated the relationship between size-selective mortality and the instantaneous mortality of a cohort of fish larvae ($Z= (\ln N_0 - \ln N_t)/t$, where N_0 is 10 000

individuals and N_t is the number of survivors after t=30 days). To evaluate the relationship between these two variables, I defined a size-selective mortality index as the absolute maximum difference between the relative cumulative frequency distribution of the growth and survival cohorts. This index is based on the statistic d_{max} of the Kolmogorov-Smirnov test for goodness of fit for discrete or continuous grouped data (Zar 1984; Sokal and Rohlf 1981). I wish to stress that in this study, it is not appropriate to calculate a probability value because I am dealing with populations and not with samples of a population.

The abundance of predator and prey is often temporally and spatially variable. The presence of pelagic fishes in nursery areas may not coincide temporally with the full developmental period of the early life stages of fishes. I ran a series of simulations in which I restricted predation by pelagic fishes to five days (Table 3.3). In these simulations, the abundance of pelagic fishes was set to 0.0192 m^{-3} which under initial conditions (i.e., no restriction on the timing of predation), 50% of the initial cohort was consumed in 30 days.

Finally, to evaluate the individual components of vulnerability (i.e., encounter and susceptibility), I manipulated vulnerability by keeping either the encounter rate or the susceptibility probability constant and independent of prey and predator sizes (Table 3.3). Due to the high level of mortality of these simulations (~96% of the initial cohort is consumed in 30 days) and to reduce the stochastic noise of the model, I increased the initial number of fish larvae to 100 000 individuals. This allowed me to better characterise the length frequency distribution of the survival cohort. I reduced the range of predator

sizes to a single class (Table 3.3) to evaluate the effects of both encounter and susceptibility without the confounding effects of variations in predator size. However, I ran other simulations where I relaxed the restriction on predator size by defining the predator population with a range of sizes as in previous simulations (Table 3.1). I condensed the presentation significantly by presenting the difference between the length frequency distributions at each larval length classes of the growth and survival cohorts (as in Crowder et al. 1994). If this value is positive, it implies that there are relatively more individuals in the growth cohort than in the survival cohort for that given length class. Therefore, fish larvae of that length class are most vulnerable to predators.

Results

Abundance of predators

The sizes of fish larvae most vulnerable to predation by crustaceans and pelagic fishes shifted towards smaller sizes as predator abundance increased (Figures 3.5a, 3.8a, respectively). As predator abundance increased, larval mortality due to these predators occurred earlier during the simulation period (Figures 3.5b, 3.8b, respectively). Peak vulnerability to predation by ctenophores and medusae appeared to be independent of predator abundance, as it always occurred early during the simulation and for small fish larvae (Figures 3.6, 3.7, respectively).

The temporal progression of the length frequency distribution of larvae subjected to different types of predation pressure showed that losses occurring early during the

development of a cohort appeared to have the greatest impact during the late larval phase (Figure 3.9). When subject to predation by invertebrates, surviving larvae were always larger than a cohort growing in the absence of predation (Figure 3.9a,b,c). The opposite is true with respect to predatory pelagic fishes (Figure 3.9d). The strongest evidence of size-selective larval mortality occured for medusae (Figure 3.9c), which fed heavily on larvae during the first six days of the simulations (Figure 3.7b) after which the larvae quickly grew out of the range of high vulnerability (Figure 3.7a). It is important to note that the evidence of size-selective mortality only appeared when total losses during 30 days exceed 50%, regardless of predator type. Furthermore, by compiling all simulations performed at different predator abundance levels, I found that the index of size-selective mortality increases with instantaneous mortality rates (Figure 3.10). In fact, there was almost a one-to-one relationship between these two variables, regardless of predator type.

Temporal variation in predation by pelagic fishes

The strongest evidence of size-selective larval mortality occurred when predation by pelagic fishes was restricted to the first five days of the simulation (Figure 3.11a). There was no difference between the length frequency distributions of the growth and survival cohorts when predation by pelagic fishes occurred for five days either at the middle or the end of the simulation (Figure 3.11b,c). Furthermore, the evidence of size-selective mortality was much stronger if predation by pelagic fishes was restricted to the first five days of the simulation (Figure 3.11a) compared to the case when predation by

pelagic fishes was not restricted and yielded a higher overall mortality rate (Figure 3.9d). If predation by pelagic fishes was restricted to the first five days of the simulation, fish larvae present at that time measured between 2 and 9 mm in length, a size range that was most vulnerable to predation by pelagic fishes (Figure 3.8).

Disentangling the effects of encounter and susceptibility

If vulnerability of fish larvae was entirely dependent on encounter, mortality increased with increasing larval length and the survival cohort was smaller in length than the growth cohort. Individuals 18 mm or longer were relatively more numerous in the growth cohort than in the survival cohort, indicating that relative mortality (based on encounter) of these individuals was higher (Figure 3.12). The evidence of size selection (expressed as the percent difference between the length frequency distributions of the growth and survival cohorts) decreases slightly with increasing invertebrate size (Figure 3.12a,b,c). There was no more than a 2% size-specific difference between the growth and survival cohorts when larvae were faced with predation by pelagic fishes (Figure 3.12d).

If vulnerability was entirely dependent on susceptibility, mortality by invertebrates decreased with increasing larval length. The survival cohort was slightly larger than the growth cohort because fish larvae less than 18 mm were most vulnerable to predation by invertebrates and by small size classes of pelagic fishes (Figure 3.13). Size-selective mortality (based on susceptibility) was similar for all three invertebrate predators and was

independent of crustaceans and medusae size classes (Figure 3.13a,c). However, it varied considerably with size classes of pelagic fishes (Figure 3.13d). Small fish larvae (less than 18 mm) had a higher susceptibility to predation by small pelagic fishes (25 and 45 mm) whereas individuals larger than 18 mm had a higher susceptibility to predation by the longest pelagic fishes (355 mm). There was no evidence of size-selective mortality (based on susceptibility) of fish larvae to predation by pelagic fishes of intermediate sizes (115 mm).

Size-dependent encounter tended to favour smaller fish larvae (Figure 3.12) whereas size-dependent susceptibility tended to favour larger fish larvae (Figure 3.13). When vulnerability was a function of both these size-dependent relationships, I found that the evidence of size-selective mortality due to predation by crustaceans decreased from ±5% to ±3% (Figures 3.12a, 3.13a, 3.14a) whereas it increased from ±4%, ±5% to >±6% for ctenophores (Figures 3.12b, 3.13b, 3.14b). The evidence of size-selective mortality due to predation by medusae (Figure 3.13c, 3.14c) or pelagic fishes (Figure 3.13d, 3.14d) remained approximately as size-dependent susceptibility would predict (i.e., ±4%). These results suggest that susceptibility is the driving force of vulnerability when 96% of the initial cohort is consumed in 30 days. When I compared these results with simulations where the predator population was characterised by a wider range of sizes, I found that the evidence of size-selective predation by crustaceans or ctenophores decreased slightly (Figure 3.14a,b). For medusae, there was very little effect of the range in medusae sizes on

the size-selective mortality of fish larvae (Figure 3.14c). There was little evidence of size-selective predation by pelagic fishes as the range of predator size increased (Figure 3.14d).

Discussion

My simulation results clearly showed that predation pressure must be substantial in order to show any evidence of size-selective losses from larval fish populations. Throughout the simulated conditions, evidence of size selection by pelagic fish predators was relatively weak. The evidence of size-selective losses appeared to be greatest for invertebrate predators relative to vertebrate predators when predation pressure was exerted throughout the simulations. The greater effect of invertebrate predators was partly because the timing of losses during the life of larvae was concentrated early in the model runs. This was made even more apparent when predation by pelagic fishes was restricted to five day periods, as only predation during the earliest part of the simulation, when larvae were smallest, produced any evidence of size-selection after a 30 day simulation. I demonstrated that this occurred because the general model of size-dependent susceptibility (Chapter II) counteracted Gerritsen and Strickler's (1977) widely used model of size-dependent encounter rates.

I found that size-selective mortality was stronger when predation by pelagic fishes was restricted to the first five days of the simulation, in contrast to predation that was restricted later during the simulation period or not at all. This contrasts with Rice et al. (1993a) argument that size-selective mortality is important later on when growth has had a

chance to create size differences among individuals. I argue that the difference between the length frequency distributions of the survival cohort and the growing cohort was enhanced with time through the action of growth because almost all of the larger fish larvae were eaten during the first five days of their developmental period. Overall mortality of fish larvae spread out over the simulation period reduced the evidence of size-selective mortality. I assume that this brief temporal overlap during the early part of the larval period may occur in the field because of the ample evidence of temporal overlap between pelagic fishes and fish eggs and larvae (Hunter and Kimbrell 1980; Santander et al. 1983; Daan et al. 1985; Cowan and Houde 1993).

In other individual-based simulation models (Rice et al. 1993a,b; 1997; Letcher et al. 1996; Cowan et al. 1996; 1997), abundance of predators were selected to yield significant predation rates comparable to field estimates of mortality rates of fish larvae. Furthermore, the predator abundance modelled was comparable to field abundance estimates of predators. However, very little is known about the pattern of size-selective predation mortality given variations in predator abundance and ultimately the overall mortality rate of the cohort of fish larvae. If the length distribution of surviving fish larvae varies with predator abundance, it could become particularly problematic if one compares different fish larvae characteristics at different abundance of predators (e.g., Cowan et al. 1996). I investigated the size characteristics of fish larvae consumed given different levels of predation pressure and found that the time when most mortality occurs changes the length distribution of fish larvae consumed by predators and consequently the length

distribution of survivors. In order to see responses of the length frequency distribution of surviving fish larvae, the impact of predation must occur during a restricted period, either by a high selection on a narrow range of larval lengths, or high mortality restricted to a few days. In the field, invertebrate predators do not feed on such a restricted size range of larvae or for a short time period (Yen 1983; Purcell 1985; Yamashita et al. 1985).

Sizes characteristics of predators have a strong influence in determining the size (Rice et al. 1993b, Letcher et al. 1996) or growth rate (Rice et al. 1993a) of surviving fish larvae. I found that the length distribution of surviving fish larvae changes with size classes of pelagic fishes. I also found that the difference between the length frequency distributions of the growth cohort and the survival cohort decreased only slightly when the predator population was characterised by a wider range of sizes. This result contradicts Rice et al.'s (1993a,b) suggestion that a wider range of predator sizes would significantly decrease the evidence of size-selective mortality of fish larvae. I suggest that this result is not due to the fact that different sizes of predators consumed different sizes of fish larvae but mostly due to differences in the timing of high vulnerability caused by the different predator sizes. High susceptibility occurs when a fish larva measures 10% of the length of a predator (Chapter II). If I compare simulation trials of different predator sizes, the time at which the cohort of fish larvae is highly susceptible to predation will differ between trials because fish larvae will measure 10% the length of the predator at different times during the simulation.

I demonstrated clearly that the encounter and susceptibility models counteracted each other. Cowan et al. (1996) used the same encounter model (Gerritsen and Strickler 1977) but their results showed much greater evidence of size-selective mortality. The difference between these results is likely due to the choice of susceptibility functions. Cowan et al. (1996) used a susceptibility function based on their own laboratory work. I used empirically-derived predation rates corrected for laboratory conditions (Chapter II). I argue, as did Rice et al. (1997), that an empirical derivation of laboratory estimates of predation rates, based on multispecies prey and predator, is probably a better generalisation of the susceptibility patterns of larval fishes. Furthermore, the parameters of such general functions are not specifically derived under a single set of experimental conditions. There is no reason to believe that one particular set of experimental conditions is likely to yield more realistic estimates of susceptibility. In addition, when prey and predators are studied in laboratory containers or enclosures, they are effectively placed in contact with each other, in contrast to field situations where spatial overlap may vary (Appendix 1 and Frank and Leggett 1982, 1985). When vulnerability is a function of both size-dependent encounter and size-dependent susceptibility, and the predator population is characterised by a wide range of sizes, I predict that the highest difference between the length frequency distributions of survivors and of growing fish larvae without predation will be less than 10%.

I seldom found notable size differences between a cohort of fish larvae surviving different predation pressures and a cohort of fish larvae growing without mortality

during the course of the simulation period (30 days). Therefore, size-selective mortality due to predation of fish larvae may therefore be difficult to evaluate in the field over the same time period (see also Miller 1997). I was able to demonstrate that this lack of size-selective mortality was partly due to the counteracting components of vulnerability. The detection of size-selective mortality will be difficult when fish larvae are growing through the dome of the vulnerability curve (see also Rice et al. 1997) and may be more easily detected when fish larvae are growing through only the down-ward slope of the vulnerability curve, as in Rice et al. (1993a). When the range of predatory fish sizes was restricted to a single size class, there was a reversal in the direction of size-selective survival of the cohort of fish larvae. Finally, there was a direct relationship between size-selective predation mortality and the instantaneous mortality rate of the cohort (Figure 3.10). This relationship clearly demonstrates that size-selective predation mortality will only be detected in cases where mortality rates are high (greater than $10\% \cdot d^{-1}$) or in cases where a population of pelagic fishes consumes a significant amount of fish larvae only during a brief period during the early development of larvae. Despite problems relating to the tremendous variability in the temporal and spatial distribution of fish larvae and their predators, I believe that with appropriate techniques, it may be possible to estimate size-specific mortality rates due to predation in the field (see also Miller 1997). However, caution should be exercised during the investigation of the individual components of vulnerability.

Table 3.1: Characteristics of the size (mm) distributions (mean, standard deviation (SD), minimum and maximum) of crustaceans, ctenophores, pelagic fishes, and medusae were calculated with the size ratio of Chapter II and were drawn at random with a random number generator. Characteristics of the initial fish larvae length (mm) distribution and growth rate ($mm \cdot d^{-1}$) distribution are also given.

Predator type		Mean	SD	Min	Max
Crustaceans		10.0	5	1.0	20.0
Ctenophores		20.0	10	1.0	40.0
Fishes		185.0	80	15.0	355.0
Medusae		55.0	27	1.0	110.0
Fish Larvae	length	3.0	0.9	1.0	5.0
	growth rate	0.5	0.08	0.2	0.8

Table 3.2: Values of the parameters of the equations of encounter radius, swimming speed, and susceptibility (see equations 3.2, 3.3 and 3.5, respectively). N.A., not applicable.

Equations		Prey	Predators			
Parameters		Larvae	Crustaceans	Ctenophores	Fishes	Medusae
Encounter radius	a	$2 / \pi^{2}$ [z]	0.1 [v]	0.5 [w]	0.8 [w]	0.5 [w]
Swimming speed	b	0 [x]	0 [v]	0 [y]	0 [x]	1.2 [y]
	c	1.0 [x]	5.0 [v]	1.16×10^{-6} [y]	1.0 [x]	0.4 [y]
	d	1 [x]	1 [v]	1.22 [y]	1 [x]	1 [y]
Susceptibility	g	N.A.	-5.6 ± 0.4	-6.1 ± 0.8	-0.25 ± 1	-7 ± 4
	h	N.A.	-1.0 ± 0.2	-2.1 ± 0.4	-1.9 ± 0.6	-1.0 ± 0.2
	i	N.A.	0	0.5 ± 0.1	-0.4 ± 0.1	0

Note: [v]Bailey and Houde (1989); Lillelund and Lasker (1971); Greene (1986) and von Westernhagen et al. (1979). [w]Cowan et al. (1996). [x]Ware (1975). [y]Cowan and Houde (1992). [z]Bailey and Batty (1983).

Table 3.3: For the three simulation trials of this study, I give the variable which was manipulated and the output variables that I considered. A_p is the abundance of predators (predator $\cdot$ m^{-3}), M is the percent of the cohort consumed by the predator in 30 days, *SMI* is the size-selective mortality index, Z is the instantaneous mortality (d^{-1}), t is day, ln(S) is the probability of susceptibility to predation, and E is the encounter rate (d^{-1}).

Simulation trials	Variable manipulated	Output variables
Predator abundance	A_p (see Table 3.4) 3 levels: M= 10%, M= 50%, M= 90%.	1) Size of larvae consumed, 2) Larvae consumed each day, 3) Length frequency distribution of survival and growth cohorts. 4) *SMI* as a function of Z.
Timing of encounters	Time when predation can occur. 3 levels: t= 1 to 5, t= 13 to 17, t= 26 to 30.	Length frequency distribution of survival and growth cohorts.
Vulnerability components*	1) Vulnerability = encounter ln(S)= 0.5, A_p so that M=96%. 2) Vulnerability = susceptibility E so that M=96%. 3) Vulnerability = $E \times$ ln(S) A_p so that M= 96%.	Difference between the size-specific frequency of individuals of the growth cohort and of the survival cohort as a function of larval fish length.

Note: *For the vulnerability components trials, I used 100 000 individuals and sizes of predators were restricted to single classes.

Table 3.4: Total abundance of predators (predator $\cdot m^{-3}$) that consumed from 10 to 90 % of the initial cohort of 10 000 fish larvae in a 30 days simulation period. The size distribution of the predator population was characterised by a normal distribution with parameters given in Table 3.1.

Normal size distribution of predator population				
Mortality (M, %)	Crustaceans	Ctenophores	Medusae	Pelagic Fishes
10	115	45	16	0.0175
30	180	70	32	0.0184
50	250	145	48	0.0192
70	330	210	75	0.0204
90	480	365	150	0.0225

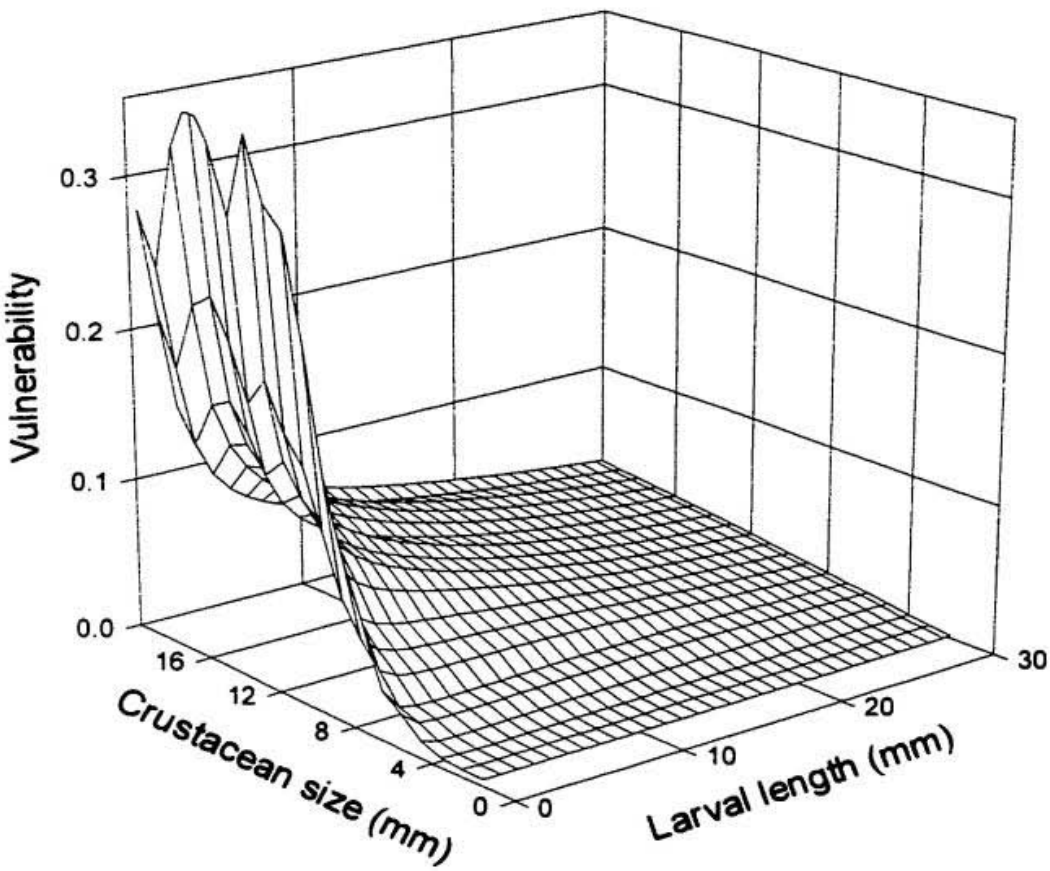

Figure 3.1: Vulnerability of fish larvae to predation by crustaceans as functions of both fish larvae length and crustaceans size. Vulnerability was calculated as the product of encounter probability (based on the formulation of Gerritsen and Strickler 1977) and susceptibility (Chapter II).

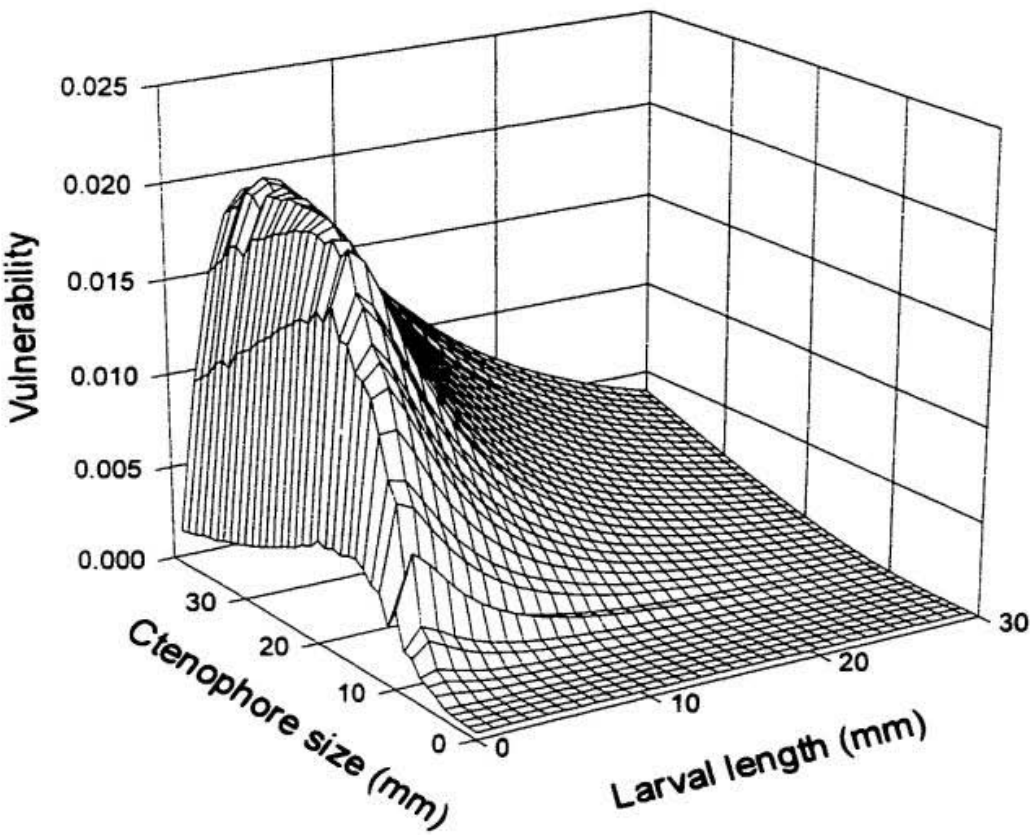

Figure 3.2: Vulnerability of fish larvae to predation by ctenophores as functions of both fish larvae length and ctenophores size. Vulnerability was calculated as the product of encounter probability (based on the formulation of Gerritsen and Strickler 1977) and susceptibility (Chapter II).

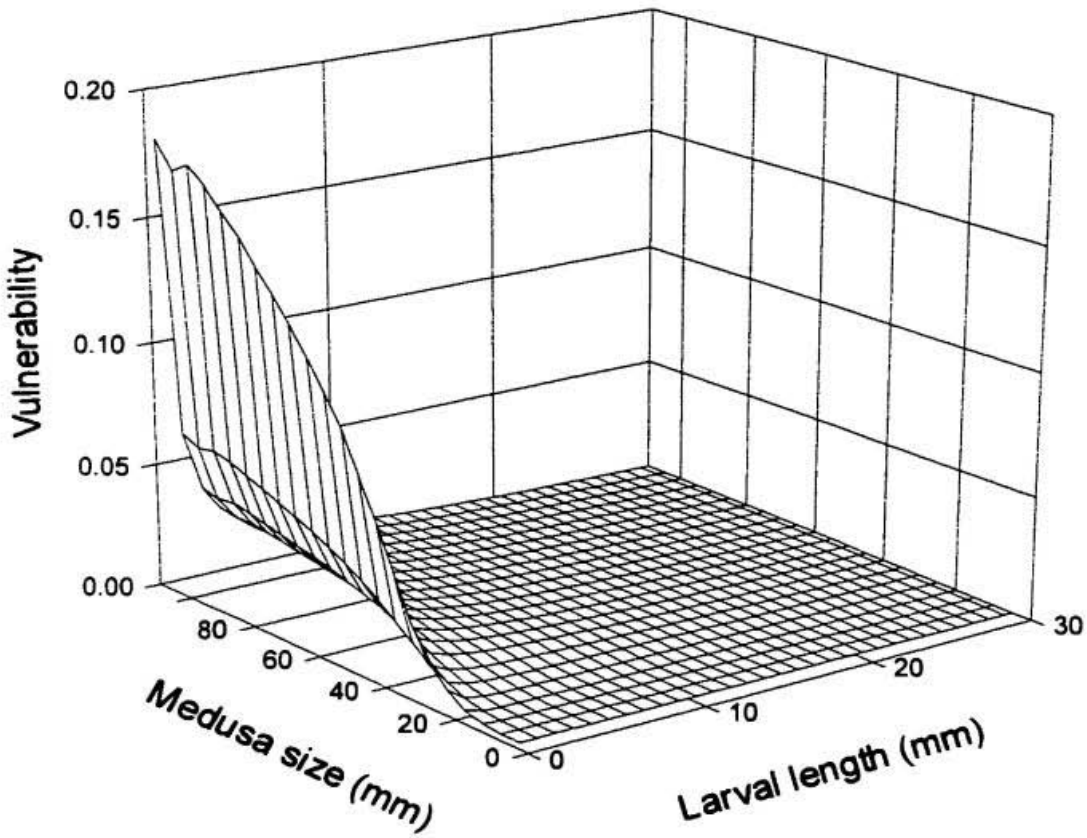

Figure 3.3: Vulnerability of fish larvae to predation by medusae as functions of both fish larvae length and medusae size. Vulnerability was calculated as the product of encounter probability (based on the formulation of Gerritsen and Strickler 1977) and susceptibility (Chapter II).

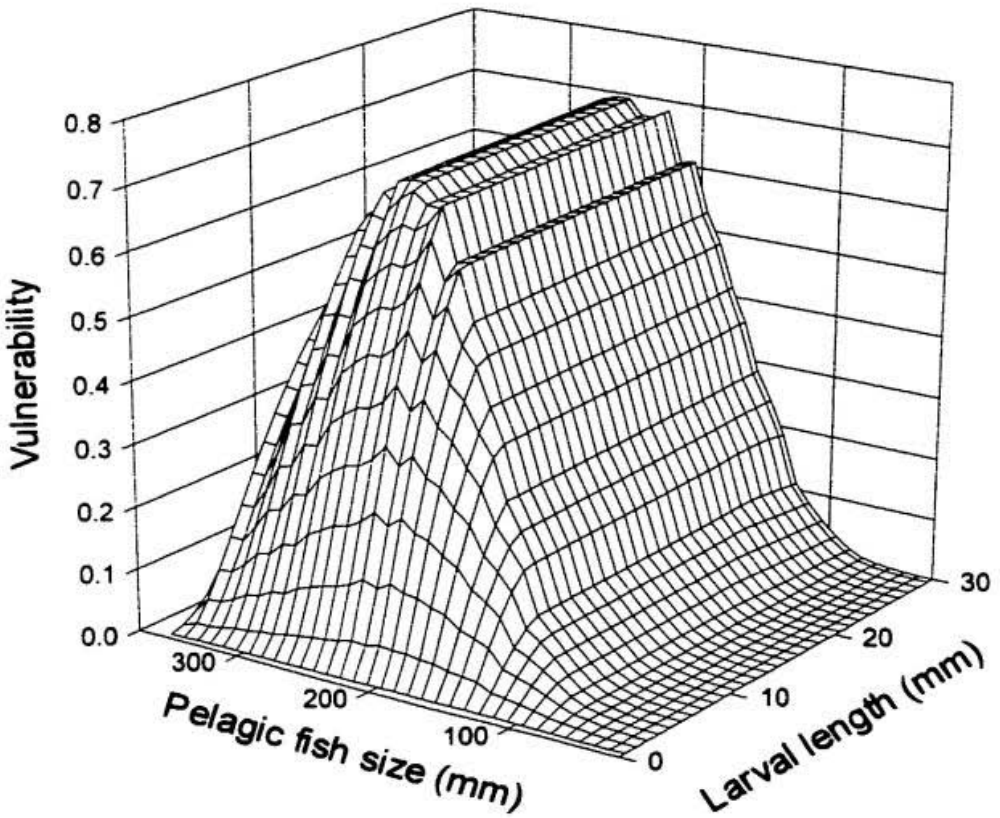

Figure 3.4: Vulnerability of fish larvae to predation by pelagic fishes as functions of both fish larvae length and size of pelagic fishes. Vulnerability was calculated as the product of encounter probability (based on the formulation of Gerritsen and Strickler 1977) and susceptibility (Chapter II).

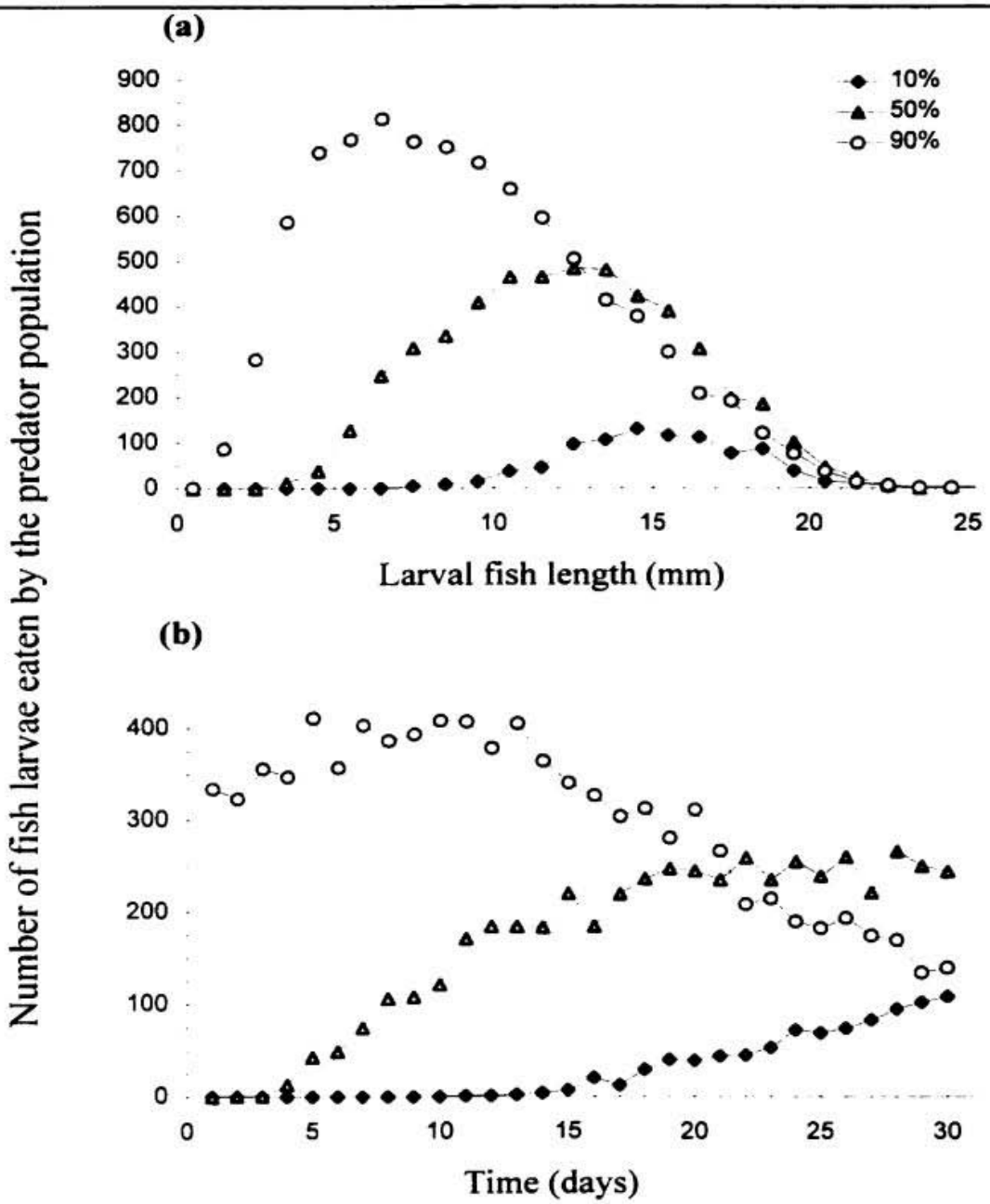

Figure 3.5: **(a)** The amount of fish larvae of specific length consumed by crustaceans and **(b)** the amount of fish larvae consumed every day by crustaceans. The abundance of crustaceans was arbitrarily chosen to yield a total mortality of 10%, 50%, and 90% of the initial cohort of 10 000 fish larvae over 30 days.

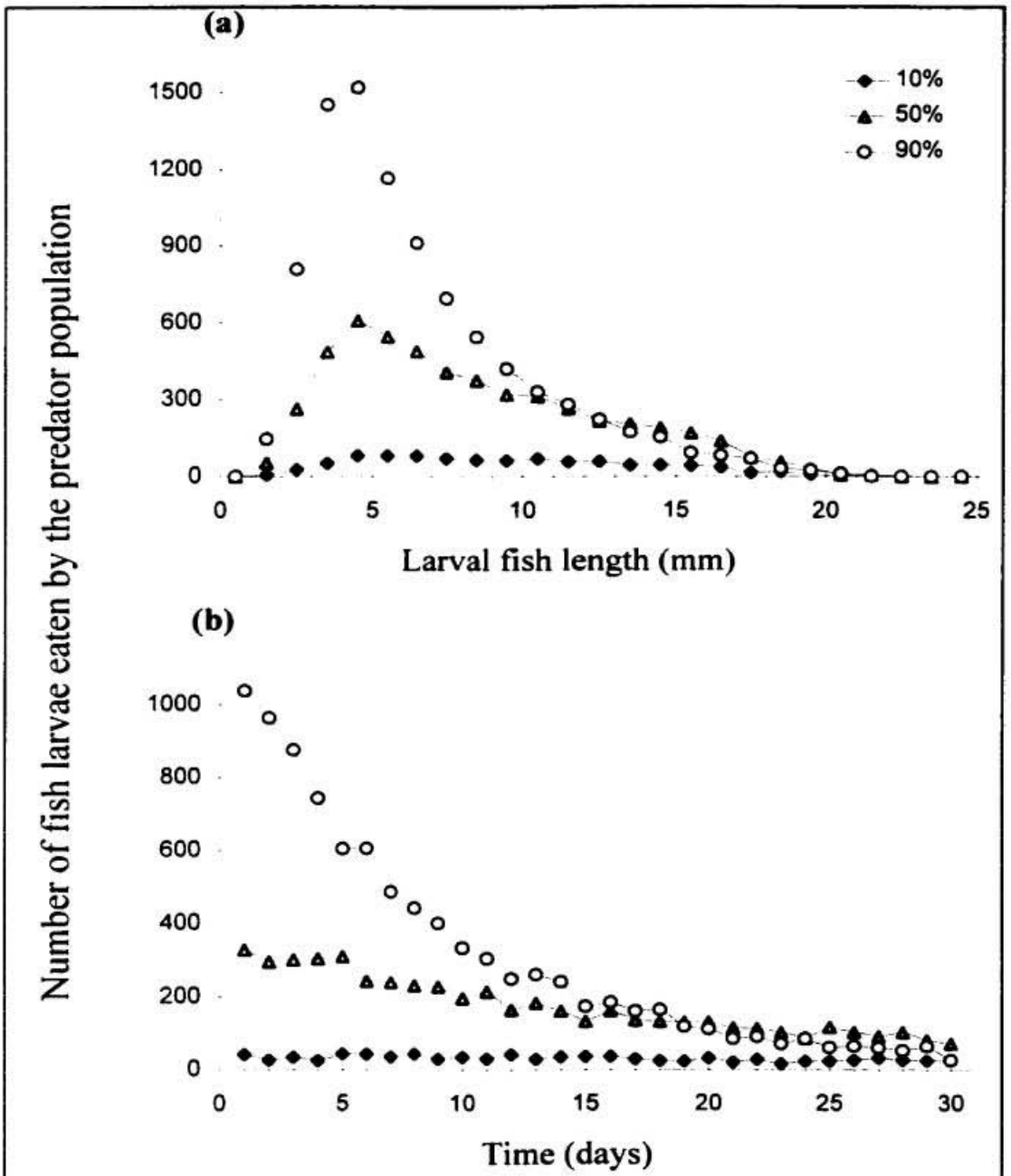

Figure 3.6: **(a)** The amount of fish larvae of specific length consumed by ctenophores and **(b)** the amount of fish larvae consumed every day by ctenophores. The abundance of ctenophores was arbitrarily chosen to yield a total mortality of 10%, 50%, and 90% of the initial cohort of 10 000 fish larvae over 30 days.

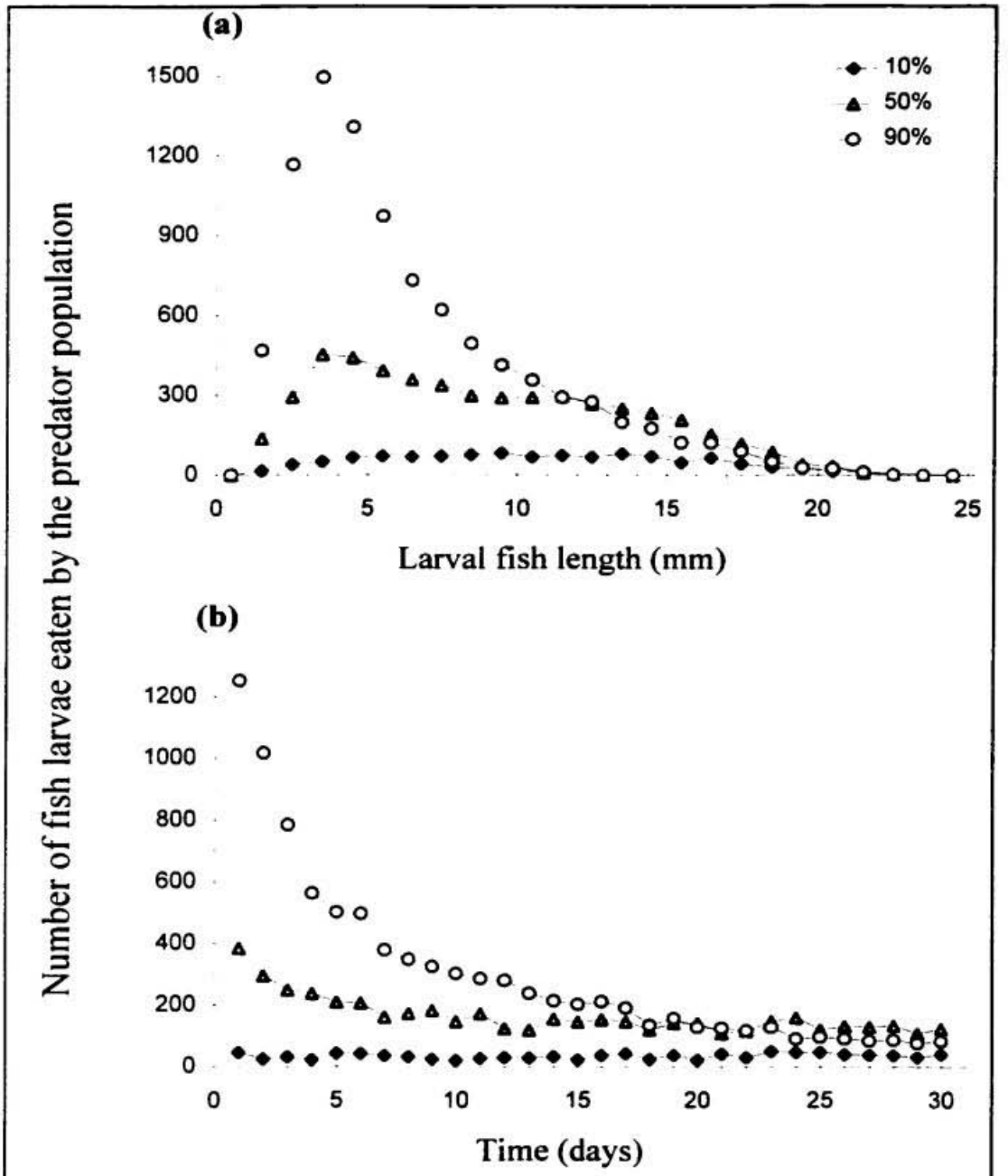

Figure 3.7: **(a)** The amount of fish larvae of specific length consumed by medusae and **(b)** the amount of fish larvae consumed every day by medusae. The abundance of medusae was arbitrarily chosen to yield a total mortality of 10%, 50%, and 90% of the initial cohort of 10 000 fish larvae over 30 days.

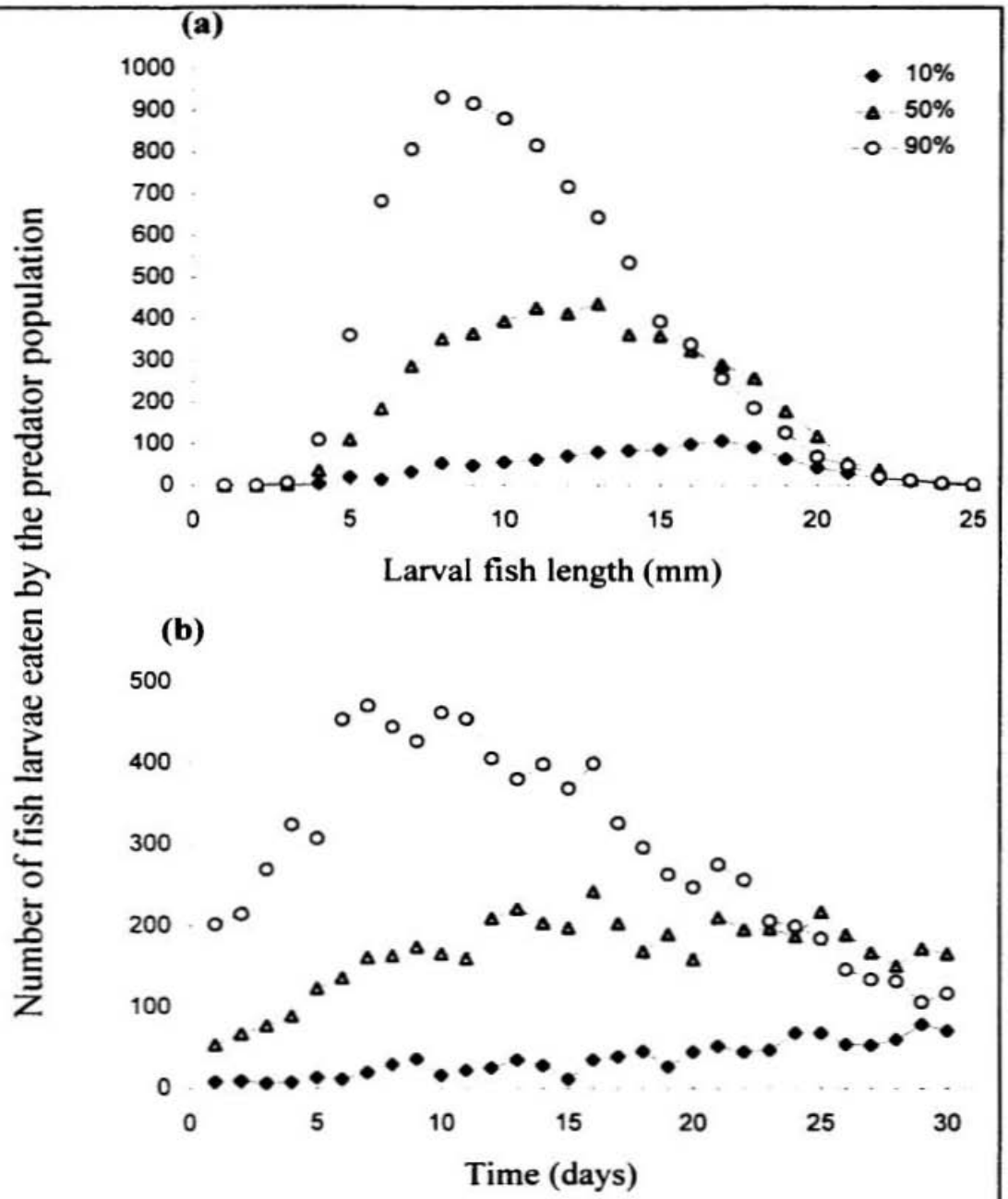

Figure 3.8: **(a)** The amount of fish larvae of specific length consumed by pelagic fishes and **(b)** the amount of fish larvae consumed every day by pelagic fishes. The abundance of pelagic fishes was arbitrarily chosen to yield a total mortality of 10%, 50%, and 90% of the initial cohort of 10 000 fish larvae over 30 days.

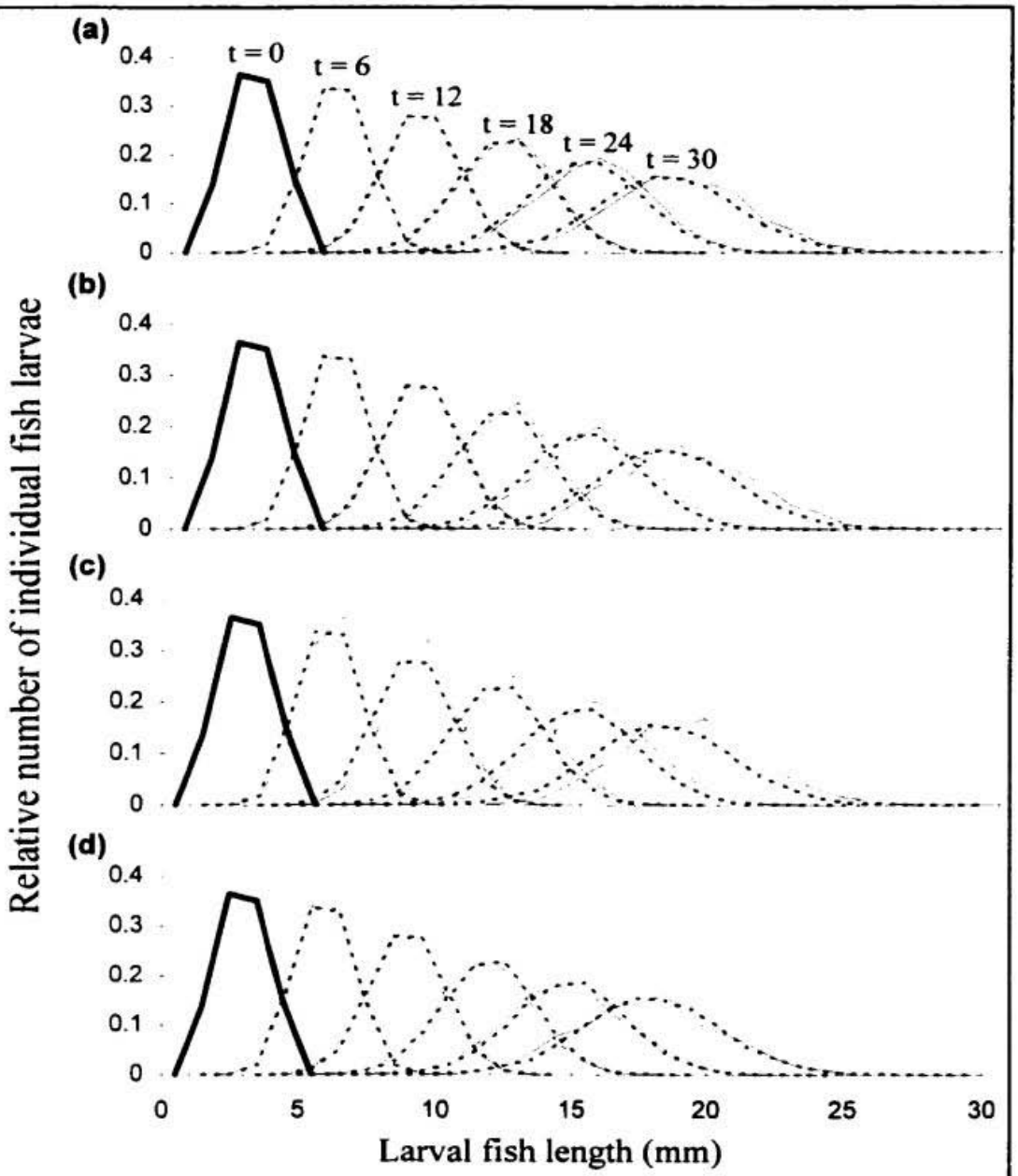

Figure 3.9: Relative length frequency distributions of the growth (no mortality, dotted lines) and survival (with predation mortality, solid lines) cohorts at time 0, and every sixth day until the end of the simulation run (day 30). The survival cohort is subjected to predation by either **(a)** crustaceans, **(b)** ctenophores, **(c)** medusae, or **(d)** pelagic fishes. The abundance of predators was arbitrarily chosen to yield mortality of 90% of the initial cohort of 10 000 individuals in 30 days.

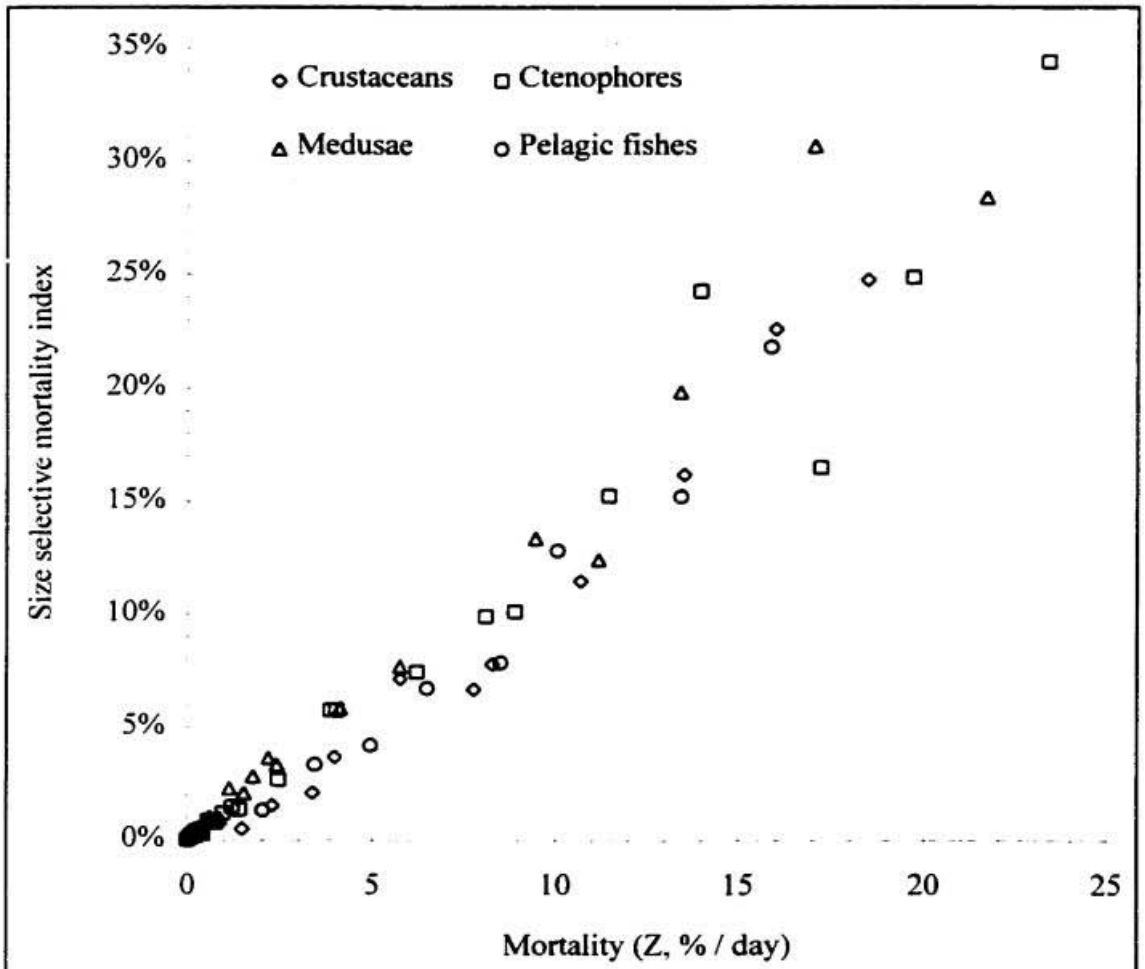

Figure 3.10: Size-selective mortality index measured as the maximum difference between the relative cumulative length frequency distributions of the growth (no mortality) and the survival (with predation) cohorts at the end of the 30 days simulation as a function of the instantaneous mortality rate (Z, % /d).

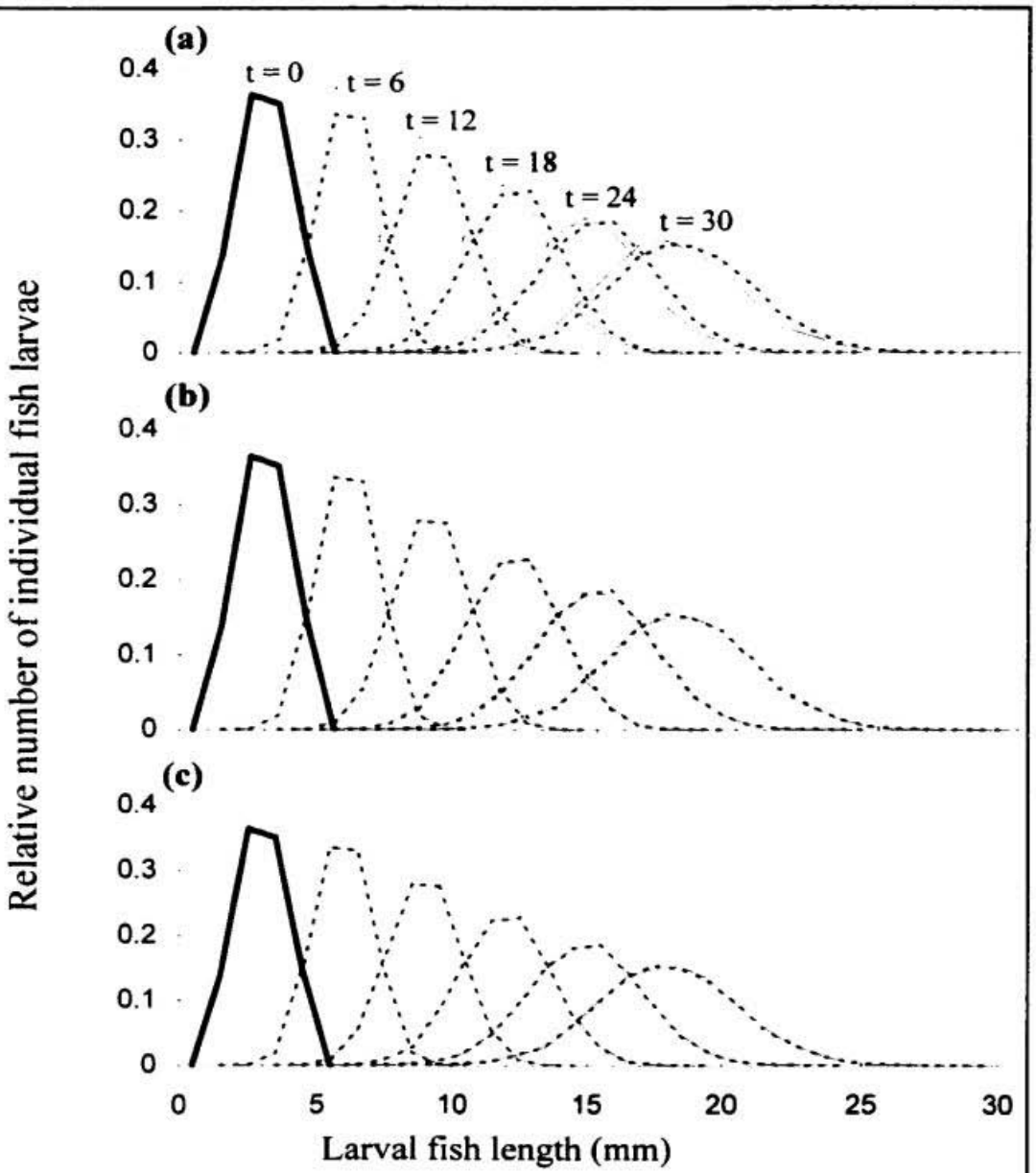

Figure 3.11: Relative length frequency distributions of the growth (no mortality, dotted lines) and survival (with predation mortality, solid lines) cohorts at time 0, and every sixth day until the end of the simulation run (day 30). Predation by pelagic fishes was restricted to five days either **(a)** early (days 1 to 5), **(b)** mid (days 13 to 17), or **(c)** late (days 26 to 30) in the simulation period. Abundance of pelagic fishes was identical to the abundance of pelagic fishes that yield mortality of 50% of the initial cohort of 10 000 individuals when predation was not restricted.

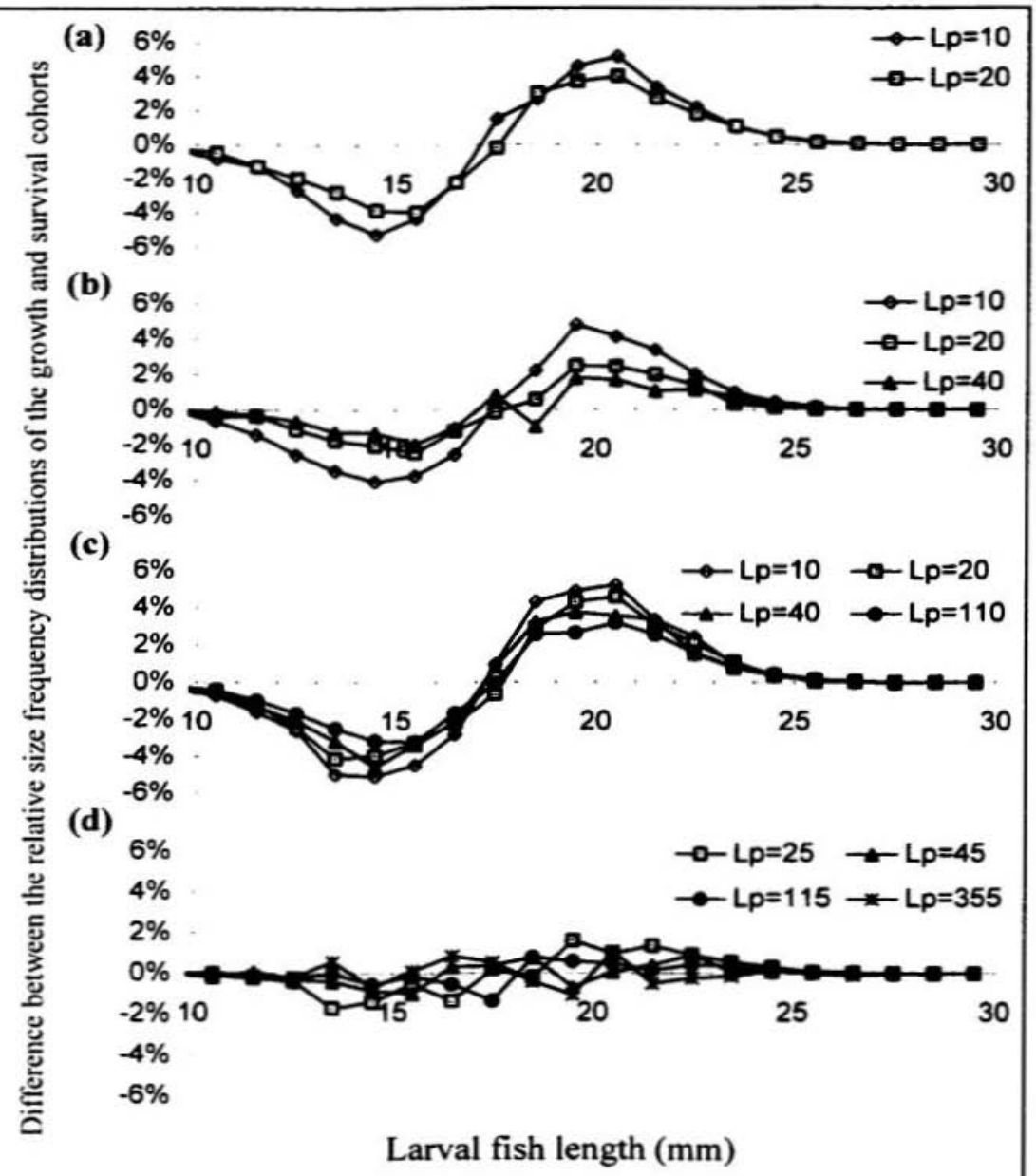

Figure 3.12: The difference between the relative length frequency distributions of the growth (no mortality) and the survival (with predation) cohorts at the end of the 30 days simulation. Vulnerability due to predation by **(a)** crustaceans, **(b)** ctenophores, **(c)** medusae, and **(d)** pelagic fishes is dependent only on encounter because susceptibility is constant. For each simulations, the predator population is composed of a single size class (Lp, mm). Abundance of predators was set to yield mortality of 96% of the initial cohort of 100 000 individuals.

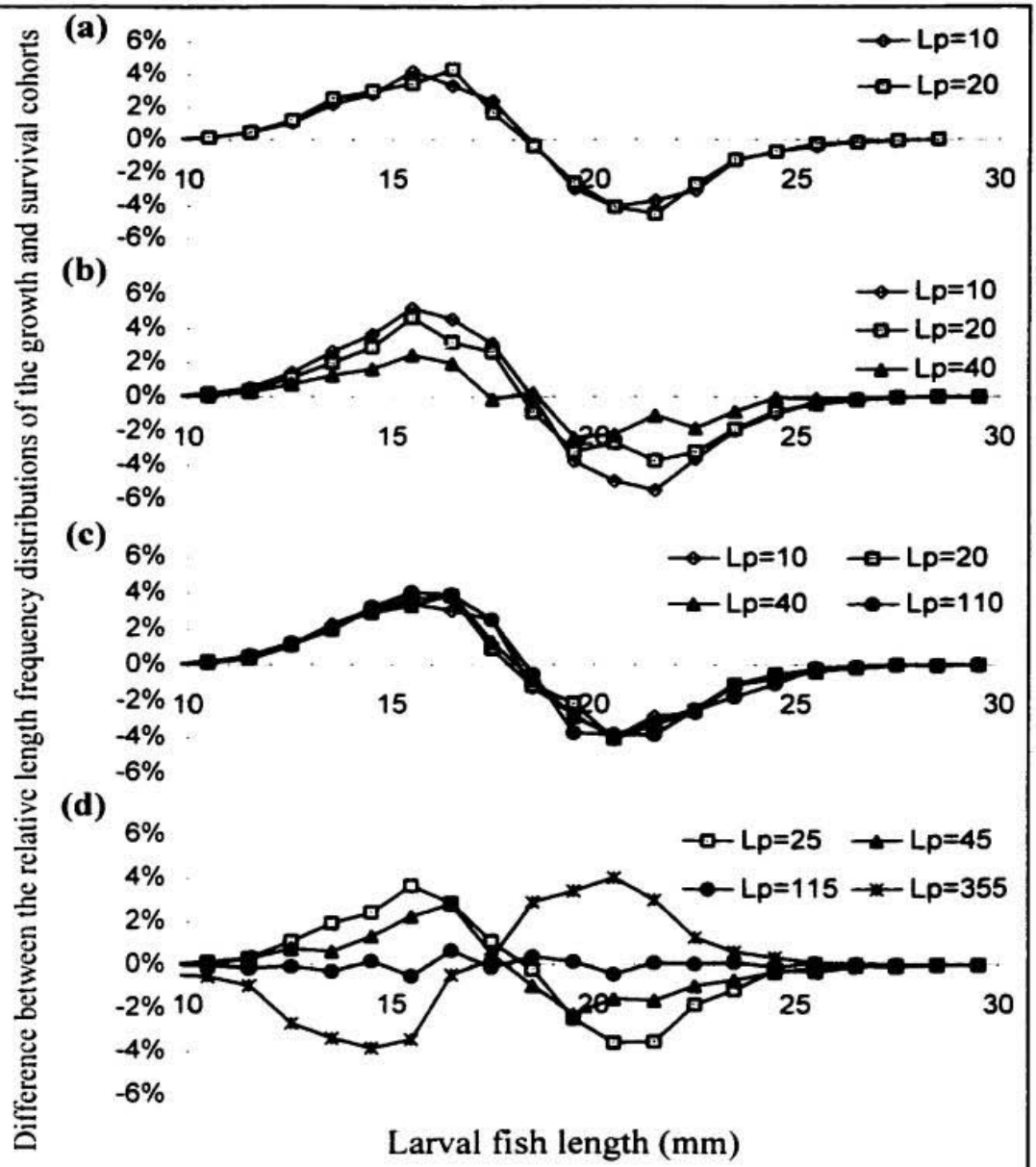

Figure 3.13: The difference between the relative length frequency distributions of the growth (no mortality) and the survival (with predation) cohorts at the end of the 30 days simulation. Vulnerability due to predation by **(a)** crustaceans, **(b)** ctenophores, **(c)** pelagic fishes, and **(d)** medusae is dependent only on susceptibility because encounter rate is constant and set to yield mortality of 96% of the initial cohort of 100 000 individuals. For each simulations, the predator population is composed of a single size class (Lp, mm).

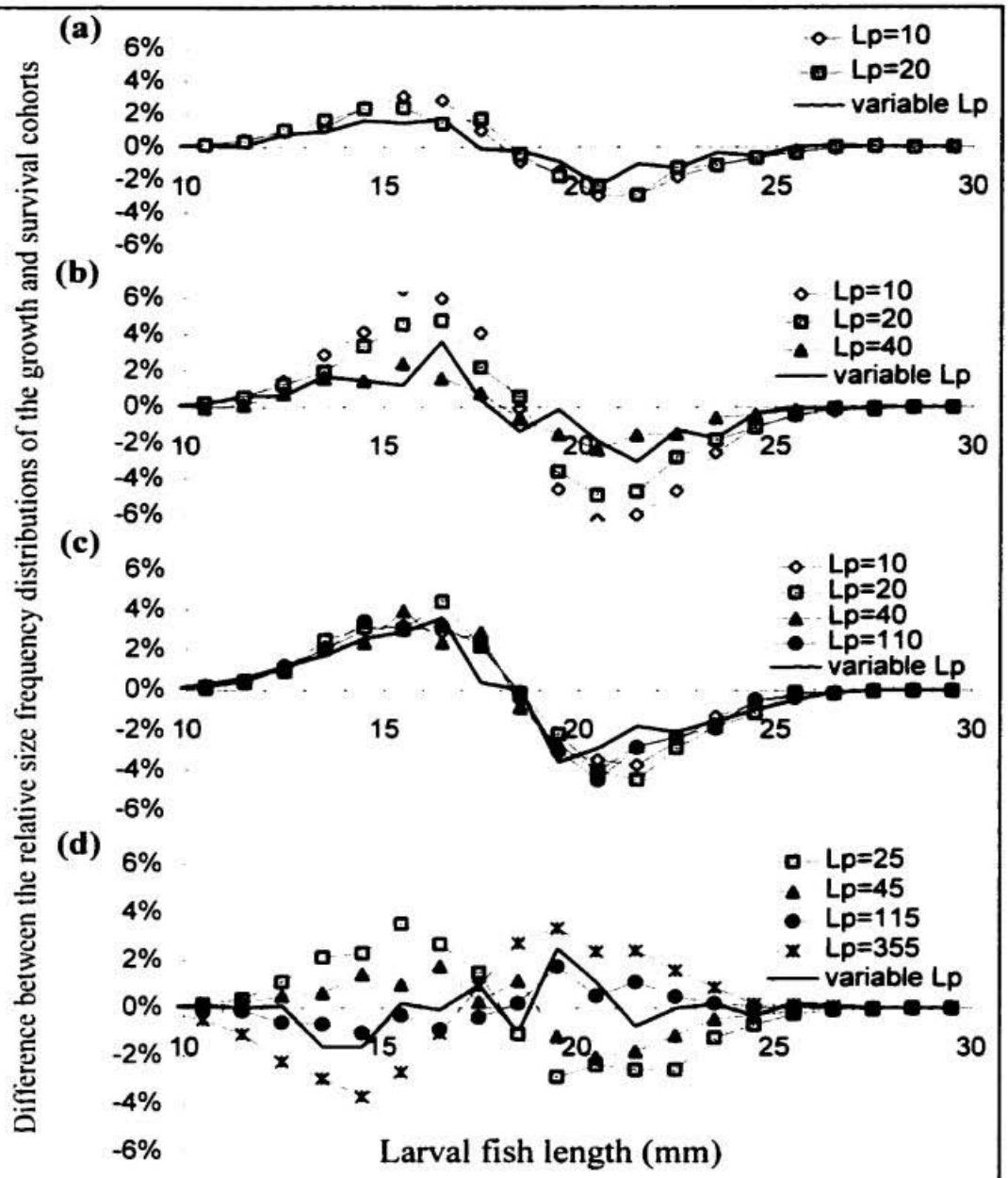

Figure 3.14: The difference between the relative length frequency distributions of the growth (no mortality) and the survival (with predation) cohorts at the end of the 30 days simulation. Vulnerability due to predation by **(a)** crustaceans, **(b)** ctenophores, **(c)** pelagic fishes, and **(d)** medusae is dependent on encounter and susceptibility but independent of predator size because the predator population is composed of a single size class (Lp, mm). However, for the four predator types, I relaxed the restriction of single predator size class (full lines, no symbols).

Chapter IV

Investigations of the effects of larval initial size and growth rate with an individual-based model of the size-selective vulnerability of fish larvae

Abstract

In Chapter III, I found that encounter rate (calculated from Gerritsen and Strickler 1977) counteracted the dome-shaped function of susceptibility (derived from Chapter II). The balance between these two processes was affected by the overall mortality rate (or abundance of predators) as well as the size characteristics of the predator population. It is important to assess how the larval characteristics may affect their survival probability and the possible interaction of this effect with predator size. In this chapter, I evaluated the extent to which mean and variation in growth rates affect the survival, length and growth rate frequency distributions of fish larvae. I also evaluated the effect of initial larval length distribution on mortality and size characteristics of fish larvae consumed by predators. There was no more than a 0.1% difference in mortality given different initial larval length distributions. The size of fish larvae consumed by invertebrate or vertebrate predators was only slightly smaller given that the initial larval length distribution was constant. The effect of mean or variance in larval growth rates on the survival rates of fish larvae and characteristics of survivors was highly dependent on predator length. I found that the pattern of size-selective mortality was reversed given predators of different sizes. This study illustrates the concept that any specific larval size or growth rate may not be a universal survival advantage.

Introduction

In previous individual-based models, the relationships for either attack or capture were derived from restricted laboratory work and species-specific observations (e.g., Cowan et al. 1996, 1997; Rice et al. 1993a,b, 1997). In Chapter II, I found empirical evidence for a general dome-shaped function with maximum predation rates when fish larvae measure 10% of the length of a predator. In Chapter III, I found that encounter rate (calculated from Gerritsen and Strickler 1977) counteracts with the dome-shaped function of susceptibility (derived from Chapter II). The balance between these two processes is affected by the overall mortality rate (or abundance of predators) as well as the size characteristics of the predator population. At this point, it is important to assess how the larval characteristics may affect their survival probability as this will also allow me to compare the model's results and predictions with another previously published individual-based simulation model (i.e., Rice et al. 1993a).

Among others, Rice et al. (1993a) investigated the effect of larval characteristics in determining their survival probability. They found that mean growth rate and variation in growth rates among individuals can interact strongly with size-dependent mortality to cause significant effects on the number, growth rates and final sizes of survivors as previously shown by Pepin (1989). Rice et al. (1993b) observed experimentally that the relative survival of fish larvae can differ substantially for cohorts of individuals differing in size. They explained that the direction of that difference can be completely reversed by a change in predator size structure (Rice et al. 1993b). In addition, Rice et al. (1997) have

demonstrated with variable size structure for both growing prey and growing predators, that the nature of the predator-prey interaction shifts as the relative sizes of predator and prey changes over time. They observed experimentally that the profitability of small prey was highest early in the experiment and by the end of the experiment, peak predation had shifted to medium-sized prey, countering the effect of selection that had occurred earlier (see also Crowder et al. 1994). This further suggests, as I found in Chapter III, that survival as well as characteristics of larval fish survivors will depend greatly upon the relative sizes of prey and predator. Thus, I should find different characteristics of surviving larvae given differences in the size characteristics of the predator population. It may be important to determine the interaction between size and growth rates of larvae with the size characteristics of the predator population. It is unknown yet if the effect of mean and variation of larval growth rates on the survival and characteristics of survivors will remain the same given differences in predator sizes. It is this interaction that I propose to investigate in this chapter.

I chose to concentrate the comparisons between this study and Rice et al.'s (1993a) work because although Cowan et al. (1996) also investigated the impacts of growth rate and initial length of fish larvae, it will be difficult to make the appropriate changes to this individual-based model to allow adequate comparisons with their results. Cowan et al. (1996) compared mean length of survivors with the mean length of fish larvae consumed by predators. With an individual-based model, it is inadequate to characterise a cohort by averaging across all individuals of the population because

population processes occur at the individual level and the variability of characteristics of individuals is highly important in regulating population processes (DeAngelis and Gross 1992).

In individual-based modeling, it may be important to incorporate initially variation in larval sizes because capturing the variability of individuals is the key to successful modeling. This may be particularly crucial if the initial variation in larval size explains the characteristics of the surviving cohort of fish larvae. In contrast, Rice et al. (1993a, 1993b) started their simulations with no variation in the initial size of larvae. The length at hatch represented the average for the species modeled.

In this chapter, I evaluated the extent to which mean and variation in growth rates affect the survival, length and growth rate frequency distributions of surviving fish larvae. I achieved this by setting the initial model conditions to the same initial conditions as Rice et al. (1993a) (Table 4.1). My intention was to evaluate how our different approaches are important when making inferences and extrapolating results from an individual-based model to general theoretical concepts. Given that our models were conceptually different, could I arrive at the same conclusions? In addition, I investigated the effect of initial larval size variation on the size of fish larvae consumed and on the timing when most mortality of individuals occurred.

Methods

Initialisation of the model

This study was designed to simulate the growth and the mortality due to predation of fish larvae in a theoretical marine system for 30 days. The initial population of fish larvae was set to 10 000 individuals and all larvae started at a length of 2 mm. Given the different distributions of larval growth rates which were identical to Rice et al. (1993a), the length frequency distribution ranged between 2.0 and 30.0 mm after 30 days if there was no mortality (Table 4.1). The predator population was composed of pelagic fishes of pre-determined lengths (Table 4.1). A predator of 25 mm in this study replicated the size ratio used by Rice et al. (1993a) whereas a predator of 90 mm replicated the predator length used in Rice et al.'s (1993a) model. In addition, I also ran a simulation with much larger predators (355 mm) as well as a population of predators with variable sizes ranging from 25 mm to 355 mm. Predators were not allowed to grow during the simulation period. The abundance of predators was chosen to yield mortality levels of about 96% of the initial cohort in 30 days (identical mortality levels to Rice et al. 1993a).

This study used the same individual-based model described in Chapter III. I present here only the changes made to the model.

Growth sub-model

Each larva was assigned a growth rate drawn at random from a normal distribution with a specified mean (μ, $mm \cdot d^{-1}$) and standard deviation (SD). Each larva

maintained their assigned growth rate throughout the entire simulation period. At the end of each day, all surviving individuals grew to a new length determined by their growth rate and were exposed to predation again on the next day.

Design of simulation hypotheses

To determine the effect of the initial length distribution on the mortality and size characteristics of fish larvae consumed by predators, I used two types of distribution. The "constant" distribution was composed of larvae of equal length, and the "variable" distribution was based on a normal distribution of larval lengths. The normal length distribution had a mean of 2.0 mm and a standard deviation of 0.1 mm. I used the constant distribution as the baseline and selected a predator abundance that generated between 95% and 98% mortality of the 10 000 individuals in 30 days (Z= $0.1 \cdot d^{-1}$, and $0.13 \cdot d^{-1}$, respectively). I maintained the same predator abundance and size distribution (see Table 4.1) and repeated the simulation with the "variable" distribution. I compared the number of fish larvae consumed by the different predator types, the size of fish larvae consumed and the time when they were consumed given the two different initial larval length distributions. The growth rate distributions of both cohorts were set to a mean of $0.4 \text{ mm} \cdot d^{-1}$ and a standard deviation of $0.08 \text{ mm} \cdot d^{-1}$.

To determine the effect of mean growth rates on the survival, size and growth rate distributions of survivors, I varied the mean of the growth rate distribution as slow (μ=0.2), intermediate (μ=0.4), and fast growth (μ=0.6) with standard deviation of

$0.08\ mm \cdot d^{-1}$. In addition, I also investigated the effect of variation about the mean growth rate by setting the mean to $0.4\ mm \cdot d^{-1}$ and the standard deviation as low (SD=0.04), intermediate (SD=0.08) and high variability in growth rates (SD=0.16). I used μ=$0.4\ mm \cdot d^{-1}$ and SD=$0.08\ mm \cdot d^{-1}$ as the baseline and selected an abundance of predatory fishes that generated about 96% mortality of the initial 10 000 individuals in 30 days. This mortality level ($Z \cong 0.1\ d^{-1}$) was identical to Rice et al. (1993a) at the same baseline conditions. I then kept the same predator abundance and size distribution (as described in Table 4.1) and repeated the simulation with either faster or slower growing cohorts and with either high or low variability in growth rates. I compared the length of fish larvae consumed by predatory fishes of different sizes and the time when fish larvae were consumed given the different larval growth regimes. I evaluated the extent to which the length frequency distribution of the surviving fish larvae differed from a larval cohort where there was no predation pressure. I also compared the growth rate distribution of the initial cohort with the growth rate distribution of the survivors to evaluate whether fish larvae of particular growth rates survived better than others.

To determine the parameters that may yield the most change in the survival of fish larvae, I varied the parameter estimates of the encounter radius (equation 3.2) and swimming speed (equation 3.3) functions of predators, as well as the parameter estimates of the susceptibility functions of different predator types. I first determined the abundance of predators required to consume 50% of the initial cohort of fish larvae in 30 days given the original parameter values of the above functions. Then, I varied the parameters of the

encounter radius function by ±10% of the original value and compared in each case the amount of fish larvae consumed given that originally 50% of the cohort was consumed in 30 days. The identical procedure was followed for the parameters of the susceptibility functions. I varied the slope estimates or the first and second order terms of the susceptibility functions (equation 3.5) within the 95% confidence intervals calculated in Chapter II. Variations in the first order term will change the position of the peak whereas variations in the second order term will change the slope of the curve.

I have presented the results as length frequency distributions of the survival cohort (with predation) and of the growth cohort (without predation) as did Rice et al. (1993a). I also computed the difference between the relative frequency of these two cohorts for each size classes. The maximum absolute difference was taken as an index of size-selective mortality (as described in Chapter III). I determined the characteristics (size and time of death) of the cohort consumed by the predators to explain some of the mechanisms behind the size-selective mortality of individuals, which permitted some discussion of the results presented in Cowan et al.'s (1996) simulation study.

Results

Effect of the initial larval length distributions

There was no difference in survival between the "variable" and the "constant" distributions (Table 4.2). The percent difference in mortality given the two types of initial length distribution was no more than 0.1%. I also found very little difference in

the length of fish larvae consumed by vertebrate predators whereas fish larvae consumed by invertebrate predators were smaller when initially all fish larvae were of equal length (Figure 4.1). Fifty percent of the mortality due to invertebrate predation occurred within the first eight days whereas 50% of the mortality due to vertebrate predation occurred within days 12 and 11 for the "constant" and the "variable" distributions, respectively (Figure 4.2). Most of the percent difference in the overall mortality of the cohorts to vertebrate predation occurred during the first five days of the simulation where the cohort of "variable" length distribution experience higher mortality levels than the cohort of "constant" length distribution (Figure 4.2).

Effect of mean and variability of larval growth rates

I found a strong interaction between the mean and variance of the larval growth rate distribution and predator length. The effect of mean or variance in larval growth rates on the survival rates of fish larvae was highly dependent on predator length (Table 4.3). A change in mean growth rate yielded a 3% to 80% difference in larval survival whereas, different variances of growth rates yielded a 6% to 400% difference in larval survival (Table 4.3). The difference in larval survival due to either a change in mean growth rates or a change in variance of growth rates tended to be much greater for a predator population of larger fishes than of smaller fishes (Table 4.3). These results indicate clearly a strong interaction between predator length and larval growth rate distribution on the survival of a cohort of fish larvae. An increase in mean larval growth

rate lead to an increase in larval survival rate to small predatory fishes but to a decrease in larval survival rate to large predatory fishes (Table 4.3). An increase in variance of larval growth rate lead to an increase in larval survival in the case of the largest predatory fishes (Table 4.3).

Changes in the mean and variance of growth rates had substantial effects on the length frequency distribution (Figures 4.3, 4.4, respectively) and the growth rate frequency distribution of survivors (Figures 4.5, 4.6, respectively). I found that predator length would not only determine the size of survivors but that the effect of mean larval growth rate on the length and growth rate frequency distributions of survivors may be reversed given different sizes of predators.

Predation by small fishes: Survivors were larger than expected by growth rates alone if predators were small (Figures 4.3, 4.4). These survivors also had faster growth rates than the larval cohort without predation mortality (Figures 4.5, 4.6). For a slow growing cohort, the pattern of size-selection was not as strong or clearly defined as for an intermediate or fast growing cohort (Figure 4.7). The pattern of size-selection was stronger and clearly defined for a cohort of intermediate variable growth rates (Figure 4.8). Fish larvae measuring less than 10 mm were most vulnerable to predation by small predatory fishes because more individuals of those size classes were consumed by the small predators (Figures 4.9, 4.10). In a slow growing cohort, predators had more time to consume fish larvae of that size class (Figure 4.11). As the mean growth of a cohort increased, small predators had less time to consume fish larvae of that size

class. Most of mortality occurred during the first 10 days for fast growing cohorts as compared to 15 days for intermediate growing cohorts or 20 days for slow growing cohorts (Figure 4.11).

Predation by large fishes: Survivors were smaller than expected by growth rates alone if predators were larger (Figures 4.3, 4.4) and had significantly lower growth rates (Figures 4.5, 4.6). The magnitude of size-selective mortality by large predators was much greater for fast growing cohorts than for slow growing cohorts but the pattern of size-selective mortality was stronger for slow growing cohorts than for fast growing cohorts (Figure 4.7). An increase in variance of growth rates amplified the evidence of size-selective mortality even though the magnitude of size-selective mortality remained the same at ±6% (Figure 4.8). Fish larvae consumed by large predators measured on average 7 mm (Figures 4.9, 4.10). The length distribution of consumed fish larvae became increasingly skewed towards larger prey sizes with increasing mean growth rate of the cohort (Figures 4.9). The time period when most of the mortality occurred was much shorter for the fast growing cohort than for the slower growing cohort regardless of the predator size (Figure 4.11).

Sensitivity analysis of parameter estimates

In all cases except crustaceans, survival of fish larvae was more sensitive to changes to the parameter of encounter radius than to changes to the parameters of swimming speed (Table 4.4). Variation in the slope of the susceptibility function for

crustaceans or medusae yielded 2% and 30% difference in survival, respectively (Table 4.4). If the peak of the susceptibility function of ctenophores or pelagic fishes was shifted toward smaller sizes, there was a decrease in survival whereas if the peak was shifted toward larger sizes of fish larvae, survival increased (Table 4.4). If the susceptibility curve was narrower around the peak (much steeper slopes), survival increased and if the peak was flatter, survival was lower. This occurred because if maximum susceptibility occurs for a wider range of sizes (flatter peak), then more fish will be consumed by the predator population and therefore, survival will be lower.

Discussion

Early life characteristics of fishes such as size and growth rate of individuals have important implications for survival to predation by fishes. The survival advantage of an individual of a specific size and growth rate will depend on the size characteristics of the predator population. In Chapter III, I found that larger larvae were more vulnerable to predation by larger fishes whereas smaller larvae are more vulnerable to predation by smaller fishes. In this study, I found that the effect of mean and variance of larval growth rates also varied according to the predator size characteristics. If Rice et al. (1993a) had chosen a different size ratio (prey length to predator length), or a larger predator size, they likely would have found different effects of growth rate distribution on the survival rate and on the characteristics of survivors. This is not to

say that their model and experiments were wrong but their conclusions are certainly restricted to the system they work with: alewife predation on bloater. From a more conceptual perspective, I found that the effect of larval growth rate was specific to the size characteristics of the predator population. We might expect then that the effect of larval growth rate may be specific to the modeled system.

Another well-modeled system is Chesapeake Bay where bay anchovy, *Anchoa mitchilli*, are consumed by a suite of predators: ctenophores, scyphomedusae and planktivorous fishes (Cowan and Houde 1990, 1992, 1993; Cowan et al. 1996, 1997). Cowan et al. (1996) found that size-selective predation by planktivorous fishes (measuring between 25 and 45 mm) was less evident for the slower growing cohort. An increase in larval mean growth rate from 0.3 to 0.5 $mm \cdot d^{-1}$ caused significant increases in size selection, survival and mean length of survivors on day 20 (Cowan et al. 1996). Their evidence for increased size selection was a greater difference between the mean length of the larvae that died on the last day with the mean length of those that were alive on the last day, which was much higher for fast growing fish larvae (Cowan et al. 1996). This may not be due to the differential encounter rates, as proposed by Cowan et al. (1996), but due to the length of fish larvae most vulnerable to predation by small fishes. In this study, the mean length of fish larvae consumed by small fishes throughout the 30 day simulation, ranges from 4 to 7 mm given different growth rates (Figures 4.9, 4.10). By characterizing the individual distribution, I demonstrated that

differential growth rates lead to different shapes of length distribution of consumed fish larvae (Figures 4.9, 4.10) but little change in the mean length of fish larvae consumed by those predators. An increasing final mean length ratio is driven by the growth rate of the cohort because a faster growing cohort will have larger individuals at the end of a simulation period whereas a slower growing cohort will have smaller individuals which would lead to a smaller final length ratio. Lastly, the final length ratio is probably not a good representation of size selection occurring in the field because the field techniques to determine the mean length of larvae that died on a particular day are indirect and prone to several compounded errors (e.g., stomach content analysis or estimation of missing larvae based on otolith reconstruction). In contrast, it is possible to assess the pattern as well as the magnitude of size selection with the size-specific difference in length frequency distribution between the cohort surviving predation and the same cohort with no predation. Nevertheless, this study reached the same conclusions as Cowan et al. (1996) regarding increasing mean larval growth rates on the size selection pattern. An increase in larval mean growth rates will lead to a decreasing size selection for any population of predatory fishes (small, large or of variable sizes). Cowan et al. (1996) found that an increase in variance of larval growth rates leads to a decrease in size selection. I found that this was the case for small predatory fishes but for larger predators or for a population of predators of variable sizes, an increase in variance of larval growth rates lead to an increase in size selection.

Furthermore, as the time period of high mortality decreased, the evidence of size-selective mortality was stronger. This was also demonstrated in Chapter III when I restricted the time when pelagic fishes could consume fish larvae. I found stronger evidence of size-selective mortality when mortality was restricted during the first seven days of the simulation trials. I have demonstrated here that a restriction on the time of high mortality from small predatory fishes can be achieved by manipulating the mean growth rate of a cohort of fish larvae.

This study implies that we can assess the importance of predation mortality on the survival rate and the characteristics of surviving fish larvae only if we gather information about the predator population as well as individual larval growth rates. The actual effect of different means or variances in growth rates will be affected by the characteristics of the predator population. It is particularly evident if we consider that for a population of predatory fishes of variable sizes, only small changes in the parameters of the encounter model and susceptibility function may lead to large differences in survival of the larval cohort (Table 4.4).

Houde (1987), Beyer (1989) and Pepin (1989 and 1991) stated that rapid larval growth may reduce the impact of a specific predator type on larval fish mortality by reducing the time spent within a high vulnerability window. However, as I suggested earlier (see Chapter II) and as did Leggett and DeBlois (1994), this is not necessarily

beneficial as an individual may pass from one predator's niche to another's. This study illustrated this concept. Any specific size or growth rate may not be a universal survival advantage. Given that a cohort of fish larvae will most likely encounter different predator populations, any individual size or growth rate may not be beneficial at any one time. A cohort of variable growth rates may experience an overall higher survival as the actual predator encountered on any given day may not be easily predicted. If predators are size-specific in terms of consuming individual fish larvae as observed in laboratory experiments (see Chapter II), then during a random encounter of a predator, some individuals of a highly variable growing cohort will be relatively less vulnerable to that predator. Size-selective removal of individuals may best be detected from length frequency distributions of such a cohort.

Table 4.1: Initial conditions (larval fish length (L_f) and number (n)) of the simulation of Rice et al. (1993a) and this study. I ran various simulations with different predators sizes. A predator of 25 mm in these simulation trials compared with Rice et al.'s (1993a) simulation because it replicated the same ratio of prey length to predator length (L_p) whereas 90 mm replicated the same predator length as they used in their study. In addition, I also ran a simulation with much larger predators as well as a population of predators with variable sizes ranging from 25 mm to 355 mm with a specified mean (μ) and standard deviation (SD).

	Rice et al. 1993a	This study
Initial cohort:	L_f = 12 mm	L_f = 2 mm
	n= 4000 or 10 000	n= 10 000
Final cohort:	L_f = [10, 74] mm	L_f = [2.003, 30.0] mm
Duration of trial:	60 days	30 days
Predator population:	L_p = 90 mm.	L_p = 25mm or 90mm or 355mm, or Variable L_p: μ= 185mm, SD= 80

Table 4.2: Number of fish larvae consumed by four different predators given two different initial length frequency distributions of fish larvae: "variable" is based on a normal distribution, and "constant" where all larvae initially have the same length. In all simulations presented here, there were initially 10 000 fish larvae and the predator abundance was set so that approximately 95 to 98% of the cohort of "constant" length distribution was consumed in 30 days. The mean growth rates was set to 0.4 $mm \cdot d^{-1}$ and standard deviation to 0.08 $mm \cdot d^{-1}$.

	Predators			
Length distributions	Crustaceans	Ctenophores	Medusae	Fishes
Constant	9723	9522	9778	9674
Variable	9735	9523	9774	9671
Difference	12	1	4	3

Table 4.3: Percent survival of a cohort of larval fish from Rice et al. (1993a, Table 4.1) as well as the percent survival of a cohort of larval fish from this study given different lengths of the predator population. The variable length refers to the population of predators with mean length of 185 mm and standard deviation of 80 mm. I chose an abundance of predator for each specified length that would yield a mortality level comparable to the baseline conditions of Rice et al. (1993a) shown in bold. I then kept the abundance of predators constant and varied the growth rates conditions either by varying mean growth rate (μ) or standard deviation (SD) of growth rates. In parentheses, the percent difference in survival given the changes in growth rate distribution.

	Rice et al. 93	This study			
μ (SD)	90mm	25 mm	90 mm	355 mm	Variable
0.2 (0.08)	0.536	3.03 (-3%)	4.69 (25%)	5.83 (80%)	5.2 (60%)
0.4 (0.08)	**3.323**	**3.11**	**3.75**	**3.24**	**3.26**
0.6 (0.08)	9.666	3.59 (15%)	1.98 (-47%)	0.79 (-76%)	1.4 (-57%)
0.4 (0.04)	3.118	3.55 (14%)	4.12 (10%)	2.1 (-35%)	3.46 (6%)
0.4 (0.08)	**3.323**	**3.11**	**3.75**	**3.24**	**3.26**
0.4 (0.16)	4.155	5.02 (61%)	8.48 (126%)	16.44 (407%)	14.13 (333%)

Table 4.4: Number of fish larvae eaten by a specific predator population given variations of the encounter rate parameters (±10% of the literature value, equations 3.2, 3.3) and variations of the susceptibility parameters (±95% confidence interval, equation 3.5). The initial cohort is composed of 10 000 individual fish larvae. *Rp* is the encounter radius of the predator, *Vp* is the swimming speed of predator, and *h* is the 1st order term and *i* the second order term of the susceptibility function (see Chapter III).

	Predator type							
	Crustaceans		Ctenophores		Medusae		Fishes	
Original values of the parameters and baseline conditions.								
Abundance (# m^{-3})	250		145		48		0.0192	
Number eaten	5054		5210		5002		5013	
Variations in the parameters of the encounter radius (R_p) and swimming speeds (V_p).								
Number eaten given:	$\pm R_p$	$\pm V_p$	$\pm R_p$	$\pm V_p$	$\pm R_p$	$\pm V_p$	$\pm R_p$	$\pm V_p$
+10%	5728	5838	6058	5711	5942	5705	9305	7931
-10%	4469	4396	4220	4633	4050	4517	0	561
% difference	±12	±14	±18	±10	±19	±12	±93	±73
Variations in the parameters of susceptibility function (*h* and *i*).								
Number eaten given:	±*h*	±*i*	±*h*	±*i*	±*h*	±*i*	±*h*	±*i*
+95% C.I.	5147	.	3712	4606	6614	.	1103	2580
-95% C.I.	4926	.	7107	6024	3573	.	5676	5625
% difference	±2	.	±33	±14	±30	.	±46	±30

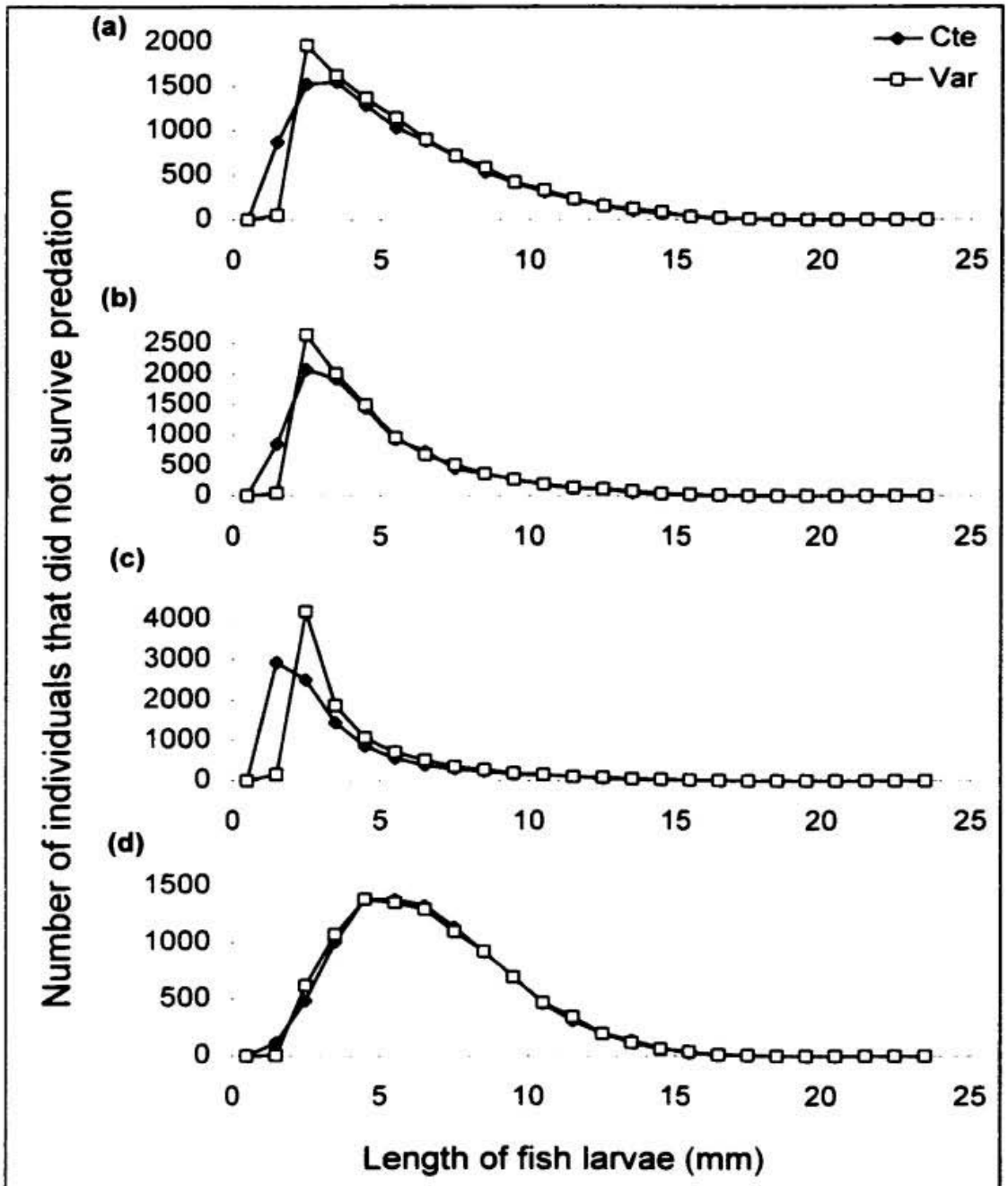

Figure 4.1: Length of fish larvae (mm) consumed by **(a)** crustaceans, **(b)** ctenophores, **(c)** medusae, or **(d)** pelagic fishes for a 30 days simulation. The initial cohort of fish larvae is composed of 10 000 individuals of either all equal length of 2.0 mm (Cte) or of variable lengths varying from 1 to 5 mm (Var).

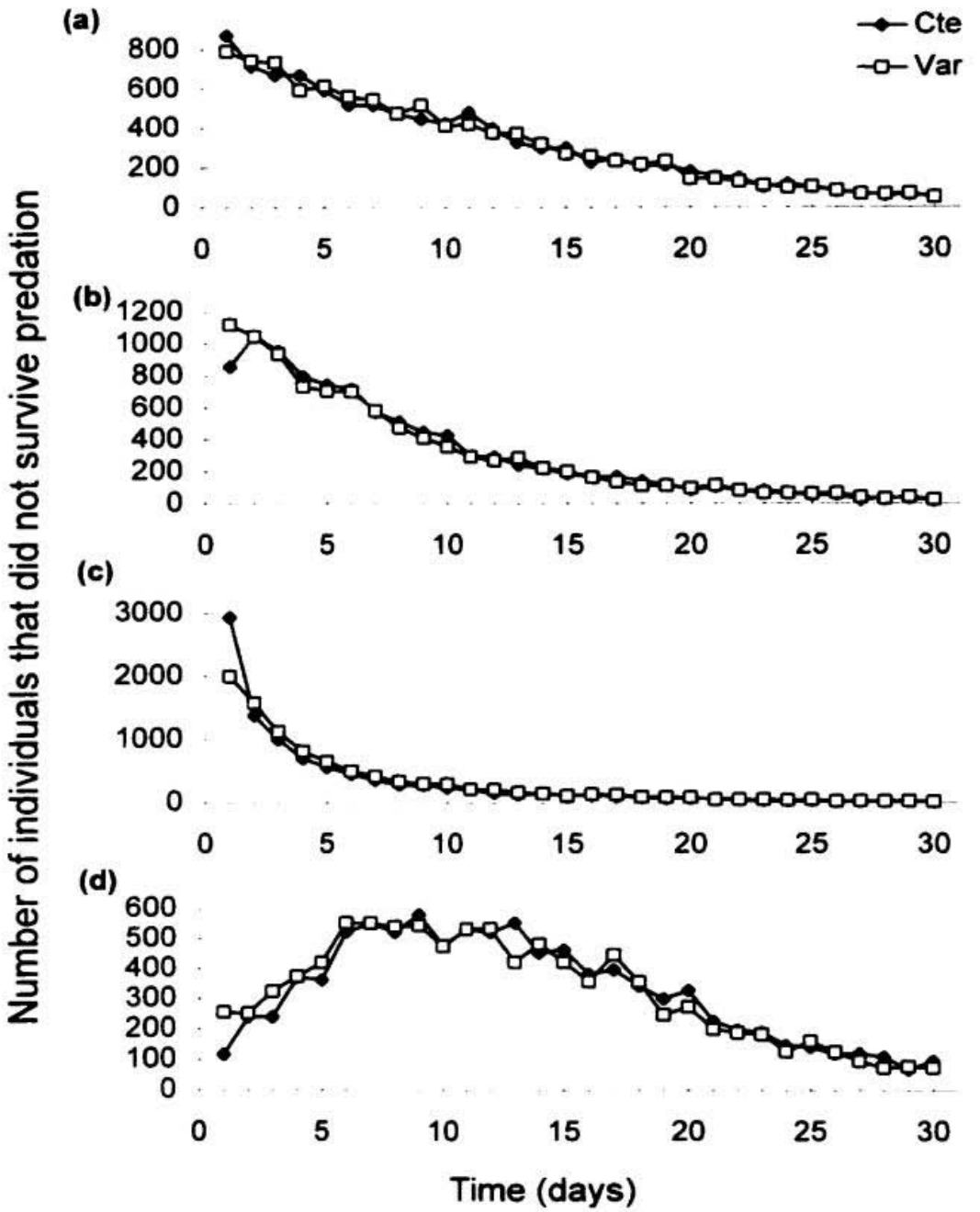

Figure 4.2: Number of fish larvae consumed each day of a 30 days simulation by **(a)** crustaceans, **(b)** ctenophores, **(c)** medusae, or **(d)** pelagic fishes. The initial cohort of fish larvae is composed of 10 000 individuals of either all equal length of 2.0 mm (Cte) or of lengths varying from 1 to 5 mm (Var).

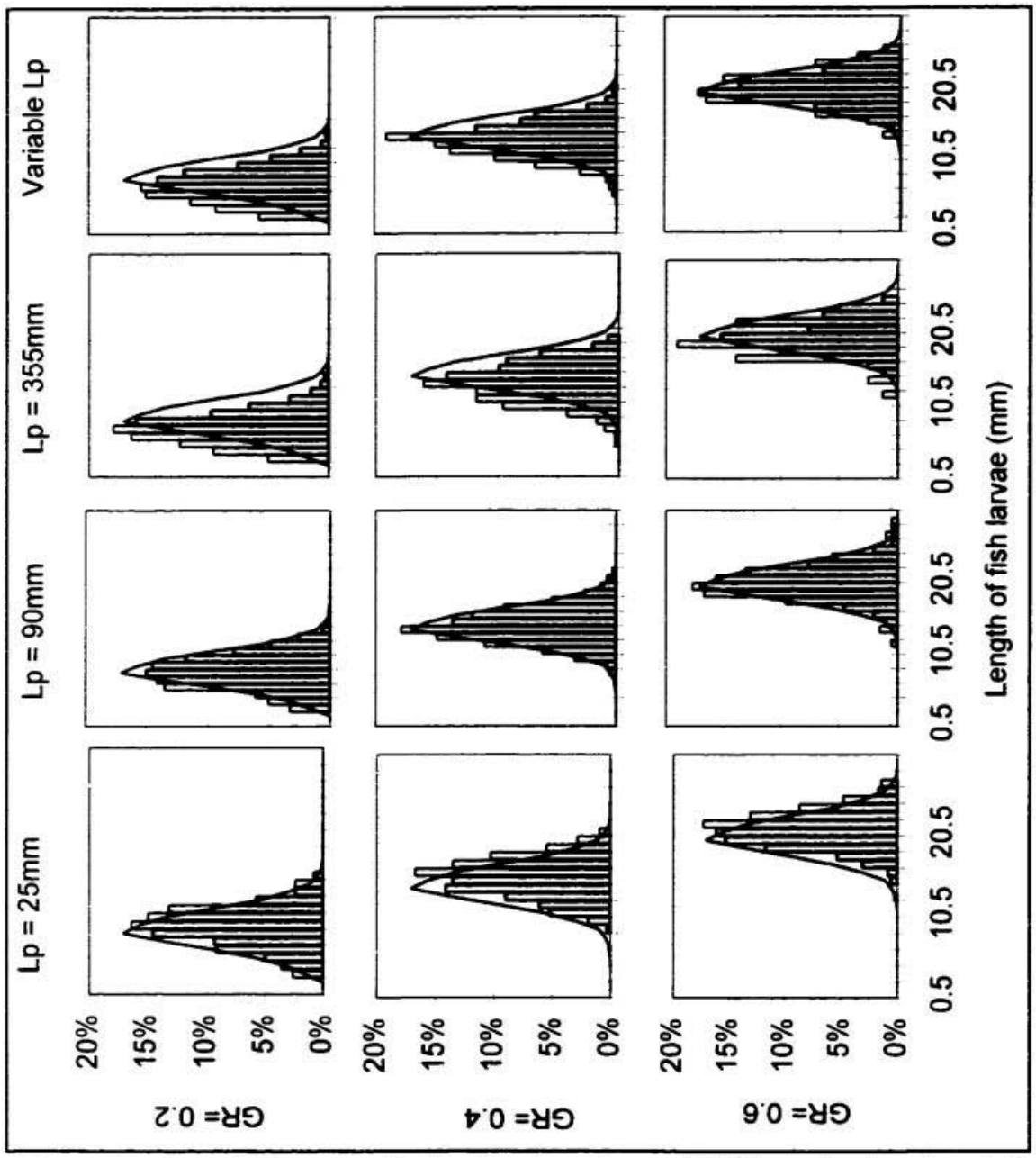

Figure 4.3: Length frequency distribution for cohorts of fish larvae having three initial mean growth rates (GR= 0.2, 0.4, 0.6 $mm \cdot d^{-1}$) after 30 days with (white bars) and without (shaded curve) predation by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm).

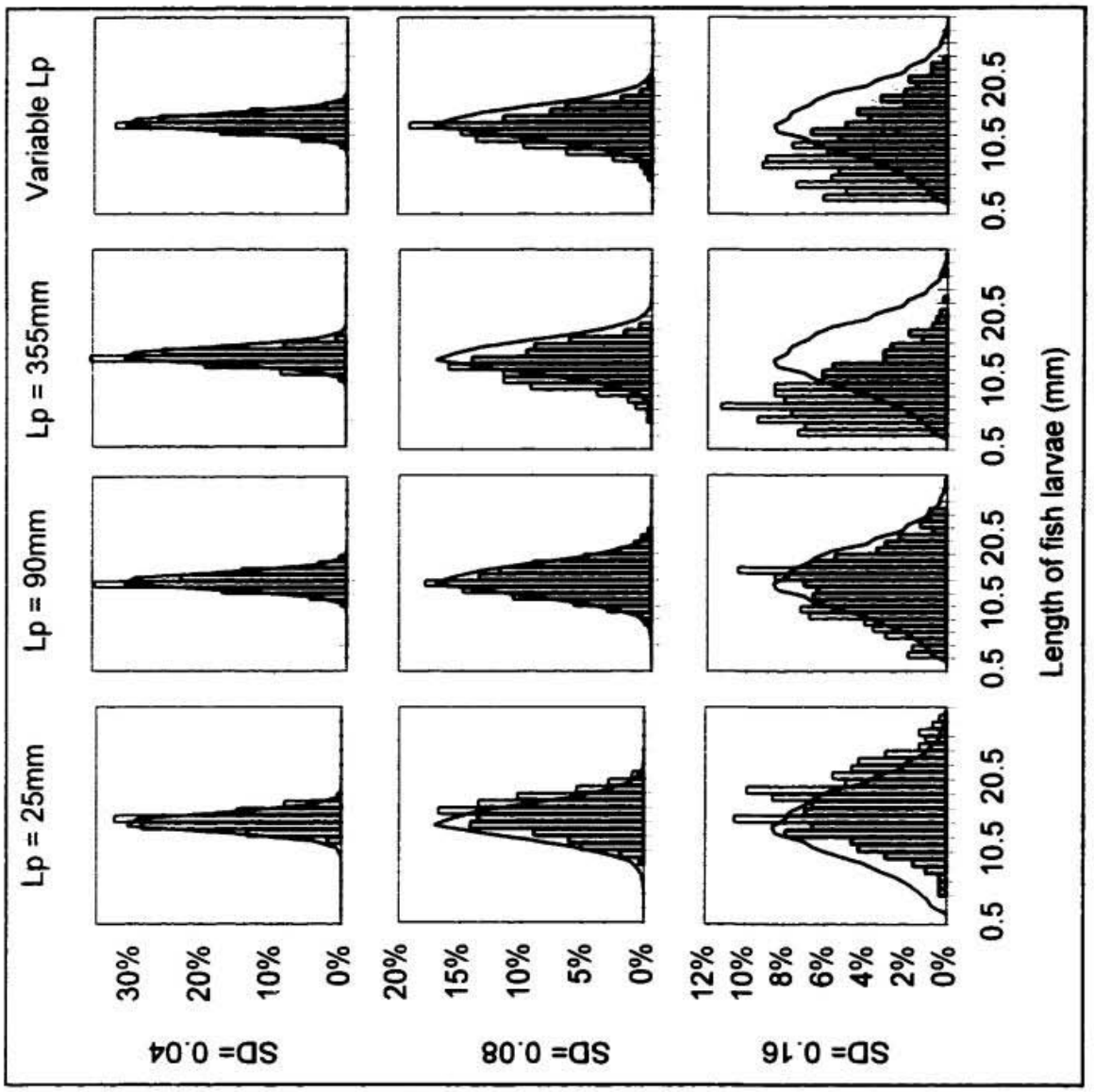

Figure 4.4: Length frequency distribution for cohorts of fish larvae having three levels of variability in growth rate among individuals (SD= 0.04, 0.08, 0.16 mm $\cdot$ d^{-1}) after 30 days with (white bars) and without (shaded curve) predation by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm).

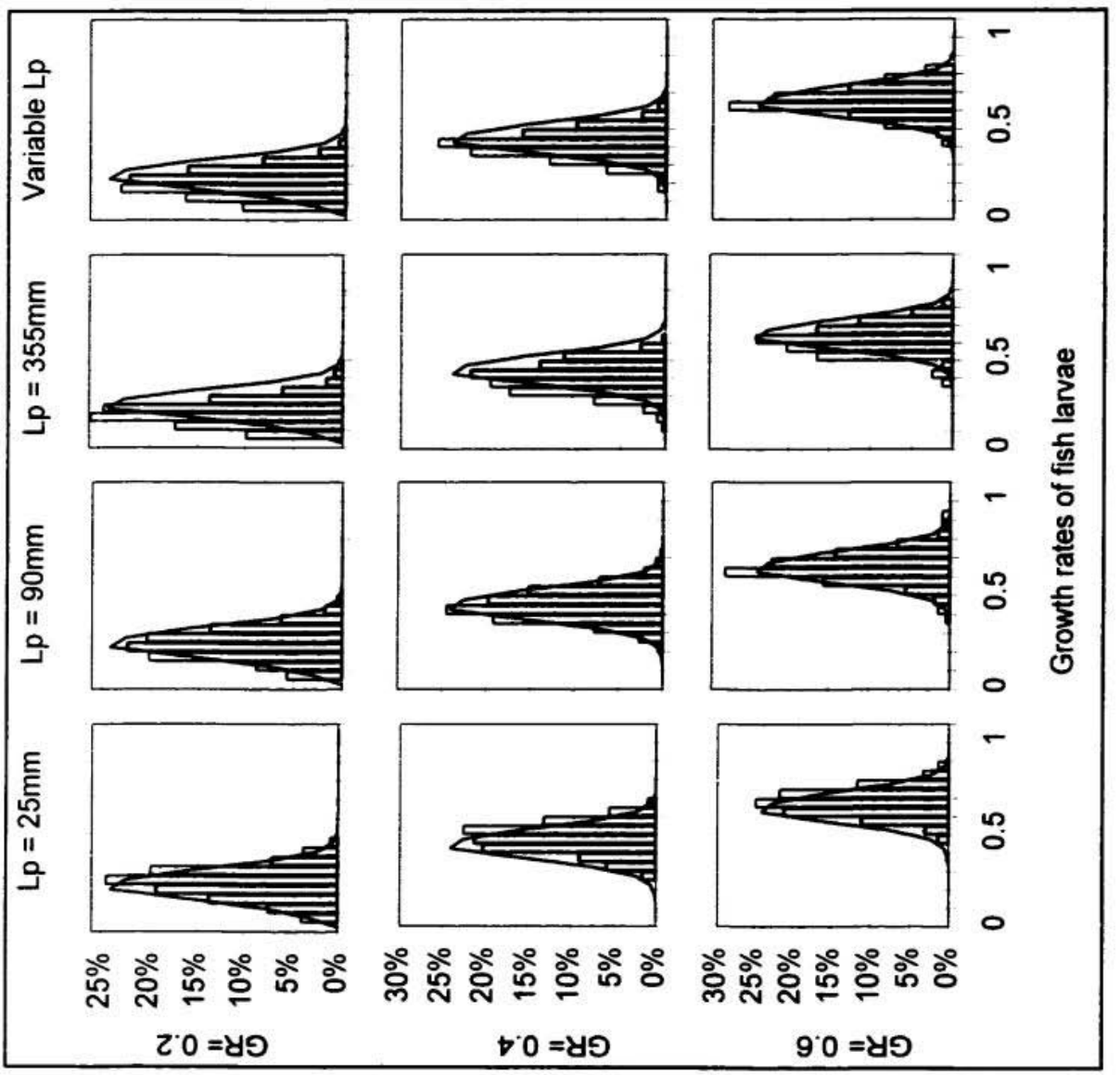

Figure 4.5: Growth rate frequency distribution for cohorts of fish larvae having three initial mean growth rates (GR= 0.2, 0.4, 0.6 $mm \cdot d^{-1}$) after 30 days with (white bars) and without (shaded curve) predation by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm).

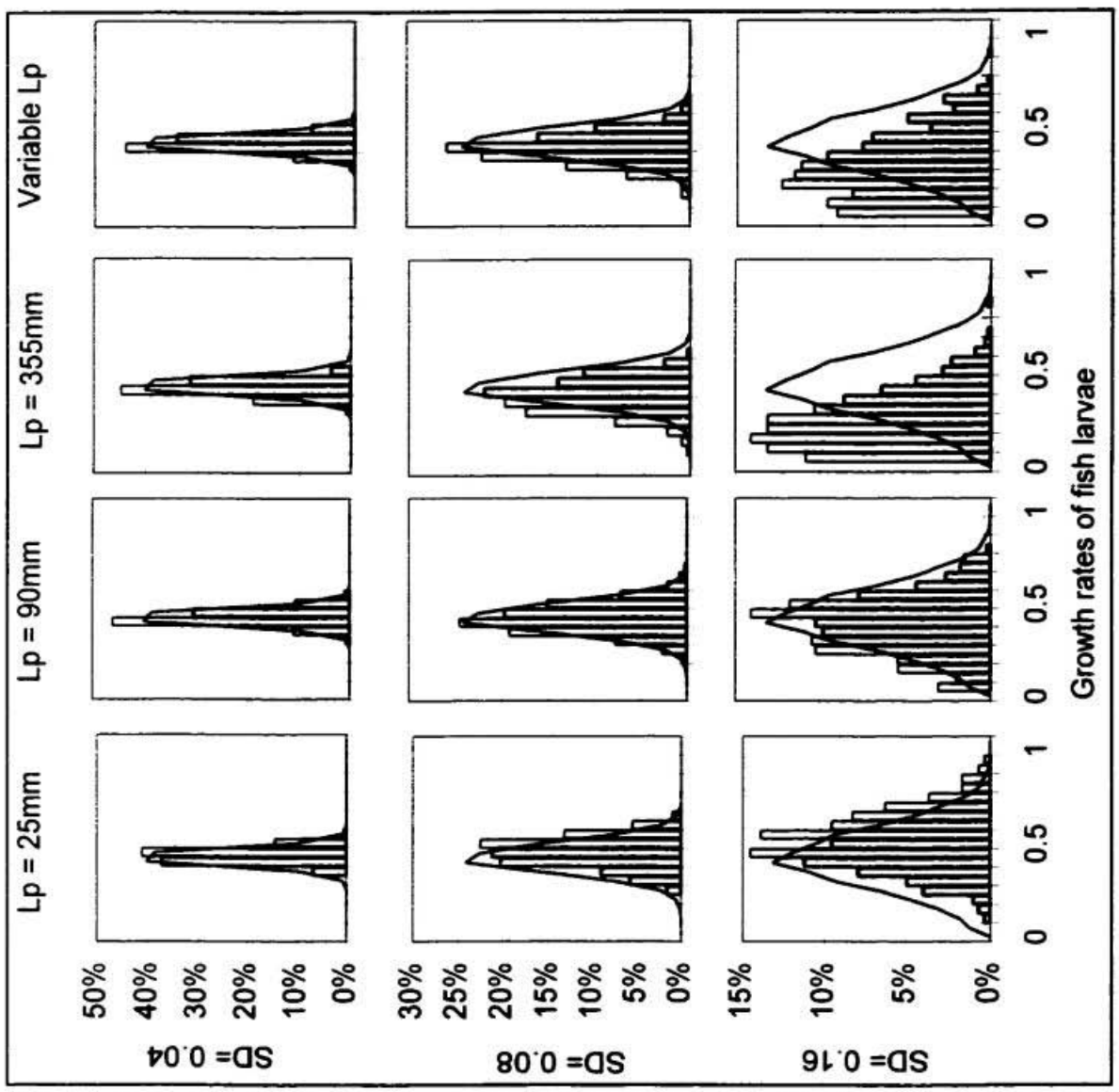

Figure 4.6: Growth rate frequency distribution for cohorts of fish larvae having three levels of variability in growth rate among individuals (SD= 0.04, 0.08, 0.16 $mm \cdot d^{-1}$) after 30 days with (white bars) and without (shaded curve) predation by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm).

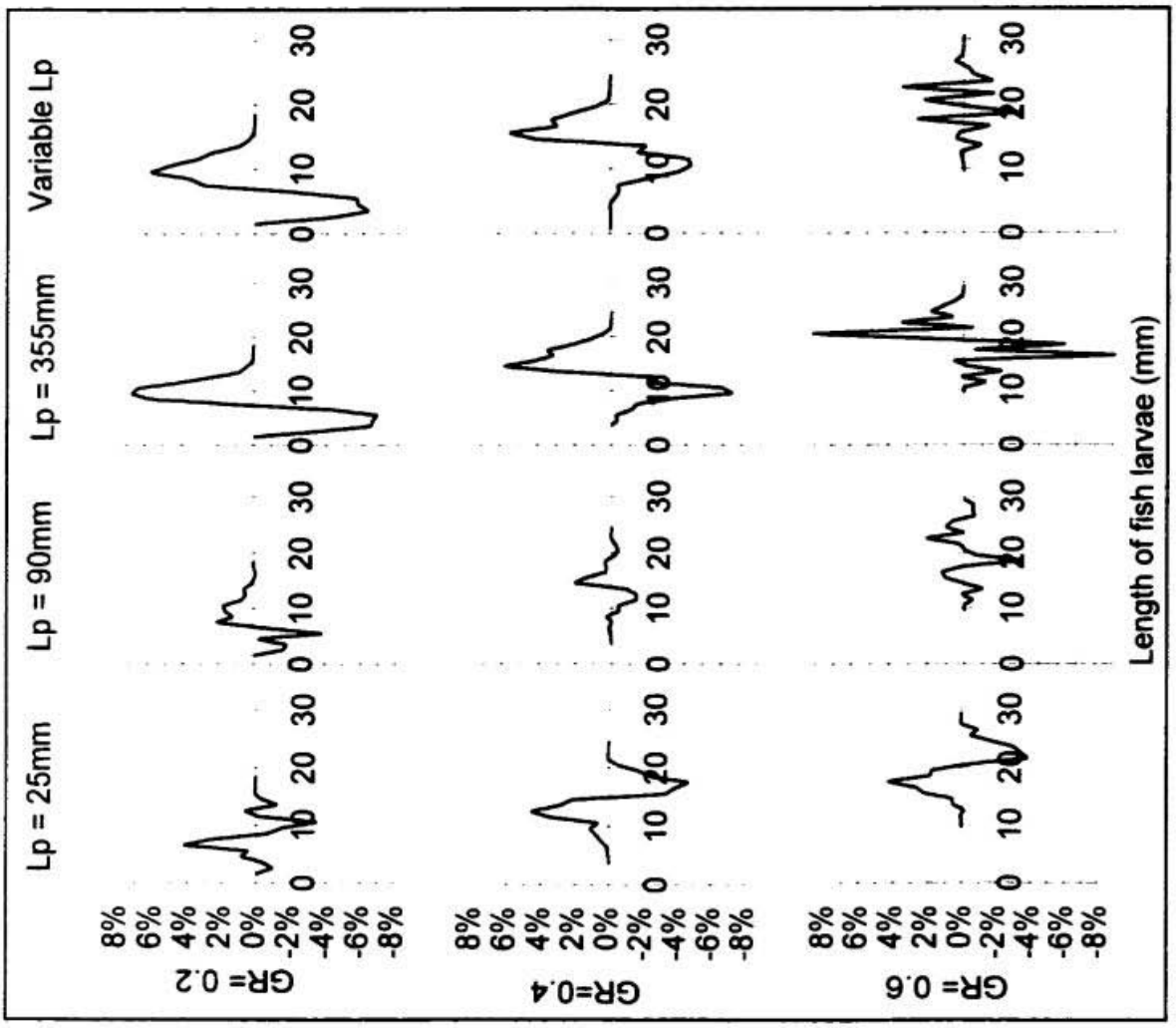

Figure 4.7: Size-selective mortality for cohorts of fish larvae having three initial mean growth rates (GR= 0.2, 0.4, 0.6 mm $\cdot$ d^{-1}) after 30 days of predation by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm). Size-selective mortality (%) measured as the difference between the frequency of individuals without predation and the frequency of individuals with predation for each particular length class at the end of the 30 days simulation period.

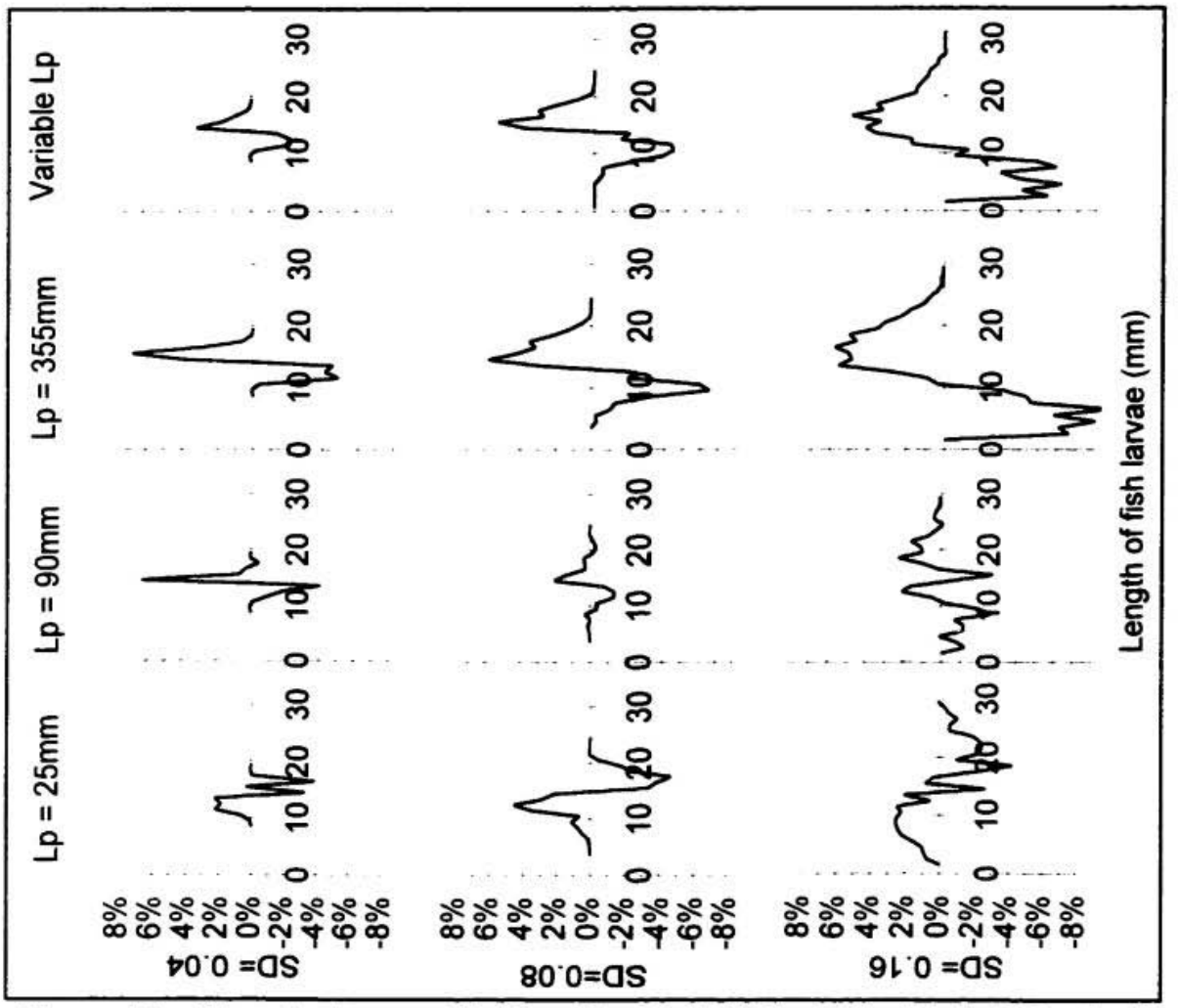

Figure 4.8: Size-selective mortality for cohorts of fish larvae having three levels of variability in growth rate among individuals (SD= 0.04, 0.08, 0.16 $mm \cdot d^{-1}$) after 30 days of predation by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm). Size-selective mortality (%) measured as the difference between the frequency of individuals without predation and the frequency of individuals with predation for each particular length class at the end of the 30 days simulation period.

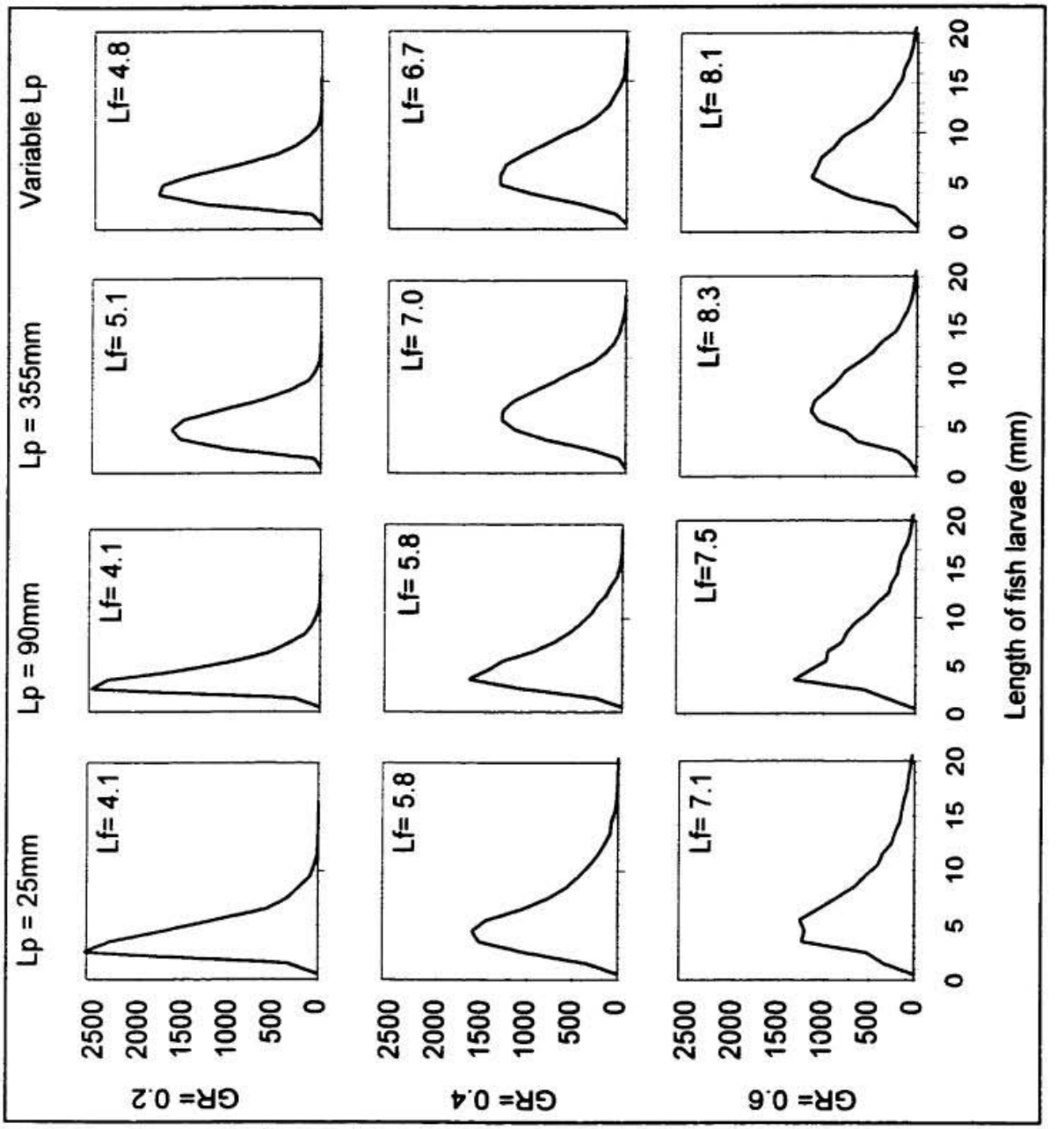

Figure 4.9: Length distributions for the consumed fish larvae having three initial mean growth rates (GR= 0.2, 0.4, 0.6 mm $\cdot$ d^{-1}) after 30 days of predation by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm).

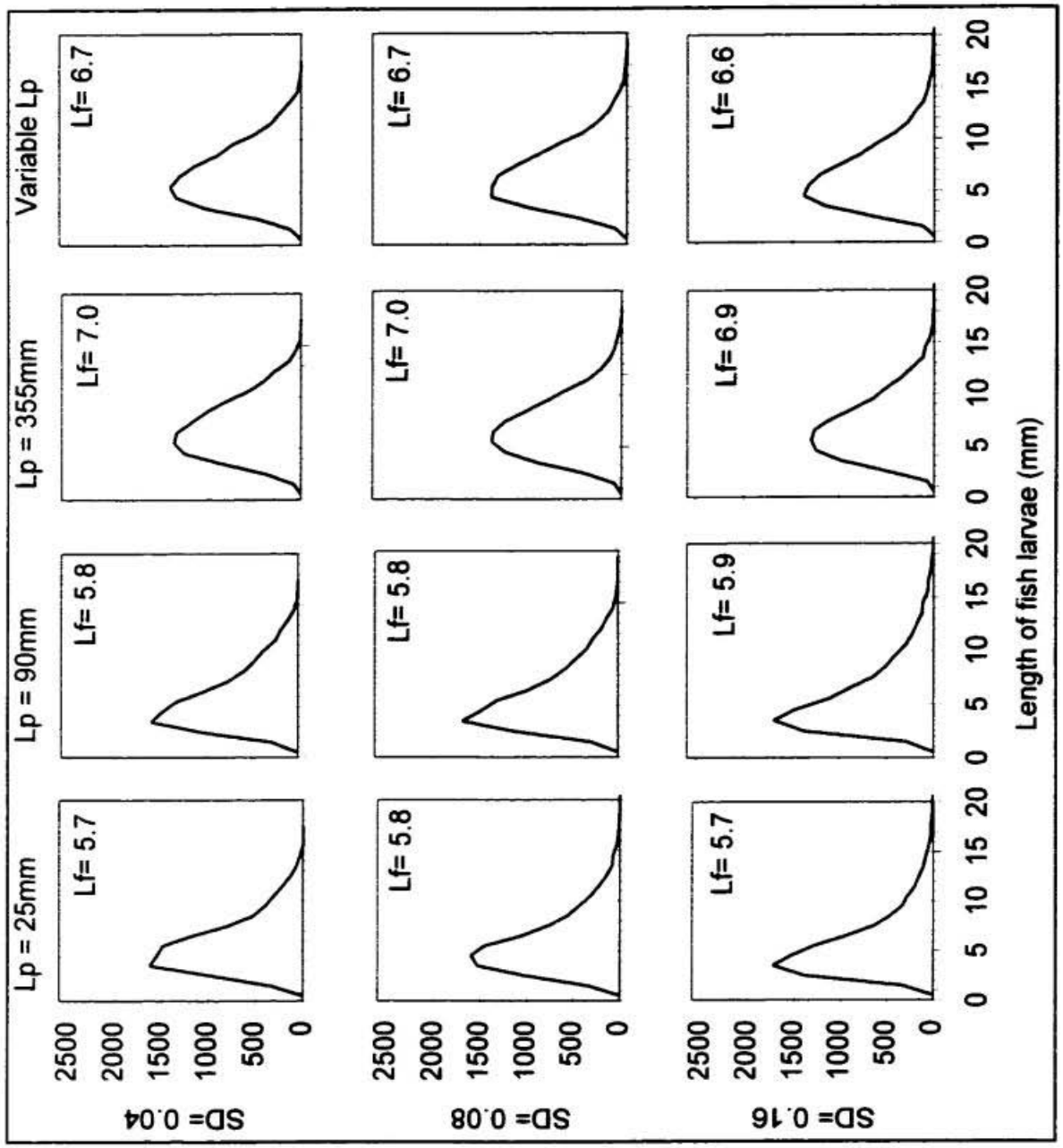

Figure 4.10: Length distributions for the consumed fish larvae having three levels of variability in growth rate among individuals (SD= 0.04, 0.08, 0.16 $mm \cdot d^{-1}$) after 30 days of predation by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm).

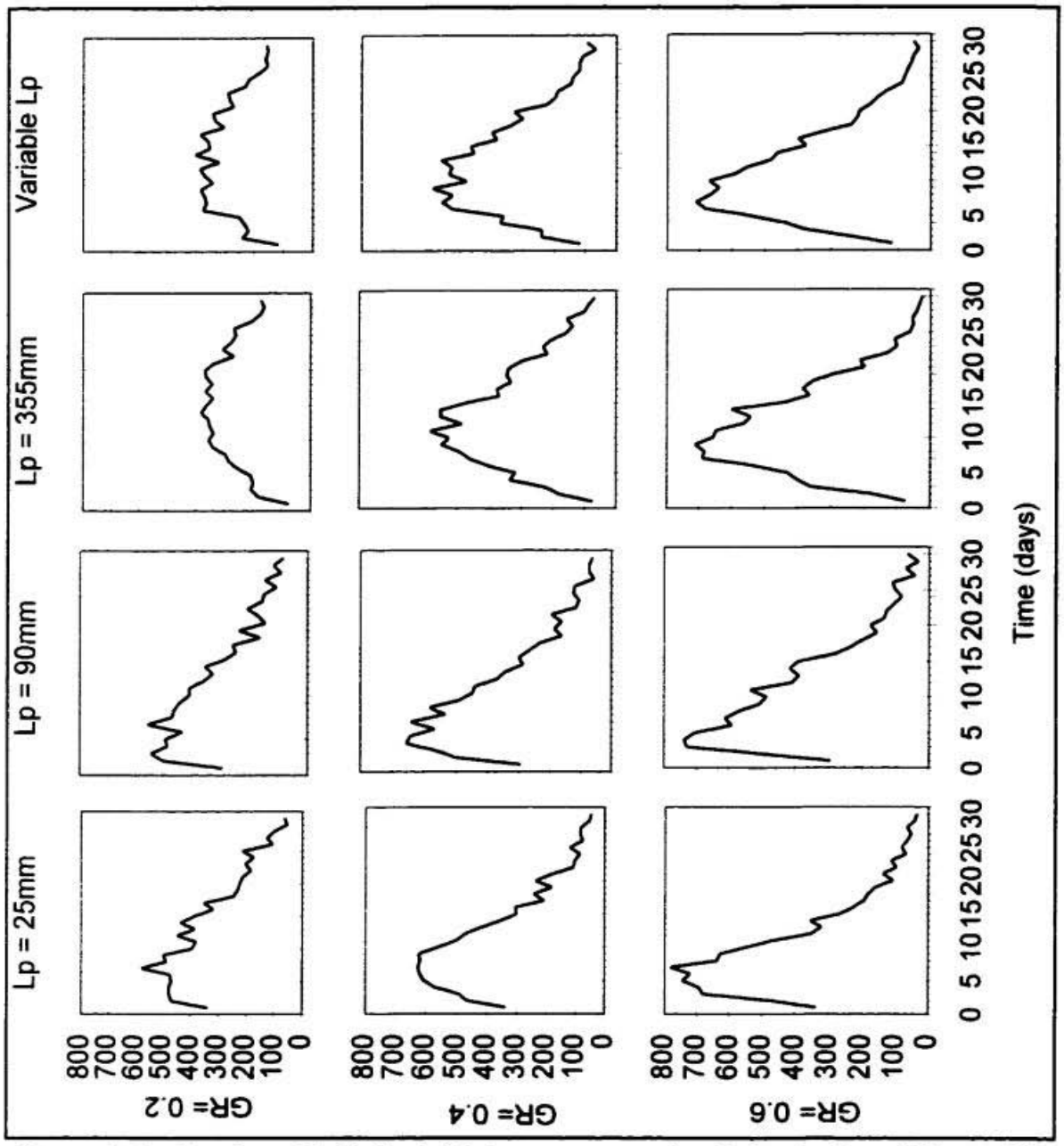

Figure 4.11: Number of individual fish larvae consumed each day by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm). The initial cohort of fish larvae had one of three initial mean growth rates (GR= 0.2, 0.4, 0.6 mm $\cdot$ d^{-1}).

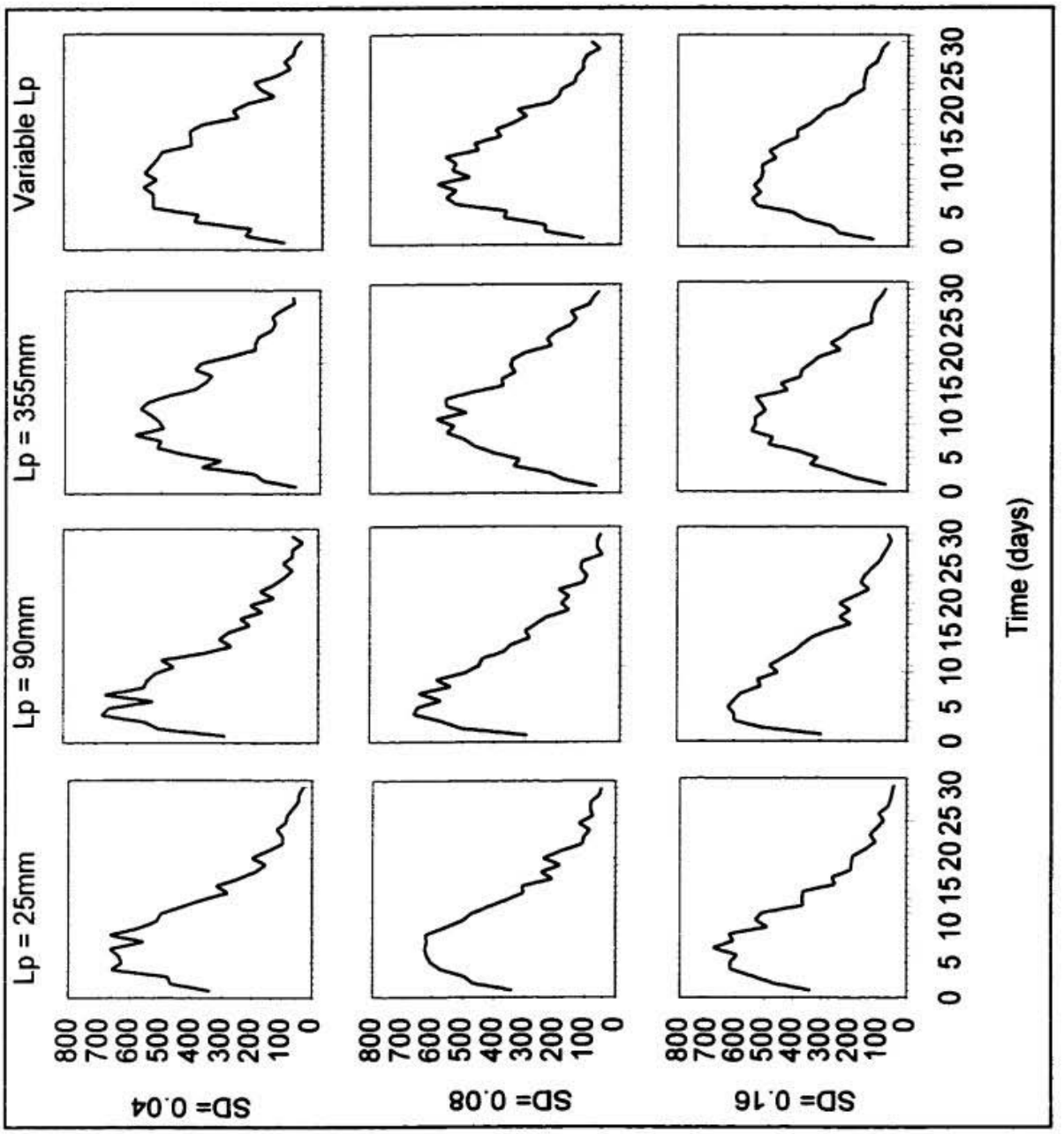

Figure 4.12: Number of individual fish larvae consumed each day by four different fish populations either all measuring (Lp) 25, 90 or 355 mm or of variable lengths (μ= 185 mm, SD= 80 mm). The initial cohort of fish larvae had one of three levels of variability in growth rate among individuals (SD= 0.04, 0.08, 0.16 mm $\cdot$ d^{-1}).

Chapter V

Modeling field length frequency distributions of fish larvae using field estimates of predator abundance and size distributions

Abstract

The goal of this study was to determine if an individual-based model could adequately and realistically simulate the growth and predation mortality of a multispecies community of fish larvae in the field. I focused on the changes in the length frequency distributions of several species of fish larvae collected in Conception Bay in 1993 and 1994. I first modeled the length frequency distribution of the field samples with the best possible estimates of mean growth rates. I then added predation mortality given the characteristics of the predator populations observed over the course of the surveys. Characteristics of the predator populations were based on surveys of the macrozooplankton community and of the adult capelin community. This study demonstrated that the larval fish community was not vulnerable to predation by macrozooplankton (average instantaneous mortality was Z= 0.04 d^{-1}) whereas fish larvae were most vulnerable to predation by the adult capelin population (Z= 0.54 d^{-1}). I demonstrated that an abundance of adult capelin ranging between 0.2 - 1.0 $\cdot 1000\ m^{-3}$ may have a substantial impact on the larval fish community. This study has significant implications for the study of larval fish survival as it demonstrated that the predictions of an individual-based model may be closely related to the accuracy and precision of the mean growth rates of the cohorts of larval fishes.

Introduction

Studies modeling the relationship between growth and mortality, particularly the role of size-selective mortality, must be supported by field research (Heath 1992). I have presented in Chapters III and IV, an individual-based model (IBM) which investigated several aspects of the prey-predator interaction involving fish larvae. In this chapter, I determined if such an IBM is capable of explaining changes in the length frequency distributions of fish larvae observed from field collections. In essence, I am asking: "Can an IBM adequately represent a sample of the true population of fish larvae? Are the model's predictions about the impact of predators supported by field observations?"

To date, few modeling studies of the vulnerability of fish larvae to predation have tried to relate their findings to field data. Rice et al. (1993, 1997) and Crowder et al. (1994) have tested their tactical models with experiments. Tactical models are testable because they describe the mechanisms operating in a particular system (Murdoch et al. 1992). In contrast, Cowan and Houde (1992) and Cowan et al. (1996, 1997) used experiments to develop their model and estimate its parameters: their tactical model is not yet supported by field observations or tested with independent experiments (i.e., experiments not used to derive parameter estimates for the model). Even if the IBM presented in previous chapters is a strategic model because it relies on empirical and general relationships (Murdoch et al. 1992), it can still be tested with field observations.

IBMs lend themselves to a process of progressive removal of particular features which should facilitate distinguishing the general mechanisms from those that account for detail rather than major dynamic features (Murdoch et al. 1992). In previous chapters, I have demonstrated some of those features in prey-predator interactions specific to larval fishes, such as characteristics of the predator population or the isolated effects of either encounter or susceptibility on the size-selective removal of individuals (Chapter III). In this chapter, I determined if the characteristics of the predator population observed coincidently with fish larvae accounted for a significant proportion of the observed changes in length frequency distributions of larvae.

Several studies have proposed methods of estimating mortality and growth rates from length frequency distributions (e.g., MacDonald and Pitcher 1979; Saila and Lough 1981; Barry and Tegner 1989; Somerton and Kobayashi 1992). A common feature of field studies of ichthyoplankton is an underlying assumption of constant daily predation rate with time (or age). This is in spite of the general recognition that mortality rates decrease with size (Peterson and Wroblewski 1984; Pepin 1991) and time (Houde 1989) and that predation is a size-selective process and major contributor of total mortality (Bailey and Houde 1989). Furthermore, vulnerability to predation of a larva is highly dependent on the characteristics and type of predators encountered throughout their early life stages (Chapters III, IV). In addition, the general consensus is that mortality and growth rates are seriously biased when they are estimated using those techniques (Lo et al. 1989; Parma and Deriso 1990; Taggart and Frank 1990;

Pepin 1993; Pepin et al. 1995). Pepin (1993) raised important questions regarding the validity and generality of the size-dependent mortality hypothesis of larval fish. He found that even though a mortality estimate based on the length-based method may support the size-dependent mortality hypothesis, the estimate changes in response to variations in the size categories sampled in a survey. Taggart and Frank (1990) and Pepin et al. (1995) argued that the time and space scales over which abundance estimates are collected can lead to biases in estimations of larval fish mortality rates. As age and size of larvae are closely coupled, and most importantly as the variation of the size of fish larvae increases with time (or age) (DeAngelis and Huston 1987; Benoît 1999), we can't rely on estimates of mortality rate.

Another method of estimating prey-predator interactions is to relate inverse spatial or temporal correlations of prey and predator abundances. The interpretation of such asynchronous oscillations has been criticized in earlier work by Frank and Leggett (1985). They argued that size distributions of prey and predators should be combined with environmental data to which the temporal and spatial distributions of the two populations may be related (Frank and Leggett 1985). Frank and Leggett (1985) believed that only in light of such data can predation be inferred from reciprocal oscillations of abundances and spatial distributions of prey-predator community. I believe that an IBM may offer a link between these field oscillations of prey-predator distributions and the potential predation mortality of fish larvae assessed in laboratory and mesocosms studies.

I focused on the changes in the length frequency distributions of several larval fish species collected in Conception Bay in 1993 and 1994 in a step-wise fashion. First, I modeled the length frequency distribution of the field samples with the best possible estimates of growth rates (individual-based growth model, IBGM). Then, I added predation mortality given the characteristics of the predator population observed over the course of the surveys (individual-based predation model, IBPM). Characteristics of the predator populations were based on surveys of the macrozooplankton and of the adult capelin population, a dominant planktivorous fish in Conception Bay. The goal was to determine if an IBM can realistically simulate the growth and predation mortality of a multispecies community of fish larvae. If the length frequency distribution of the cohort modeled with predation provided a better fit to the length frequency distribution observed in field collections than that predicted by growth rates alone, then I could conclude that size-selective predation was having a significant impact on the characteristics of survivors. An advantage in working with a multispecies assemblage of fish larvae is that it allowed discrimination of processes occurring due to the size of fish larvae from those occurring due to the early life history strategies of the different species.

The IBPM was based on three processes: encounter between individual fish larvae and a population of predators, susceptibility of fish larvae to the encountered individual predator, and the growth of fish larvae. The IBGM was based on a single process: growth of fish larvae. I used the IBM developed in Chapters III and IV. Even

though the details of the models are not species-specific, I modeled each species individually because they exhibited substantial differences in terms of abundance, length and growth rates.

Methods

Study site, survey design and sampling procedures

Conception Bay is located on the north-east coast of Newfoundland, Canada (Figure 5.1) and is approximately 50 km long and 20 km across at the mouth with a maximum depth in the center of about 300 m and a total surface area exceeding 1000 km^2. The Bay is influenced by the inshore arm of the Labrador current as well as wind forcing on time scales of 5 to 15 days (deYoung and Sanderson 1995; Laprise and Pepin 1995). Stratification is primarily due to salinity with some thermal effect in the upper 10 to 20 m. Subzero temperatures are typically found below 50 m. Mixed layer depths range from about 10 to 40 m.

The surveys were designed to estimate the abundance of ichthyoplankton populations as well as the invertebrate predator community at regular intervals and provide simultaneous observations of water properties (temperature and salinity). In 1993, 5 to 9 stations were sampled each day. In 1993, we tried to cover the entire Bay in a single day. The 6 sampling periods were separated by 2 to 4 days (12, 14-16, 19, 21-22, 26 and 29-30 July 1993). In 1994, 13 to 16 stations were sampled over a 2 day period. In 1994, stations at the bottom of the Bay (i.e., F1, BRs, CT2s and CT3s) were

sampled on one day and the stations at the mouth of the Bay (i.e., US and CW lines) on the following one (Figure 5.1). Stations BI1 and CN3 were sampled only if time permitted. The 5 sampling periods were separated by 3 to 6 days (12-13, 19-21, 24-25, 29-30 July, and 4-5 August 1994).

All sampling was conducted during daylight hours to avoid potential bias associated with diurnal variations in net avoidance. Ichthyoplankton and macrozooplankton samples were obtained using a 4 m^2 Tucker trawl equipped with sections of 1000, 570, and 333 μm mesh Nitex. At each station, a single oblique tow of approximately 15 min. was made at 1 $m \cdot s^{-1}$. The net was lowered to 40 m and retrieved at rates of 0.25 $m \cdot s^{-1}$ and 0.064 $m \cdot s^{-1}$. Maximum tow depth was chosen to include the mixed layer in which >95% of the larval fish reside (Frank and Leggett 1982; deYoung et al. 1994). I was unable to assess the effect of non-sampled predators on larval fish survival and will discard them. On deck, the net was washed and the samples were preserved in 2% buffered formaldehyde. Salinity and temperature profiles were obtained at each net station to within 5 m of the bottom using a Seabird-25 conductivity-temperature-depth (CTD) meter. The conductivity and temperature sensors were calibrated under laboratory and field conditions.

Ichthyoplankton and macrozooplankton were sorted and identified to species or the lowest taxonomic level possible by the Atlantic Reference Centre (Huntsman Marine Science Centre, St. Andrews, NB). Subsampling of an individual taxon was performed for samples in which numbers of that species exceeded 200 individuals per

stage (i.e., eggs or larvae) using a beaker technique (van Guelpen et al. 1982). The length frequency distribution for each larval fish species and sample was estimated by measuring up to 200 larvae. Standard length was measured to the nearest millimeter using a dissecting microscope and a graded background. Abundance (number per 1000 cubic meters) was calculated for each larval fish species for every 1 mm length interval for each sample. Abundance estimates were corrected for the number of larvae that could not be measured due to damage (<5%). Size of crustaceans was estimated with literature values for specific taxon captured (Squires 1990) whereas diameter of medusae was measured from a subsample in the laboratory to the nearest millimeter using an imaging system (Bioscan OPTIMAS® 4.10). The size of crustaceans varied between 1 and 10 mm whereas the diameter of medusae varied between 1 and 50 mm.

I selected species of fish larvae based on their length frequency distribution and if they were captured more than twice and were relatively abundant in Conception Bay in one or both years sampled. I separated species into separate cohorts when I could identify a bimodal distribution or if the same length frequency distribution occurred twice during the survey and was accompanied by a sharp increase in abundance.

Pelagic fishes are important predators of fish larvae (Chapter II). The most important population of pelagic fishes of Conception Bay coinciding with the survey was adult capelin (*Mallotus villosus*). Aerial estimates of capelin schools conducted since 1982 have been used as an index of inshore abundance of mature capelin in Conception Bay (Nakashima 1995). Spawning of adult capelin takes place primarily on

the beaches of the western shore of the Bay (Templeman 1948). The school areas were estimated from digital imagery data collected by the Compact Airborne Spectrographic Imager (specific details can be found in Nakashima 1995). In 1993 and 1994, the aircraft (De Havilland Beaver), survey time (30 June - 28 July 1993 and 2 July - 4 August 1994), altitudes flown, and flight time were similar (Nakashima 1995). For each transect flown, the total surface areas of individual capelin schools were estimated. The abundance index of capelin was estimated by dividing the sum of the total surface areas of the schools observed on the inner and outer transects of Conception Bay with the total area of the surveys in Conception Bay (4.66×10^7 m^2).

Production

The observed abundance of fish larvae was primarily due to two processes: production and mortality. Immigration was included in production and emigration was included in mortality. Usually, larvae are assumed to be retained in the Bay for a longer period (deYoung et al. 1994) than our survey and the flux of larvae at the mouth of the bay is independent of their size (Pepin et al. 1995). Pepin et al. (1995) estimated an average flux at the mouth of the bay of about 3% d^{-1} (in and out). For the purpose of this study, I assumed that even though this flux may bias estimates of mortality rates due to biological processes (Pepin et al. 1995), it will not affect the overall length frequency distribution of fish larvae. If the abundance of fish larvae increased with time, it implies that there are more fish larvae being produced than dying. The

abundance of larvae was corrected for this production. I assumed that fish larvae caught in later samples could not have decreased in size. I made the appropriate corrections for each species by deleting all size-classes caught in later samples that were smaller than the smallest size-class of the initial cohort. I also deleted any size-classes that could not be explained by the smallest growth increment calculated based on the first estimate of growth rates. The corrected length distributions for each selected larval fish species are presented in panels (**a**) in Figures 5.4 to 5.13.

Estimation of growth

As a first step, I estimated the mean growth rate of a cohort with a linear regression of mean length of the cohort over time. This estimate of growth rate assumes that all individuals within a cohort have the same growth rate and hence that there is no variability in growth among individuals. This assumption is an inaccurate representation of individual larval growth (Benoît 1999) but this method allows a first estimate of the magnitude of mean growth rate of the cohort. It was this estimate that I first used in the IBGM.

A more realistic approach to evaluating the growth rate of the cohort was to use a modified version of the IBM presented in Chapters III and IV. I assumed that growth rates were either normally or log-normally distributed and that each larva was assigned a growth rate at random from this distribution. At this stage, I did not include predation. I modified the model so that it followed the length frequency distribution of growing

fish larvae with no mortality (IBGM). I visually compared the length frequency distribution of this modeled cohort with the length frequency distribution of the field cohorts. I adjusted the mean and standard deviation of the growth rate distribution to align and overlap the mode and spread of both distributions. With the IBGM, I assessed how a distribution of individual growth rates could predict the changes of the length frequency distribution observed in the field when the assumption of equal growth rates among individuals was relaxed.

One problem associated with this procedure is that I was purposely seeking to minimize discrepancies between the projected and the observed length frequency distributions by adjusting different parameters of the growth rate distributions. These discrepancies were the actual mortality I wished to evaluate. However, when I added predation in the IBM, any further minimization of discrepancies between the two length frequency distributions were due to predation beyond that explained by growth rates alone. I realize that there are draw-backs for using this approach due to the severe effect of under- or over-estimating the growth rate distribution on the conclusion about size-selective mortality (Miller 1997) but in the absence of independent measures of individual growth rates or mean growth rate of cohorts, it was the only option available.

Initialization of the individual-based predation model

I used the conditions observed during the surveys of Conception Bay to set the parameters of the IBPM. The initial number of fish larvae was set to 10 000 regardless

of the species-specific abundance estimated. The initial length frequency distribution of selected species was based on the first sample collected. The larval growth rates were based on the IBGM method described above. The abundance of predators was based on field estimates during the same time interval and the size distribution of predators was generated with a random number generator and based on the size range collected in the macrozooplankton tows and known lengths of mature adult capelin (Jangaard 1974; Winters 1982; Sager et al. 1989; Carscadden et al. 1997). The shape of the size distribution of predators was assumed as a normal distribution about a mean size.

Inverse method to estimate mortality due to predation by pelagic fishes

Given that the abundance index derived from aerial surveys was arbitrarily defined, I estimated the abundance of capelin required to inflict a mortality level comparable to previous field estimates of fish larvae mortality in Conception Bay (Pepin 1993). This inverse method was used only when the IBPM based on aerial estimates of abundance of adult capelin predicted unrealistically high mortality rates ($Z > 0.8 \cdot d^{-1}$) which occurred more often in 1993 than in 1994. I converted the length-based mortality estimates (Pepin 1993) such that: Z (instantaneous mortality, d^{-1}) = M (length-based mortality, mm^{-1}) × G (species-specific mean growth rate estimated with the IBGM, $mm \cdot d^{-1}$).

Analyses

Given the initial length frequency distribution, I extrapolated the distributions that should have been present in later samples in the absence and presence of predators. I wished to evaluate how well the IBGM and the IBPM could predict the future length frequency distributions of fish larvae. The length frequency distributions of the modeled cohorts and the field cohort are presented in panels **(b)** in Figures 5.4 to 5.13. To evaluate how the projections in time based on the initial sample compared with the actual future sample, I first computed the difference at each size class between the length frequency distributions of the field cohorts with the modeled cohorts (panels **(c)** in Figures 5.4 to 5.13). If the difference between the observation and prediction was positive, it implied that a certain proportion of a specific size class of the field cohort was not explained by growth rates alone (IBGM) or by the addition of predation (IBPM). Ideally, the goal was to reduce to zero the difference between the length frequency distributions observed in field collections and predicted from the IBPM.

Second, I performed a one-tailed variance ratio test. The null hypothesis is that the length frequency distribution is less variable if predicted by the IBPM than by the IBGM. The test was computed:

$$F=\frac{(\sum_i (RF_{field} - RF_{IBGM})^2)/df}{(\sum_i (RF_{field} - RF_{IBPM})^2)/df} \qquad (5.1)$$

where RF is the relative length frequency for each size-classes (i) of the field cohort and the modeled cohorts (IBMs), df is the degrees of freedom ($df = i$-1). One will

recognize that the numerator and denominator of this equation are the mean square errors for both IBMs. The significance level was set to α=0.05.

Third, I combined all probabilities from these tests of significance based on a technique developed by Fisher (Sokal and Rohlf 1981, p. 779). This combined test provides an assessment of whether adding predation improved significantly the prediction of the length frequency distribution observed from the field samples. The actual computation is based on the fact that $-2\times\ln(P)$ is distributed as $\chi^2_{[2]}$. The resulting sum, $-2\Sigma\ln(P)$, is compared to χ^2 with degrees of freedom equal to twice the number of separate tests (Sokal and Rohlf 1981).

Results

Environmental conditions of Conception Bay

The vertical profile of water temperature was constant through time in 1993 whereas in 1994 there was a rapid warming of the top 15 m of water after July 25 (Figure 5.2). The daily water temperature (averaged over depth) increased over the survey period from 4.5°C to 6.7 °C in 1993 and 4.1°C to 10°C in 1994. These trends translated into a wider range of water temperatures in the surface 40 meters in 1994 than in 1993 (Figure 5.2).

The abundance of invertebrate predators in the Bay was very different in the two years of study (Figure 5.3). The average abundance of the invertebrate community was approximately one order of magnitude greater in 1994 than in 1993 (crustaceans:

1182.1 and 152.3 · 1000m^{-3}, $t_{0.05(2)17}$= 6.31, p<0.001; medusae: 286.3 and 64.7 · 1000m^{-3} $t_{0.05(2)17}$=10.75, p<0.001). There was little temporal variation of invertebrate predator abundance within study periods as the average abundance was more or less constant across surveys.

Spawning by adult capelin co-occurred with the 1993 survey but only during the first seven days of the 1994 survey (Figure 5.3). The aerial integrated index of adult capelin abundance was twice as high in 1993 than in 1994 ($2.3 \cdot 10^{-3}$ and $1.2 \cdot 10^{-3}$, respectively).

Estimation of the growth rate distribution

The estimates of mean growth rate of fish larvae in 1994 were consistently lower than larval growth rates in 1993 (Table 5.1) even though the water temperature was on average higher in 1994 than in 1993 (Figure 5.2). These estimates fell within a range of larval growth rates observed in other field and laboratory studies (Table 5.2).

In general, the IBGM was able to track changes in the length frequency distribution of most cohorts of fish larvae. The length frequency distribution of *Hippoglossoides platessoides*, *Pseudopleuronectes americanus*, and *Ulvaria subbifurcata* in 1994 were fit better if the growth rates were log-normally distributed (Table 5.1, Figures 5.5, 5.10, and 5.13, respectively). This was due to the skewness of the length frequency distribution at the end of our survey. The IBGM was much better for tracking the length frequency distributions of *Clupea harengus*, *Liparis atlanticus*,

and *Pseudopleuronectes americanus* caught in 1994 than in 1993 (Figures 5.4, 5.6, and 5.10, respectively). This may be due to the wide range of length classes caught in 1994 relative to 1993.

Adding predation mortality in the individual-based model

Given the field abundance of invertebrate predators (Figure 5.3), I found that crustaceans and medusae did not have a significant impact on the survival of any species of fish larvae in 1993 as the highest instantaneous mortality predicted by the IBPM was 0.004 d^{-1} (Table 5.3). This low mortality due to invertebrate predation has no application on eggs because the survey concentrated on fish larvae. However, in 1994, the instantaneous mortality due to predation by crustaceans was significant for *Liparis gibbus* at 0.10 d^{-1} while predation by medusae was still insignificant for all larval species at 0.0004 d^{-1}. In general, the higher mortality rates predicted in 1994 was primarily due to the higher observed abundance of crustaceans. Given the high field abundance of invertebrate predators, it is surprising that the predicted mortality for larval fishes were so low. This was due in part to the relative size of prey and predator which translated into a low overall vulnerability to predation. In most cases, the predicted mortality due to invertebrate predation was too low (Table 5.3) for any size-selective effect to be detected from length frequency distributions (except for *Liparis gibbus*, Figure 5.7). There was some indication that larvae of *L. gibbus* surviving predation by crustaceans were smaller than expected by growth rates alone (Figure 5.7).

Predictions of mortality due to invertebrate predators were much lower than previous estimates of mortality in Conception Bay (Pepin 1993), except *Clupea harengus* (1994) and *Liparis gibbus* (Table 5.3).

Given the observed field abundance of adult capelin and the duration of spawning (Figure 5.3), I found that in general, the IBPM predicted that most fish larvae could be consumed by adult capelin (Table 5.3). The predicted mortality due to predation by adult capelin was much higher in 1993 than in 1994, mostly because the population was twice as abundant in 1993 (Figure 5.3). In 1993, predicted instantaneous mortality rates were all higher than $0.5\ d^{-1}$, except for *Pseudopleuronectes americanus* and *Pleuronectes ferrugineus* (Table 5.3). However, these predictions were still higher than the mortality rates estimated previously in Conception Bay (Pepin 1993), except for *P. americanus*. In 1994, the IBPM predicted that all 10 000 fish larvae of *Clupea harengus* and *Liparis gibbus* were consumed by adult capelin (Table 5.3). Otherwise, mortality predictions by the IBPM were similar to the mortality estimated previously for *Hippoglossoides platessoides*, *Liparis atlanticus*, and *Ulvaria subbifurcata*. For all larval fish species (except for *Pleuronectes ferrugineus* in 1993, Figure 5.9), the IBPM predicted that survivors to capelin predation were smaller than predicted by growth rates alone.

The mean square errors of the IBPMs were lower than the IBGM for six larval fish species out of nine in 1993 and for seven out of eight in 1994 (Table 5.4). This implies that the difference between the length frequency distributions observed in the

field and predicted by the IBPM is closer to zero than if predation is not included (IBGM). The better fit by the IBPM in 1993 was largely due to the predicted impact of adult capelin whereas in 1994, the size-selective impact of crustaceans was more apparent. For most larval fish species, there was a good proportion of the most abundant size class observed in field distributions that could not be explained by growth rates or by predation estimated in this study (e.g., Figures 5.4, 5.6). There may be other factors coming into play such as immigration or unaccounted production. Even if adding predation provided a better fit to the field length frequency distribution, the improvement was not statistically significant (except, *Pleuronectes ferrugineus*, 1994) (Table 5.4).

Inverse method to estimate predation by adult capelin

The IBPM predicted for several larval fish species that all 10 000 individuals could be consumed by the adult capelin population (Table 5.3). The abundance of adult capelin derived by the inverse method ranged from 0.18 to $1.0 \cdot 1000 m^{-3}$ (Table 5.5) which is realistic enough to generate the same order of mortality estimated previously in Conception Bay (Pepin 1993). Under these calculated abundance levels of adult capelin, surviving fish larvae were also smaller than predicted by growth rates alone (Figures 5.4, 5.7, 5.11). This inverse method generated three lower mean square error for the IBPM than the mean square error of the IBGM for 1993 and one more for 1994 (Table 5.4). When the abundance of adult capelin was estimated with the aerial surveys

and thus mortality was extremely high ($Z > 0.8\ d^{-1}$), the IBPM was unable to predict the length frequency distribution of *Mallotus villosus* (Figure 5.8) and of *Ulvaria subbifurcata* (cohort 2, Figure 5.12). However, when mortality was decreased with the inverse method, the mean square error was slightly lower (Table 5.4). Thus, the IBPM was better able to predict the changes of their length frequency distribution observed in the field.

General results

Larval fish species of Conception Bay were not vulnerable to predation by crustaceans or by medusae in both survey years but were most vulnerable to predation by adult capelin. For most larval fish species, the IBPM predicted that the smallest individuals tend to survive predation by both invertebrates (when significant) and adult capelin. The IBPM was better able to predict the changes of the length frequency distribution of most larval fish species found in Conception Bay. However, even when I combined all probability values from the one-tailed variance ratio test, I found that there was no statistical evidence to conclude that the length frequency distribution was less variable predicted by the IBPM than by the IBGM (Table 5.6). Furthermore, as the range and mean length of larval fish species increased, the mortality due to adult capelin predicted by the IBPM also increased (Table 5.3). This suggests that as fish larvae grow, they become more vulnerable to predation by adult capelin and that larger

larval fish species are more vulnerable to predation by adult capelin than smaller larval fish species.

Discussion

This study demonstrated that adding predation by adult capelin to an IBM decreased the variability between the length frequency distributions predicted by the model and observed in the field for all larval fish species except *Clupea harengus* (1993), *Ulvaria subbifurcata* (cohort 2, 1993) and *Pseudopleuronectes americanus* (1993, 1994). The IBPM predicted quite well the length frequency distribution for abundant larval fish species with a wide range of sizes and for cohorts composed of larger individuals. In general, the IBPM also did better when the time between the initial and final samples was longer (i.e., the 1994 survey). However, it is important to note that adding predation in the IBM was not a statistical significant improvement in predicting changes in the length frequency distribution of larval fish species in Conception Bay.

This study demonstrated that even under the highest observed abundance of invertebrate predators, size-selective removal of individuals could not be detected from length frequency distributions observed in the field. This was due primarily to the low overall mortality rates imposed by these predators on the cohorts of fish larvae present in Conception Bay in 1993 and 1994. Although for three larval fish species in 1994 (*Clupea harengus*, *Liparis gibbosus*, and *Mallotus villosus*), including predation by

crustaceans decreased the variability of the length frequency distribution predicted by the model. In contrast, this study showed that adult capelin have the potential, given gross estimates of adult capelin abundance based on aerial surveys, to consume significant amounts of fish larvae in Conception Bay. I demonstrated, with the inverse method, that previously observed mortality rates (Pepin 1993) could be attained with a relatively less abundant community of adult capelin. These previously observed mortality rates may explain significant changes in the length frequency distributions of fish larvae in Conception Bay in 1993 and 1994.

Miller (1997) argued that it is imperative to critically assess our ability to detect phenotypic selection (in this case, size-selective mortality) with field data. While he investigated the use of mean-variance approaches, residual analysis techniques and assessment of phenotypic reconstruction, I have used IBMs to predict the changes in the length frequency distributions of a multispecies larval fish community in Conception Bay. Miller (1997) effectively demonstrated that the mean-variance techniques could only detect delayed size-selective mortality as it relied on a sufficient period of growth between sampling periods. I found that even with longitudinal data (reconstruction of the length frequency distribution with a growth rate estimate), size-selective mortality is more easily predicted when there is a longer period of time between the first and last samples (1994 survey). In earlier results, I demonstrated that the evidence of size-selective mortality was stronger if mortality occurred during a restricted period which could be due to either restricted spatial and temporal overlap

between prey and predator (Chapter III) or differential growth rates among individuals or cohorts (Chapter IV). In addition, Miller's (1997) simulations suggested that the problem of error propagation is particularly acute when there is high mortality between successive censuses and recommended that the timing of sampling should be adjusted to scale with the expected mortality rate. I have shown in Chapter III as well as in this study, that the overall losses due to predation must be particularly high in order to detect significant departures from length frequency distributions of surviving fish larvae. Furthermore, scaling sampling protocol to the overall mortality observed by the cohorts requires prior knowledge of that mortality which may not be realistic.

The IBPM is based on the encounter model of Gerritsen and Strickler (1977) which assumes random spatial distribution of prey and predator. Under this assumption, I have identified specific conditions under which we might expect size-selective mortality of individuals. Pelagic fishes, especially capelin, are highly aggregated on the beaches and along the coasts of the bays (Templeman 1948). This feature is exploited in the aerial survey methodology used to assess biomass of spawning capelin in Newfoundland (Nakashima 1995). It is possible that the spatial distribution of predators may influence the size-selective mortality of fish larvae. Williamson and Stoeckel (1990) investigated how the spatial distribution of prey and predator may affect the predation risk of zooplankton and found that risk from some predators could be consistently under-estimated if a random distribution of predators is assumed. Under random spatial distribution of pelagic fishes, I found that capelin have the potential to

consume unrealistically high amounts of fish larvae. However, I demonstrated that given previously published estimates of mortality rates for the larval fish species of Conception Bay (Pepin 1993), lower abundance of pelagic fishes may still consume significant amounts of fish larvae. I propose that future studies should investigate how a non-random spatial distribution of pelagic fishes affects the characteristics of surviving fish larvae. McGurk (1986) proposed a model of mortality-patchiness interaction for fish eggs and larvae by including a spatial patchiness based on Lloyd's index for fish eggs and larvae. The spatial distribution of predators as well as the timing of their encounters with cohorts of fish larvae may be important factors of larval survival as suggested by Williamson et al. (1989) for zooplankton prey-predator interactions. If the spatial distribution of prey and predator have the potential to affect the overall mortality of a population of prey, it may also affect the size-selective nature of mortality and our ability to detect the size-selective removal of individuals (as in Chapter III).

DeAngelis and Huston (1987) identified that if a narrowing of the size distribution is not due to deceleration growth rates, then it can be due to stabilizing size-selective mortality. In 1993, *Liparis atlanticus*, *Pseudopleuronectes americanus*, *Stichaeus punctatus* (cohort 1), and *Ulvaria subbifurcata* (cohort 2) all displayed a narrowing of size distribution. Unfortunately, I cannot rule out decelerating growth rate as an explanation.

Miller (1997) argued that a growth rate derived from larvae experiencing both growth and phenotypic selection may be a biased estimate and that the conclusions

reached in studies where this bias may occur could potentially be misleading. To investigate the potential of growth rate bias, I varied the estimate of mean growth rate of *Hippoglossoides platessoides* by ±10%. I have chosen *H. platessoides* because the IBPM (with adult capelin) gave the best results in terms of predicting the length frequency distribution (lowest mean square error) and because the range of sizes was much larger than for other larval fish species. I compared the new predictions of both models given the changes of the mean estimate of growth rate with the length frequency distribution observed in the field (Figure 5.14). The one-tailed variance ratio test would have been significant if the mean growth rate was under-estimated by 10% (Table 5.7). However, if the mean growth rate was over-estimated by 10%, the addition of predation would not have provided a better prediction of the observed length frequency distribution. It would seem then that extremely precise estimates of mean growth rate are important in attempting to detect size-selective removal of individual fish larvae from changes of the length frequency distributions. Some may argue that otolith can provide accurate estimates of individual growth rates (e.g., Campana 1990). Pepin and Dower (*unpublished data*) calculated an error of ±3% of mean growth rates for *Ulvaria subbifurcata*. However, Miller et al. (1999) demonstrated that otolith-based attempts to backcalculate the size of cod larvae may be prone to substantial error. To date, there is no reason to believe that this error propagation may not occur for other larval fish species. At the very least, if I had an independent estimate of mean growth rates, I could have had an indication about the direction of size-selective mortality of

individuals and if the prediction of the IBPM was heading in the right direction. There is no reason to believe that the gross estimates of mean growth rates used in this study are inaccurate. These estimates are well within the range of previous ones. Sampler bias is also an unlikely contributor to error. The Tucker trawl is a proven effective sampling gear for a multispecies community of fish larvae (Pepin and Shears 1997). In Conception Bay, most larval fish species hatch at a mean length greater than the lower length caught by the Tucker trawl (i.e., 2 mm, Pepin and Shears 1997) (Table 5.8). Furthermore, the IBPM predicted that larger individuals were more vulnerable to predation but these individuals were also more effectively sampled by the Tucker trawl (Pepin and Shears 1997). So if the prediction was wrong, I would have expected to have catch these larger fish larvae using this sampling gear.

This study has significant implications for the study of larval fish survival. It appears that including predation in an IBM provided better predictions of the length frequency distribution of survivors, but these were not statistically significant. The predictions were closely related to the accuracy and precision of the mean growth rates of the cohorts of larval fishes. Furthermore, there was strong evidence that the mortality due to predation by invertebrates may not be substantial enough to allow detection of size-selective removal of individuals. However, mortality due to predation by adult capelin may lead to dramatic effects on the characteristics of surviving fish larvae.

Table 5.1: Estimate of mean (μ) and standard deviation (SD) of the growth rate distribution of several species of fish larvae in Conception Bay in 1993 and 1994. For *Stichaeus punctatus* and *Ulvaria subbifurcata*, I identified two cohorts. The growth rate was estimated with an individual-based simulation model which allowed variation in individual growth rates of a species-specific cohort (IBGM). The best distribution (either N, normal or L, log-normal) of growth rates is also stated.

	1993			1994		
	μ	SD	Type	μ	SD	Type
Clupea harengus	0.2	0.05	N	0.08	0.03	N
Hippoglossoides platessoides	.	.	.	0.3	0.5	L
Liparis atlanticus	0.08	0.005	N	0.04	0.005	N
Liparis gibbus	.	.	.	0.07	0.05	N
Mallotus villosus	0.15	0.03	N	0.1	0.03	N
Pseudopleuronectes americanus	0.15	0.05	N	0.07	0.5	L
Pleuronectes ferrugineus	0.12	0.03	N	0.06	0.04	N
Stichaeus punctatus	0.39	0.05	N	.	.	.
Cohort 2	0.44	0.05	N	.	.	.
Ulvaria subbifurcata	0.05	0.01	N	0.09	2.0	L
Cohort 2	0.15	0.05	N	.	.	.

Table 5.2: Literature values of growth rates for the larval fish species collected in Conception Bay in 1993 and 1994. Minimum and maximum larval length from which the growth rates were calculated or measured are also indicated as well as the temperature of the laboratory (L) or the field study (F).

Species	Growth rate ($mm \cdot d^{-1}$)	Length (mm)	Temp. (°C)	Reference (Field, Laboratory)
C. harengus	0.27 - 0.35	6 - 16	9.5	(L) Gamble et al. (1981) [a]
	0.14 - 0.41	6 - 50	7.8	(L) Gamble et al. (1985) [a]
	0.11 - 0.42	<15 - 23	8 - 14	(L) Geffen (1982) [a]
	0.07, 0.23, 0.35	7.4 - 14.3	10.6-11.2	(F) Heath and Rankine (1988)
	0.14, 0.17, 0.26	7.2 - 18.0	8.0	(F) Munk et al. (1986) [a]
	0.23 - 0.64	9 - 16	.	(F) Peltonen (1990) [a]
H. platessoides	0.34	3 - 15	4.7 [b]	(F) Pepin et al. (1995)
	0.33	4 - 11	2.2 [b]	(F) Pepin et al. (1995)
L. atlanticus	0.07	3 - 8	4.7 [b]	(F) Pepin et al. (1995)
L. gibbus	0.29	7 - 19	2.2 [b]	(F) Pepin et al. (1995)
M. villosus	0.13 - 0.25	1st feed	4 - 14 [c]	(L) Frank and Leggett (1986)
	0.20 - 0.35	5 - 33	8	(F) Jacquaz et al. (1977) [a]
	0.09	6.3 - 6.0	5 - 11	(L) Williams et al. (1996)
P. americanus	0.14	6.6 - 8.6	14.5	(L) Laurence et al. (1978) [a]
	0.16	3.5 - 6.0	14.5	(F) Pearcy (1962) [a]
	0.09	3.7 - 8.3	5 - 11	(L) Williams et al. (1996)
P. ferrugineus	0.08 - 0.19	11.8 - 22.1	7	(L) Benoît (1999)
	0.12 - 0.36	12.6 - 32	11	(L) Benoît (1999)
	0.14 - 0.41	13.4 - 26	13	(L) Benoît (1999)
S. punctatus	0.25	10 - 18	2.2 [b]	(F) Pepin et al. (1995)
U. subbifurcata	0.12	6.3 - 11.5	5 - 11	(L) Williams et al. (1996)
	0.38	4 - 13	4.7 [b]	(F) Pepin et al. (1995)
	0.17	4 - 11	2.2 [b]	(F) Pepin et al. (1995)

Note: [a] from Pepin (1991), [b] Laprise and Pepin (1995), [c] Frank and Leggett (1982).

Table 5.3: Instantaneous mortality rates of a cohort of 10 000 fish larvae (Z, d^{-1}) predicted from the individual-based predation model. I also give the range of mortality estimates previously reported for Conception Bay (Pepin 1993). Growth rates were estimated with the individual-based growth model. A mortality estimate of '0' implies that no fish larvae were consumed by the predator population during the simulation whereas a mortality estimate of '∞' implies that all 10 000 fish larvae were consumed by the predator population during the simulation. A dot indicates that the species was not captured that year or was not selected for analysis (see Methods for selection criteria). N/A, information not available.

		1993				1994			
	Pepin (93)	Length	Crusta	Medu	Capelin	Length	Crusta	Medu	Capelin
C. harengus	1 - 3%	8 - 19 mm	0.0006	0	∞	12 - 24 mm	0.03	0.0001	∞
H. platessoides	20 - 26%	.	.	.	.	4 - 24 mm	0.009	0.00007	0.16
L. atlanticus	3 - 6%	3 - 6 mm	0	0	0.51	3 - 8 mm	0.00006	0	0.04
L. gibbus	1 - 6%	.	.	.	.	22 - 29 mm	0.10	0.0004	∞
M. villosus	12 - 26%	3 - 11 mm	0	0	0.83	4 - 13 mm	0.0006	0	0.09
P. americanus	10 - 20%	1 - 5 mm	0	0	0.18	2 - 6 mm	0.000007	0	0.02
P. ferrugineus	N/A	1 - 4 mm	0	0	0.15	1 - 9 mm	0.00002	0	0.01
S. punctatus	10 - 16%	8 - 18 mm	0.0005	0.00001	∞	.	.	.	.
Cohort 2		15 - 25 mm	0.004	0.00004	∞				
U. Subbifurcata	4 - 22%	4 - 9 mm	0	0	0.97	4 -17 mm	0.002	0.00001	0.13
Cohort 2		4 - 9 mm	0	0	0.89				

Table 5.4: Mean square errors calculated for the individual-based growth model (IBGM) and the individual-based predation models (Medusae, Crustaceans, and Fish). Inv. Fish refers to the inverse method of estimating capelin predation. The one-tailed variance ratio test for the hypothesis that the length frequency distribution is less variable if predicted by the IBPM than by the IBGM (*P*-values are in parantheses). NA, not applicable because the IBPM predicted no mortality would occur or mortality rates higher than 0.8 d^{-1}. A dot indicates that the species was not captured or was not selected for analysis.

	1993					1994				
	IBGM	Medusa	Crusta	Fish	Inv. Fish	IBGM	Medusa	Crusta	Fish	Inv. Fish
C. harengus	0.0047	NA	0.0049 (0.53)	NA	0.0066 (0.71)	0.0043	0.0042 (0.48)	0.0041 (0.46)	NA	0.0069 (0.78)
H. platessoides	.	.	.	.	.	0.001	0.001 (0.5)	0.0009 (0.38)	0.0006 (0.1)	NA
L. atlanticus	0.0873	NA	NA	0.0752 (0.45)	NA	0.0363	NA	0.0363 (0.5)	0.0104 (0.13)	NA
L. gibbus	.	.	.	.	.	0.0152	0.015 (0.49)	0.0121 (0.37)	NA	0.0128 (0.4)
M. villosus	0.0030	NA	NA	0.0587 (0.9998)	0.0024 (0.38)	0.0397	NA	0.035 (0.44)	0.0541 (0.65)	NA
P. americanus	0.0499	NA	NA	0.0613 (0.59)	NA	0.0193	NA	NA	0.0229 (0.56)	NA
P. ferrugineus	0.0123	NA	NA	0.0041 (0.15)	NA	0.0719	NA	0.0719 (0.5)	0.0165 (0.03)	NA
S. punctatus	0.0156	0.0156 (0.5)	0.0165 (0.53)	NA	0.0135 (0.41)	.	.	.	.	.
Cohort 2	0.0171	0.0171 (0.5)	0.0170 (0.5)	NA	0.0170 (0.49)					
U. subbifurcata	0.0474	NA	NA	0.0137 (0.1)	0.0272 (0.28)	0.0084	0.0084 (0.5)	0.0082 (0.48)	0.0056 (0.25)	NA
Cohort 2	0.0149	NA	NA	0.0380 (0.86)	0.0179 (0.59)					

Table 5.5: Estimated abundance of adult capelin ($1000m^{-3}$) calculated with an individual-based predation model needed to generate instantaneous mortality rates estimated previously in Conception Bay (Pepin 1993). NA, the inverse method is not applicable because the field abundance observed during our survey predicted mortality rates lower than $80\% \cdot d^{-1}$. A dot, the species were not collected or not selected for analysis for that year.

	Abundance of capelin (# / $1000m^3$)	
	1993	1994
C. harengus	0.27	0.55
L. gibbus	.	0.215
M. villosus	0.72 - 1.0	NA
S. punctatus	0.285 - 0.333	.
Cohort 2	0.183 - 0.212	
U. subbifurcata	0.495 - 0.81	NA
Cohort 2	0.48 - 0.82	

Table 5.6: Fisher's technique for combining all probabilities from the one-tailed variance ratio test for each predator type (Table 5.4). The combination of all *P*-values is distributed as χ^2 with (2×the number of probabilities) degrees of freedom (*df*).

Predator type	χ^2	*df*	*P*
Medusae	11.47	14	0.65
Crustaceans	17.07	22	0.76
Fishes	41.92	36	0.23

Table 5.7: Hypothetical variation (±10%) of mean growth rate ($mm \cdot d^{-1}$) of *Hippoglossoides platessoides* and the effect of bias in mean growth rate on the one-tailed variance ratio test (*F*-ratio). The mean square error is calculated as: Σ [RF(field)-RF(model)]^2/*df*, where RF is the relative frequency distribution observed in the field and predicted by the model (with or without predation) and *df* is the degrees of freedom (number of size classes -1).

Growth rate	MSE(growth)		MSE(predation)	*F*-ratio	*df*	*P*
0.27	0.00084	<	0.0014	0.59	20	0.87
0.3	0.00098	>	0.00056	1.76	22	0.1
0.33	0.00132	>	0.00038	3.00	24	0.005

Table 5.8: Mean hatch sizes (mm) and mean sizes at the start of the juvenile stage (mm) for the fish larvae species collected in Conception Bay during the 1993 or 1994 surveys. N/A, information was not available. Data were from Scott and Scott (1988) except when indicated.

Species	Hatch (mm)	Juvenile (mm)
C. harengus	4 - 10 6.5[a]	N/A
H. platessoides	4 - 6	18 - 34 > 25 [b]
L. atlanticus	N/A	N/A
L. gibbus	N/A	N/A
M. villosus	5 - 5.5[c]	N/A
P. americanus	2.9[c] 3.6[d,e] - 3.8[e]	7.5 - 8.3 [f]
P. ferrugineus	2 - 3.5	11.6 - 16[b]
S. punctatus	9.9 - 22.4[g]	N/A
U. subbifurcata	6.6[h]	18.4

Note: [a] Klinkhardt (1986), [b] Van Guelpen (1980), [c] Pepin (1991), [d] Klein-MacPhee et al. (1984), [e] Buckley (1982), [f] Chambers and Leggett (1987), [g] range of length of larvae from Grigor'ev (1993), [h] Williams et al. (1996).

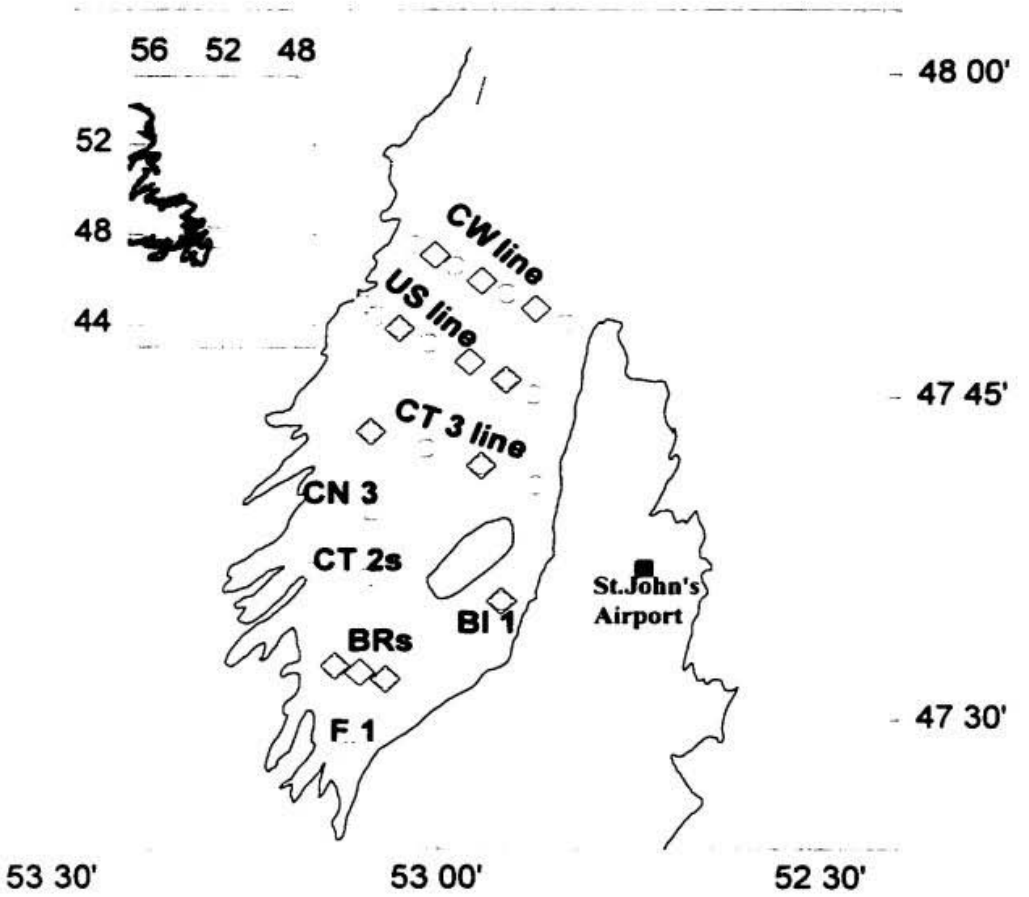

Figure 5.1: Survey stations of 1993 (diamonds) and 1994 (circles) in Conception Bay, Newfoundland, Canada. St. John's airport is indicated as a reference.

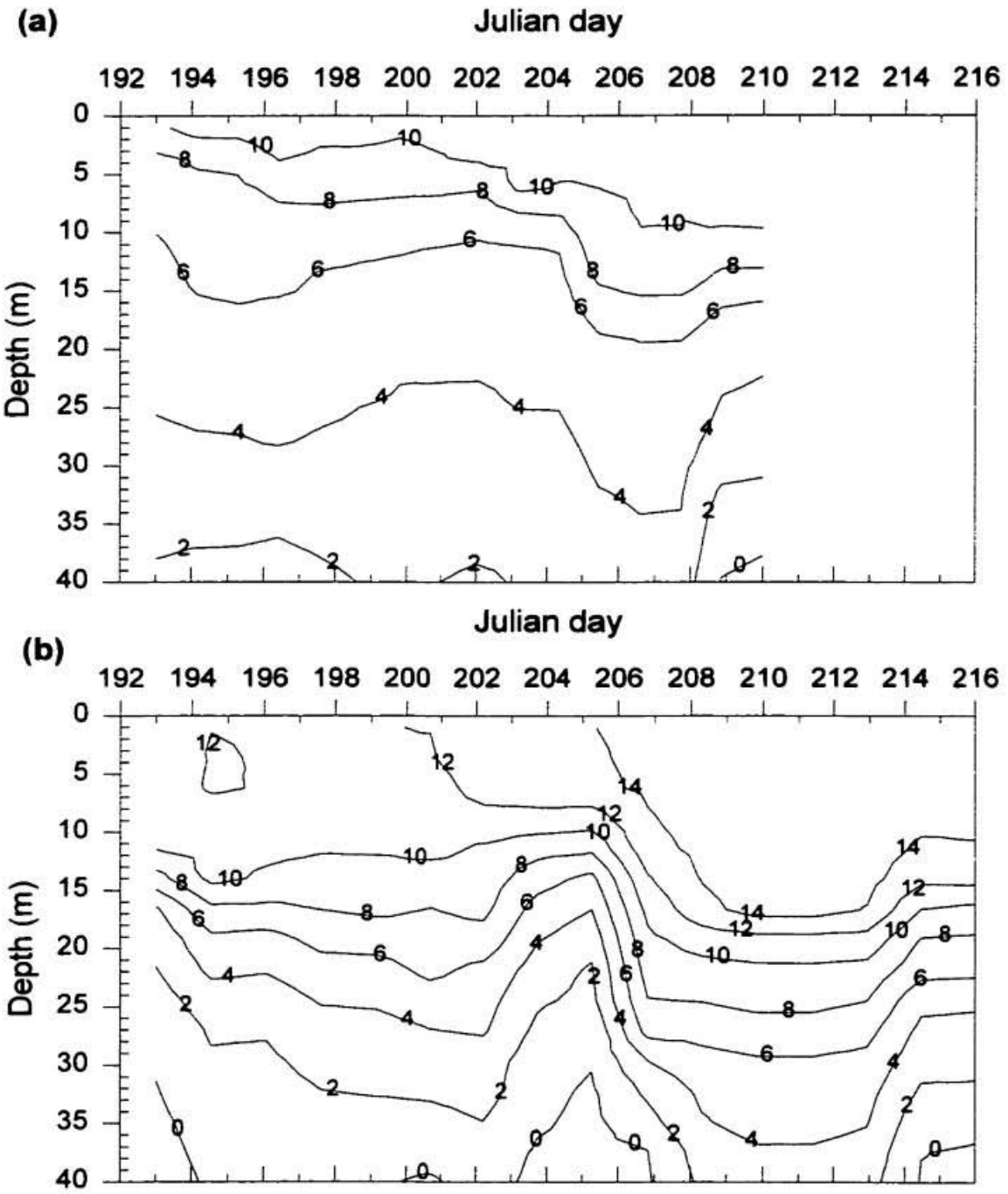

Figure 5.2: Average water temperature at each 1m depth interval each throughout the **(a)** 1993 survey and **(b)** 1994 survey of Conception Bay. The water temperatures were measured at a fixed station in the middle of the Bay.

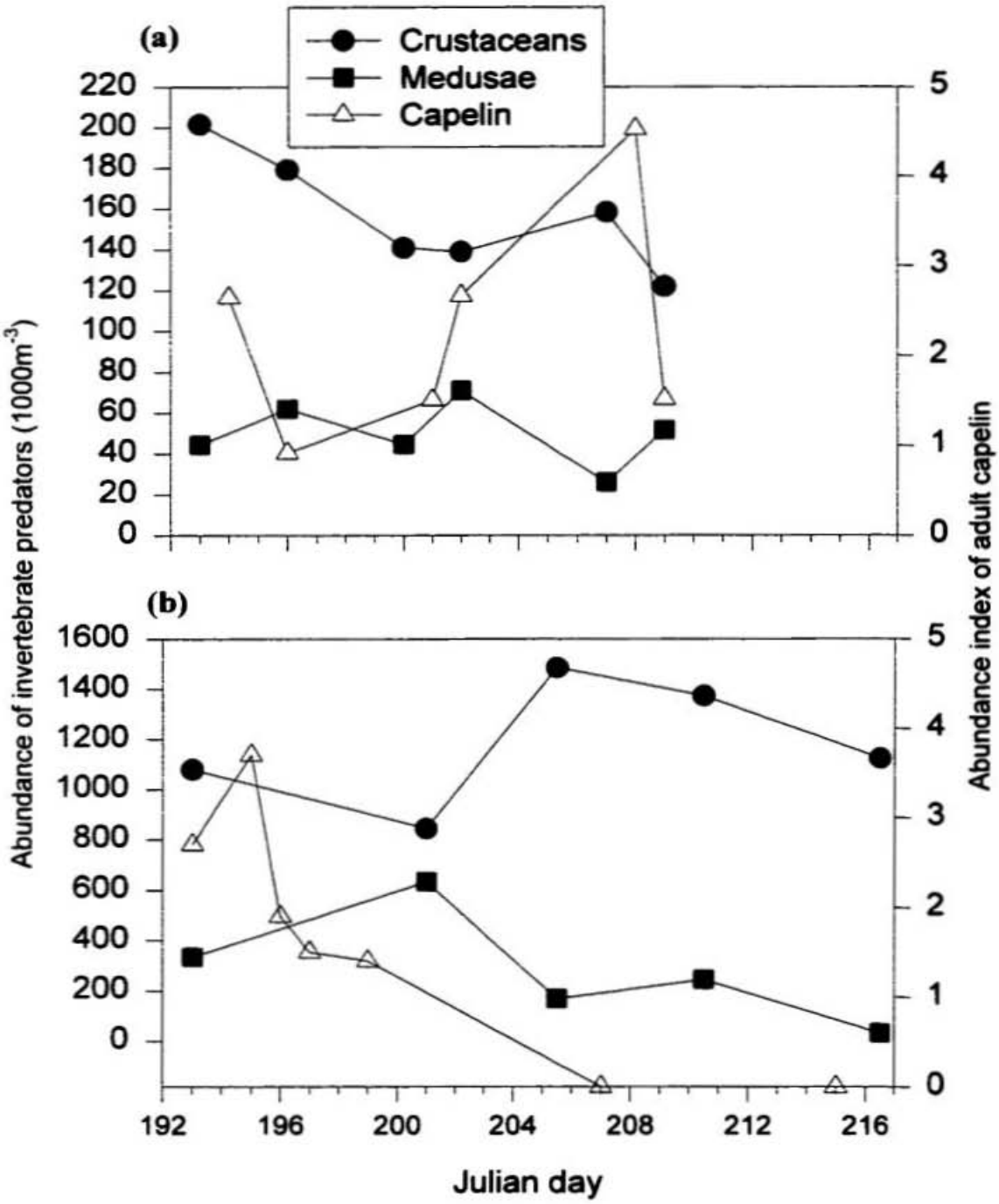

Figure 5.3: Predator abundance of invertebrate predators (1000 m^{-3}) and abundance index of adult capelin (based on aerial surveys) in Conception Bay in **(a)** 1993 and **(b)** 1994.

Title for Figures 5.4 to 5.13

(a) Length distributions of the cohort of fish larvae caught in 1993 and 1994. Julian days of samples (T) are given above each panel. **(b)** Length frequency distribution of the cohort observed on the last day of the surveys and the length frequency distribution modelled by the individual-based growth model (IBGM, thin line) and the individual-based predation model (IBPM). The length frequency distribution modelled by the IBPM are based on either the survivors of predation by crustaceans (dashed lines), medusae (dash-dotted lines) or capelin (dotted lines). **(c)** The size-specific difference between the relative frequency on the last day of surveys and the relative frequency modelled by either the IBGM or the IBPM. A positive difference implies that the model was unable to resolve that particular size class wheras a negative difference implies that the model predicts a size class to be observed in field samples when in fact it was not the case. If the model is perfect, there will be no difference between what is observed in field samples and what is predicted by the model and the percent difference will be zero.

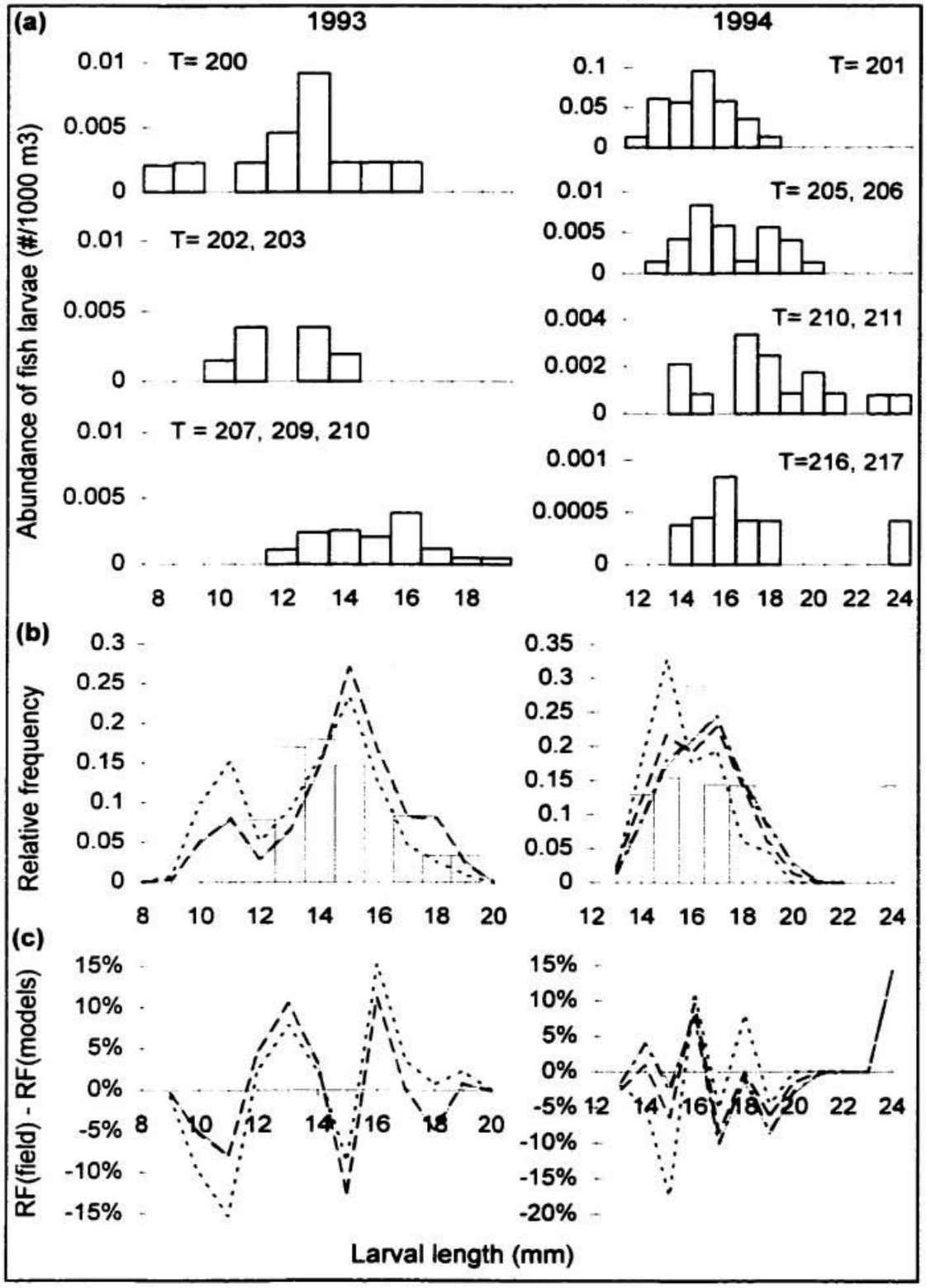

Fig. 5.4: *Clupea harengus*

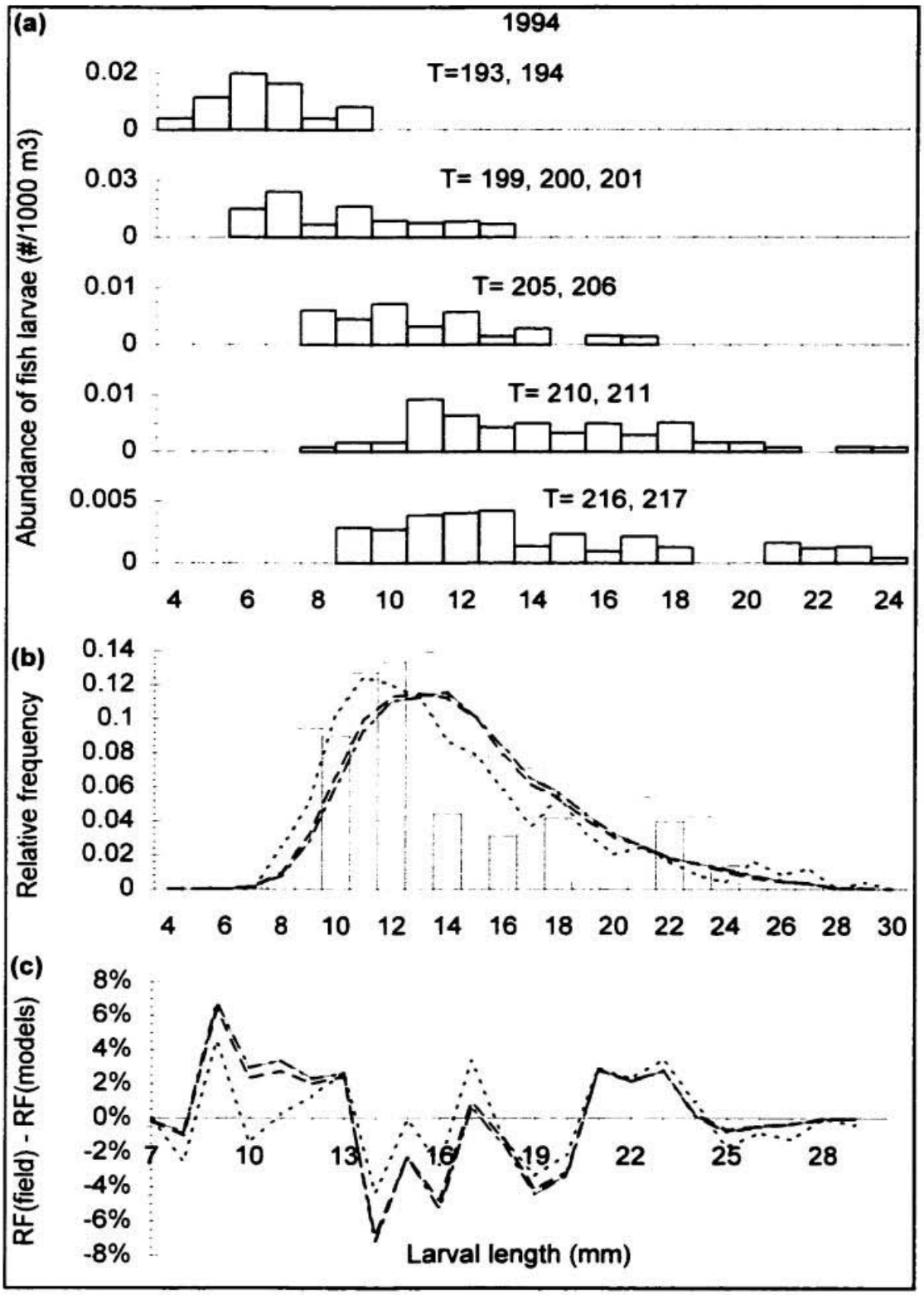

Fig. 5.5: *Hippoglossoides platessoides*

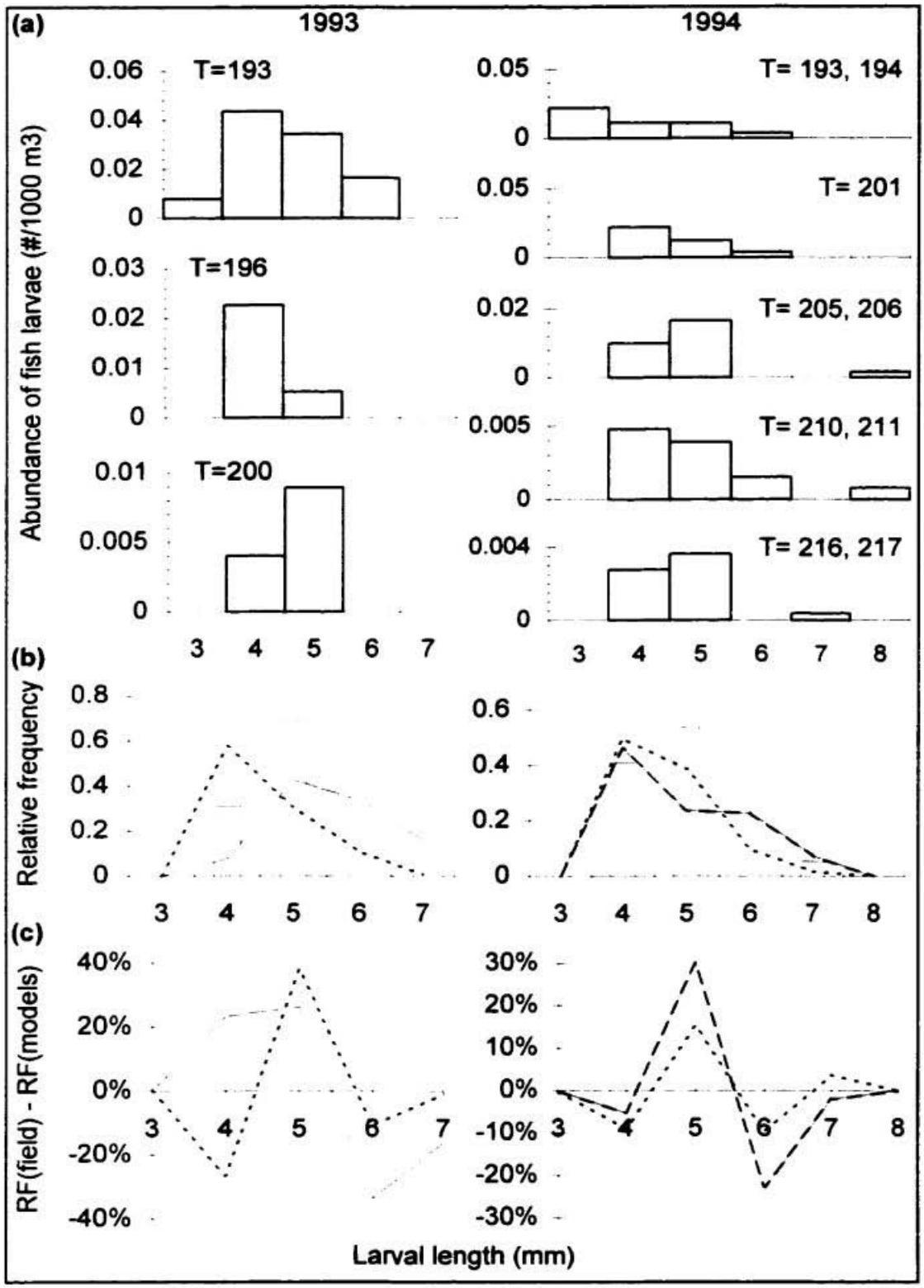

Fig. 5.6: *Liparis atlanticus*

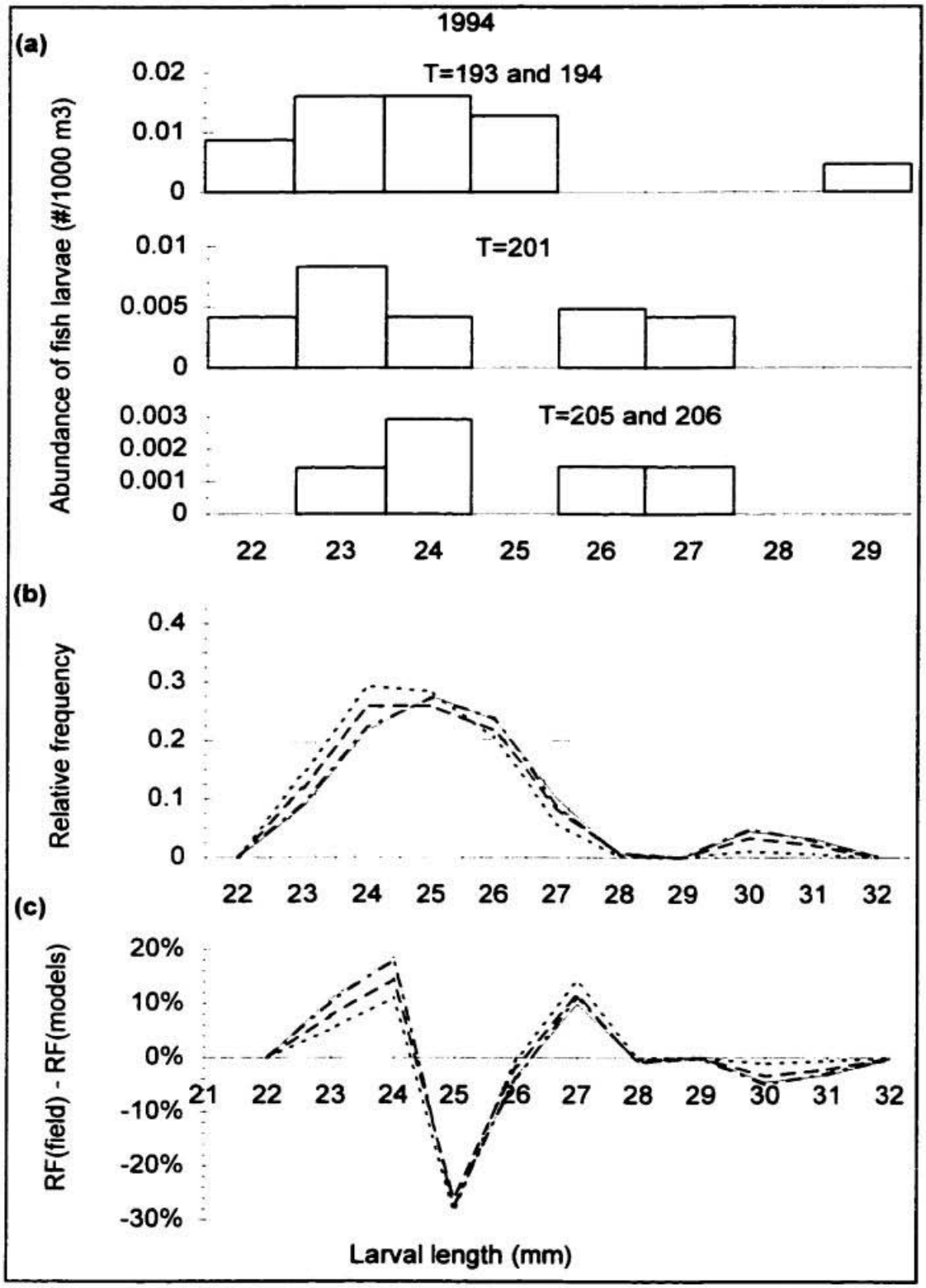

Fig. 5.7: *Liparis gibbus*

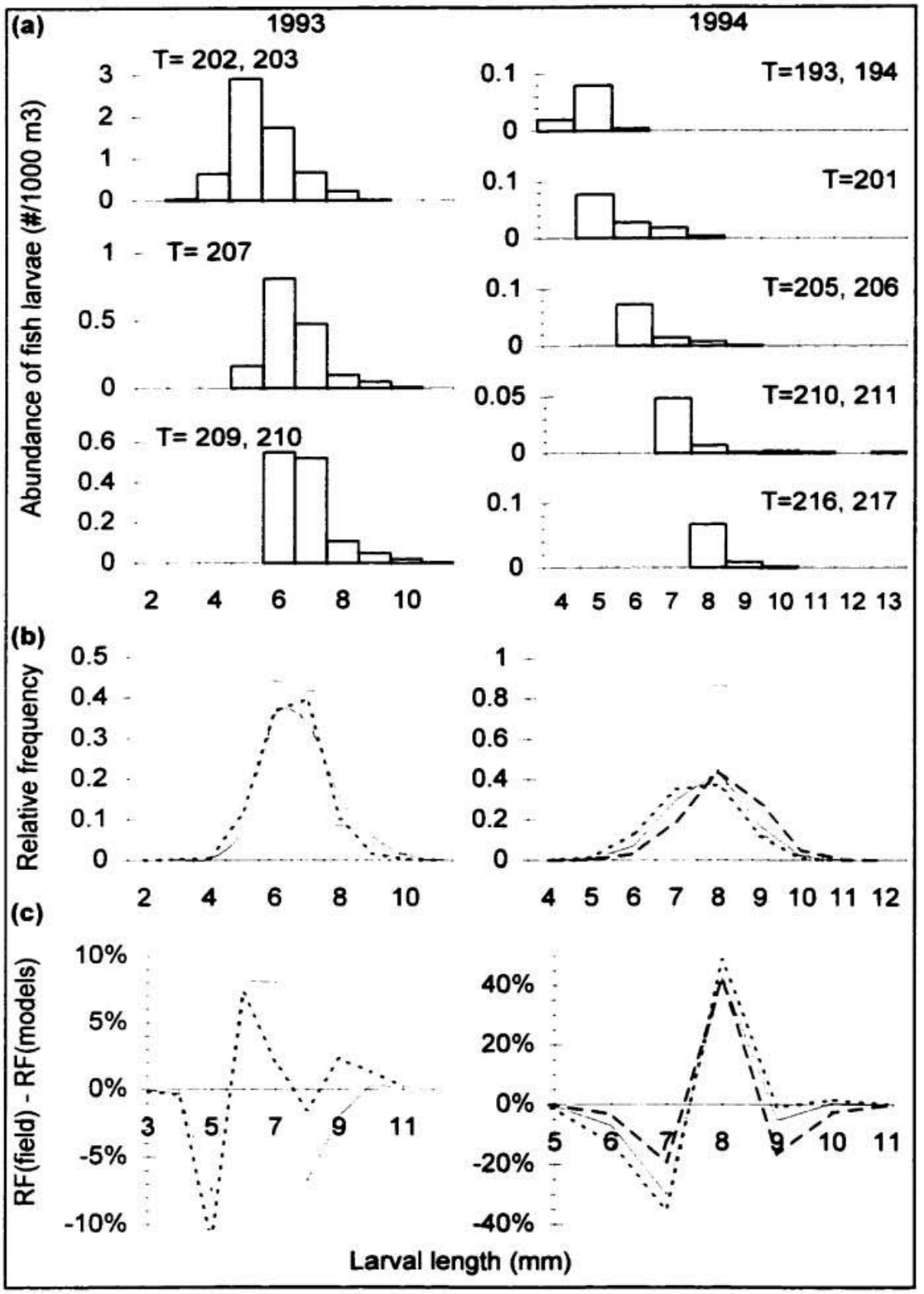

Fig. 5.8: *Mallotus villosus*

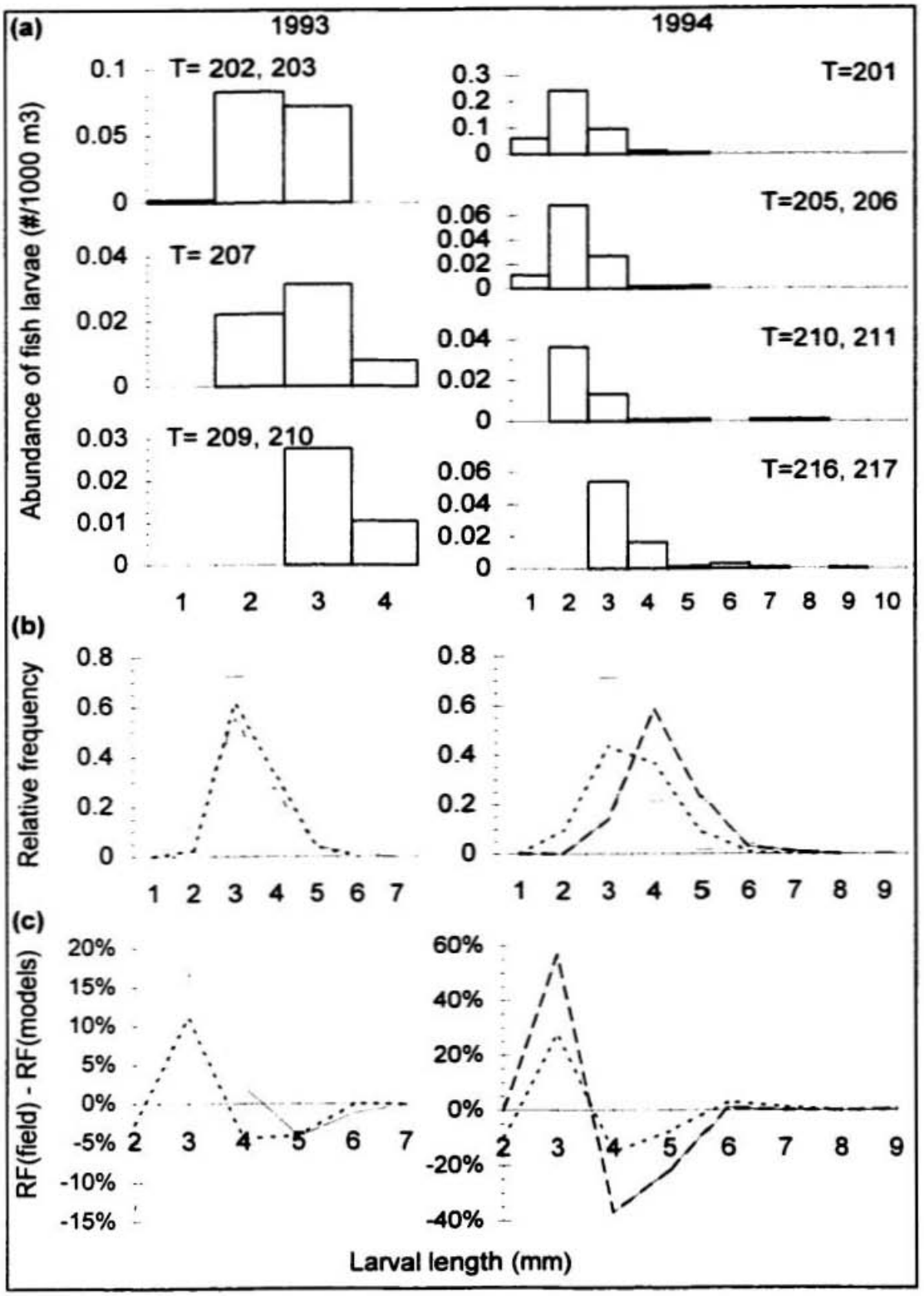

Fig. 5.9: *Pleuronectes ferrugineus*

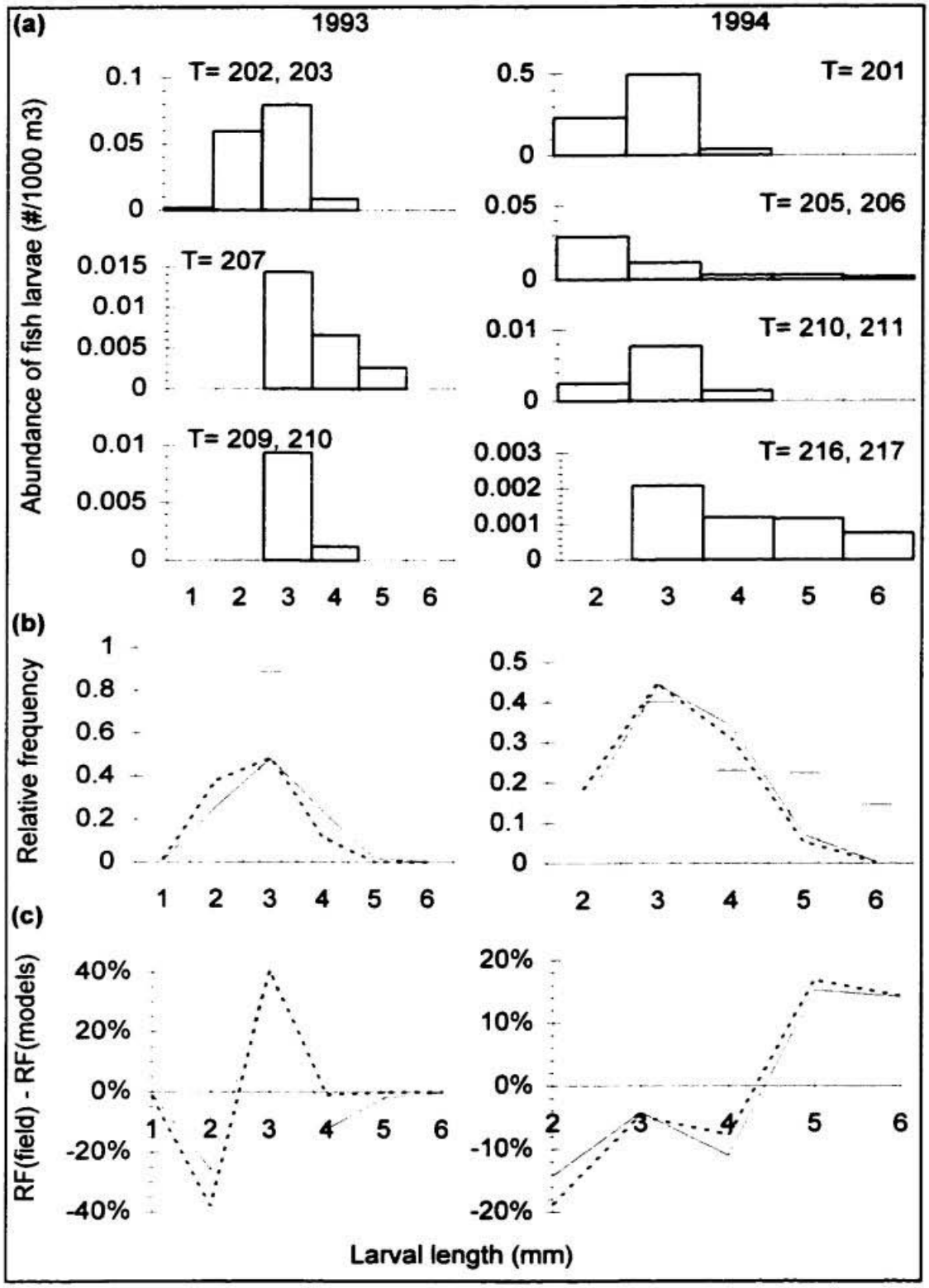

Fig. 5.10: *Pseudopleuronectes americanus*

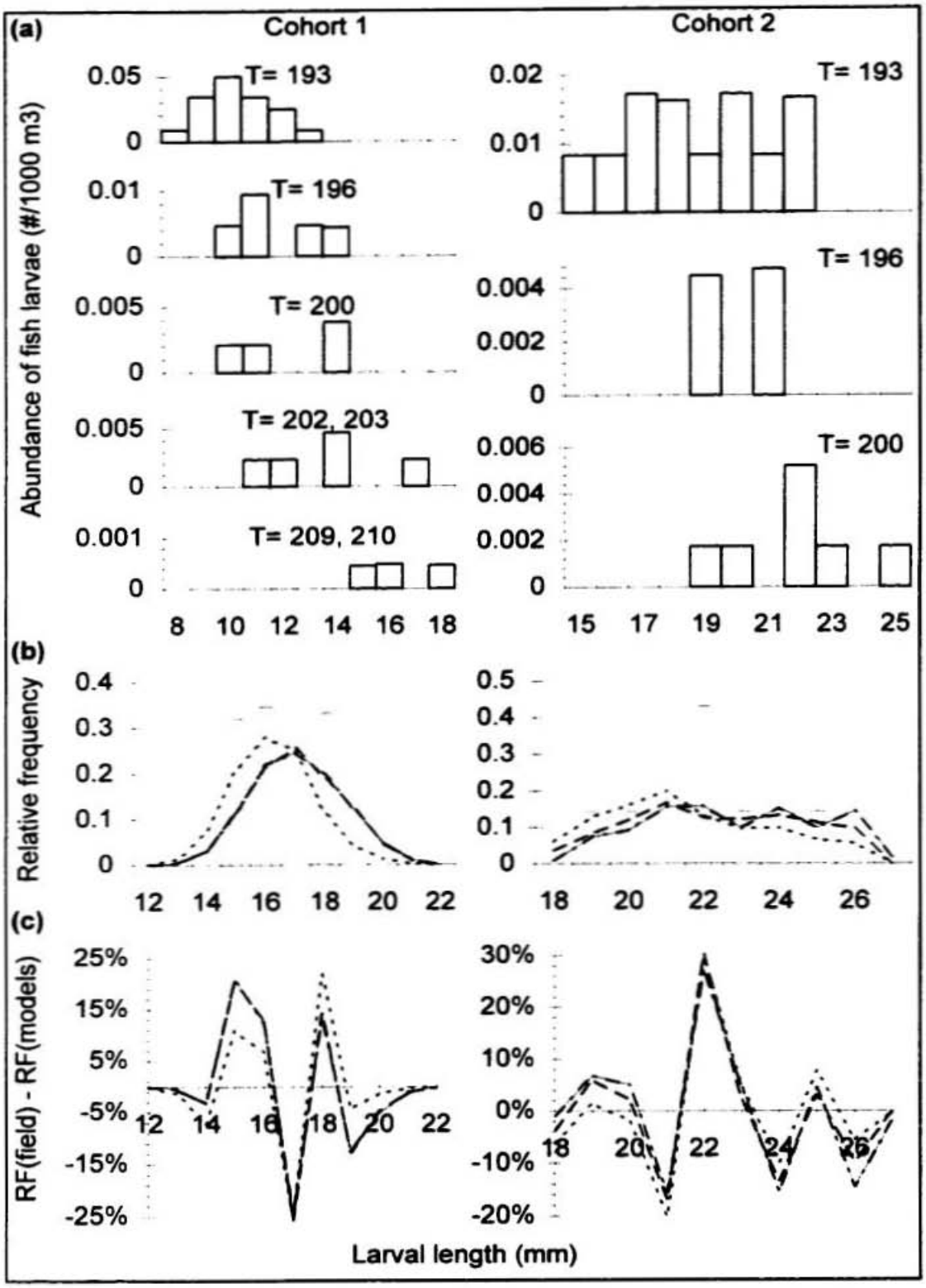

Fig. 5.11: *Stichaeus punctatus*

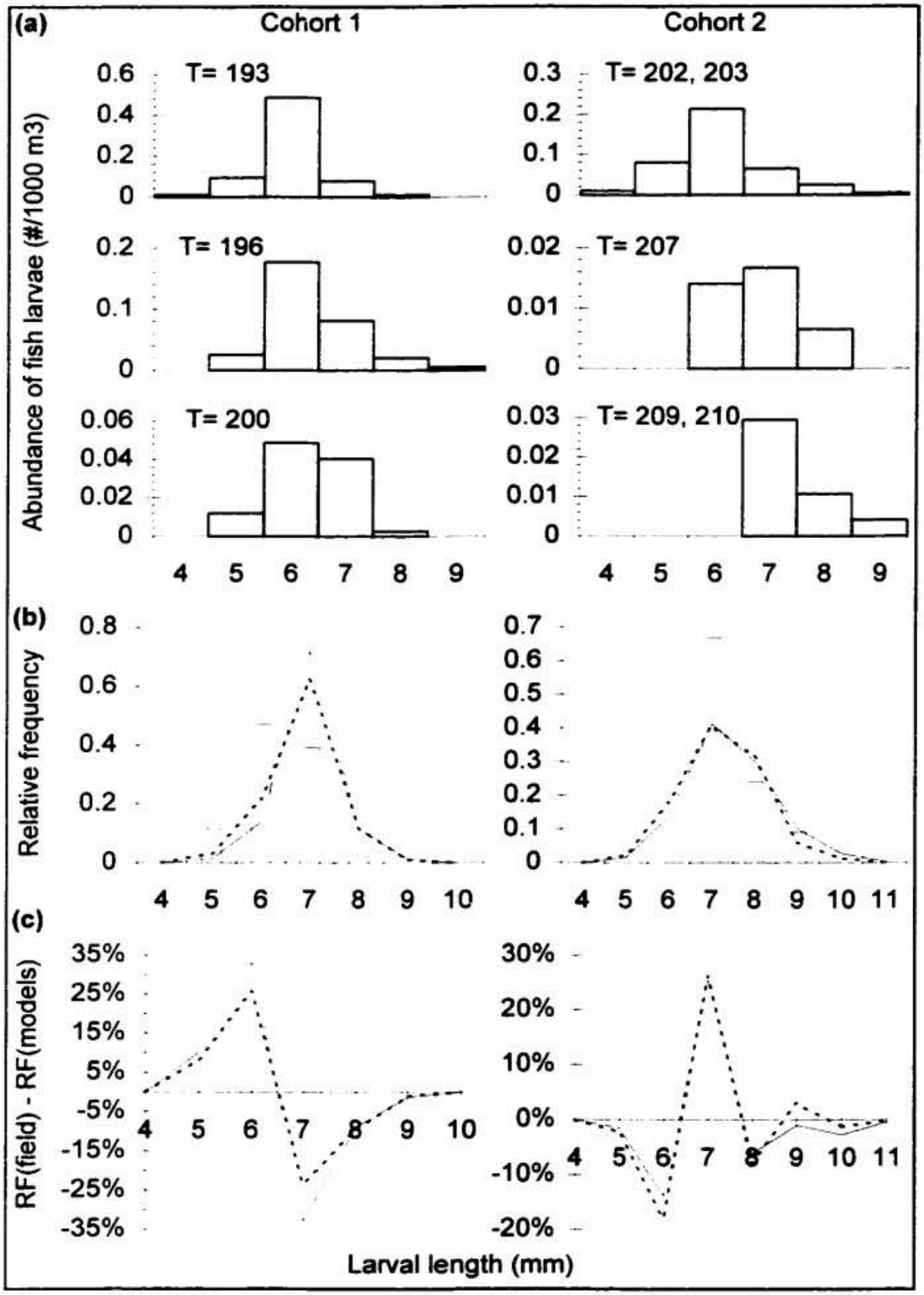

Fig. 5.12: *Ulvaria subbifurcata* 1993

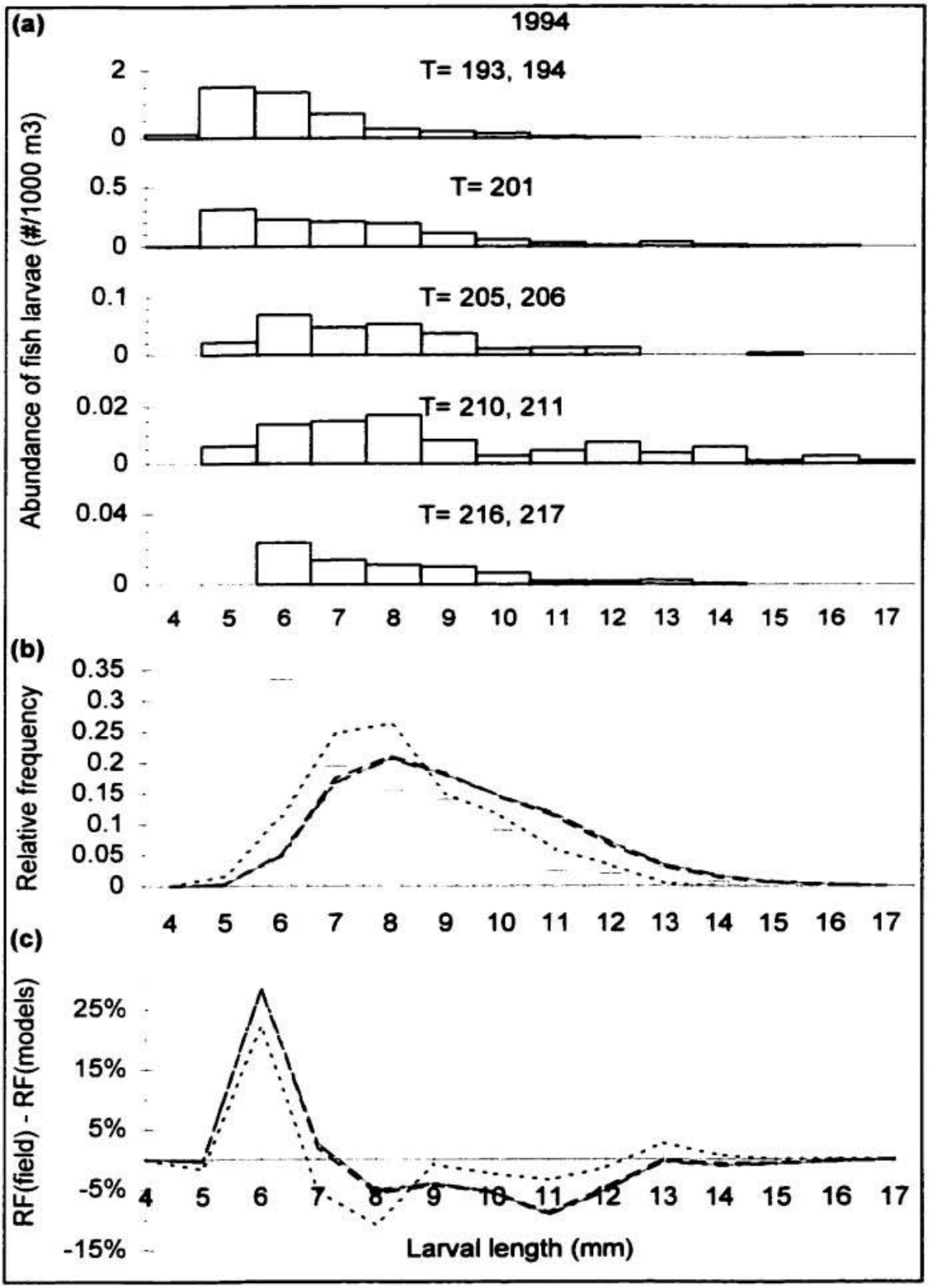

Fig. 5.13: *Ulvaria subbifurcata* 1994

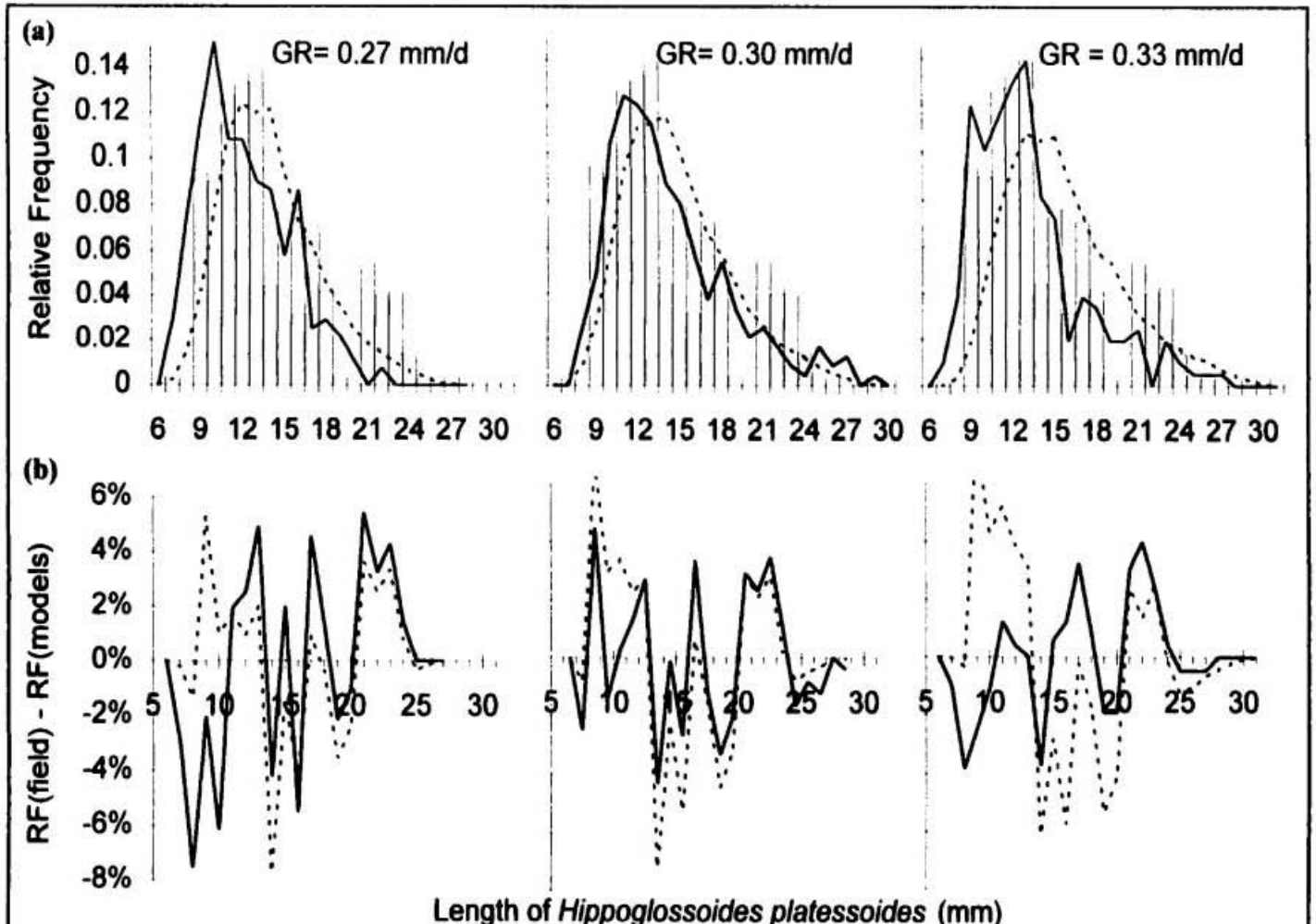

Figure 5.14: **(a)** Length frequency distributions of the modeled and field cohorts and **(b)** difference at each size classes between modeled and field cohorts given variations of 10% of the mean estimated growth rates of *Hippoglossoides platessoides*.

Chapter VI: **Conclusion**

From the introduction...

I originally intended to investigate the following question: Which larvae are better able to survive predation? Or, is it that size of fish larvae has little effect on their survival and survival is mostly affected by external factors? I have come to realize that the answer to this question is less important when I investigate how reliable the answer is or, in other words, the reliability of the conclusions. Are we actually able to detect size-selective removal of individuals from field samples? When one or two individuals survive out of a million, does it really matter how big they are and how fast they are growing? What is the actual difference in probability of survival of two individuals of different sizes? How is this difference affected by the precision of measurements of size of larvae? Ultimately, is this difference in survival significant? May we expect to detect such difference from field samples using current statistical analytical tools and sampling protocols?

What I have found in previous chapters

General empirical models predict that fish larvae measuring 10% the size of predators are most susceptible to predation. This pattern seems constant across a variety of experimental conditions and for at least four different types of predators.

When the encounter model of Gerritsen and Strickler (1977) was combined with a general empirical susceptibility model, I found that the size-dependent components of the two counteract each other. The encounter model predicted that larger fish larvae would encounter more predators than smaller larvae whereas the susceptibility model predicted that these larger individuals were less susceptible to predation than smaller ones. The detection of size-selective removal of individuals and thus the balance between these two models was closely related to the overall mortality of the cohort as well as the size of predators. I found that the predator characteristics (such as abundance and size) were most important in determining the number and length of survivors. Furthermore, differential timing of encounter between a cohort of fish larvae and the predator population may result in a significant size-selective removal of individuals at lower overall mortality rates suffered by the cohort.

I have demonstrated that the effect of the larval characteristics (such as length and growth rate) in determining the number, length and growth rate of survivors depends on the characteristics of the predator population. This implies that growing faster or being larger does not necessarily translate into a universal survival advantage

because it depends on the characteristics of the predator population. For example, if the predator population is composed of large pelagic fishes, growing faster or being larger may be a serious survival disadvantage.

Analysis of time-series samples from ichthyoplankton surveys demonstrated that the individual-based model was able to explain, for some larval fish species, changes of the length frequency distribution observed in the field in terms of the predatory fish population co-occurring in Conception Bay. In Conception Bay, fish larvae were not vulnerable to invertebrate predation and smaller fish larvae were better able to survive predation by adult capelin. The predictions of the model were highly sensitive to mean growth rate estimates.

The general theoretical framework of the early life history of fishes

The length of fish larvae at hatch is less variable than their length during the late larval period or early juvenile period. This spread of sizes is due mostly to growth rate variation among individuals (DeAngelis and Huston 1987, Benoît 1999). If the growth rates of individuals are distributed normally, we would expect that the variance of the size distribution of these individuals increases with time (DeAngelis and Huston 1987).

Mortality is time-dependent and decreases exponentially with it (Houde 1987). Mortality during the egg and yolk-sac stages are much higher than at the late larval and juvenile stages. Predation is a size-selective process. Detecting the effect of size-

selective predation requires variability of sizes among individuals within a cohort. Furthermore, the magnitude of the impact will influence our ability to detect the effect of size-selective mortality. I have shown theoretically in Chapter III that detecting size-selective removal of individuals from length frequency distributions requires a minimum larval mortality of at least 0.1 d^{-1}. If only 1% of individuals are removed from the population, it will be difficult to determine the size-selective nature of mortality. In contrast, if 50% to 95% of individuals are removed then the nature of mortality can more easily be assessed. For example, if nearly all of the 50 smallest individuals remain, we might conclude that mortality due to predation was highest for the largest individuals of the population, but if only one individual is removed, even if it is the largest individual, it will be difficult to establish with certainty that mortality due to predation selects the largest individuals. In addition to this "number" effect, there is also the importance of time. In order to detect size-selective removal of individuals, there must be enough time between the initial and final samples of individuals for the effect of variations in growth rates to be compounded in the length distribution of survivors (Chapter V but see Miller 1997).

Therefore, I argue that to detect size-selective removal of individuals from a population, three fundamental conditions must be met: 1) the population must be composed of individuals of variable sizes; 2) the overall mortality of the population must be high; and 3) the sampling time interval must be sufficient for the effect to be detectable. How variable the sizes of individuals need to be, how much mortality is

high enough and the amount of time needed will depend mostly on the difference in survival between different sizes of individuals, the precision of the size measurements, and the growth rates of individuals.

A simple example

If we take for granted that mortality is time-dependent, we might want to investigate how, under this mortality regime, we can potentially differentiate two different selection processes of surviving individuals. I define two theoretical cohorts of fish larvae. Both suffer time-dependent mortality but differ in terms of how surviving individuals are selected. Survivors are either picked at random or size-selectively by a population of predatory fishes of variable sizes. I chose to concentrate on the predatory fishes because as I have demonstrated in Chapter V, they seem to be major predators of ichthyoplankton in Conception Bay. I can then assess how well the selective nature of mortality can be inferred from samples of these two populations of survivors.

Let's define a theoretical parent population of yellowtail flounder larvae at hatch: 10 000 individuals at time zero, with mean length of 2.19 mm (SD=0.12) and with a mean growth rate of 0.23 $mm \cdot d^{-1}$ (SD=0.06) (Benoît and Pepin 1999). Metamorphosis of yellowtail flounder occurs between 75 and 120 days after hatch (Benoît and Pepin 1999). I will ignore that there is variability among individuals in the timing of the juvenile stage and will assume that the population has reached

metamorphosis 90 days after hatch. Based on Houde's (1987) hypothesis of time-dependent mortality, I define an instantaneous mortality rate of 0.1 d^{-1} for the first 30 days (day 1 to 30), of 0.055 d^{-1} during the following 30 days (day 31 to 60), and of 0.04 d^{-1} during the last 30 days of the larval stage (day 61 to 90).

Now, let's define 2 cases of selective mortality. For the first scenario, survivors are chosen at random whereas for the second scenario, survivors are size-selectively chosen by a predatory fish population of variable sizes (as defined in previous chapters). The mortality rates translate to 500 or 499 survivors on day 30, 370 or 367 on day 60 and 275 or 276 on day 90 (Table 6.1). The magnitude of mortality is identical but the process of selection of survivors is different. The length frequency distribution of these two populations are significantly different from one another after 30, 60 and 90 days (based on Kolmogorov-Smirnov goodness of fit for continuous distributions: D=0.14 > $D_{0.05}$=0.09, D=0.21 > $D_{0.05}$=0.1, and D=0.29 > $D_{0.05}$=0.12, respectively). The mean length of both populations are also significantly different after 60 and 90 days ($t_{0.05(2)735}$=4.5, p<0.001 and $t_{0.05(2)549}$=5.8, p<0.001, respectively) but not after 30 days ($t_{0.05(2)997}$= 1.8, p=0.07). Therefore, I would be confident that a population where removal of individuals is random is different from a population where removal of individuals is size-selective.

The question now is: "if I take a sample from each population, can I differentiate between these two populations?" Figure 6.1 demonstrates how a single random sample of 10% of the population compares with the population during the

larval stage. At any time, random samples are not statistically different from its true population (Table 6.1). However, discrepancies between the length frequency distribution of the true and sample populations range from 1 to 8% (Figure 6.2). In Chapter III, at comparable levels of mortality (95% of the 10 000 individuals in 30 days, or 0.10 d^{-1}), where vulnerability to predation was based on size-dependent encounter and susceptibility, I found that the evidence of size-selective removal of individuals varied between 1% and 6%. Under non-biased random sampling of a population of fish larvae during the first 30 days of the early life stages, there is no chance of statistically detecting size-selective removal of individuals and this, even under the strongest size-selective mortality. In addition, we will notice that there is no statistical difference between mean length ($t_{0.05(2),113}$= 1.5, P> 0.1, and $t_{0.05(2),75}$= 1.4, P> 0.1) and length frequency distributions (based on Kolmogorov-Smirnov statistics: D=0.19 < $D_{0.05}$= 0.25, and D=0.26 < $D_{0.05}$= 0.31) of the two samples on days 30 and 60. Even though the random samples came from two populations with different selective processes, we would have concluded incorrectly that these two samples came from the same population. This implies that we cannot expect to statistically differentiate between a population of fish larvae where removal of individuals is size-selective from another where removal of individuals is random. We can detect differences between the mean length of the two samples only at the onset of the juvenile stage (day 90) ($t_{0.05(2),54}$= 4.2, P< 0.001) and conclude that the two samples came from clearly different populations (D=0.49 > $D_{0.05}$= 0.36).

Miller et al. (1995) presented the first accurate field examination of the initial variability in egg and larval sizes in a natural population. They suggested that there is considerable potential for phenotypic selection among individual cod eggs and larvae of different sizes on the Scotian Shelf (Miller et al. 1995). However, the above analysis demonstrated that unless extremely precise measurements of egg and larval sizes are made (± 0.1 mm), detection of size-selective removal of individuals is unlikely. Measurements in this thesis were ±1 mm. Furthermore, Pepin et al. (1998) demonstrated that changes in body length of larval fish due to handling and preservation are neither uniform nor consistent among individuals within narrow 1 mm length intervals. Pepin et al.'s (1998) study implies that precise measurements of larval fish may not be accurate representation of the actual size of larval fish.

The exercise presented here conflicts somewhat with the results of Chapter V where I found that for some species, information about the predator population of pelagic fishes co-occurring with the early life history of fishes could explain the changes of the length frequency distribution observed in field collection of natural populations. Even if I found that qualitatively, adding predation improved the fit between the model's predictions and the field observations, only one of the one-tailed variance tests was statistically significant. Furthermore, combining probabilities did not indicate a statistically significant trends of improvement due to the addition of predation in the individual-based model.

To further investigate this issue, I calculated the statistical power of such comparisons of length frequency distributions. The theoretical *F*-ratio required for significance at $\alpha = 0.05$ decreases sharply with increasing degrees of freedom (Figure 6.3). This implies that during the 30 days in my simulations, where the length frequency distribution of the cohort increases from 2 to 10 size classes, the variance in the growth model would have to be three times that of the predation model in order to show significant improvement. Even at metamorphosis (day 90, 25 size classes), the variance of the growth model must be more than twice that of the predation model. The goal of the analysis of Chapter V was to first set the growth rate as to minimize the discrepancies between the field observations and the model's predictions (i.e., mean square error as close to zero as possible). This goal has serious restrictions on the power of the analyses. If I had an independent estimate of growth rate (even if it's an estimate of the cohort's mean growth rate), I might have been able to achieve a difference in variance required for statistical significance at the α level. However, this estimate of the mean growth rate of the cohort must be very precise as I have demonstrated that the analysis was highly sensitive to small variations in mean growth rate (Chapter V).

Regardless of this shortcoming, it is important to note that even if not statistically significant, there seems to be a general consensus that smaller fish larvae survive better to predation by adult capelin of variable sizes and that the addition of predation did improve the model's predictions. However, is this faint signal real?

Assumptions of the simulation models

It may be important to re-iterate the assumptions and conditions of the simulation model used in this thesis: 1) random movements and spatial distributions of prey and predators; 2) number of encounters per day follows a Poisson distribution; and 3) laboratory-derived predation rates are estimates of field susceptibility probabilities given that an encounter took place.

At this point, are we confident that the mathematical derivation of encounter 4rates based on Gerritsen and Strickler's (1977) study is directly applicable or relevant to field situations? Are we confident in the empirically-derived susceptibility functions derived in Chapter II? I argue that the laboratory derivation of predation are reliable estimates of field susceptibility because the empirical patterns appeared to be consistent for a variety of species, and they were derived from a broad range of different laboratory experiments. Furthermore, the pattern of size-selection is, to some extent, independent of predator type (Chapter II).

The mathematical derivation of encounter rates assumes random spatial distribution and movement of predators and prey (Gerritsen and Strickler 1977). Spatial aggregations of pelagic fishes (e.g., schools of spawning fishes) can have significant repercussions on the survival of fish larvae (e.g., William 1991). To determine

specifically if the mathematical derivation of encounter is applicable to the analysis of field data, I suggest that empirical evidence from field observations is needed. This evidence cannot be gathered from laboratory work because even the largest enclosures and mesocosms restrict the value of encounter by enclosing prey and predator in the same body of water, except for ctenophores (see Appendix 1). We need to estimate the actual field encounter rates between cohorts of fish larvae and predator populations and compare these with the encounter rates calculated with the formulation of Gerritsen and Strickler (1977). When we do so, we should consider that any given predator does not necessarily encounter a given larva but rather encounters a cohort of fish larvae. This latter consideration may imply only a slight modification of the Gerritsen and Strickler (1977) model. At the very least, the encounter radius and swimming speed should be calculated for a cohort of fish larvae and not for an individual larva. The encounter radius should be related to the number of individuals of the cohort as well as the average dispersal of individuals from one another within the cohort. I suspect that early during the larval stage, the average swimming speed of a larval cohort will be related to the average speed of the water mass within which the cohort is contained. As individuals grow, their individual swimming speed may become more important in the approximation of actual field encounters with potential predators. Therefore, swimming speed of the larval cohort will most likely be related to the oceanographic characteristics of their environment.

Alternatively, one could try to devise a new mathematical derivation of encounter between fish larvae (or any planktonic animals) and their predators. I suspect that in the field, encounters between fish larvae and their predators will likely be a function of prey and predator densities as well as spatial and temporal overlap of their distributions (as defined by Williamson et al. 1989). Williamson et al. (1989) argued that in cases where prey and predators are patchily distributed, spatial overlap, which is a function of prey and predator densities at specific locations, must be incorporated in the estimation of predation rate. Williamson and Stoeckel (1990) evaluated the importance of this estimate of spatial overlap and demonstrated clearly that predation risk was underestimated under the assumption of uniform prey and predator densities. Quantification of spatial overlap between prey and predator should be integrated in the encounter model used by larval fish ecologists similarly to the encounter model used by limnologists. This simple addition could approximate more realistically the computer intensive calculations of physical oceanographic models (e.g., Heath 1994, Hinckley et al. 1996) of larval fish dispersal which I think may eventually be used to estimate encounters between fish larvae and their predators.

Concluding remarks

I believe this thesis to be an important contribution to the theoretical framework of early life history of fishes as it poses serious questions about the effectiveness of current sampling protocols and statistical analytical tools in the investigation of size-selective predation mortality. Furthermore, it describes the specific conditions under which size-selective removal of individual fish larvae may best be detected in natural populations. It also pinpoints the areas where future studies may have the best impact in advancing knowledge in the characteristics of survivors approach. I agree with Dr. J.A. Rice (Stages, Sept. 1998) when in his keynote address at the Larval Fish Conference (Ann Arbor, MI), he argued that: " The utility of the process-oriented approach will be greatly enhanced if we also begin to ask what it can tell us about the nature of uncertainty and limits on predictability of recruitment." He predicted that: "Ironically, the same analyses that show that a particular process may have significant effects on interannual variation in survival may also demonstrate that we are unlikely to be able to quantify these effects in the field." He concluded that: "In such cases, it may be more useful to focus on the magnitude and consequences of uncertainty than on trying to predict specific effects on survival."

Table 6.1: Number of survivors to either time-dependent mortality or size-selective mortality of the population with mean length (mm) and standard error (SE) of the population as well as the number sampled from the population and the mean estimate of length and standard error of the sample.

	Time-dependent mortality			Size-selective mortality		
	T=30	T=60	T=90	T=30	T=60	T=90
Number of survivors	500	370	275	499	367	276
Mean length ± SE	9.10±0.05	16.0±0.1	23.0±0.2	8.9±0.1	15.0±0.2	20.4±0.4
Number sampled	61	33	29	54	44	27
Mean length ± SE	9.0±0.1	16.2±0.5	23.8±0.6	8.6±0.3	15.1±0.7	18±1

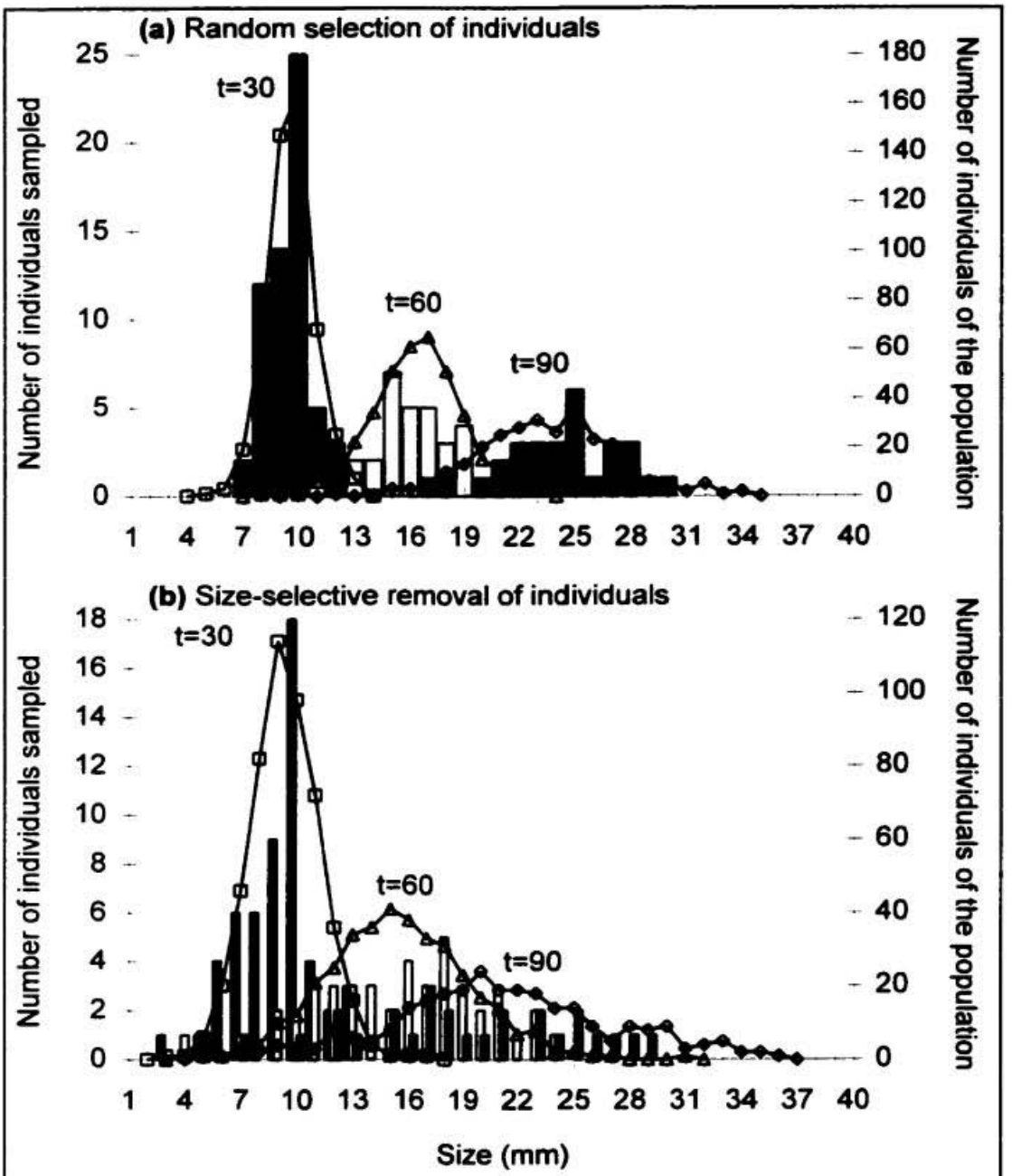

Figure 6.1: Length distribution of the population and of the random sample from that population given that the individuals are either **(a)** randomly selected or **(b)** selected by a population of pelagic fishes of variable sizes (as defined in previous chapters). The distributions are given at three time periods: 30, 60 and 90 days.

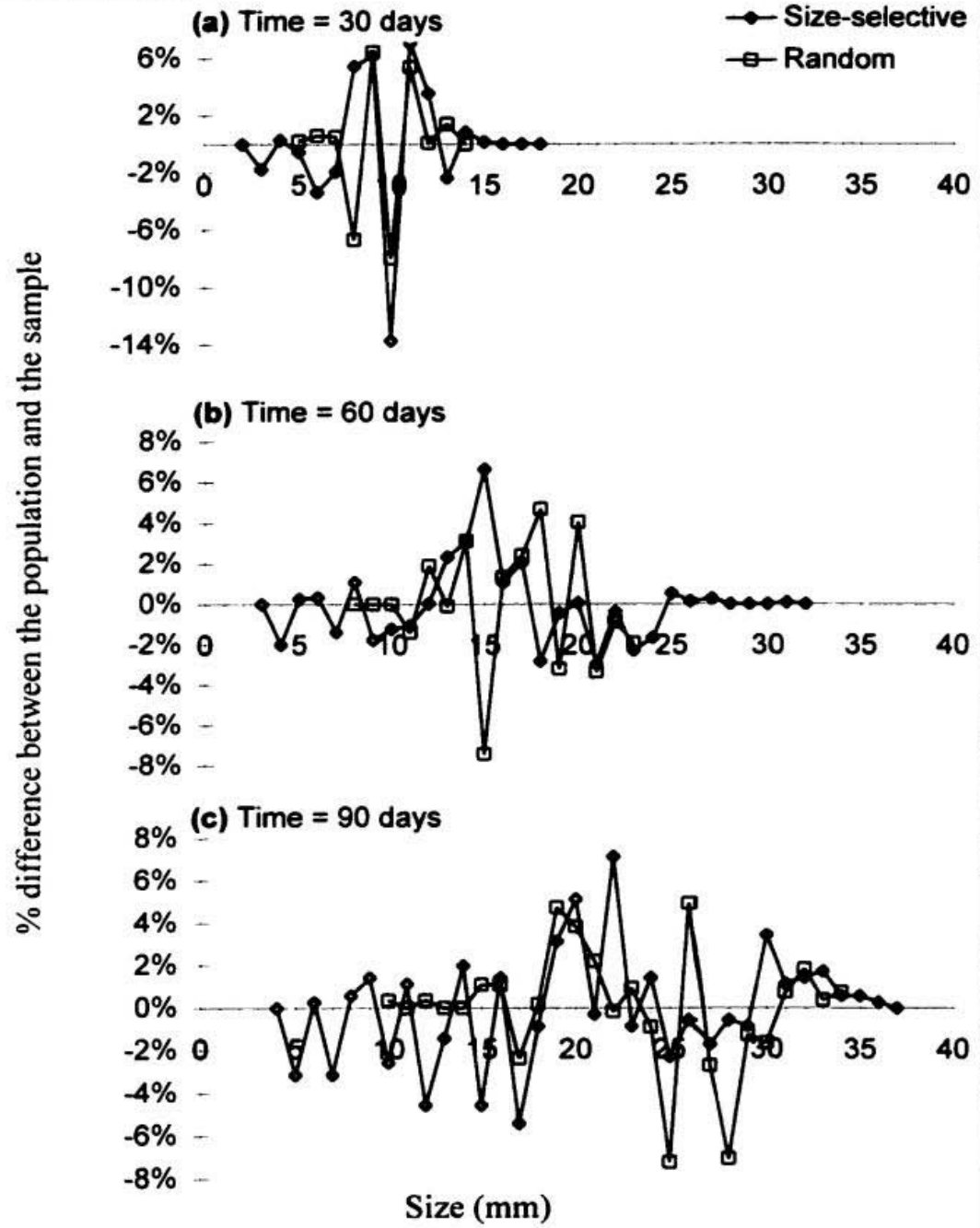

Figure 6.2: Size-specific percent difference between the population and the random sample at three different periods during the early development of yellowtail flounder: **(a)** 30 days, **(b)** 60 days, and **(c)** 90 days. Surviving individuals were either randomly selected or selected based on their size.

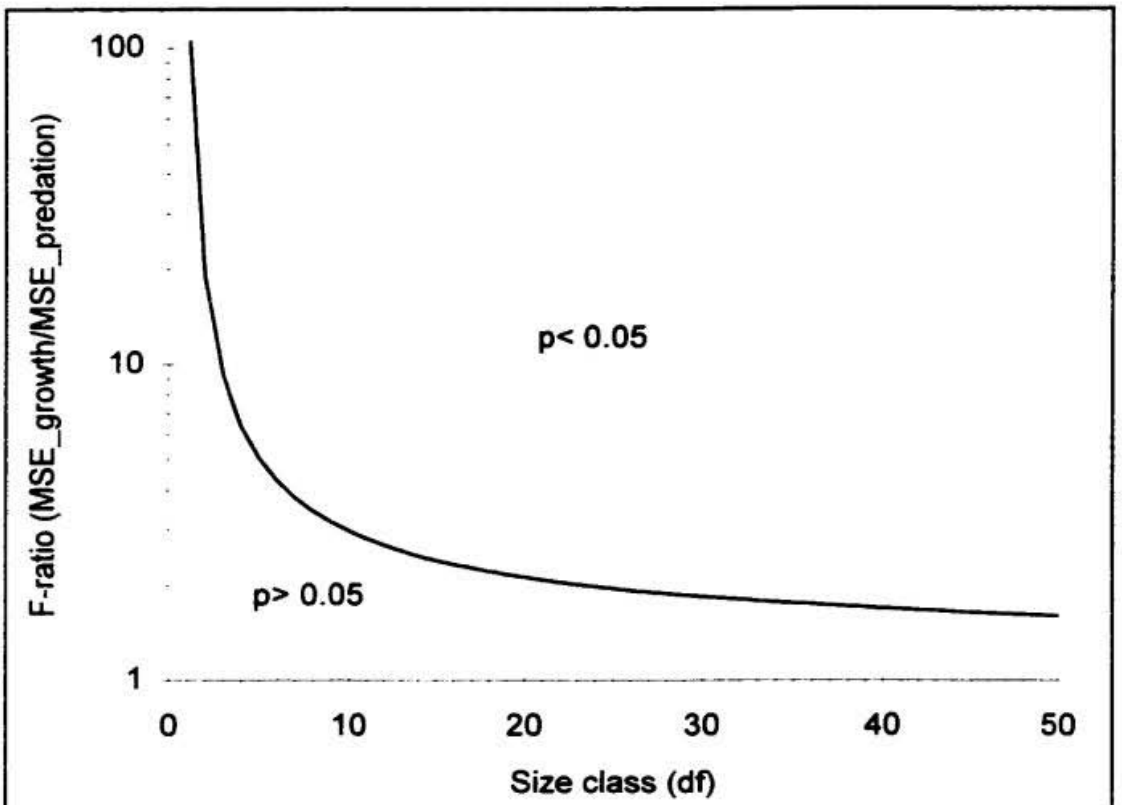

Figure 6.3: One-tailed variance ratio test (F-ratio = mean square error of individual-based growth model - mean square error of individual-based predation model) computed for different degrees of freedom (size classes) at the a-level of 0.05.

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Chapter VI

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Appendix 1

In Chapter II, I derived empirical predation rates based on multi-specific experimental laboratory work. Can I assume that these laboratory and empirically-derived predation rates are susceptibility functions in field analyses? Under experimental conditions used to date to quantify laboratory predation rates, can I assume that prey and predator are enclosed in the same body of water? In other words, encounter rates, according to Gerritsen and Strickler (1977) formulation must be ≥ 1.0. This would imply that predation rates measured from laboratory experiments are actually, susceptibility functions on a broader temporal and spatial scale (i.e., in the ocean). The averaged experimental conditions that may affect predation rates via the encounter process were as follows:

Variables (Units)	Container volume (m^3)	Duration of experiment (h)	N_p (#)	L_p (mm)	L_f (mm)
Crustaceans	0.00755	20.8	3.9	9.95	7.23
Ctenophores	0.0172	5.78	1.4	18.8	3.2
Fishes	3.55	7.48	2.7	113.1	12.9
Medusae	1.7065	20.67	4.8	33.8	8.35

where N_p is the number of predators used on average in experimental studies of predation and L_p and L_f are the size of predators and size of fish larvae, respectively.

According to Gerritsen and Strickler (1977) model of encounter (equation 3.1) and if I substitute the averaged value from the table above, I find that the encounter rates during the mean duration of laboratory experiments were: $E_{crustaceans} = 20.0$ encounters, $E_{ctenophores} = 3.0$ encounters, $E_{fishes} = 34.9$ encounters, and $E_{medusae} = 2.0$ encounters.

I can safely assume that under averaged experimental conditions, fish larvae and their predators are enclosed in the same body of water because encounter rates (according to Gerritsen and Strickler 1977) is greater than one. This implies that the measured predation rates of averaged experimental conditions are actually measures of susceptibility to predation. If I use these predation rates at greater spatial and temporal scales such as those in the field, I should assume that these predation rates estimates are in fact, estimates of the function of susceptibility.

Appendix 2

This program was written in FORTRAN 77 and ran on the High Performance Computing system at Memorial University of Newfoundland, 1995 - 1999.

MAIN.f

```
c       This is the main program.

        CALL INITIALIZE
        CALL MC_LOOP
        end
```

INITIALIZE.f

```
      SUBROUTINE INITIALIZE

c     This is the subroutine is used to initialize the variables.
c     This is the baseline, no changes.
c     This model simulates the predation mortality of a population of fish larvae
c     confronted to several populations of predators (up to four). The populations
c     are defined in terms of size distributions.
c     The predation mortality follows the Gerritsen and Strickler (1977) encounter
c     model, the susceptibility are calculated according to the empirical model of
c     Paradis et al. (1996). Several encounters can occur in a day and follows the
c     Poisson distribution as explained in Pepin (1989). The growth rate of fish larvae
c     follows one of the algorithm explained in Rice et al. (1987), ie. either the
c     constant growth or the random walk with memory.

c     DECLARE VARIABLES
c     x is the individual prey
c     d is the individual predator of specific length
c     p is the index for the predator type, p=1,maxp
c     j is the index for the predator length, j=1,maxj(p)
c     i is the index for the prey length, i=1,maxi
c     k is the index for the combined predator type and length on a single dimension.
c     dumx is a dummy variable to replace index of individual prey, ie. x.
c     dumd is the dummy variable to replace index k.
c     dump is the dummy variable to replace index p.
c     p_de(d) is the index which prings back the k to the individual predator.
c     nbenc is the index for the number of encounters [0-3].
c     volume is the water body of the simulation, 50 000 m^3
c     pmaSSy and pmbSSy are the parameters of the prey swimming speed function.
c     pmaRDy is the parameter of the prey reactive distance function.
c     ld_cont(d,p) is the predator length in the input datafile.
c     ld(j,p) is the predator length set on a set scale.
c     nd(j,p) is the number of individual predator of the length interval j and of type p.
c     SSy(i) is the swimming speed calculated for each prey length.
c     SSd(p,j) is the swimming speed calculated for each predator type and length.
c     RDy(i) is the reactive distance calculated for each prey length.
c     RDd(p,j) is the reactive distance calculated for each predator type and length.
c     pmaSSd(p) and pmbSSd(p) are the parameters of the swimming speed function
c           of predators, given their lenght.
c     pmaRDd(p) is the parameter of the reactive distance function of predator type,
c           given their length.
```

```
c       pmaTS(p), pmbTS(p) and pmcTS(p) are the parameters of the vulnerability
c            function, specific to predator type.
c       maxCER(i) is the last cumulative encounter rate. It is used to calculate it on a
c            relative scale. Then, the maxCER is equal to 1.
c       fact(nbenc) is the factorial value of nbenc.
c       maxCPr(i) is the last cumulative probability of encounter.
c            It is used to calculate it on a relative scale.
c       ER(i,j,p) is the encounter rate calculated for each prey length and predator type
c            and length.
c       TS(i,j,p) is the susceptability rate calculated for each prey length and predator
c            type and length.
c       CER(i,k) is the cumulative encounter rate between prey lenght i and predator
c            length j of type p (on a combined scale=k)
c       Pr(i,k,nbenc) is the probability of 'nbenc' encounters between prey length i and
c            predator length j of type p (ie. k).
c       CPr(i,nbenc) is the cumulative probability of 'nbenc' encounters for prey length i

        IMPLICIT NONE
        INCLUDE 'input.h'
        INCLUDE 'limits.h'
        INCLUDE 'global.h'

        INTEGER type_p
        INTEGER*4 p,j,i,y,k,d,dumx,dumd,dump
        INTEGER*4 tempo,nbenc,maxenc,maximum_encounters
        REAL*8 maximum, tmp_var
        REAL*8 pmaSSy,pmbSSy,pmaRDy
        REAL*8 conc(maxj_LIM,p_LIM)
        REAL*8 SSy(maxi_LIM),SSd(maxj_LIM,p_LIM)
        REAL*8 RDy(maxi_LIM),RDd(maxj_LIM,p_LIM)
        REAL*8 pmaSSd(p_LIM),pmbSSd(p_LIM),pmaRDd(p_LIM)
        REAL*8 pmaTS(p_LIM),pmbTS(p_LIM),pmcTS(p_LIM)
        REAL*8 pmaTSCow(p_LIM),pmbTSCow(p_LIM),pmcTSCow(p_LIM)
        REAL*8 pmdTSCow(p_LIM)
        REAL*8 maxCER(maxi_LIM)
        REAL*8 CER(maxi_LIM,maxk_LIM)
        REAL*8 Pr(0:enc_LIM)
```

```
c       HERE IS THE LIST OF INPUT FILES AND THEIR VARIABLES
c       startvar.dat =  maxx, maxi, maxt, maxp, incr_y
c                       maxj(p), type_p
c       prey.dat = dumx,ly_cont(x)
c       pred.dat = p,j,ld(j,p),conc(j,p)
c       fnssd.dat = dump,pmaSSd(p),pmbSSd(p)
c       fnrdd.dat = dump,pmaRDd(p)
c       fnssy.dat = pmaSSy,pmbSSy
c       fnrdy.dat = pmaRDy
c       fnts.dat = dump,pmaTS(p),pmbTS(p),pmcTS(p)
c       THE LIST OF RESULTS FILES AND THE TITLES OF THEIR COLUMNS
c       er.dat = ly(i), ld(j,p), er(i,j,p)
c       ts.dat = ly(i), ld(j,p), ts(i,j,p)
        OPEN(97,file='data/results/er.dat',status='unknown')
        CLOSE(97,status='delete')
        OPEN(94,file='data/results/ts.dat',status='unknown')
        CLOSE(94,status='delete')

c       Retrieve the number of individual prey, the number of length, the number of
c       combined predator type and length and the number of length per predator type
c       and the time period of the simulation and the number of predator types and the
c       increment of length for the prey distribution.
        OPEN(2,file='data/startvar.dat',status='unknown')
        READ(2,*)maxx,maxi,maxt,maxp,incr_y
        do p = 1,maxp
          READ(2,*)maxj(p),type_p
        enddo
        CLOSE(2)

c       Set the scale for prey length
        i=1
        ly(i)= incr_y/2.D0
        do i=2,maxi
          ly(i)=ly(i-1)+incr_y
        enddo

c       Retrieve the information about prey distribution.
        OPEN(4,file='data/distributions/prey.dat',status='unknown')
        do x = 1,maxx
          READ(4,*)dumx,ly_cont(x)
          ly_init(x) = ly_cont(x)
        enddo
        CLOSE(4)
```

```
c       Set the position of the individual's length on our i scale
        do 100 x = 1,maxx
         do i = 1,maxi
          if (ly_cont(x).le.ly(i)+incr_y/2.D0) then
           i_de_x(x,0)=i
           goto 100
          endif
         enddo
100     continue

c       Set the time to zero.
        t=0
c       Compute the distributions of prey.
        do i = 1,maxi
         dist(i,t)=0
        enddo
        do x = 1,maxx
         dist(i_de_x(x,t),t)=dist(i_de_x(x,t),t)+1
         alive(x) = .true.
        enddo

c       Retrieve the information about predator size distributions
        OPEN(7,file='data/distributions/pred.dat',status='unknown')
        do p = 1,maxp
         do j = 1,maxj(p)
          READ(7,*)p,j,ld(j,p),conc(j,p)
         enddo
        enddo
        CLOSE(7)

c       Compute predator swimming speed and reactive distance
        OPEN(8,file='data/fnssd.dat',status='unknown')
        OPEN(9,file='data/fnrdd.dat',status='unknown')
        do 103 p = 1,maxp
         READ(8,*)dump,pmaSSd(p),pmbSSd(p)
         READ(9,*)dump,pmaRDd(p)
         if (maxp.eq.1) then
          if (type_p.eq.4) then
           do 104 j = 1,maxj(p)
            SSd(j,p)=pmaSSd(p)*(ld(j,p)**(pmbSSd(p)))
            RDd(j,p)= pmaRDd(p)*ld(j,p)
104        continue
          else
```

```
              do 1045 j= 1,maxj(p)
                SSd(j,p)= pmaSSd(p)+((ld(j,p))*(pmbSSd(p)))
                RDd(j,p)= pmaRDd(p)*ld(j,p)
1045          continue
             endif
            else
             do 105 j=1,maxj(p)
              if (p.eq.2) then
                SSd(j,p)=pmaSSd(p)*(ld(j,p)**(pmbSSd(p)))
                RDd(j,p)=pmaRDd(p)*ld(j,p)
               else
                SSd(j,p)= pmaSSd(p)+((ld(j,p))*(pmbSSd(p)))
                RDd(j,p)= pmaRDd(p)*ld(j,p)
               endif
105          continue
            endif
103       continue
          CLOSE(8)
          CLOSE(9)

c         Compute prey swimming speed and reactive distance
          OPEN(10,file='data/fnssy.dat',status='unknown')
          OPEN(11,file='data/fnrdy.dat',status='unknown')
          READ(10,*)pmaSSy,pmbSSy
          READ(11,*)pmaRDy
          do i= 1,maxi
            SSy(i)= pmaSSy*(ly(i))**(pmbSSy)
            RDy(i)= pmaRDy*ly(i)
          enddo
          CLOSE(10)
          CLOSE(11)

c         Compute encounter rate
          OPEN(97,file='data/results/er.dat',status='unknown', access='append')
          do 106 p = 1,maxp
           do 107 j = 1,maxj(p)
            do 108 i = 1,maxi
             if (SSy(i).lt.SSd(j,p)) then
              ER(i,j,p)= conv*(pi/3.D0)*DH*conc(j,p)*(DBLE(10)**(-9))*
     *                   ((RDy(i) + Rdd(j,p))**2)*
     *                   (((SSy(i)**2)+3.D0*(SSd(j,p)**2))/SSd(j,p))
             else
```

```
            ER(i,j,p)= conv*(pi/3.D0)*DH*conc(j,p)*(DBLE(10)**(-9))*
     *                  ((RDy(i) + RDd(j,p))**2)*
     *                  (((SSd(j,p)**2)+3.D0*(SSy(i)**2))/SSy(i))
           endif
           if (ER(i,j,p).ne.0) then
            WRITE(97,*)ly(i),ld(j,p),ER(i,j,p)
           endif
108      continue
107     continue
106   continue
      CLOSE(97)

c     Compute maxk, j and p on a single scale
      maxk=0
      do p=1,maxp
       maxk = maxk+maxj(p)
      enddo

c     Compute cumulative encounter rates
      do 109 i = 1,maxi
        k = 0
        do 110 p = 1,maxp
          do 111 j = 1,maxj(p)
            k = k+1
              if (k.eq.1) then
                CER(i,k) = ER(i,j,p)
              else
                CER(i,k) = CER(i,k-1) + ER(i,j,p)
              endif
              if (k.eq.maxk) then
                maxCER(i) = CER(i,k)
              endif
111       continue
110     continue
109   continue

c     Compute relative cumulative encounter rates (RCER)
      do 112 i=1,maxi
       if (maxCER(i).eq.0) goto 112
        do 113 k=1,maxk
          RCER(i,k)= CER(i,k)/maxCER(i)
113     continue
112   continue
```

```
c       Check to see if enc_LIM is large enough!
        OPEN(75,file='data/results/max-enc.dat',status='unknown', access='append')
        max_enc = enc_LIM
        maximum_encounters = 0
        do p = 1,maxp
          do j = 1,maxj(p)
            do i = 1,maxi
              maximum = 0.D0
              do nbenc = 0,enc_LIM
                if(nbenc.eq.0) then
                 if(ER(i,j,p).ne.0) Pr(nbenc) = DEXP(-ER(i,j,p))
                  if(ER(i,j,p).eq.0) Pr(nbenc) = 1.D0
                  else
                   Pr(nbenc) = Pr(nbenc-1)*ER(i,j,p)/DBLE(nbenc)
                  endif
                  if(Pr(nbenc).gt.maximum) then
                    maximum = Pr(nbenc)
                    if(nbenc.gt.maximum_encounters) then
                     maximum_encounters = nbenc
                    endif
                  endif
              enddo
            enddo
          enddo
        enddo
        WRITE(75,*)maximum_encounters
        CLOSE(75)
        if (maximum_encounters.gt.(0.8*max_enc)) then
         WRITE(6,*)'warning!! enc_LIM is too small!'
         stop
        endif

c       Open and read datafile to calculate the TS function.
        OPEN (12,file='data/fnts.dat',status='unknown')
        do p=1,maxp
         READ(12,*)dump,pmaTS(p),pmbTS(p),pmcTS(p)
        enddo
        CLOSE(12)

c       Compute susceptibility rates based on Paradis et al. 1996 the predation rates in
c       Paradis et al. (1996) units are h-1 must convert on per day, considering there is
c       only 13h/24h of daylights in a day (DH)
```

```
c       Save the susceptibility rates in a file
        OPEN(94,file='data/results/ts.dat',status='unknown',
     *     access='append')
        do p=1,maxp
         do j=1,maxj(p)
          do i=1,maxi
           if (type_p.eq.1) then
            TS(i,j,p)= ((DH*24.D0)*(exp(pmaTS(p) +
     *          pmbTS(p)*(dlog(ly(i)/ld(j,p))) +
     *          pmcTS(p)*(dlog(ly(i)/ld(j,p)))**2)))
            if (TS(i,j,p).ge.1.0) then
              TS(i,j,p)= 0.99999999
            endif
            WRITE(94,*)ly(i),ld(j,p),TS(i,j,p)
           else
            TS(i,j,p)= ((exp(pmaTS(p) +
     *                  pmbTS(p)*(dlog(ly(i)/ld(j,p))) +
     *                  pmcTS(p)*(dlog(ly(i)/ld(j,p)))**2)))
            if (TS(i,j,p).ge.1.0) then
              TS(i,j,p)= 0.99999999
            endif
            WRITE(94,*)ly(i),ld(j,p),TS(i,j,p)
           endif
          enddo
         enddo
        enddo
        CLOSE(94)

c       Initialization of some variables to create results files: dead.dat and meal.dat
        do i = 1,maxi
         do p = 1,p_LIM+1
          death_bin(i,p) = 0
         enddo
        enddo
        do p = 1,maxp
         do j = 1,maxj(p)
          meal_bin(j,p) = 0
         enddo
        enddo
        return
        end
```

```
      SUBROUTINE MC_LOOP
c     This is the main MC loop.
c     This is the baseline, no restriction on time
c     Here is a list of the variables
c     seed is the number needed to start off the random number generator
c     i is the index for the prey length
c     j is the index for the predator length
c     k is the combined index of predator type and length
c     p is the index for the predator type
c     predateur is the chosen predator type which encounters a particular prey
c     longueur is the chosen predator length which encounter a certain prey
c     interval is the place where the x is.??? length interval of prey x
c     number_encouter is the number of encounters chosen for that specific prey in
c           that day.
c     index_enc is the index of the number of encounters chosen
c     nbenc is the index of the number of encounters [0-3]
c     ly_noPR(x) is the length of prey x at the end of experiment but given they did
c           not suffer from predation mortality. This is the 'control'
c     dist_noPR(x) is the size distribution given there are no predation mortality, ie.
c           control.
c     random_E(x) is the random number chosen for x, to determine if there was an
c           encounter and with what predator type and length.
c     random_S(nbenc*x) is the random number chosen for x, to determine if the
c           attack and capture between x and the encountered predator was
c           successfull. This is in a loop because there might be more than one
c           encounter.
c     random_P(nbenc*x) is the random number chosen for x, to determine how many
c           encounters occured in that day for that prey.

      IMPLICIT NONE
      INCLUDE 'input.h'
      INCLUDE 'limits.h'
      INCLUDE 'global.h'

      INTEGER*4 rencontre,surv(0:maxt_LIM),died
      INTEGER*4 i,j,k,p,predateur,longueur,interval,temp
      INTEGER*4 number_encounter,index_enc,nbenc
      INTEGER*4 i_of_x(maxx_LIM)
      REAL*8 ly_noPR(maxx_LIM,0:maxt_LIM)
      REAL*8 dist_noPR(maxx_LIM,0:maxt_LIM)
      REAL random_E(maxx_LIM)
```

```
      REAL random_S(enc_LIM)
      REAL random_P(maxx_LIM)
      REAL*8 tmp,tmp_number

c     Declare variables for the gaussian random number generator
      INTEGER*4 iteration
      REAL etc,range
      REAL random,gaussian
      REAL*8 mean
      REAL sigma,stddev
      INTEGER seed2,seed

      external random,gaussian
      common/one/ seed2
      common/two/ etc
      common/three/sigma
      common/four/range

      INTEGER*4 index,bin(-200:200)
      REAL*8 temp_gauss,x_max,x_min

c     Open results file to save the results of the simulation run.
c     statsize.dat = x  ly_cont(x)  GR(x)
c     control.dat = ly(i)  dist_noPR(i)
c     statcont.dat = x  ly_noPR(x)  GR(x)
c     timedead.dat = t died
c     sizedead.dat= ly(i) death_bin(p=1) death_bin(p+1) death_bin(p=4)
c     dist_rel.dat = ly(i) reldist(t=0) reldist(t+6) reldist(t=30)
c     sizemeal.dat= ld(j,p=1) meal_bin(j,p=1)..ld(j,p=4) meal_bin(j,p=4)
c     nb-dead.dat= (t-1) (temp-1)

      OPEN(96,file='data/results/statsize.dat',status='unknown')
      CLOSE(96,status='delete')
      OPEN(95,file='data/results/control.dat',status='unknown')
      CLOSE(95,status='delete')
      OPEN(94,file='data/results/statcont.dat',status='unknown')
      CLOSE(94,status='delete')
      OPEN(93,file='data/results/timedead.dat',status='unknown')
      CLOSE(93,status='delete')
      OPEN(92,file='data/results/sizedead.dat',status='unknown')
      CLOSE(92,status='delete')
      OPEN(91,file='data/results/dist_rel.dat',status='unknown')
      CLOSE(91,status='delete')
```

```
      OPEN(90,file='data/results/sizemeal.dat',status='unknown')
      CLOSE(90,status='delete')
      OPEN(89,file='data/results/nb-dead.dat',status='unknown')
      CLOSE(89,status='delete')
      OPEN(88,file='data/results/sizeselect.dat',status='unknown')
      CLOSE(88,status='delete')

c     Do initialization for the random number generator
      temp=0
      seed = 456732
      CALL RLUXGO(3,seed,0,0)

c     Retrieve from a data file the standard deviation of the normal growth curve.
      OPEN(1,file='data/pm_gr.dat',status='unknown')
      READ(1,*)mean,stddev
      CLOSE(1)

c     Initialization of the growth random number generator.
      sigma = stddev
      seed2 = 7642315
      range = 1
      etc = range/2147483648.1

c     Compute the growth rate (GR(x)) from the ran.# generator
      do x=1,maxx
       tmp = DBLE(gaussian())
       GR(x)=mean+tmp
       if (GR(x).le.0) then
        GR(x)=dabs(GR(x))
       endif
      enddo
      rencontre = 0
      surv(0) = maxx
      died = 0

c     Start counting time
      do 100 t=1,maxt
        index_enc = 0

c     At midnight -1 and at midnight +1 minute, the distributions are the same
      rencontre = 0
      surv(t) = 0
      died = 0
```

```
      do x = 1,maxx
        i_de_x(x,t) = i_de_x(x,t-1)
      enddo
      do i = 1,maxi
        dist(i,t)=dist(i,t-1)
      enddo

c     To determine if or how many encounters:
      CALL RANLUX(random_E,maxx)

c     To determine which predator:
      CALL RANLUX(random_P,maxx)

       do 101 x = 1,maxx
        if(.not.alive(x)) goto 101
        i = i_de_x(x,t)
         k=0
         predateur=0
         longueur=0
         interval=0
         do 102 p = 1,maxp
          do 103 j = 1,maxj(p)
             k = k+1
             if (RCER(i,k).ge.random_P(x)) then
c     Encounters who?
                  predateur = p
                  longueur = j
                  interval = i
                  goto 1035
c     You have chosen the predator, go below to pick the # encounters
               endif
103         continue
102       continue

c     That prey did not encounter, make it grow then next prey.
          goto 105
```

```
c       Pick the number of encounters with the chosen predator.
1035      tmp_number = 0.D0
          do 104 nbenc = 0,max_enc
            if (nbenc.eq.0) then
             if(ER(i,j,p).eq.0) tmp_number= 1.D0
              if(ER(i,j,p).ne.0) tmp_number= dexp(-ER(i,j,p))
              else
               tmp_number = tmp_number*ER(i,j,p)/DBLE(nbenc)
              endif
               if (tmp_number.ge.random_E(x)) then                    !encounter(s)
                  number_encounter = nbenc
               if (number_encounter.eq.0) then
                 goto 105                                          !!! no encounter !!!
               endif
                if (number_encounter.ne.0) then
                   rencontre = rencontre+1
                   goto 106 !!! x encounter(s) occur !!!
             endif
            endif
104       continue
c       Number of encounters determined (nbenc) and .neq. 0, the type of predator and
c       the size is also determined.
c       That predator is given nbenc chances of eating that prey.
c       To determine if or not alive:
106     CALL RANLUX(random_S,number_encounter)
          do 107 index_enc = 1,number_encounter
            if (TS(interval,longueur,predateur).ge.random_S(index_enc)) then
             dist(i_de_x(x,t),t)=dist(i_de_x(x,t),t)-1
             alive(x) = .false. !prey killed
             death_bin(i_de_x(x,t),predateur) = death_bin(i_de_x(x,t),predateur)+1
             death_bin(i_de_x(x,t),p_LIM+1) = death_bin(i_de_x(x,t),p_LIM+1)+1
             meal_bin(longueur,predateur) = meal_bin(longueur,predateur)+1
             died = died+1
             temp=temp+1
             goto 101                                        !next individual prey
            endif
107       continue
          goto 105
c       Prey survived the nbenc encounters with p,j then make it grow:
105       CALL GROWTH
          surv(t) = surv(t)+1
          goto  101
101     continue                                              !end the individual x loop
```

```
      do 108 x=1,maxx
       ly_noPR(x,t)=ly_init(x)+(t*GR(x))
       do 109 i=1,maxi
        if (ly_noPR(x,t).le.ly(i)+incr_y/2.D0) then
         i_of_x(x)=i
         goto 108
        endif
109    continue
108    continue

      do i=1,maxi
       dist_noPR(i,t)=0
      enddo
      do x=1,maxx
       dist_noPR(i_of_x(x),t)=dist_noPR(i_of_x(x),t)+1
      enddo

      OPEN(93,file='data/results/timedead.dat',status='unknown',
     *        access='append')
      WRITE(93,*)t,died
      CLOSE(93)

100   continue                                          !end the time t loop

c     Save the actual number of individuals consumed by the predators
      OPEN(89,file='data/results/nb-dead.dat',status='unknown', access='append')
      WRITE(89,*)t-1,temp
      CLOSE(89)

c     Save the size distribution in a histogram format.
      OPEN(91,file='data/results/dist_rel.dat',status='unknown')
      do i=1,maxi
        WRITE(91,2000)ly(i),(dist(i,t)/DBLE(surv(t)), t = 0,maxt,maxt/3)
      enddo
      CLOSE(91)

c     Save the control experiment, distribution of prey given no predation.
      OPEN(95,file='data/results/control.dat',status='unknown')
      do i=1,maxi
       WRITE(95,2000)ly(i),(dist_noPR(i,t)/DBLE(maxx),t = 0, maxt,maxt/3)
      enddo
      CLOSE(95)
```

```
c       Save the index of size-selective mortality per size class.
        OPEN(88,file='data/results/sizeselect.dat',status='unknown')
        do i=1,maxi
        WRITE(88,2000)ly(i),((dist_noPR(i,t)/DBLE(maxx))-
     *          (dist(i,t)/DBLE(surv(t))),t = 0,maxt,maxt/3)
        enddo
        CLOSE(88)
2000    FORMAT(1F9.3,4F10.4)

c       Save the size frequency at the time of death of fish larvae
        OPEN(92,file='data/results/sizedead.dat',status='unknown')
        do i=1,maxi
          WRITE(92,1100)ly(i),(death_bin(i,p),p = 1, p_LIM+1,1)
        enddo
        CLOSE(92)
1100    FORMAT(1F9.3,5I8)

c       Save the # of fish larvae eaten by specific predator length.
        OPEN(90,file='data/results/sizemeal.dat',status='unknown')
        do j=1,maxj_LIM
          WRITE(90,1200)ld(j,1),meal_bin(j,1),ld(j,2),meal_bin(j,2),
     *          ld(j,3),meal_bin(j,3),ld(j,4),meal_bin(j,4)
        enddo
        CLOSE(90)
1200    FORMAT(1F9.3,1I8,1F9.3,1I8,1F9.3,1I8,1F9.3,1I8)

c       Save the dist.dat file in a format to perform stats.
        OPEN(96,file='data/results/statsize.dat',status='unknown')
         do x=1,maxx                    !!! i'm at time t=maxt
          if (alive(x)) then            !!! since i'm out of time loop
            WRITE(96,*)x,GR(x)
          endif
         enddo
        CLOSE(96)

c       Save the control.dat file in a format to perform stats.
        OPEN(94,file='data/results/statcont.dat',status='unknown')
        do x=1,maxx                     ! i'm at time t=maxt
         WRITE(94,*)x,GR(x)                     ! since out of time loop
        enddo                                   ! ly_noPR(x) at maxt
        CLOSE(94)
        return
        end
```

GROWTH.f

```
      SUBROUTINE GROWTH
c     This is the subroutine for the growth component.

      IMPLICIT NONE
      INCLUDE 'input.h'
      INCLUDE 'limits.h'
      INCLUDE 'global.h'
      INTEGER*4 i,prey

      dist(i_de_x(x,t),t)=dist(i_de_x(x,t),t)-1
      ly_cont(x)=ly_cont(x)+GR(x)
c     find new interval
      do i=i_de_x(x,t),maxi
       if (ly_cont(x).le.ly(i)+incr_y/2) then
        i_de_x(x,t)=i
        dist(i,t)=dist(i,t)+1
        goto 101
       endif
      enddo
101   continue
      return
      end
```

```
c       *This is used to declare input variables.              *
c       maxx = is the maximum number of individual prey (x)
c       maxi = is the maximum number of prey length (i)
c       maxt = is the maximum days the simulation will run (t)
c       maxp= is the number of predator types (p).
c       maxk = is the combination of predator type and predator length,
c              this index combines both index on the same scale.

        INTEGER*4 maxx,maxi,maxt,maxp,maxk
        COMMON/gi1/maxx,maxi,maxt,maxp,maxk
```

```
c       *This file contains the declarations for the parameters.*
c       maxx_LIM is the number of space needed for the maximum number of
c             individual prey (maxx) to fit in.
c       p_LIM is the number of space needed for the number of types of predators used
c             in the simulation.
c       maxi_LIM is the number of space needed for the maximum number of  prey
c             length (maxi).
c       maxj_LIM is the number of space needed for the maximum numbers of predator
c             length (maxj(p)).
c       maxk_LIM is the number of space needed for the maximum number of
c             combined predator type (p) and length (j), (maxk).
c       maxt_LIM is the number of space needed for the maximum number of days
c             (maxt).
c       enc_LIM is the number of space needed for the maximum number of
c             encounters in a day, allowed by the model (maxenc=5)
c       temp_LIM is the space for the combination of k and nb_enc which is k*5
c       pi = 3.1416...
c       DH = 13/24, the number of daylight in a 24 hours period.
c       conv = 86400 seconds in a day.  This is to convert my encounter
c             rates from seconds to days.

        INTEGER*4 maxx_LIM,p_LIM,maxi_LIM,maxj_LIM,maxk_LIM,maxt_LIM
        INTEGER*4 enc_LIM,temp_LIM

        REAL*8 pi,DH,conv

        PARAMETER(maxx_LIM = 10000)
        PARAMETER(p_LIM = 1)
        PARAMETER(maxi_LIM = 50)
        PARAMETER(maxj_LIM = 120)
        PARAMETER(maxk_LIM = p_LIM*maxj_LIM)
        PARAMETER(maxt_LIM = 90)
        PARAMETER(enc_LIM = 1000)
        PARAMETER(temp_LIM = 2000)
        PARAMETER(pi = 3.141592654D0)
        PARAMETER(DH = 13.D0/24.D0)
        PARAMETER(conv = 60.D0*60.D0*24.D0)
```

```
c       *This file contains the variables declarations for global variables.*
c       t= is to count the time (in days)
c       x= is the identification of individual prey.
c       incr_y= increment of prey length
c       alive: tell whether the prey is alive or dead.
c       i_de_x(x,t) is to replace (or substitute) de index i (index of prey length).
c       This is used mostly when i want to look up which specific individual has i of ly
c       ly(i) is the prey length actual value, it's index is i.
c       ly_init(x) is the initial prey length actual value.
c       ly_cont(x) is the prey length at time 0 in INITIALIZE
c       dist(i_de_x(x),t) is the number of prey of a length i
c       maxj(p) is the maximum number of lengths for each predator types.
c       TS(i,j,p) is the susceptibility rate between prey and predator encountered.
c       GR(x) is the growth rates of individual prey.

        INTEGER*4 t,x,max_enc
        REAL incr_y
        LOGICAL alive(maxx_LIM)
        INTEGER*4 i_de_x(maxx_LIM,0:maxt_LIM)
        INTEGER*4 death_bin(maxi_LIM,p_LIM+1)
        INTEGER*4 meal_bin(maxj_LIM,p_LIM+1)
        REAL*8 fact(0:enc_LIM)
        REAL*8 ld(maxj_LIM,p_LIM)
        REAL*8 maxj(p_LIM),ly_init(maxx_LIM)
        REAL*8 ly(maxi_LIM),dist(maxx_LIM,0:maxt_LIM),ly_cont(maxx_LIM)
        REAL*8 RCER(maxi_LIM,maxk_LIM)
        REAL*8 TS(maxi_LIM,maxj_LIM,p_LIM),TSCow(maxi_LIM,p_LIM)
        REAL*8 GR(maxx_LIM)
        REAL*8 ER(maxi_LIM,maxj_LIM,p_LIM)

        COMMON/g1/t,x,max_enc
        COMMON/g6/alive
        COMMON/g2/i_de_x
        COMMON/g10/death_bin,meal_bin,ld
        COMMON/g4/ly,dist,maxj,ly_init,ly_cont
        COMMON/g3/RCER
        COMMON/g5/TS,TSCow
        COMMON/g7/GR
        COMMON/g8/incr_y
        COMMON/g9/ER
        COMMON/g11/fact
```