Environment and Longevity: The Demography of the Growth Rate

MARC MANGEL

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One of the cornerstones of demography is the mortality rate $m(x,t)$ of individuals of age x at calendar time t. However, from the perspective of evolutionary theory in general (Stearns and Hoekstra 2000) or biodemography more specifically (Gavrilov and Gavrilova 1991; Carey 2001; Carey and Judge 2001) the mortality rate is an evolved quantity, the result of natural selection acting on patterns of growth, behavior, and reproduction.

Our understanding of aging has been enormously advanced by genetic studies in the laboratory, to the point that it appears we will be able to understand all aspects of life span and aging with genetic tools. But aging takes place outside the laboratory and is also very much a phenotypic process. Recent studies, showing that the "public mechanisms" affecting longevity (Martin et al. 1996) are common to a wide variety of organisms (Guarente and Kenyon 2000; Strauss 2001; Zhang and Herman 2002), suggest that we need to understand the role of environment to master our understanding of aging and life span. Furthermore, it is likely that our understanding will be advanced the most when we are able to link proximate (mechanistic) and ultimate (fitness/selection–related) approaches to life-history questions, thereby "dissecting" the life history (Kirkwood 1992; Thorpe et al. 1998).

In this chapter, I develop a model that links environment, growth rate, and life span in the context of a Darwinian framework. In the model, the mortality rate emerges as the result of adaptive processes associated with growth and reproduction. These connections have important demographic implications. For example, it is likely that most organisms evolved in a world with fluctuating food supply, so that deeply embedded in the evolutionary history of organisms lies the ability to survive periods of food shortage. Such survival mechanisms could include adjustments to the growth rate or to reproductive output. This may be one of the causes of the association between caloric restriction and extension of life span, as suggested by Masoro and Austad (1996).

The approach that I use is based on life-history theory (Roff 1992; Stearns 1992). This field of evolutionary biology focuses on natural selection, constraint, adaptation, and Darwinian fitness. R. A. Fisher (1958 [1930]) described life-history theory as asking how environment and age affect the allocation of resources to reproduction. McNamara (1993) described life-history theory through a set of interconnected questions: What is the intrinsic rate of increase of a population that follows a given strategy and how does one find it? What is the current reproductive value of an organism and how does one calculate it? How does one decompose reproductive value into current and future reproductive success? How much effort should an organism put into current reproduction at the expense of future reproduction? How is individual behavior linked to population growth rates? What should an organism maximize over its lifetime?

The work outlined here is motivated in part by the remarkable longevity of the Pacific rockfish, which I describe next.

Longevity and growth in Pacific rockfish

The rockfish (*Sebastes* spp.) are remarkable because of their longevity (Table 1; also see Reznick et al. 2002 and the web site maintained by the Pacific Fisheries Management Council «http://www.psmfc.org/habitat/rockfish_lifespan.html»). Many rockfish species live in the California Current of the Pacific Ocean, which flows eastward toward North America and then southward along the coast. This environment is characterized by seasonal vari-

**TABLE 1   Maximum estimated ages of some commercially important rockfish *Sebastes***

Species	Common name	Maximum age (years)
S. aleutianus	Rougheye rockfish	140
S. alutus	Pacific ocean perch	90
S. borealis	Shortraker rockfish	120
S. brevispinis	Silvergray rockfish	80
S. crameri	Darkblotched rockfish	47
S. entomelas	Widow rockfish	58
S. flavidus	Yellowtail rockfish	64
S. paucispinis	Bocaccio	36
S. pinniger	Canary rockfish	75
S. proriger	Redstripe rockfish	41
S. reedi	Yellowmouth rockfish	71
S. variegatus	Harlequin rockfish	43
S. zacentrus	Sharpchin rockfish	45

SOURCE: Leaman and Beamish (1984).

ability (Chelton et al. 1982; Lynn and Simpson 1987) and long-term fluctuations (MacCall 1996; Watanabe and Nitta 1999) in which different environmental regimes persist at times on the order of decades before an abrupt transition occurs in environmental characteristics (particularly a switch between warm and cold water temperature and differential levels of food). Seasonally unpredictable environments are associated with high variation in spawning success, with such success being often associated with long life span. Murphy (1968) and Mann and Mills (1979) demonstrated a strong correlation between variation in spawning success (maximum/minimum observed reproductive success) and reproductive life span; see Figure 1. Long reproductive life span must perforce be correlated with long life span, and we are thus led to wonder how long life span is achieved.

In general, rockfish grow slowly. The growth and maturity of fish are traditionally measured using the von Bertalanffy equation describing size at age (Haldorson and Love 1991). This description involves asymptotic size ($L_\bullet$), growth rate (k), and a parameter that describes initial size (t_0).[1] The growth rates for rockfish are low ($k = 0.08$ to 0.19 per year) compared to shorter-lived fish of similar size (for example, $k = 0.3$ to 0.4 for cods; Leaman and Beamish 1984). The description of the life history is completed by inclusion of mortality rate M.

FIGURE 1   Relationship between variation in spawning success (fecundity)(often environmentally induced) and reproductive life span for a variety of species of fish
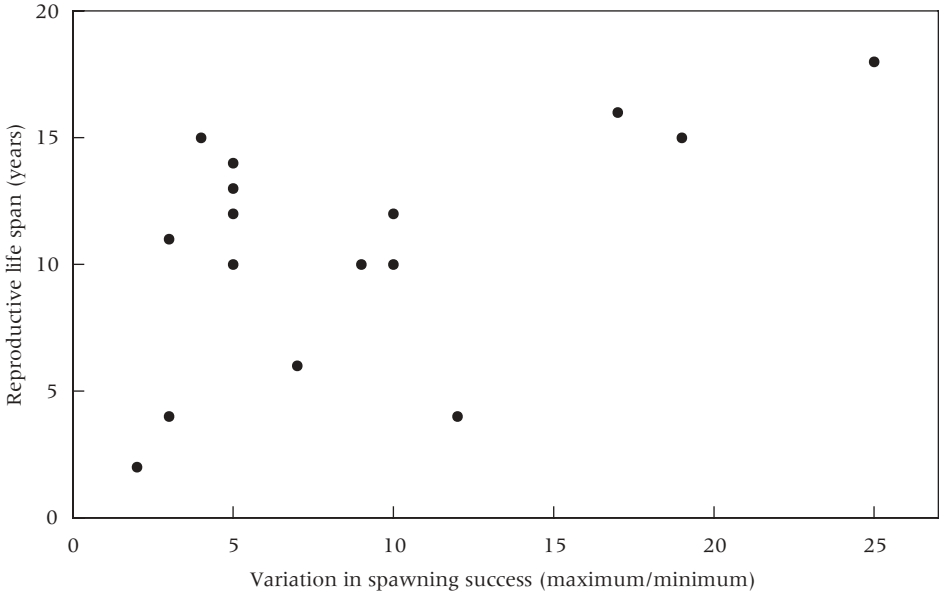
SOURCE: Redrawn from Mann and Mills (1979).

The fishery scientist R. J. H. Beverton explored the relationship between asymptotic size, growth constant, mortality rate, and life span (Beverton and Holt 1959; Beverton 1963, 1987, 1992) and created a theory relating growth, maturity, and longevity (or GML). Beverton's theory is reviewed by Mangel and Abrahams (2001); the work reported here is an extension of the line of research started by Beverton.

The importance of facultative growth (metabolic choice) for demography

Work with the nematode worm *C. elegans* (Wolkow et al. 2000; Cowen 2001; van Voorhies 2001) suggests that metabolism and longevity are separately regulated, but clearly connected. In common with terrestrial organisms, fish use oxygen to fuel growth and reproduction. In the California Current, oxygen concentration varies by a factor of nearly 10 (Lynn and Simpson 1987; Boehlert and Kappenman 1980); faster growth occurs in the northern parts of the range of the fish, where water temperatures are lower and oxygen content is higher. Growth rate and reproductive success generally decline as dissolved oxygen declines (Stewart 1967). Indeed, some authors (Pauly 1981; Bakun 1996) have proposed that oxygen may be an alternative currency to food in the ecological budget of fish and that understanding the role of oxygen is crucial to understanding all aspects of fish life history (Pauly 1998).

It has long been understood from both empirical (Pettersson and Bronmark 1993) and theoretical (Clark and Mangel 2000; Mangel and Stamps 2001) perspectives that organisms commonly make tradeoffs between mortality risk and foraging success. Ricklefs et al. (1994) hypothesized that rapid growth may be inversely correlated with functional maturity and muscle development, and Ricklefs and Scheuerlein (2001) have shown that in zoo animals the best explanatory variable for the initial mortality rate and the rate of aging in a Weibull model is postnatal growth. Here, I explore the tradeoff between rapid growth, internal damage resulting from oxidative processes, fitness consequences (von Schantz et al. 1999), and the demography of life span (see Figure 2). I assume that faster growth increases the prevalence of reactive oxygen species (ROS). There is evidence that ROS can damage proteins, DNA, and lipids (Tolmassof 1980; Stadtman 1992; Ames 1993; Barja et al. 1994; Pollack and Leeuwenburgh 1999; Ozawa 1995; Shigenaga 1994; Lane et al. 1996; Brewer 2000; van Voorhies 2001; Van Remmen and Richardson 2001). There is also evidence of direct associations between oxidative damage and life span (Sohal et al. 1993; Agarwal and Sohal 1994; Harshman and Haberer 2000) and between longevity and low levels of free radical production *in vivo* (Barja et al. 1994).

Furthermore, reproduction is associated with increased use of oxygen. In some cases, gestating fish used about 35 percent more oxygen per mass

FIGURE 2   Maximum longevity in 46 species of fishes in the family *Scorpaenidae* by maximum depth
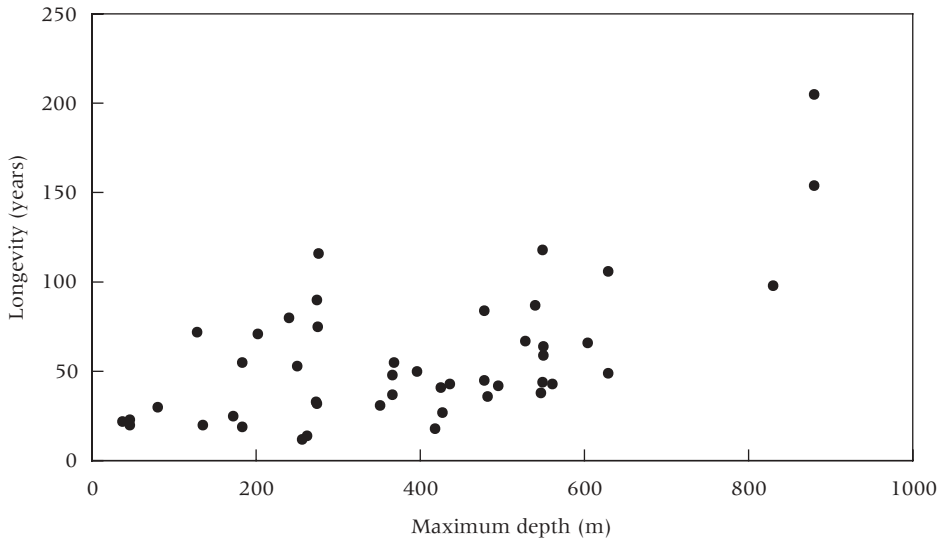
SOURCE: Redrawn from Cailliet et al. (2001).

than nongestating fish (Boehlert et al. 1991). Hopkins et al. (1995) found that metabolic rates of incubating yellowtail rockfish (*Sebastes flavidus*) were 82 percent higher than those of spent females and 101 percent higher than those of nonreproductive males. Whether these differences represent oxygen consumption by embryos or females' providing resources to embryos, they surely represent a potential source of oxidative stress.

Oxidative stress is intimately linked to reproductive fitness (von Schantz et al. 1999); indeed, Sibly and Calow (1986) concluded that evolution proceeded toward maximization of ATP production, but not necessarily minimization of heat per ATP produced. There is evidence for the anticipation of oxidative damage (Novoseltsev et al. 2000), individual regulation of oxygen consumption (Quetin and Ross 1989), and within-individual variation in oxidative stress according to calendar time, age, and physiological or reproductive state (Rikans and Hornbrook 1997; Zielinski and Pörtner 2000; Blount et al. 2001; Filho et al. 2001; Royle et al. 2001; Salmon et al. 2001). I define responses that vary within individuals according to state or time as facultative (Mangel and Clark 1988; Clark and Mangel 2000). Because the facultative response refers to growth rate, which is intimately tied to metabolism, I will also refer to it as metabolic choice.

In order to understand how facultative growth and life span are related, I return to the von Bertalanffy description of growth and dissect it into components. One assumes that:

—Weight ($W(t)$) and length ($L(t)$) are allometrically related by $W(t) = cL(t)^3$.

—The increment in weight is determined by the difference between anabolic and catabolic terms, in which the anabolic term is proportional to surface area (hence $L(t)^2$), food availability, and oxygen; and the catabolic term is proportional to mass (hence $L(t)^3$) and oxygen that the fish uses.

—Asymptotic size is determined by the weight at which anabolic and catabolic terms balance. Larger values of the metabolic choice parameter (a) lead to faster growth, larger size at age, and smaller age at maturity.[2] If predation is size dependent (a common feature in the marine environment; McGurk 1986, 1996), then faster growth will lead to higher survival rates from the viewpoint of predation.

To capture the effects of ROS, I introduce another state variable that measures damage accumulated at age[3]—measured, for example, in terms of lipid peroxidation (Matsuo and Kaneko 1999), oxidized amino acids, or DNA (Leeuwenburgh et al. 1999). Since oxidative damage accumulates as a function of metabolism, the dynamics of damage are related to metabolism. The reason for introducing oxidative damage caused by ROS is that survival from one age to the next depends upon both size (bigger size and thus faster growth are better) and accumulated damage (slower growth is better). Thus, there is an interplay between the short-term and long-term survival processes.[4]

This formulation is sufficient to compute the survival to a specific age for an individual using a fixed value of the metabolic choice parameter. In general, predation-related survival is an increasing function of the metabolic choice parameter, and damage-related survival is a decreasing function of the parameter. Thus, a fixed high metabolic rate allows the fish to grow rapidly, escaping size-dependent predation and reaching age at maturity sooner. But a fixed high metabolic rate also leads to increased levels of oxidative damage, thus decreasing long-term survival. Survival, and thus life span, depends upon the balance of these factors. In Figure 3, I show life span (defined as the age at which survival from birth falls to less than one in 10 million) when the metabolic parameter is fixed throughout an individual's life. We see, for example, that in the base case maximum life span is about 48 years, achieved by using a fixed metabolic parameter of 0.6; when the repair rate is doubled, the corresponding values are 79 years and $a = 1.2$. Thus, although doubling the repair rate allows individuals to grow at a faster rate, with concomitant benefits, it does not double life span.

On the other hand, whereas fast growth may be advantageous at small size (to help the organism escape size-dependent predation), there is little advantage to fast growth at larger sizes and a definite cost. We are thus led to ask what pattern of growth (and thus ROS production) maximizes the

FIGURE 3   Life span (age at which survival probability drops to less than 1 in 10 million) among fish as a function of a fixed metabolic parameter _a_. When there is a balance between predation-related survival and damage-related survival, life span peaks for an intermediate value of the metabolic parameter.
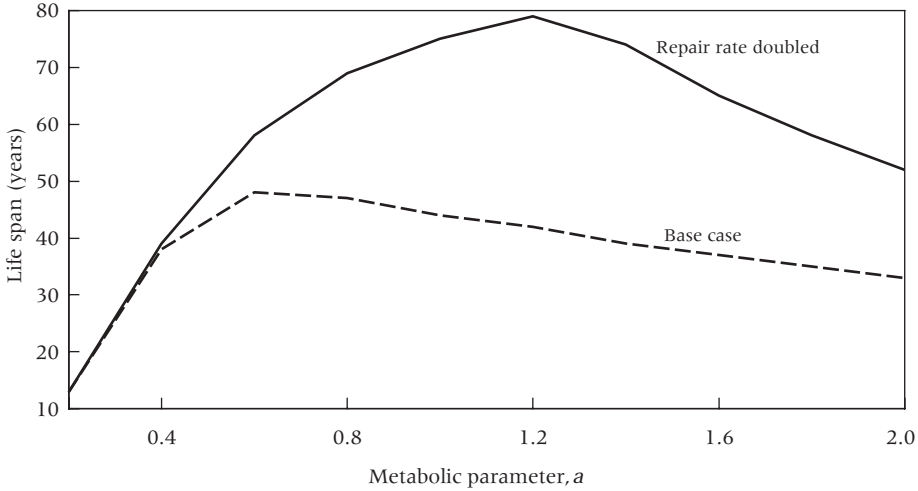

long-term number of descendants (equivalent to _r_ of the Euler–Lotka equation) and what is the effect on life span?

To characterize reproduction, I assume that a fish is capable of reproduction once its size exceeds 60 percent of asymptotic size (Beverton 1992) and that the amount of reproduction is determined allometrically by length. Reznick et al. (2002) argued that this increasing fecundity with age separates fish from birds or mammals and has allowed the evolution of such remarkably long lives. The optimal pattern of growth can be determined by the method of stochastic dynamic programming (Mangel and Clark 1988; Clark and Mangel 2000; McNamara 2000). The output of such an analysis is a "decision matrix" that gives the optimal value of the parameter of metabolic choice for each size and damage level. Age dependence is an implicit rather than explicit relationship. Thus, aging, senescence, and longevity are emergent properties in this approach (Waldorp 1992; Jazwinski et al. 1998; Goodwin 2001; Mangel 2001), rather than constitutive properties of the model.

The theory leads to the prediction of age-dependent growth and damage, through the parameter of metabolic choice (Figure 4a), and thus the dynamics of length (Figure 4b), survival (Figure 4c), and mortality (Figure 4d). The theory thus provides a method for computing life span and for deriving the mortality rate as a result of the adaptive processes of growth and reproduction. The life span, again defined as the age at which survival

FIGURE 4   A theory that allows facultative growth (metabolic choice) allows prediction of the optimal age-dependent value of the metabolic parameter (panel a) and the length (panel b), survival (panel c), and mortality (panel d) dynamics. This theory thus allows the prediction of the mortality rate as a result of adaptive processes of growth and reproduction.
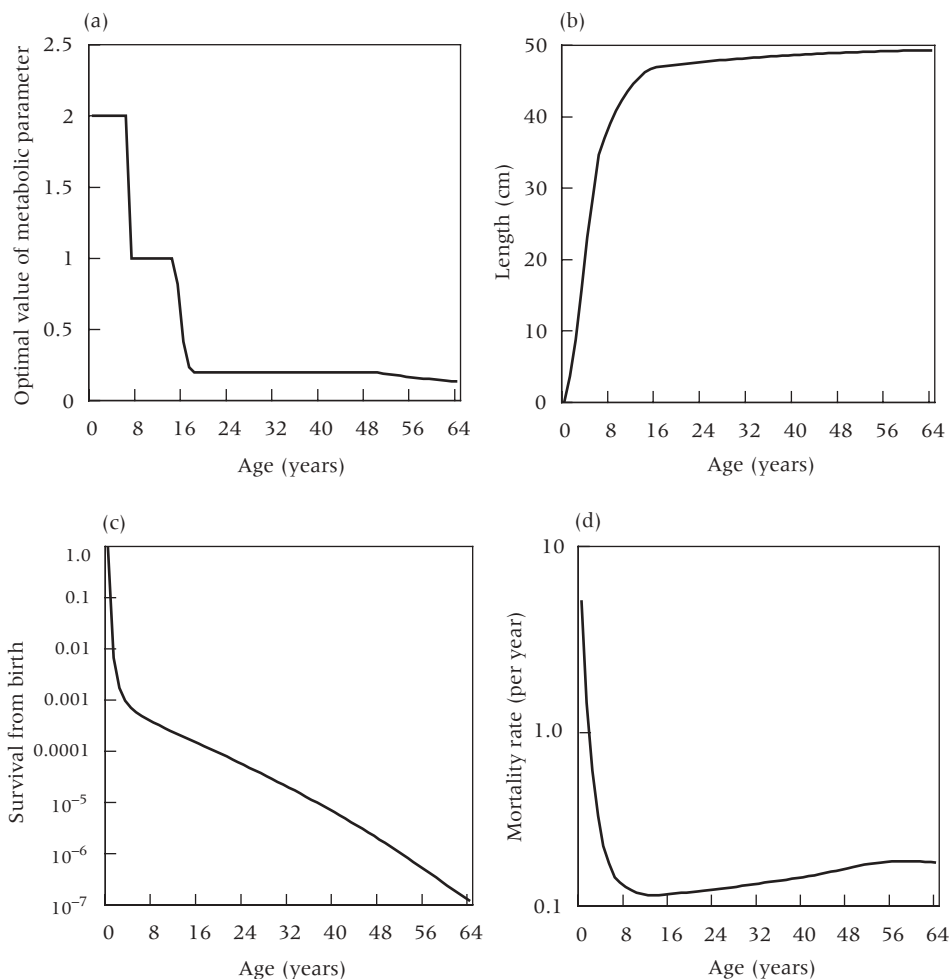
probability from birth drops below one in 10 million, is 65 years; with a doubled repair rate the life span is 88 years. The precise factors affecting life span include size-dependent mortality, damage, and food availability. Because mortality is size dependent, small fish experience high predation rates, but these decline as the fish grow older. In all of these cases, facultative growth leads to longer life span than occurs with a fixed growth parameter. It is important to recognize, however, that life span, like the mortality rate

itself, is the outcome of natural selection maximizing expected reproductive success, rather than a direct target of natural selection per se.

Discussion

The models introduced here are conceptual ones, intended to facilitate exploration of the relationship between environment and longevity and to illustrate how the perspective of evolutionary ecology allows us to predict the mortality rate as a function of growth and reproduction. To convert these models to applied, calculational tools (*sensu* Mangel et al. 2001) requires extensions that include temperature dependence and density dependence of growth (Ylikarjula, Heino, and Dieckmann 1999; Lorenzen 2000; Mangel and Abrahams 2001).

Even so, a strong qualitative prediction of this theory is an ontogenetic shift with age toward slower growth (and thus to generally deeper and oxygen-poorer water). That is, a behavioral trait is predicted on the basis of physiological processes in a demographic context. Various species of fish fit this pattern. Chilipepper rockfish (*Sebastes goodei*) and splitnose rockfish (*Sebastes diplorproa*) show an ontogenetic shift toward deeper water and lower oxygen concentrations as they get bigger (Vetter and Lynn 1997). Shortspine thornyhead (*Sebastolobus alascanus*), longspine thornyhead (*Sebastolobus altivelis*), and dover sole (*Microstomus pacificus*) have the same pattern but move even deeper and into the oxygen-minimum zone (Vetter et al. 1994; Vetter and Lynn 1997). Similarly, juvenile sablefish (*Anoplopoma fimbria*) (Pallas) are found at the surface in oxygen- (and predator-) filled regions of the ocean, but adult sablefish are found in the subsurface zone of low oxygen concentration (Bakun 1996; Sogard and Olla 1998). The study of these patterns, known as bathymetric demography (Jacobson and Hunter 1993; Jacobson and Vetter 1996), has important implications for fishery management. In this regard fish, because they live in a much-enlarged three-dimensional world, allow exploration of demographic ideas that terrestrial organisms do not.

It is also clear that not all long-lived marine species live in deep water (sturgeon and yelloweye rockfish, for example, do not) and that several short-lived species are found in deep water (the lantern fish, for example). According to Donald Gunderson (personal communication), data on thornyheads suggest that oxygen, temperature, and depth do little to explain differences in mortality across species or regions. These differences may be a result of the balance of production of ROS and repair. However, the arguments presented here could be adapted to other mechanisms that connect rate of growth and damage.

The models show the importance of considering the organism in its environment, the role of natural selection in shaping life span in the con-

text of environment, and the need for broad integration of different levels of biological organization for a full understanding of longevity.

Notes

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1 If $L(t)$ denotes the length of a fish at age t, the von Bertalanffy description of growth is $L(t) = L_\cdot(1-\exp(-k(t-t_0)))$. This equation is the solution of the differential equation

$$\frac{dL}{dt} = k(L_\cdot - L(t))$$

and satisfies the difference equation

$$L(t+1) = L(t)\exp(-k) + L_\cdot(1 - \exp(-k))$$

as can be verified directly from substitution into the differential equation.

2 In a discrete time formulation the weight dynamics are

$$W(t+1) - W(t) = aY[O_2]L(t)^2 - ma[O_2]L(t)^3$$

where a is the parameter of metabolic choice and can be interpreted as the fraction of ambient oxygen flowing across the gills (Waller et al. 2000) or a correlate of depth; Y is food

availability; $[O_2]$ is the concentration of oxygen in the water; and m determines catabolic costs. Length dynamics in discrete time follow directly from this since it is assumed that weight is proportional to length cubed.

3 The discrete time dynamics for damage in the nonreproductive period are

$$D(t+1) = D(t) + kma[O_2]L(t)^3 - r$$

where k is a parameter converting metabolism to damage and r is the damage repair rate. When the fish is reproductively mature, additional damage accumulates, associated with the numbers and quality of offspring (more or higher-quality offspring imply higher rates of accumulation of damage).

4 I assume here that the two processes are independent, so that given $L(t) = l$ and $D(t) = d$

$$\text{Prob}\{\text{survive to age } t+1\} = \exp\{-m/l\}\exp\{-(d/d_c)^2\}$$

where m and d_c are parameters characterizing size- and damage-dependent mortality respectively. Given the dynamics of size and damage, this probability can be iterated from $t = 0$, when survival probability is unity, into the future. The mortality rate is then computed from the survival probability in the standard way.

References

Agarwal, S. and R. S. Sohal. 1994. "DNA oxidative damage and life expectancy in house-flies," *Proceedings of the National Academy of Sciences USA* 91: 12332–12335.

Ames, B. N., M. K. Shigenaga, and T. M. Hagen. 1993. "Oxidants, antioxidants and the degenerative disease of aging," *Proceedings of the National Academy of Sciences USA* 90(17): 7915–7922.

Bakun, A. 1996. *Pattern in the Ocean: Oceanic Process and Marine Population Dynamics.* La Jolla, CA: California Sea Grant.

Barja, G., S. Cadenas, C. Rojas, M. Lopez-Torres, and R. Perez-Campo. 1994. "A decrease of free radical production near critical targets as a cause of maximum longevity in mammals," *Comparative Biochemistry and Physiology* 108B: 501–512.

Beverton, R. J. H. 1963. "Maturation, growth, and mortality of clupeid and engraulid stocks in relation to fishing," *Rapport Conseil International Exploration de la Mer* 154: 44–67.

———. 1987. "Longevity in fish: Some ecological and evolutionary considerations," in A. D. Woodhead and K. H. Thompson (eds.), *Evolution of Longevity in Animals*. New York: Plenum, pp. 161–185.

———. 1992. "Patterns of reproductive strategy parameters in some marine teleost fishes," *Journal of Fish Biology* 41 (Supplement B): 137–160.

Beverton, R. J. H. and S. J. Holt. 1959. "A review of the life-spans and mortality rates of fish in nature, and their relationship to growth and other physiological characteristics," *CIBA Foundation Colloquium on Ageing* 54: 142–180.

Boehlert, G. W. and R. F. Kappenman. 1980. "Variation of growth with latitude in two species of rockfish (*Sebastes pinniger* and *S. diploproa*) from the Northeast Pacific Ocean," *Marine Ecology-Progress Series* 3: 1–10.

Blount, J. D. et al. 2001. "Antioxidants, showy males and sperm quality." *Ecology Letters* 4: 393–396.

Boehlert, G. W., M. Kusakari, and J. Yamada. 1991. "Oxygen consumption of gestating female *Sebastes schlegeli*: Estimating the reproductive costs of livebearing," *Environmental Biology of Fishes* 30: 81–89.

Brewer, G. J. 2000. "Neuronal plasticity and stressor toxicity during aging," *Experimental Gerontology* 35: 1165–1183.

Cailliet, G. M. et al. 2001. "Age determination and validation studies of marine fishes: Do deep-dwellers live longer?" *Experimental Gerontology* 36: 739–764.

Carey, J. R. 2001. "Insect biodemography," *Annual Review of Entomology* 46: 79–110.

Carey, J. R. and D. S. Judge. 2001. "Principles of biodemography with special reference to human longevity," *Population: An English Selection* 13: 9–40.

Chelton, D. B., P. A. Bernal, and J. A. McGowan. 1982. "Large-scale inter annual physical and biological interaction in the California Current," *Journal of Marine Research* 40: 1095–1125.

Clark, C. W. and M. Mangel. 2000. *Dynamic State Variable Models in Ecology. Methods and Applications*. New York: Oxford University Press.

Cowen, T. 2001. "A heady message for life span regulation," *Trends in Genetics* 17: 109–113.

Filho, D. W. et al. 2001. "Seasonal changes in antioxidant defenses of the digestive gland of the brown mussel (*Perna perna*)," *Aquaculture* 203: 149–158.

Fisher, R. A. 1958 [1930]. *The Genetical Theory of Natural Selection* 2nd edition [1st edition]. New York: Dover Press.

Gavrilov, L. A. and N. S. Gavrilova. 1991. *The Biology of Life Span: A Quantitative Approach*. Chur, Switzerland: Harwood Academic Publishers.

Goodwin, B. 2001. *How the Leopard Changed Its Spots. The Evolution of Complexity*. Princeton, NJ: Princeton University Press.

Guarente, L. and C. Kenyon. 2000. "Genetic pathways that regulate ageing in model organisms," *Nature* 408: 255–262.

Haldorson, L. and M. Love. 1991. "Maturity and fecundity in the rockfishes, *Sebastes* spp., a review," *Marine Fisheries Review* 53(2): 25–31.

Harshman, L. G. and B. A. Haberer. 2000. "Oxidative stress resistance: A robust correlated response to selection in extended longevity lines of *Drosophila melanogaster*?" *Journal of Gerontology: Biological Sciences* 55A: B415–B417.

Hopkins, T. E., M. B. Eldridge, and J. J. Cech. 1995. "Metabolic costs of viviparity in yellowtail rockfish, *Sebastes flavidus*," *Environmental Biology of Fishes* 43: 77–84.

Jacobson, L. D. and J. R. Hunter. 1993. "Bathymetric demography and management of Dover sole," *North American Journal of Fisheries Management* 13: 405–420.

Jacobson, L. D. and R. D. Vetter. 1996. "Bathymetric demography and niche separation of thornyhead rockfish: *Sebastolobus alascanu* and *S. altivelis*," *Canadian Journal of Fisheries and Aquatic Sciences* 53: 600–609.

Jazwinski, S. M., S. Kim, C-Y. Lai, and A. Bengura. 1998. "Epigentic stratification: The role

of individual change in the biological aging process," *Experimental Gerontology* 33: 571–580.

Jensen, A. 1996. "Beverton and Holt life history invariants result from optimal trade-off of reproduction and survival," *Canadian Journal of Fisheries and Aquatic Sciences* 53: 820–822.

Kirkwood, T. B. L. 1992. "Comparative life spans of species: Why do species have the life spans they do?" *American Journal of Clinical Nutrition* 55: 1191S–1195S.

———. 1977. "Evolution of ageing," *Nature* 270: 301–304.

Kirkwood, T. B. L. and M. R. Rose. 1991. "Evolution of senescence: Late survival sacrificed for reproduction," *Philosophical Transactions of the Royal Society of London B*: 15–24.

Lane, M. A. et al. 1996. "Calorie restriction lowers body temperature in Rhesus monkeys, consistent with a postulated anti-aging mechanism in rodents," *Proceedings of the National Academy of Sciences USA* 93(9): 4159–4164.

Leaman, B. M. 1991. "Reproductive styles and life history variables relative to exploitation and management of *Sebastes* stocks," *Environmental Biology of Fishes* 30: 253–271.

Leaman, B. M. and R. J. Beamish. 1984. "Ecological and management implications of longevity in some northeast Pacific groundfishes," *International North Pacific Fisheries Commission Bulletin* 42: 85–97.

Leeuwenburgh, C., P. A. Hansen, J. O. Holloszy, and J. W. Heinecke. 1999. "Oxidized amino acids in the urine of aging rats: Potential markers for assessing oxidative stress in vivo," *American Journal of Physiology (Regulatory Integrative Comparative Physiology)* 276: R128–R135.

Lorenzen, K. 2000. "Population dynamics and management," in M. C. M. Beveridge and B. J. McAndrew (eds.), *Tilapias: Biology and Exploitation.* Dordrecht: Kluwer Academic Publishers, pp. 163–226.

Lynn, R. J. and J. J. Simpson. 1987. "The California Current system: The seasonal variability of its physical characteristics," *Journal of Geophysical Research* 92: 12947–12966.

MacCall, A. 1996. "Patterns of low-frequency variability in fish populations of the California current," *California Cooperative Fishery Investigations (CalCOFI) Reports* 37: 100–110.

Mangel, M. 2001. "Complex adaptive systems, aging, and longevity," *Journal of Theoretical Biology* 213: 559–571.

Mangel, M. and M. V. Abrahams. 2001. "Age and longevity in fish, with consideration of the ferox trout," *Experimental Gerontology* 36: 765–790.

Mangel, M. and C. W. Clark. 1988. *Dynamic Modeling in Behavioral Ecology.* Princeton, NJ: Princeton University Press.

Mangel, M., O. Fiksen, and J. Giske. 2001. "Theoretical and statistical models in natural resource management and research," in T. M. Shenk and A. B. Franklin (eds.), *Modeling in Natural Resource Management. Development, Interpretation, and Application.* Washington, DC: Island Press, pp. 57–72.

Mangel, M. and J. Stamps. 2001. "Trade-offs between growth and mortality and the maintenance of individual variation in growth," *Evolutionary Ecology Research* 3: 583–593.

Mann, R. H. K. and C. A. Mills. 1979. "Demographic aspects of fish fecundity," *Symposia of the Zoological Society of London* 44: 161–177.

Martin, G. M., S. N. Austad, and T. E. Johnson. 1996. "Genetic analysis of ageing: Role of oxidative damage and environmental stresses," *Nature Genetics* 13: 25–34.

Masoro, E. J. and S. N. Austad. 1996. "The evolution of the antiaging action of dietary restriction: A hypothesis," *Journal of Gerontology: Biological Sciences* 51A: B387–B391.

Matsuo, M. and T. Kaneko. 1999. "Lipid peroxidation," in B. P. Yu (ed.), *Methods in Aging Research.* Boca Raton, FL: CRC Press, pp. 571–606.

McGurk, M. D. 1986. "Some remarks on 'Model of monthly marine growth and natural mortality for Babine Lake sockeye salmon (*Oncorhynchus nerka*)' by Furnell and Brett," *Canadian Journal of Fisheries and Aquatic Sciences* 43: 2535–2536.

———. 1996. "Allometry of marine mortality of Pacific salmon," *Fishery Bulletin* 94: 77–88.

McNamara, J. M. 1993. "State-dependent life history equations," *Acta Biotheoretica* 41: 165–174.

———. 2000. "A classification of dynamic optimization problems in fluctuating environments," *Evolutionary Ecology Research* 2: 457–471.

Murphy, G. I. 1968. "Pattern in life history and environment," *American Naturalist* 102: 391–403.

Novoseltsev, V. N., J. R. Carey, P. Liedo, J. Novoseltseva, and A. I. Yashin. 2000. "Anticipation of oxidative damages decelerates aging in virgin female medflies: Hypothesis tested by statistical modeling," *Experimental Gerontology* 35: 971–987.

Ozawa, T. 1995. "Mitochondrial DNA mutations associated with aging and degenerative diseases," *Experimental Gerontology* 30: 269–290.

Pauly, D. 1980. "On the interrelationships between natural mortality, growth parameters, and mean environmental temperature in 175 fish stocks," *Journal of the International Council for the Exploration of the Seas* 39(2): 175–192.

———. 1981. "The relationships between gill surface area and growth performance in fish: A generalization of von Bertalanffy's theory of growth," *Meeresforschung* 28: 251–282.

———. 1998. "Tropical fishes: Patterns and propensities, " *Journal of Fish Biology Supplement A* 53: 1–17.

Pettersson, L. B. and C. Bronmark. 1993. "Trading off safety against food: State dependent habitat choice and foraging in crucian carp," *Oecologia* 95: 353–357.

Pollack, M. and C. Leeuwenburgh. 1999. "Molecular mechanisms of oxidative stress in aging: Free radicals, aging, antioxidants and disease," in C. K. Sen, L. Packer, and O. Hanninen (eds.), *Handbook of Oxidants and Antioxidants in Exercise.* Amsterdam: Elsevier Science B.V., pp. 881–923.

Preston, S. H., P. Heuveline, and M. Guillot. 2001. *Demography: Measuring and Modeling Population Processes.* Oxford: Blackwell.

Quetin, L. B. and R. M. Ross. 1989. "Effects of oxygen, temperature and age on the metabolic rate of the embryos and early larval stages of the Antarctic krill *Euphausia superba* Dana," *Journal of Experimental Marine Biology and Ecology* 125: 43–62.

Reznick, D., C. Ghalambor, et al. (2002). "The evolution of senescence in fish," *Mechanisms of Ageing and Development* 123: 773–789.

Ricklefs, R. E., R. E. Shea, and I-H. Choi. 1994. "Inverse relationship between functional maturity and exponential growth rate of avian skeletal muscle: A constraint on evolutionary response," *Evolution* 48(4): 1080–1088.

Ricklefs, R. E. and A. Scheuerlein. 2001. "Comparison of aging-related mortality among birds and mammals," *Experimental Gerontology* 36: 845–857.

Rikans, L. E. and K. R. Hornbrook. 1997. "Lipid peroxidation, antioxidant protection and aging," *Biochimicia Biophysica Acta* 1362: 116–127.

Roff, D. A. 1992. *The Evolution of Life Histories.* New York: Chapman and Hall.

Royle, N. J., P. F. Surai, R. J. McCartney, and B. K. Speake. 1999. "Parental investment and yolk lipid composition in gulls," *Functional Ecology* 13: 298–306.

Royle, N. J., P. F. Surai, and I. R. Hartley. 2001. "Maternally derived androgens and antioxidants in bird eggs: Complementary but opposing effects?" *Behavioral Ecology* 12: 381–385.

Salmon, A. B., D. B. Marx, and L. G. Harshman. 2001. "A cost of reproduction in *Drosophila melanogaster*: Stress susceptibility," *Evolution* 55(8): 1600–1608.

Shigenaga, M. K., T. M. Hagen, and B. N. Ames. 1994. "Oxidative damage and mitochondrial decay in aging," *Proceedings of the National Academy of Sciences USA* 91(23): 10771–10778.

Sibly, R. M. and P. Calow. 1986. *Physiological Ecology of Animals.* Oxford: Blackwell Scientific Publications.

Sogard, S. M. and B. L. Olla. 1998. "Behavior of juvenile sablefish, *Anoplopoma fimbria* (Pallas), in a thermal gradient: Balancing food and temperature requirements," *Journal of Experimental Marine Biology and Ecology* 222: 43–58.

Sohal, R. S., S. Agarwal, A. Dubey, and W. C. Orr. 1993. "Protein oxidative damage is associated with life expectancy of houseflies," *Proceedings of the National Academy of Sciences USA* 90: 7255–7529.

Stadtman, E. R. 1992. "Protein oxidation and aging," *Science* 257: 1220–1224.

Stearns, S. C. 1992. *The Evolution of Life Histories.* New York: Oxford University Press.

Stearns, S. C. and R. F. Hoekstra. 2000. *Evolution.* New York: Oxford University Press.

Stewart, N. E., D. L. Shumway, and P. Doudoroff. 1967. "Influence of oxygen concentration on the growth of juvenile largemouth bass," *Journal of the Fisheries Research Board of Canada* 24: 475–494.

Strauss, E. 2001. "Growing old together," *Science* 292: 41–43.

Thorpe, J. E., M. Mangel, N. B. Metcalfe, and F. A. Huntingford. 1998. "Modelling the proximate basis of salmonid life-history variation, with application to Atlantic salmon, *Salmo salar* L.," *Evolutionary Ecology* 12: 581–600.

Tolmassof, J. M., T. Ono, and R. G. Cutler. 1980. "Superoxide dismutase: Correlation with life-span and specific metabolic rate in primate species," *Proceedings of the National Academy USA* 77(5): 2777–2781.

Van Remmen, H. and A. Richardson. 2001. "Oxidative damage to mitochondria and aging," *Experimental Gerontology* 36: 957–968.

van Voorhies, W. A. 2001. "Metabolism and life span," *Experimental Gerontology* 36: 55–64.

Vetter, R. D. and E. A. Lynn. 1997. "Bathymetric demography, enzyme activity patterns, and bioenergetics of deep-living scorpaenid fishes (genera *Sebastes* and *Sebastolobus*): Paradigms revisited," *Marine Ecology Progress Series* 155: 173–188.

Vetter, R. D., E. A. Lynn, and A. S. Costa. 1994. "Depth zonation and metabolic adaptation in Dover sole, *Microstomus pacificus,* and other deep-living flatfishes: Factors that affect the sole," *Marine Biology* 120: 145–159.

von Schantz, T., S. Bensch, M. Grahn, D. Hasselquist, and H. Wittzel. 1999. "Good genes, oxidative stress and condition-dependent signals," *Proceedings of the Royal Society of London Series B* 266: 1–2.

Waldorp, M. M. 1992. *Complexity.* New York: Touchstone Books.

Waller, U., E. Black, D. Burt, C. Groot, and H. Rosenthal. 2000. "The reaction of young coho *Oncorhynchus kisutch* to declining oxygen levels during long-term exposure," *Journal of Applied Ichthyology* 16: 14–19.

Watanabe, M. and T. Nitta. 1999. "Decadal changes in the atmospheric circulation and associated surface climate variations in the northern hemisphere winter," *Journal of Climate* 12: 494–510.

Wolkow, C. A., K. D. Kimura, M-S. Lee, and G. Kuvkun. 2000. "Regulation of *C. elegans* life-span by insulinlike signaling in the nervous system," *Science* 290: 147–150.

Ylikarjula, J., M. Heino, and U. Dieckmann. 1999. "Ecology and adaptation of stunted growth in fish," *Evolutionary Ecology* 13: 433–453.

Zhang, Y. and B. Herman. 2002. "Ageing and apoptosis," *Mechanisms of Ageing and Development* 123: 245–260.

Zielinski, S. and H.-O. Pörtner. 2000. "Oxidative stress and antioxidative defense in cephalopods: A function of metabolic rate or age?" *Comparative Biochemistry and Physiology Part B* 125: 147–160.