Chapter 3

Mortality

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3.1 Introduction

Teleost fishes have high fecundities and so they must experience high mortality rates to prevent explosive increases in numbers. In many populations, the number of eggs spawned annually or the number of newly hatched larvae may exceed 1×10^{12}. Declines in numbers during egg and larval stages therefore must be precipitous, if juvenile and adult populations are to be maintained at levels that can be supported by an ecosystem. The summed losses, or mortalities, experienced by a population are considered relative to age (age-specific) or ontogenetic state (stage-specific). Natural mortality rates are highest in early life, gradually declining during larval and juvenile stages, and often becoming more or less stable after fishes reach maturity. Fishes may die from many causes, but predation is usually the principal agent of death. Other causal factors are poor nutrition, disease, and unfavorable environmental conditions. Human-related causes of mortality, for example, fishing, contaminants, altered water quality, deteriorating or lost habitats, and power-plant impingement or entrainment may be important sources of mortality in many freshwater and marine fishes.

The range in natural mortality rates of juvenile and adult fishes is variable and strongly related to the life span of a species. Small, short-lived fishes, such as many anchovies and atherinids, may sustain annual mortality rates after recruitment that range from 50% to 90%, making them essentially annual fishes because few individuals live more than 1 year. On the other hand, large and long-lived fishes such as sharks, sturgeons, and swordfish, may experience natural mortality rates of 10% per year or less. There are many medium-sized, slow growing fishes that are long-lived, such as the Pacific rockfishes (*Sebastes* spp.) or striped bass (*Morone saxatilis*) that also have low natural mortality rates. Knowing the natural mortality rate of a population is important, especially for populations that also are subjected to significant mortality from fishing. Despite this need, the natural mortality rates of most fishes are poorly estimated or unknown. It is a difficult and sometimes costly task to estimate natural mortality rates, which vary with age or size, in addition to being inversely correlated with the life span of a species. For heavily exploited species such as Atlantic cod (*Gadus morhua*) and Atlantic herring (*Clupea harengus*), or freshwater percids, fishing mortality rates may be as much as five times higher than the natural mortality rates. Such high mortality rates can destabilize a population and jeopardize the sustainability of a fishery or cause failures in recruitment followed by collapses of stocks.

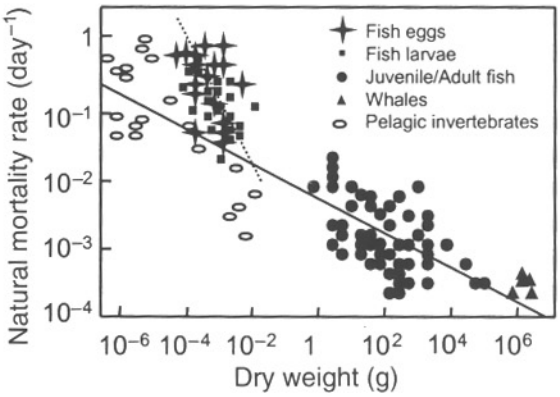

Figure 3.1 Relationship between mortality rate (day^{-1}) and dry weight for marine organisms. Dotted line represents the regression line and slope for fish eggs and larvae, which is significantly higher than the slope of the regression for marine organisms in general. The overall slope is −0.25, while the slope for fish eggs and larvae is −0.85 (reproduced from McGurk 1986 with permission of Inter-Research).

It has been demonstrated that the natural mortality rates of marine organisms in pelagic ecosystems, ranging from the smallest invertebrate larvae to whales, are strongly size-dependent and decline approximately as $M = 0.0053W^{-0.25}$ (McGurk 1986), where W is individual weight. The power relationship expressed here is believed to be the consequence of predation in size-structured aquatic ecosystems. Natural mortality rates of juvenile and adult fishes fit this picture reasonably well, but for eggs and larvae, the exponent tends to be even less than −0.25, indicating a higher than expected mortality rate during these stages and a rapid decline in M with ontogeny (Figure 3.1). The high and variable mortality rates observed for the smallest stages of fishes (Table 3.1) suggest that predation, a primary cause of death, is also high and variable, and potentially a major contributor to fluctuations in levels of recruitment that characterize fish year classes (see Chapter 4).

3.1.1 Survivorship curves

Mortality of a population is usually expressed as, and estimated from, the decline in numbers in relation to age. This relationship, in which numbers of survivors are plotted

Table 3.1 The average relationship between M and W for five species of fishes during the larval stage.

Species	Relationship
American shad	$M = 1.724W^{-0.392}$
Northern anchovy	$M = 1.073W^{-0.353}$
Bay anchovy	$M = 2.284W^{-0.318}$
Walleye pollock	$M = 3.874W^{-0.622}$
Striped bass	$M = 4.875W^{-0.424}$

From Houde (1997).

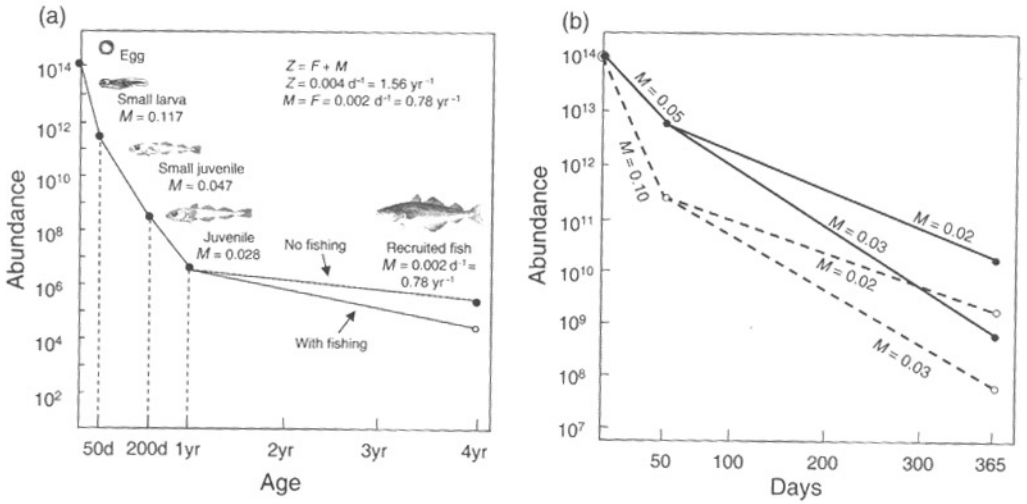

Figure 3.2 Conceptual illustrations of survivorship in a fish cohort from the egg stage through recruited stages. (a) Stage-specific mortality rates for four designated life stages. Effects of fishing are illustrated for the recruited stage. (b) Effects on survival of variability in mortality rates during the larval stage (0–50 days) and in the juvenile stage (50–365 days). Order-of-magnitude variability in survival results from modest changes in mortality rates (M = instantaneous natural mortality, F = instantaneous fishing mortality, Z = instantaneous total mortality).

against age, is a survivorship curve. Fishery scientists often derive this relationship as a "catch curve" in which the slope of the $\log_e$ abundance of surviving individuals plotted against age (often in years) is the age-specific (or instantaneous) mortality rate. In early life stages, the initial slope (steepness) of the survivorship curve and subsequent changes in the slope during early life illustrate the effects of coarse controls that can determine the fate of a cohort (Figure 3.2a). Even small changes in the slope of the survivorship curve (that is, mortality rate) early in life, when numbers are high, can generate very substantial differences in abundances by the end of the larval stage* (Figure 3.2b). It is easy to demonstrate by simple simulations that very high loss rates early in life, from whatever cause, can coarsely control the fate of a cohort's abundance later in life, resulting in a failed or poor recruitment. The converse, however, is not true; low mortality rates during the egg and larval stages do not guarantee high survivorship at recruitment, if mortality rates during the juvenile stage are elevated even modestly above average levels during this life stage, which may be hundreds of days in duration (Figure 3.2b).

3.1.2 Estimating mortality

Survivorship curves generally provide the basis for mortality estimation in fishes. This is true whether we are estimating annual mortality rates of recruited fishes or the losses

* Here, "stage" is used in a broad sense to represent major phases of a fish's life history (such as, embryonic, larval, and juvenile), rather than as a more instantaneous depiction of ontogenetic state as in Chapter 1.

during egg, larval, and juvenile stages. The catch curves that are fitted to abundance-at-age data provide estimates of losses and the rates of mortality. It is important to accurately estimate mortality rates in any stage to understand how factors such as fishing and natural mortality rates act to control population abundances. It is particularly important, but also especially difficult, to estimate mortality in larval stages. A major impediment to obtaining good estimates of larval mortality is our poor ability to obtain representative samples of larvae that are patchily distributed in both freshwater and marine habitats. The open nature of many aquatic environments, in which complex hydrography and larval behavior interact to disperse, transport, and distribute larvae over broad regions, challenges the ability of fishery scientists to estimate larval mortality.

3.1.3 The components of mortality

The losses in a population, which are proportional to the mortality rate, are expressed as

$$-\mathrm{d}N = M \cdot N_t \, \mathrm{d}t \tag{3.1}$$

where N is the number of individuals in a population, M is the natural mortality rate, and t refers to age. Solving

$$-M \, \mathrm{d}t = \frac{1}{N_t} \cdot \mathrm{d}N \tag{3.2}$$

and

$$N_t = N_0 \cdot \mathrm{e}^{-M \cdot t} \tag{3.3}$$

Mortality rates can be partitioned into component causes. For post-juvenile and adult stages, natural mortality M and fishing mortality F (for species that are fished) are the two components that contribute to total mortality Z ($= F + M$). For unfished species and for life stages on which no fishing takes place, $F = 0$ and $Z = M$. These additive "instantaneous" rates (per unit time) are related to more conventional, conditional loss rates (fraction dying per unit time) by the following relationships

$$A = 1 - \mathrm{e}^{-Z} \tag{3.4}$$

$$m = 1 - \mathrm{e}^{-F} \tag{3.5}$$

$$n = 1 - \mathrm{e}^{-M} \tag{3.6}$$

where A is the total mortality, that is, the proportion dying during a specified period, m is the proportion expected to die from fishing if no other causes of mortality were operating, and n is the proportion expected to die from natural mortality in the absence of fishing. For species or early life stages with $F = 0$, the conditional total mortality simplifies to $A = n = 1 - \mathrm{e}^{-M}$. In some cases, the natural mortality rate, M, can be partitioned into its constituent components, for example, predation (M_1), and other causes (M_2), although in most cases, the fractional mortality attributable to specific natural causes is unknown.

Natural mortality of organisms can arise from many sources, but predation is usually the primary cause in aquatic ecosystems. Additional sources of mortality can include starvation, disease, or effects of poor water quality and other unfavorable environmental conditions.

For early life stages of fishes, the role of nutrition and its effect on growth rate, and therefore size-at-age, can be crucial since predation may be size-specific and size-selective in size-structured aquatic ecosystems. Interactions between growth rate and mortality rates experienced by young fishes are believed to be among the more important variables that determine recruitment success in marine fishes. Fishing may become an important source of mortality to juvenile and adult fishes, but larval stages are seldom exposed to fishing mortality. In a few notable exceptions, fisheries are directed at larval stages of anchovies and sardines or newly transformed eels in the Mediterranean Sea and western Pacific Ocean.

3.1.4 Concepts of compensation and density dependence

Natural mortality rates may be rather constant with respect to age and size in fishes that survive early life and a single value for M is often assigned to older life stages after the age at recruitment. But during the larval and early juvenile stages, mortality rates are highly variable and responsive to environmental variability, shifts in predation pressure, or prey availability. To compensate for high variability in mortality and its propensity to destabilize populations, fish stocks have a compensatory reserve that can be expressed through density-dependent mortality acting during early life. This regulatory mechanism results in proportional increases of mortality rates in relation to abundance of the young stages themselves or abundance of adult spawners. If density dependence is operating, mortality rates increase in proportion to abundance, a result of competition for resources or an increase in specific predation rates on the population. While direct evidence for density-dependent, compensatory mortality of young fishes in the sea remains rather uncommon, models imply that even small amounts of density dependence can be very important in stabilizing recruitments (see Chapter 4) and regulating abundances of fish stocks. Without such a mechanism, these stocks would be expected to fluctuate wildly as mortality rates in early life shifted up or down in response to annual variability in environmental conditions.

3.2 Larval mortality: concepts and relationships

Mortality rates in fishes decline with size and age during early life. Initially, rates may be very high, exceeding 50% day^{-1} in some species and commonly exceeding 10% day^{-1}. For example, in a review of mortality estimates (Houde & Zastrow 1993), the mean mortality rate for marine fish larvae was $M = 0.24$, a rate equivalent to a loss of 21.3% day^{-1}. The mean mortality rate for freshwater fish larvae is lower than that for marine fish larvae, but freshwater larvae still die at a high rate, on average at $M = 0.16$, equal to 14.8% day^{-1}. The higher mortality rate for marine larvae probably is a consequence of their much smaller average size and higher vulnerability to a more diverse assemblage of predators. Although significantly different for marine and freshwater larvae, such high mortality rates in both groups lead to rapid losses from cohorts in early life. If mortality rates did not decline during ontogeny, few individuals would remain alive at the end of early life stages to contribute to recruitment. For marine species, only 180 individuals are expected to survive the larval stage (>99.9% mortality) from an initial cohort of one million larvae under conditions of average mortality rates ($M = 0.24$) and larval stage durations (36 days). In the case of an

"average" freshwater species, lower mortality ($M = 0.16$) is estimated over a typical 20.7 day larval stage, but 96.4% are expected to die. It seems clear that mortality rates must decline during ontogeny and growth to maintain recruitment levels and insure stabilization of stock abundance (see Chapter 4).

Mortality and growth rates of marine fish larvae are strongly coupled during early life. Species with high mortality rates also have high growth rates, and both of these rates are strongly and positively correlated with temperature. Consequently, species from temperate and high latitudes die and grow at slower rates than species from tropical habitats or species that spawn under summer conditions. The larval stage is protracted for most species from cold environments, a consequence of slow growth rates, thus spreading the stage-specific mortality over a longer time frame. Despite the differences in daily rates for species from warm or cold environments, the stage-specific mortalities are similar because of the strong concordance between mortality and growth rates. For example, gadid species such as cod and haddock (*Melanogrammus aeglefinus*; Figure 1.3p, n), which live in high latitude seas and spawn at temperatures less than 10°C, spend approximately 100 days in the larval stage while tropical gobies (Gobiidae) and damselfishes (Pomacentridae) that spawn at temperatures greater than 25°C spend only 25 days in the larval stage. Yet, average survival rates at the end of their respective larval stages are similar because mortality and growth rates are strongly correlated, increasing at the same average rate with respect to temperature (approximately 0.01 per °C) (Figure 3.3).

Where data are available, it can be demonstrated that mortality rates decline regularly as development and growth of larvae occur, although the rate of decline with respect to body size or age is highly variable among species. The relationships describing the declines in M with respect to weight (W) for five species (including anadromous, estuarine, and marine species) during the larval stage (Table 3.1) ranged from $W^{-0.318}$ to $W^{-0.622}$. In all of

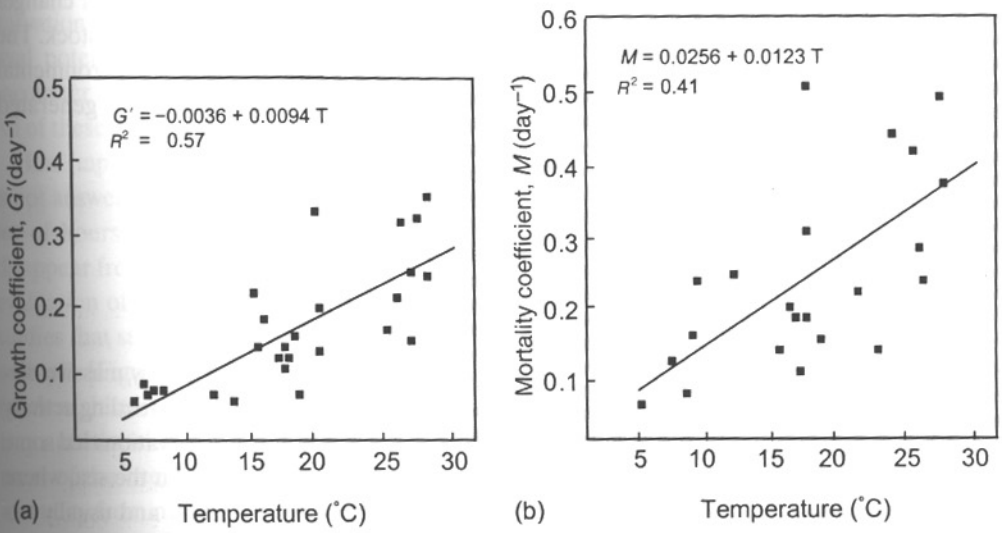

Figure 3.3 Relationships between G', the weight-specific growth rate, and M, the instantaneous mortality rate, with respect to temperature for several species of marine and freshwater fish larvae (derived from Houde 1989).

Table 3.2 The relationships between mortality rate (M) and dry weight (W, in μg) for 6 year classes of American shad and 7 year classes of walleye pollock larvae.

Species	Year	Fitted model
American shad	1979	$M = 4.48W^{-0.564}$
	1980	$M = 0.97W^{-0.319}$
	1981	$M = 1.13W^{-0.339}$
	1982	$M = 1.92W^{-0.381}$
	1983	$M = 0.78W^{-0.292}$
	1984	$M = 33.29W^{-0.889}$
Walleye pollock	1985	$M = 1.72W^{-0.522}$
	1986	$M = 1.68W^{-0.457}$
	1987	$M = 2.43W^{-0.515}$
	1988	$M = 68.59W^{-1.207}$
	1989	$M = 4.38W^{-0.661}$
	1990	$M = 1.31W^{-0.456}$
	1991	$M = 13.52W^{-0.820}$

From Houde (1997).

the cases, declines in M were more rapid than the $W^{-0.25}$ predicted from size-spectrum theory. As one example of declines in mortality, the averaged ontogenetic declines in mortality rates for observations made over 7 years on walleye pollock (*Theragra chalcogramma*, Figure 1.3o) were 5 days old, $M = 0.21$; 12 days old, $M = 0.11$; 37 days old, $M = 0.04$. The mortality rates themselves and the ontogenetic declines in the rates during the larval stage can differ annually in response to variable environmental conditions and predation that larvae encounter from year to year (Table 3.2). Annual variability is important because even relatively small, subtle changes in stage-specific mortality rates can generate major changes in abundances of juvenile survivors and ultimately the level of recruitment to the stock. The ability to estimate stage-specific mortality and to relate variability in it to environmental factors is a key requirement for understanding how recruitment variability is generated (see Chapter 4).

3.3 Causes of early life mortality

3.3.1 Starvation and nutritional deficiencies

Laboratory experiments on feeding by larval fishes often are characterized by massive die-offs at the termination of the yolk-sac stage when larvae are obliged to begin feeding actively on plankton, rather than subsisting solely on their yolk reserves. Such observations led some scientists to propose that similar mass starvations might occur commonly in the sea, where suitable kinds and amounts of planktonic prey were believed to be patchy and usually too dilute to support high survival rates. The Critical Period Hypothesis of Johan Hjort emerged from such observations (Figure 3.4). Massive and sudden mortalities of larvae at the first-feeding stage, taken to be evidence supporting the hypothesis, have been

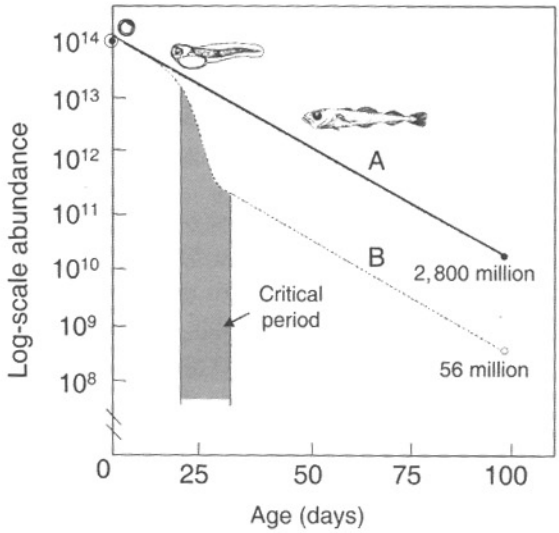

Figure 3.4 Survivorship curves showing effect of a "critical period" on abundance of survivors at 100 days post-hatching. Both populations have an initial mortality rate of $M = 0.10$ day^{-1}. The population that experiences high losses in the "critical period," although recovering to experience the initial $M = 0.10$ day^{-1} rate after the "critical period," is 50 times less abundant at 100 days post-hatching.

observed in some cases. Temporal and spatial matches between larval hatching and the proliferation (blooms) of planktonic prey (the Match/Mismatch Hypothesis of David Cushing [1990]) promote high survival of some larval populations, such as Norwegian cod. On average, the concentration of prey suitable as larval food seems too low to readily support survival of fish larvae, leading to the hypothesis that only under conditions where aggregations of prey occur, for example, in depth-stratified layers, can larvae feed successfully and avoid starvation (the Stable Ocean Hypothesis of Reuben Lasker). On fine- and microscales, survival potential of larvae is elevated in the presence of microturbulence that increases encounter rates of larvae with prey (as proposed by Brian Rothschild and Thomas Osborn). All of these hypotheses to explain recruitment variability are discussed in the next chapter.

How important is starvation as a major cause of mortality to fish larvae? The question is not answered easily because larvae that are poorly nourished can be weakened in a matter of hours, becoming highly vulnerable to predation. Consequently, such larvae may disappear from populations at an accelerated rate and may not occur in collections to allow evaluation of their poor nutritional condition. Nevertheless, there is evidence from many studies that starvation-related mortality of larvae (in Atlantic herring and Atlantic cod, for example [Figure 1.3m, p]), is important. While measures of larval nutritional condition (such as, histological measures, RNA/DNA analysis, or lipid indices) may accurately portray nutritional status of sampled survivors, the indices may not provide a reliable assessment of the overall condition of a larval population or the probability that starvation and mortality from nutritional deficiencies had played a major role. In field collections, we can presume that a larger fraction of larvae than is actually observed was susceptible to starvation mortality. Starvation and nutrition-related mortalities will selectively remove poorly nourished larvae, which are more vulnerable to predation. Selective predation on slower

growing, but healthy individuals also occurs, potentially independently of larval nutritional condition. However, such selective mortality can be the consequence of relatively low food intake and, as such, is related to nutrition. Selective mortality not only obscures starvation as the source of mortality but it also may cause shifts in observed size distributions, age structure, and apparent growth rates of survivors, greatly complicating interpretation of dynamics in early life.

During starvation, larval fishes approach a point of nutritional deficit referred to as the "point of no return," which can occur in a matter of hours (see Chapter 1) and leads to death. Juvenile fishes are much less susceptible to mortality from starvation than larvae. Nevertheless, they too can suffer elevated mortality during long periods of starvation or under poor nutritional conditions, especially if they grow slowly and have high vulnerability to predators. Research to date has provided only threads of evidence that survival and growth of larval stages are regulated by the abundances of larvae present that compete for limiting prey resources. But, there is a high probability that such density-dependent mortality, which is regulated by competition for limited food, will increase during the late larval and juvenile stages (Figure 3.5).

In temperate freshwater and marine environments, overwinter mortality of young-of-the-year individuals may occur. Such mortality is usually size dependent, with smaller and poorly conditioned individuals being more susceptible to mortality from energetic deficiencies or predation as the winter proceeds. Size-selective overwinter mortality has been reported for many species, including smallmouth bass (*Micropterus dolomieu*), white perch (*Morone americana*), striped bass, and Atlantic silverside (*Menidia menidia*). Such mortality of

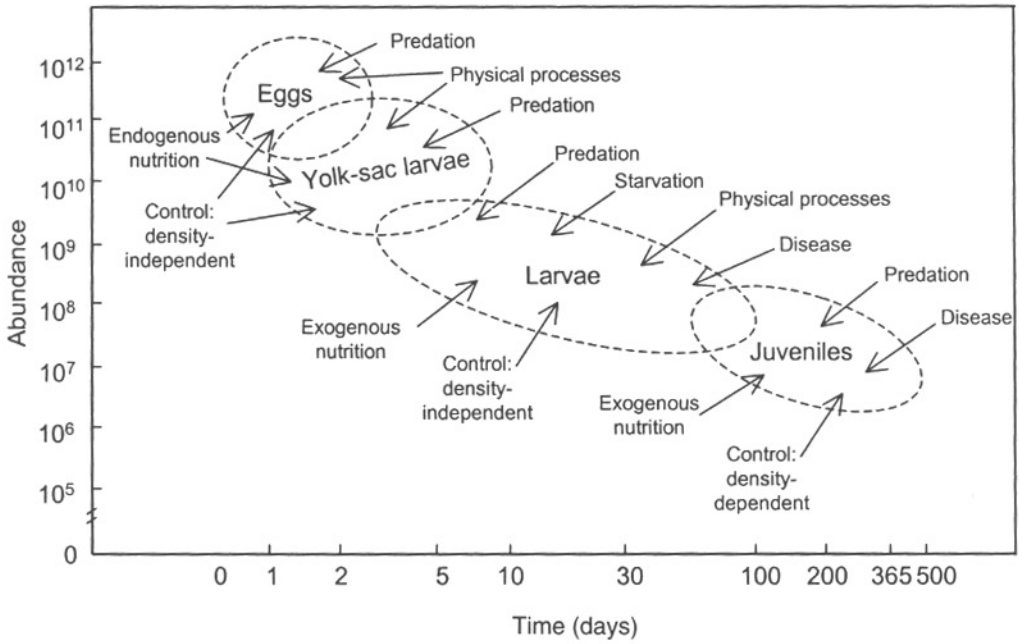

Figure 3.5 Survivorship curve conceptualizing the recruitment process in fishes, including factors that affect mortality and growth. Hypothesized mechanisms of control are indicated (reproduced from Houde 1987 with permission of the American Fisheries Society).

overwintering young-of-the-year fish can selectively alter the size distribution of survivors that will contribute to recruitment as well as the abundance of recruits. In some cases, extreme winter mortalities of young-of-the-year fishes can occur near the range boundaries of tropical and subtropical fishes. For example, annually variable overwinter mortality of Atlantic croaker (*Micropogonias undulatus*) in mid-Atlantic estuaries of North America is believed to be a strong controller of its year-class size.

3.3.2 Predation

Predators inflict a heavy toll on the young stages of fishes and probably are the biggest single cause of mortality. As noted above, predation losses may be linked to nutritional deficiencies that increase vulnerability of young fishes to predators. Predation on fish eggs is size-specific and predation on larvae may be both size-specific and growth-rate dependent. As larvae grow and develop, swimming ability and predator-detection capabilities improve. These developmental factors generally lead to lower predation mortality as ontogeny proceeds (see Chapter 1).

Many organisms, both vertebrates and invertebrates, prey on young fishes. Important predators include juvenile and adult fishes, jellyfishes (ctenophores and medusae), chaetognaths, euphausiids, and insects. The diversity of predators in marine ecosystems apparently is broader than that in freshwater ecosystems. For example, cnidarians and ctenophores, represented by many taxa in marine ecosystems and demonstrated to be major consumers of fish eggs and larvae, are hardly represented in freshwaters. Insects, on the other hand, are essentially confined to freshwater ecosystems and tidal freshwaters of estuaries. Some organisms, not usually considered as predators on early life stages, may be important predators under some circumstances. For example, copepods, amphipods, hydroids, birds, and amphibians all consume young stages of fishes. Some of these predators, such as birds, are significant agents of mortality on small juvenile fishes in both fresh waters and marine waters.

There is a general lack of information on the community of predators that consumes fish eggs and larvae, especially in quantifying effects of specific predators, which makes it difficult to evaluate taxa and their roles as predators. Stated simply, in any region and moment of time, there not only is an array of predator species and sizes that can consume eggs and larvae, but also variable amounts of alternative prey which must be considered in evaluating predation on early life stages and its consequences to a population. Research to date, including modeling approaches, rarely has been able to partition mortality of young stages of fishes among the array of predator taxa and sizes that are present. We are quite confident that predation is a major source of mortality to young fishes, but we are lacking sufficient detail to understand how the process operates in a complex community of potential predators and prey.

Most predators on young fishes feed either by active pursuit or by ambush. Cruising raptorial predators such as small pelagic fishes actively pursue larval fish prey. Cruising, filter-feeding fishes, for example, sardines (*Sardinops* spp.) and some anchovies in marine waters, feed by filtering pelagic eggs or small larvae from the water. Drifting jellyfishes may ambush eggs or larvae by entangling them in tentacles or may employ cruising, filtering behavior to encounter eggs and larvae (for example, lobate ctenophores). Fishes and invertebrates in both freshwater and marine vegetation may be ambush predators or may actively pursue

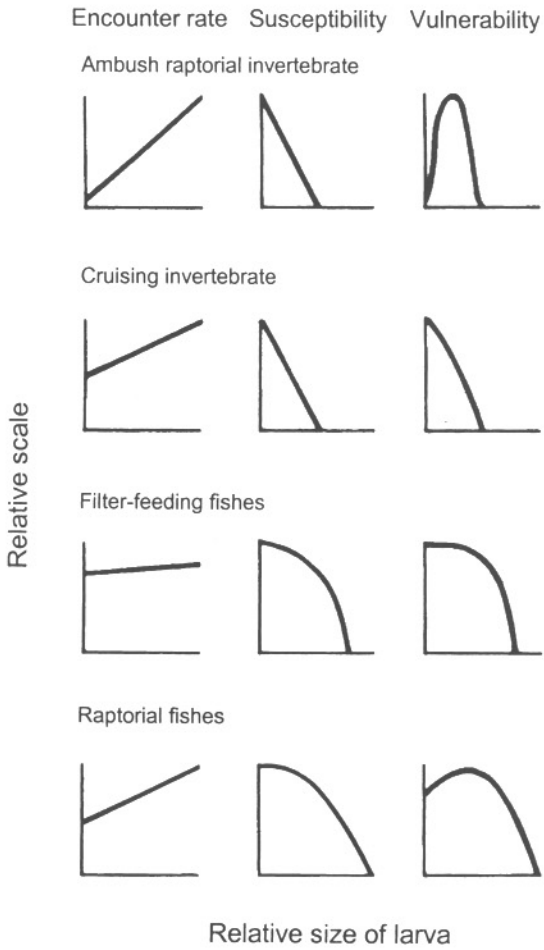

Figure 3.6 Conceptual models showing encounter rates, susceptibility, and vulnerability of larval fishes to predation. The dome-shaped vulnerability curves illustrate the hypothesized trends in mortality relative to larval size for predators of specific size and predatory behavior (reproduced from Bailey & Houde 1989 with permission of Academic Press).

young fishes. Demersal predators, primarily fishes, may ambush newly settling young that descend to reef or hard-substrate habitats at the end of the pelagic stage.

The relative vulnerability of larval fishes to many kinds of predators can be represented and illustrated principally by dome-shaped curves in relation to larval size (Figure 3.6). The susceptibility of larvae to attack and capture by a particular type and size of predator usually declines as larvae grow, a function of increases in swimming speeds, improved ability to detect predators, and better avoidance and escape capability (see Chapter 1). Vulnerability, which represents the net effect of ontogenetic changes in encounter probability and susceptibility, may increase for intermediate size larvae (Figure 3.6), at least with respect to a particular predator of specific size and capability. Eventually, larger larvae become increasingly more adept at detecting predators and avoiding attacks, thus reducing their vulnerability despite the possibility that encounter rates may continue to increase.

Laboratory experiments have confirmed the high vulnerability of fish eggs and larvae to a variety of predators. The size-specific nature of predation is also illustrated by laboratory experiments which indicate that predators, independent of taxon, tend to consume larval prey that is preferentially about 10% of their body size (Paradis *et al.* 1996). Modeling studies have demonstrated that variability in predation mortality can control or regulate the recruitment process and temporal variability in recruitment success of fishes. Despite the wealth of laboratory research and the proliferation of models, predation remains difficult to detect or evaluate in the sea. Eggs and especially larvae are soft-bodied and are either destroyed upon consumption, making them unrecognizable, or are digested quickly. As a consequence, eggs and larvae may go undetected or be under-represented as prey unless sophisticated immunoassays are conducted on gut contents of suspected predators.

Mortality from cannibalism represents a special kind of predation. Cannibalism on eggs is common in some clupeoid fishes, either by incidental filter-feeding or by selective consumption. A significant fraction of egg and yolk-sac-larva mortality (>20%) in sardines (*Sardinops* spp.) and anchovies (*Engraulis* spp.) in upwelling ecosystems can be accounted for by this type of cannibalism. Cannibalism also is common among fishes that practice parental care (for example, Cichlidae, Gasterosteidae) and apparently becomes significant when parents judge the reproductive value of a clutch to be low, opting to cannibalize it and promote their own nutrition, which potentially results in more successful spawning in the future. Cannibalism also may occur in many species where metamorphosing larvae settle onto a substrate already occupied by older and larger conspecifics. Sibling cannibalism, in which larvae prey upon sibling larvae is reported in many taxa, including freshwater esocids (Figure 1.3e) and characids and marine scombrids (Figure 1.3s). It is beyond the scope of this chapter to critically evaluate the role of cannibalism, except to note that such mortality is density-dependent and thus can serve as a mechanism to regulate survival and recruitment level (see Chapter 4).

3.3.3 *Physics: transport, retention, and dispersal*

Water currents that disperse or transport young stages of fishes into environments unfavorable for survival can be a significant source of mortality. Variability in physical properties of aquatic ecosystems, operating on fine- to basin-wide scales (meters to 1000 km) is strongly dependent on wind and weather patterns that vary on time scales from days to seasons. Fine-scale variability in water-column properties, especially stratification and its effect on the stability of vertical distributions of fish eggs and larvae, their predators, and prey plays a critical role in controlling conditions that determine survival of early life stages. For some species, especially clupeoid fishes in upwelling regions of marine ecosystems, the relaxation of upwelling under low wind conditions is associated with enhanced larval survival and growth. In this circumstance, the stability of the water column under calm conditions allows formation of layers or patches of plankton prey at concentrations suitable for larval feeding, enhancing the probability that larvae will encounter sufficient prey to survive. The breakdown of water-column stability under stormy conditions can disperse larval prey and produce poor feeding conditions, leading to mortality of anchovy and sardine larvae (Figure 3.7).

Hypotheses dealing with recruitment processes historically have focused on transport and retention mechanisms as the critical factors affecting survival and growth of larval

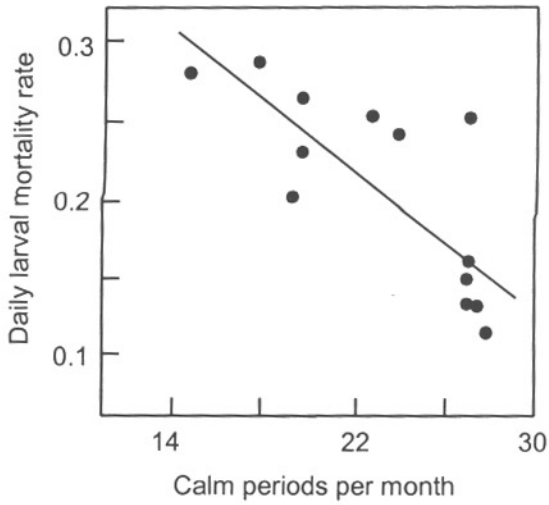

Figure 3.7 Daily mortality rate (day^{-1}) of northern anchovy larvae in relation to calm periods per month in the California Current over a 13-year period. Larval mortality rate declines substantially under calm conditions when water-column stratification occurs that presumably promotes larval feeding success (reprinted from Peterman, R.M. & Bradford, M.J 1987. Wind speed and mortality rate of a marine fish, the northern anchovy (*Engraulis mordax*). *Science* **235**, 354–356. Copyright 1987 with permission from the American Association for the Advancement of Science).

fishes during the pre-settlement stages. Aberrant transport or poor retention will lead to high mortality. Retention mechanisms in estuaries or frontal systems of dynamic marine ecosystems are particularly important to retain larvae in regions where they have a reasonable chance to survive. In recent years, major national and international research programs have attempted to unravel the physical mechanisms that promote survival of early life stages (for example, FOCI for walleye pollock, GLOBEC and OPEN for Atlantic cod, and SABRE for Atlantic menhaden [*Brevoortia tyrannus*]). The complexities of aquatic ecosystems and physical processes therein have required that dual emphasis, distributed between observational science and coupled biological–physical modeling, be coordinated to understand how physics and biology interact to support survival of young fishes.

While it is quite clear that physics at meso- and broader scales (one to hundreds of kilometers) plays a role in controlling levels of mortality of larval stages, physics at finer scales, for example, on millimeter to meter scales, also can be important. Turbulence at those scales plays a role in controlling contact rates between larval fishes and their prey. Rates of contact, controlled by microscale turbulence, can directly influence the nutritional status of larvae and can directly affect their vulnerability to predation. Modeling studies, laboratory experiments, and some field observations on the role of fine-scale turbulence in promoting larval feeding success, and thus growth and survival, have proliferated in recent years. They have attempted to explain how survival of fish larvae in the sea is possible under conditions where average prey levels are thought to be lower than necessary for larval survival. In the case of Atlantic cod larvae, it is apparent that survival is maximized under moderate wind conditions that generate microturbulence sufficient to enhance encounter rates between

larvae and prey but which is not so turbulent that larvae are unable to capture prey that they encounter (MacKenzie & Kiørboe 2000).

3.3.4 Water quality and nursery habitats

Egg and early larval stages of fishes are surrounded and enveloped by their fluid environment and unable to escape their immediate surroundings because they are immobile (eggs) or swim poorly. This makes them vulnerable to poor water quality, whether a consequence of human activities or natural phenomena. Mortality from poor water quality is perhaps most threatening to young fishes in spatially restricted freshwater environments and in estuarine tributaries that serve as spawning sites for many anadromous fishes. Release of contaminants and toxic materials, either as chronic or episodic inputs, can be lethal to eggs and larvae of fishes or may prevent successful spawning by adults. Other water-quality factors may act in more subtle ways. For example, increased loadings of nutrients such as nitrogen and phosphorous can lead to eutrophication of many fresh waters, estuaries, and coastal ecosystems, which can deplete dissolved oxygen, leading to hypoxia or anoxia that is lethal to fish eggs and larvae. Shifts in ecosystem structure during eutrophication may transform macrophyte-dominated ecosystems into phytoplankton-based ecosystems that lack aquatic vegetation that is critical to shelter juvenile fishes from predation in freshwater and coastal systems. Another threat to eggs, larvae, and juvenile fishes is the acidification of many fresh waters and tidal tributaries that has developed in recent decades as a consequence of industrial combustion of fossil fuels and acid rains that result. Low pH, in the range of 5.0–6.5, has made many fresh waters unsuitable for survival of eggs and larvae of salmonids, and causes mortality of the eggs and larvae of anadromous shads and river herrings in their nursery streams. Low pH events may be episodic, occurring immediately after heavy rains from runoff into poorly buffered streams or ponds, or may be chronic when related to acidification from mining operations or other sources of persistent acidified runoff.

Natural variability in water quality may be caused by weather, precipitation, and runoff events or can be tied to geology and soils in drainage basins. In some ecosystems, these factors present risks to survival or increase the variability in survival of early life stages of fishes. Temperature, nutrient status, levels of alkalinity, conductivity, salinity in estuaries, pH, dissolved oxygen, and turbidity all can vary substantially both temporally and spatially, and on time scales from hours to decades. Lethal levels of these factors, especially as pulsed events, may occur uncommonly on continental shelves and in the open sea, but they can occur more frequently in small estuarine and freshwater ecosystems where water quality and environmental conditions are subject to rapid change.

Water quality can act chronically or episodically to control survival of early life stages. For example, temperature, a primary controller of physiological activity in fishes, can act chronically by controlling growth rates and stage durations, thus indirectly influencing survival potential. In the case of plaice (*Pleuronectes platessa*) eggs, which require many days to hatch at ambient temperatures in the North Sea, egg mortality rate is directly related to water temperature (Figure 3.8). In years of low temperatures, plaice eggs and young experience high survival that supports high recruitments. In fresh waters and in estuaries, heated effluents (episodic or chronic effects) from power plants can be lethal, either directly or indirectly, in addition to causing mortality by entrainment and mechanical damage. Sudden and episodic

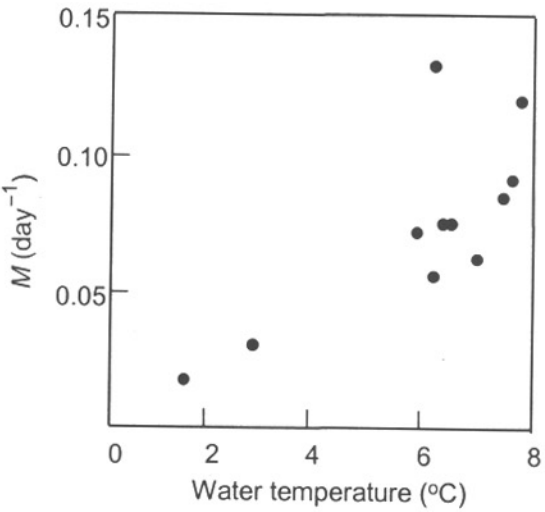

Figure 3.8 Daily mortality rate (day^{-1}) of plaice eggs in the Southern Bight of the North Sea in relation to average water temperature during egg development for an 11-year period (data from Harding *et al.* 1978, figure modified from Van der Veer *et al.* 2000 with permission of Academic Press).

changes in temperatures of nursery habitats that occur naturally are common in temperate regions, especially during transition seasons when many freshwater and anadromous fishes spawn. Such episodic events often are accompanied by other water-quality changes that can be fatal to egg and larval stages or the prey of larvae. For example, striped bass eggs and larvae (Figure 1.3q) in Chesapeake Bay tributaries may experience partial or complete mortality when water temperatures drop below 12°C, which occurs after the passage of weather fronts and cold rains during the April spawning season. Those same weather fronts and rains also can substantially reduce pH, conductivity, and alkalinity levels, thus magnifying the effects of temperature stress and increasing the risk of mortality to eggs and larvae. Chapter 7 provides an extended discussion of spawning and nursery habitat.

3.3.5 Diseases and parasites

Diseases and parasites are agents of mortality to fishes in all life stages. In juvenile and adult fishes, especially in aquaculture and sometimes in natural environments, effects of diseases are recognized, if not quantified, and epizootics sometimes are reported as major controllers of abundance. Except in aquaculture, we seldom consider death of fish eggs and larvae from diseases or parasites as serious sources of mortality. Bacterial and viral diseases certainly must take a toll of young fishes. Parasites of eggs and larvae, including dinoflagellates, protozoans, helminths, and copepods have been reported regularly, although mortality rates and population-level consequences are unevaluated. Under laboratory conditions, external parasites such as copepods of the genus *Caligus* are obvious sources of mortality to cultured larvae. In the wild, diseased, parasitized, and poorly conditioned larvae and eggs disappear rapidly from populations, either through selective predation or decomposition of dead bodies, leading to a poor appreciation of disease or parasites as sources of mortality to early life stages of fishes.

Genetic disorders leading to abnormal development and death during early life are known and reported, primarily from laboratory research and aquaculture experiences. In polluted waters, incidences of morphological abnormalities in eggs and larvae may be elevated, for example, in winter flounder (*Pleuronectes americanus*) in some coastal areas, that are clear expressions of contaminant effects, but the levels of mortality and implications for population-level control are largely unknown.

3.3.6 Interacting factors

It is a simplification to assign conditional probabilities to single causes of mortality without understanding how potentially lethal factors interact. As an example, we are still unable to easily separate the fraction of young stages that die from starvation from those that die from predation. Poorly fed, slowly growing larvae are more vulnerable to predators. In laboratory experiments and in model simulations, such larvae are selectively eaten by predators and have a relatively low probability of survival. Despite such observations and strong intuition, it remains problematic or impossible to quantify the proportional mortalities from starvation or predation in natural ecosystems. There is no denying that the interaction between nutritional state and vulnerability to predation must be important. Low prey levels lead to slow growth, longer stage durations, and greater probability of being consumed during an early life stage. In the case of the bloater (*Coregonus hoyi*, Figure 1.3b), a modeling analysis demonstrated how levels of growth and the variable potential for growth in a larval population modified the effects of predation on survival (Rice *et al.* 1993). High growth rates, and especially variable growth rates, reduced the risk to bloater larvae of size-selective predation and promoted recruitment potential (see Chapter 11).

Physical and biological processes also interact as they affect survival probability. In many cases, failed retention, unfavorable transport, or poor environmental conditions (for example, temperature, pH, hypoxia) may act directly to kill eggs and larvae, but these conditions also will affect the predators and prey of early life stages, creating a complex web of interactions that have important implications for survival of early life stages. Finally, effects of contaminants or poor water quality may alter behavior of larvae, thus impeding feeding and reducing growth rates or making larvae more vulnerable to predation.

Cascading effects can increase the risk of mortality to eggs and larvae in stressed aquatic ecosystems. For example, excess nutrients can lead to eutrophication, which may result in low dissolved oxygen, harmful algal blooms, losses of aquatic vegetation, and probable increases in some larval predators, such as jellyfishes, as the trophic state of the ecosystem shifts. Evaluating and partitioning the effects of such multiple sources of mortality is difficult and seldom accomplished, except in modeling research, which is now contributing valuable insight into the complex and interacting processes that generate mortality in the early life of fishes.

3.4 Estimating larval mortality

Obtaining accurate and precise estimates of mortality rates for any life stage of fishes can be a formidable task. The general pattern of survivorship curves in early life is known

(Figures 3.2 and 3.5), but the levels of abundance and loss rates for a particular life stage can seldom be estimated with certainty. The magnitude of required effort and the cost of the estimation task are greatest in large ecosystems, especially those which are essentially unbounded and subject to significant losses through dispersal and translocation, in addition to losses from mortality. The possibility of success in determining mortality improves in embayments, estuaries, and freshwater habitats, where dispersal losses are minor or can be accounted for by sampling the entire system. Successful estimation of mortality depends upon accurate determination of abundances and dependable assignment of individuals to age classes or stages. Fortunately, most fish larvae and many juveniles can be accurately aged from daily increments in otoliths (see Chapter 2), a great advantage in determining age-specific mortality.

3.4.1 Catch curves and survivorship

As noted earlier, the rate of loss of individuals from a population by death expresses the mortality rate. Age-specific losses often are estimated from a "catch curve" in which abundances-at-age of survivors are plotted against age (Figure 3.2). A log-linear regression equation of $\log_e$ abundances on age then can be fitted to the data to estimate the regression coefficient (slope), which is the instantaneous mortality rate

$$\ln N_t = \ln N_0 - M \cdot t \tag{3.7}$$

where N_t is abundance-at-age t (often expressed in days for early life stages, but in years for older fishes), N_0 is an estimate of abundance at the beginning of the stage, and the slope M estimates the instantaneous mortality rate, usually expressed as day^{-1} for fish eggs or larvae but year^{-1} for recruited fishes. The cumulative mortality over a period of t days is $M \cdot t$, the survival rate is

$$S = e^{-M \cdot t} \tag{3.8}$$

and the proportion of a population dying in a time period is

$$A = 1 - e^{-M \cdot t} \tag{3.9}$$

The hypothetical survivorship curve (Figure 3.2a) and tabulated summary (Table 3.3) illustrate a survivorship analysis based on catch curves for a typical marine fish species. Initially, mortality rates of a newly spawned cohort are high, frequently greater than 10% day^{-1} and sometimes more than 50% day^{-1}. Rates generally decline during early life stages. Cumulative mortalities ($M \cdot t$) usually are highest during the egg–larval or juvenile stages when more than 99.5% of individuals may perish. In the illustrated example, the natural mortality rate of cohorts after recruitment is 52% annually ($M = 0.002$ day^{-1}), a high rate but 58 times lower than the daily rate during the egg–larval stage. Imposition of fishing mortality certainly has a substantial effect on abundances of recruited age classes (Figure 3.2a), but it is early life mortality rates and their variability that are the dominant factors affecting the dynamics, variability, and fates of recruiting cohorts (see Chapter 4).

An example of the application of a catch-curve approach, based upon intensive sampling of the entire Chesapeake Bay ecosystem and its population of bay anchovy (*Anchoa mitchilli*), a species that is essentially confined in the bay during its egg, larval, and juvenile stages, serves to illustrate the approach. Two bay-wide surveys provided data for a

Table 3.3 Instantaneous mortality rates (M, F, Z), cumulative mortalities (cum. Z), and percent mortalities within each larval stage during ontogeny of a typical marine fish ($Z = F + M$). See Figure 3.2a.

Stage/age	M (day^{-1})	F (day^{-1})	Z (day^{-1})	Cum. Z	Deaths (%)
Egg/larvae (0–50 days)	0.1170	–	0.1170	5.85	99.71
Early juvenile (50–200 days)	0.0470	–	0.0470	7.05	99.91
Late juvenile (200–365 days)	0.0280	–	0.0280	4.62	99.01
Recruited stage (no fishing) (1–4 year)	0.0020	–	0.0020	2.19	88.81
Recruited stage (fishing) (1–4 year)	0.0020	0.0020	0.0040	4.38	98.75

catch-curve analysis on bay anchovy larvae during June and July 1993. In this example, larval mortality was significantly higher in June than in July (Figure 3.9). The higher mortality in June ($M = 0.41$, 33.6% day^{-1}), compared to the lower mortality in July ($M = 0.23$, 20.5% day^{-1}) was attributed to high predation by abundant jellyfish predators in June. The differences in mortality rates indicated that the vast majority of recruited bay anchovy in 1993 was derived from cohorts that originated from July spawning.

3.4.2 *Models and data*

The catch-curve method described above assumes that an equal proportion of a population dies at each age included in the analysis. If the proportional mortality rate is changing

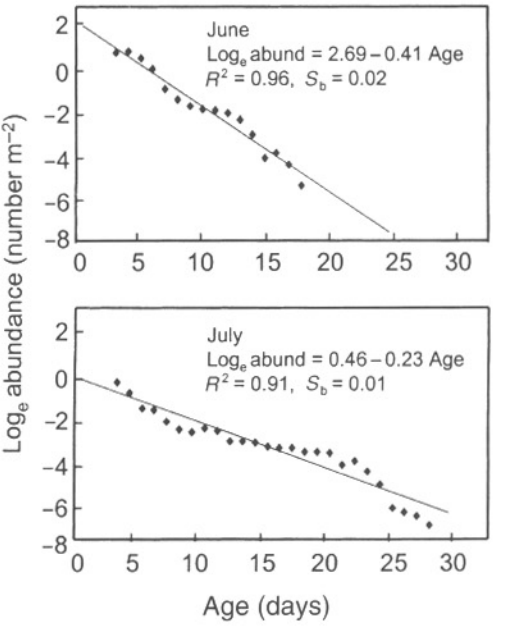

Figure 3.9 Catch curves of log$_e$ abundance vs. age (in days) for larval bay anchovy in Chesapeake Bay during June 1993 and July 1993. The regression coefficients (slopes) are estimates of M in each case (from Rilling & Houde 1999).

during a stage, this assumption may not hold and the simple log-linear model may be inadequate to estimate age-specific mortality rates in the life stage. Other models, such as the Pareto model, may describe the decline in numbers more accurately (Lo 1986), as in larval northern anchovy (*Engraulis mordax*) off the California coast. An example and comparison of the Pareto and log-linear models fitted to data for larval striped bass in the Nanticoke River, Maryland, demonstrates the utility and improvement in statistical fit (higher R^2) provided by the Pareto model (Figure 3.10). The Pareto model, which can be fitted to data by an iterative least squares method, assumes that mortality is a power function of age (or size). It is

$$\ln N_t = \ln N_0 + \beta \cdot t^{\alpha} \tag{3.10}$$

where β estimates the overall rate of decline and α describes the particular shape of the survival curve, depicting how mortality rate changes with age or size. Survival rates for any period (or size range) can be calculated by estimating the respective abundances N_t and N_{t-i}, and solving for $S = N_t/N_{t-i}$, where i represents the time interval in which mortality rate is estimated. In cases where the mortality rate declines substantially in relation to age (or size), especially at the youngest ages, as in the Nanticoke River striped bass example (Figure 3.10), the Pareto model will be a better choice to estimate age-specific (or size-specific) mortality rates.

In many instances, estimated mortality rates of pelagic fish eggs or larvae will be confounded by unaccounted losses due to dispersal out of the sampling area. In this circumstance, estimated mortality rates will be too high and must be adjusted for the non-mortality losses. Models that include terms to account for diffusive and advective losses in open, dynamic ecosystems, such as that of Helbig & Pepin (1998), may be necessary to separate dispersive losses from deaths.

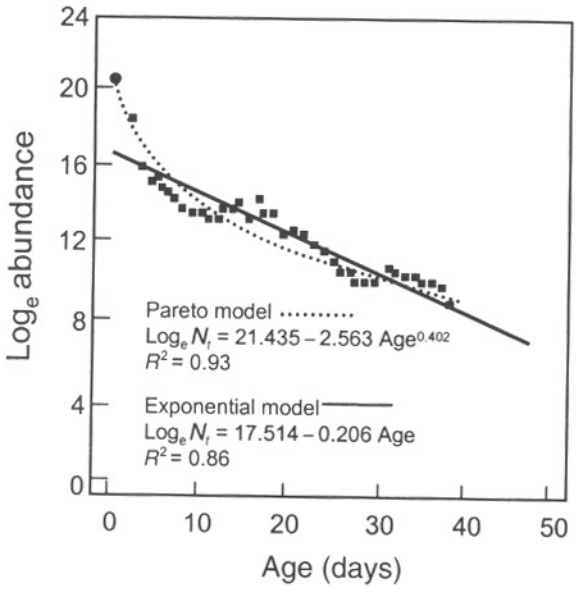

Figure 3.10 Catch curve for larvae of striped bass from the Nanticoke River, 1992. A linear $\log_e$-exponential model and a non-linear Pareto model are fitted to the data for comparison. In this case, the Pareto model provides a better fit (modified from Kellogg *et al.* 1996).

Stage-specific mortality

In many cases, our objective is to estimate the mortality that occurs within a life stage or between size groups to evaluate stage-specific dynamics during early life. Here, the linked dynamics of growth and mortality must be considered and can be expressed by evaluating the ratio M/G', where G' is the instantaneous growth rate in weight. This ratio, sometimes referred to as the "physiological mortality rate," can serve as an index of cohort dynamics and productivity that integrates mortality and growth processes (Beyer 1989, Houde 1996). It also can be used to evaluate the potential of cohorts to generate biomass during early life. Stage-specific survival is

$$S = \frac{N_s}{N_{s-1}} = \left(\frac{W_s}{W_{s-1}}\right)^{-M/G'} \tag{3.11}$$

where W_s and W_{s-1} are weights of fish at respective stages (see also Equation 2.9). Relative cohort biomasses with respect to life stages also can be expressed in relation to M/G'

$$\frac{B_s}{B_{s-1}} = \left(\frac{W_s}{W_{s-1}}\right)^{(1-[M/G'])} \tag{3.12}$$

Only when $M/G' < 1.0$ does cohort biomass increase between successive stages $s-1$ and s. It is observed that the index M/G' and mortality rates (M) generally decline during the early life of fishes. For most marine species and many freshwater species as well, M/G' is >1.0 during the smallest larval stages, signifying that cohort biomasses are declining. As ontogeny proceeds, M/G' eventually declines to <1.0 and a cohort's biomass will begin to increase (see Section 2.5.6).

Variability in the M/G' index and its rate of decline with ontogeny can be used to compare survival potential of larval cohorts among years or to compare different species during early life (Figure 3.11). The size at which $M/G' = 1.0$, and its variability among years for an individual species or among species in comparative analyses, describes the stage-specific survival pattern of a cohort with respect to ontogeny. Such patterns and variability can help to interpret how ontogenetic variability in stage-specific survival and growth relates to recruitment success or failure.

3.4.3 Projecting and predicting

The patterns, levels, and variability of cohort mortality in young fishes translate into successful or failed recruitment. Stage-specific mortality rates and cumulative mortality rates during the egg, larval, and early juvenile stages have been used to forecast cohort survival and recruitment potentials (see for example, Bailey *et al.* 1996, Houde 1997). It is important to remember that mortality rates during the earliest life stages are, at best, uncertain predictors of recruitment. Estimates of abundance and mortality rates in natural bodies of water often are inaccurate or have low precision. On the other hand, even relatively crude estimates of larval mortality may serve to predict potential recruitment, if there is considerable annual variability in the mortality rates and if mortality estimates are available for a series of years or for several cohorts

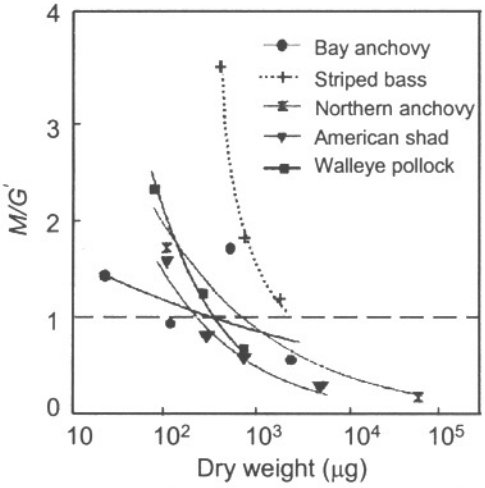

Figure 3.11 Relationships between M/G' and $\log_{10}$ body weights for five species of marine and estuarine fish larvae. Data points are averages for cohorts and year classes that had been studied (reproduced from Houde 1997 with permission of Academic Press).

during a single year. Higher than average mortality rates (or cumulative larval mortalities) generally can successfully predict low recruitment potential for a cohort, but lower than average larval mortality rates cannot reliably predict high recruitments because variable mortality in the relatively long juvenile stage can still generate high cumulative mortality (for example, see Table 3.3). Consequently, only in years when larval stage survival is low can one confidently use mortality in the larval stage to predict levels of recruitment.

Although it is risky to predict recruitment from extrapolated estimates of larval mortality rates and abundances, such estimates can provide important insights into the dynamic processes and their variability within the larval stage. Evaluation of mortality rates in relation to environmental variability and oceanographic factors in studies of larval stage dynamics (see Chapter 5) also may provide valuable insights into processes that control recruitment. An excellent example is provided by the FOCI Program, in which larval and early juvenile mortality rates of walleye pollock were estimated for many years in the Gulf of Alaska. During the 1980s, larval mortality rates (M) and cumulative larval stage mortalities ($M \cdot t$) were inversely correlated with levels of walleye pollock recruitment. In years after 1989, the relationship between larval mortality and recruitment failed when the ocean environment underwent a major change in response to climate variability. In the 1990s, the cumulative mortality ($M \cdot t$) of walleye pollock during the early juvenile stage increased progressively and juvenile stage mortality then became a better predictor of recruitment (Bailey 2000). More information on the FOCI Program is provided in Section 4.8.2.

3.5 Issues and implications

Are survivors of the egg and larval stage favored by some selected qualities that elevated their chances of survival or were they simply lucky? This question has intrigued fishery scientists for nearly three decades. For most typical marine teleosts, mortality exceeds 99%

during the egg and larval stages. For freshwater species, mortality is lower, but still usually exceeds 95% before the juvenile stage. In some research, it is clear that survivors had grown at exceptional rates or that they had experienced better than average nutritional condition (Campana 1996, Meekan & Fortier 1996). Such observations do not necessarily answer the question of whether survivors were predisposed to survive or just lucky. There is increasing evidence, however, that selection for survival favors larval genotypes or phenotypes that grow fast or behave appropriately to reduce their probability of being preyed upon. Alternatively and additionally, the selection process could operate on adults rather than early life stages, favoring survival of eggs and larvae of adults that spawned at favorable sites and times (the Match/Mismatch Hypothesis). These alternative possibilities remind us that there are important dependencies on physical properties of aquatic environments, close linkages among life stages, and feedback mechanisms that act together to regulate or control survival during early life.

Obviously, there are many sources of early-life mortality. Some may act essentially independently of each other, for example, episodic weather events, massive contaminant releases, or harmful algal blooms. But, it is likely that most agents of mortality interact in complex ways to affect mortality and growth rates through the relatively small (and difficult to measure) effects on dispersal patterns, predation mortality, or nutritional deficiencies. It is easy to imagine how greater or less than expected mortality might result from interactions of factors; for example, unfavorable dispersal could deliver larvae to poor feeding areas, making them more susceptible to predation. Under the best of conditions, it is a challenge for fishery scientists to estimate mortality rates with accuracy and precision. Detecting small, but decisive changes or variability in mortality rates, which is essential to understand cohort fates, is difficult under any circumstances and usually cannot now be accomplished with confidence in large or complex aquatic ecosystems. Partitioning the total mortality rate into its constituent components, for example, predation, starvation, and disease, represents yet another challenge that compounds the problem. Simulation modeling convincingly assures us that small changes or variability in mortality and growth rates during early life can control a cohort's fate, but actually estimating rates and slight changes in them is a formidable task.

It is not the magnitude of early life mortality rates so much as variability in the rates that is critical in generating recruitment successes or failures. Small variability in larval mortality rates, for example, 15% above or below average daily rates, when projected over a 100-day period can generate order-of-magnitude differences in recruitment levels. Only the most comprehensive and well designed surveys of eggs and larvae can hope to estimate abundances with sufficient precision to detect such differences in daily mortality. Given the large numbers of eggs spawned by teleost fishes and the high mortality rates that are common in early life, we should not be surprised that order-of-magnitude variability in recruitment occurs. Rather, the fact that even higher variability is seldom seen is perhaps more surprising.

Compensation, exercised through density-dependent mortality, must regulate mortality during early life, thus contributing to stabilization of stock abundance. Unfortunately, we rarely identify or quantify density-dependent mortality in studies on egg and larval stages. Survival of those stages seems to be controlled primarily by coarse, density-independent variability in mortality. Even a small amount of density-dependent mortality in early life stages could provide sufficient compensation to dampen variability and regulate recruitment levels. In the late larval and juvenile stages, density-dependent mortality is recognized more

frequently and is demonstrated to be an effective regulator of survival during the relatively long juvenile, pre-recruit stage (Van der Veer *et al.* 2000).

Will new technologies, analytical procedures, or models improve our ability to estimate age- and stage-specific abundances, and thus mortality rates of young fishes? Improvements in sampling technology certainly took place during the 1970s and 1980s. Depth-specific sampling with large, multiple-opening-closing nets equipped with environmental monitoring systems became common and helped greatly to estimate abundances, the key to estimating mortality and its relationship to oceanographic factors. Analytical procedures did not advance so rapidly in the same period, although more sophisticated statistical testing and estimation of variances became common, often confirming that our estimates were imprecise. The biggest advances in exploring how environmental factors relate to mortality were made in modeling the dynamics of early life. Coupled biophysical models became more sophisticated in recent years. Individual-based models, emphasizing biological responses and individual variability, proliferated and are being linked to spatially explicit, hydrodynamic models to characterize and identify mortality factors that determine the fate of cohorts. Consequently, we are able to evaluate and estimate mortality, and the processes that generate it, better than we were able two decades ago. This is particularly true in small, enclosed ecosystems (lakes and estuaries), but also in the coastal ocean where the oceanography is now reasonably well understood.

3.6 Conclusions

In the absence of fishing, most temporal variability in the abundances and recruitments of fish stocks results from variability in cohort survival during early life. Coarse controls on survivorship during egg and larval stages, and then fine-tuning in the juvenile, pre-recruit stage, are the processes that control and regulate recruitment. The interplay of episodic and subtle factors, the relative intensities of density-independent control and density-dependent regulation, the relative roles of predation and nutrition, and the presence of other potentially important causes of mortality all must be considered to fully appreciate how variability in mortality during early life shapes recruitment success. Advances in sampling technology, analytical capability, and modeling applications are improving our ability to relate early life processes to recruitment and to the overall population dynamics of fish stocks. It is often stated that the average fish larva is a dead larva, implying that we should emphasize analysis of unique characteristics of relatively rare survivors. To be sure, much can be learned from that approach, but identifying causes of mortality and estimating mortality rates in early life are the essential steps that we must employ to evaluate the processes and variability in them that determine levels of recruitment to adult stocks.

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