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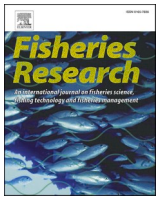

Size- and age-dependent natural mortality in fish populations: Biology, models, implications, and a generalized length-inverse mortality paradigm

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ABSTRACT

Natural mortality rates (M) in fish populations vary with body size and age, often by orders of magnitude over the life cycle. Traditionally, fisheries models and stock assessment methods have treated M as constant in the recruited stock, but that axiom has been challenged on empirical and theoretical grounds, and by practical assessment needs. Reviewing biological considerations, empirical generalizations, and theoretical models of size- and age-dependent natural mortality in fish populations, I show how multiple strands of evidence lead to a coherent new M paradigm best described as 'generalized length-inverse mortality' (GLIM). GLIM holds that mortality declines inversely with body length throughout much of the juvenile and adult phases of the fish lifecycle. Deviations from the length-inverse pattern may occur in older ages due to senescence and in early juveniles due to density-dependence. GLIM is strongly supported by empirical meta-analyses of mortality-size relationships and is also broadly consistent with multi-species and ecosystem models of predation mortality. Whether operationalized in closed functional form or through multi-species modeling of predation and residual mortality, GLIM provides a new 'standard M′ for fish population modeling and stock assessment applications. Consequences of mis-specifying size- and age-dependent M in stock assessment applications vary from moderate in many cases to severe under certain conditions, but even moderate consequences can be quantitatively significant in stock assessment and management. Further research is indicated with regards to senescence and to the representation of residual or non-predation mortality (M1) in multi-species and ecosystem models.

1. Introduction

Natural mortality is a key process in fish population dynamics that remains challenging to quantify (Maunder and Piner, 2015). Modeling of the natural mortality rate (M) forms part of all age and size-based fisheries assessment methods, from Beverton and Holt's (1957) yield-per-recruit model to today's integrated assessment models (Methot and Wetzel, 2013). Traditionally, fisheries models and stock assessment methods have adopted a highly simplified representation of the life cycle, with a recruited stage where natural mortality is described explicitly as a constant rate, and a stock recruitment relationship that implicitly accounts for juvenile mortality (and the environmental and density-dependent influences upon it). Many stock assessment applications (Brodziak et al., 2011) and standard texts on the subject (e.g. Hilborn and Walters, 1992; Quinn and Deriso, 1999; Haddon, 2011) treat M in the recruited stock as constant. However, evidence for size- and age-dependent M in juvenile and adult fishes has become all but insurmountable (Vetter, 1988; McGurk, 1986; Lorenzen et al., 2022). Likewise, limits to the 'constant M′ paradigm for the recruited stock have become increasingly apparent in fisheries assessment applications. For example, some fisheries exert heavy fishing pressure on juvenile stages, which must then be explicitly modeled in assessments (Abella et al., 1997; SEDAR, 2018; Punt et al., 2019). At the same time, due to the proliferation of marine reserves, populations where truly 'old' fish are well-represented are bound to become more common (Berkeley et al., 2004) and consideration of senescence effects more relevant. Last but not least, current interest in size-based and trait-based population dynamics modeling and in environmentally-driven variation challenge the 'constant M′ paradigm from a fundamental scientific perspective (Holsman et al., 2016; Andersen, 2019; Plagányi et al., 2022).

Research on size- and age-dependent M has developed from three different perspectives: Comparative empirical studies of mortality rates in relation to body size (Peterson and Wroblewski, 1984; McGurk, 1986; Lorenzen, 1996, 2000), modeling of predation mortality in multi-species and ecosystem models (Pope, 1991; Hollowed et al., 2000; Walters and Martell, 2004), and interest in the evolutionary ecology of senescence in fishes (Beverton and Holt, 1959; Beverton et al., 2004; Purchase et al., 2022). Fish evolutionary (Mangel and Abrahams, 2001; Mangel et al.,

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2010; Jørgensen and Holt, 2013) and community (Gislason et al., 2008; Blanchard et al., 2009) ecologists have long dispensed with the constant M axiom and adopted more realistic size- and/or age-dependent models. Models of size- or age-dependent M in fishes have been developed in closed form (Chen and Watanabe, 1989; Caddy, 1991; Lorenzen, 2000) and in the form of predation mortality models that typically have no closed form and are either data-driven (e.g. multi-species virtual population analysis (MSVPA), Magnusson, 1995; Pope, 1991) or emerge from complex simulation models (Hall et al., 2006; Walters and Martell, 2004). Over the past two decades, closed-form age-dependent M models have increasingly found their way into practice in fish population modeling (Lorenzen, 2005; Ahrens et al., 2020) and stock assessments (McKechnie et al., 2017; ICCAT, 2018; SEDAR, 2018). Likewise, predation mortality models have been operationalized for stock assessments in the most data-rich regions, principally the North Atlantic (Curti et al., 2013; Pope et al., 2021) and the North Pacific (Hollowed et al., 2000; Adams et al., 2022). Flexibility in specifying M models is viewed as an essential feature for next-generation stock assessment packages (Punt et al., 2020). However, modeling and assessment scientists are often unsure what M model to choose and when to diverge from the still-engrained constant M assumption. The purpose of this review is to encourage better and more consistent representation of size-and age--dependent M in fish population models. I synthesize current biological understanding, empirical analyses, and theoretical models of natural mortality in fish populations and show how multiple strands of evidence lead to a coherent and practical, 'generalized length-inverse mortality' (GLIM) paradigm.

2. Biology of natural mortality in fishes

2.1. Conceptual framework

Natural mortality rates in fish populations vary over the life cycle, often by orders of magnitude and show some clear patterns of regularity. To better understand these patterns, it is useful to consider basic components the lifetime mortality curve, the role of intrinsic and extrinsic factors in shaping this curve, and the occurrence of density-dependence. Mortality over the lifetime of animals is generally expected to show a 'bathtub' pattern of declining morality with age in early life and increasing mortality later in life, with an intermediate minimum that is sometimes described as a constant or 'quiescent' phase (Carnes et al., 1996, 2006; Engelman et al., 2017). A lifetime M pattern of this type is shown in Fig. 1 for laboratory-reared zebrafish (*Danio rerio*). To further analyze such mortality patterns, it is useful to consider the biology and ecology of extrinsic and intrinsic causes of mortality, their action over the course of the organism's life cycle, and the evolutionary forces shaping these patterns (Carnes et al., 1996). Extrinsic causes of mortality are those that originate outside of the organism, such as abiotic challenges, predation pressure, competition, and exposure to pathogens. Intrinsic causes are those that originate within the organism such as senescence, physiological resilience, cost of reproduction, anti-predator responses, competitive ability, and disease resistance/immunity. Extrinsic and intrinsic causes of mortality may interact, which complicates interpretation of combined patterns (Koopman et al., 2015). For example, predation mortality results from the interaction between extrinsic predation pressure and intrinsic habitat selection and anti-predator responses. Finally, we need to consider where and how

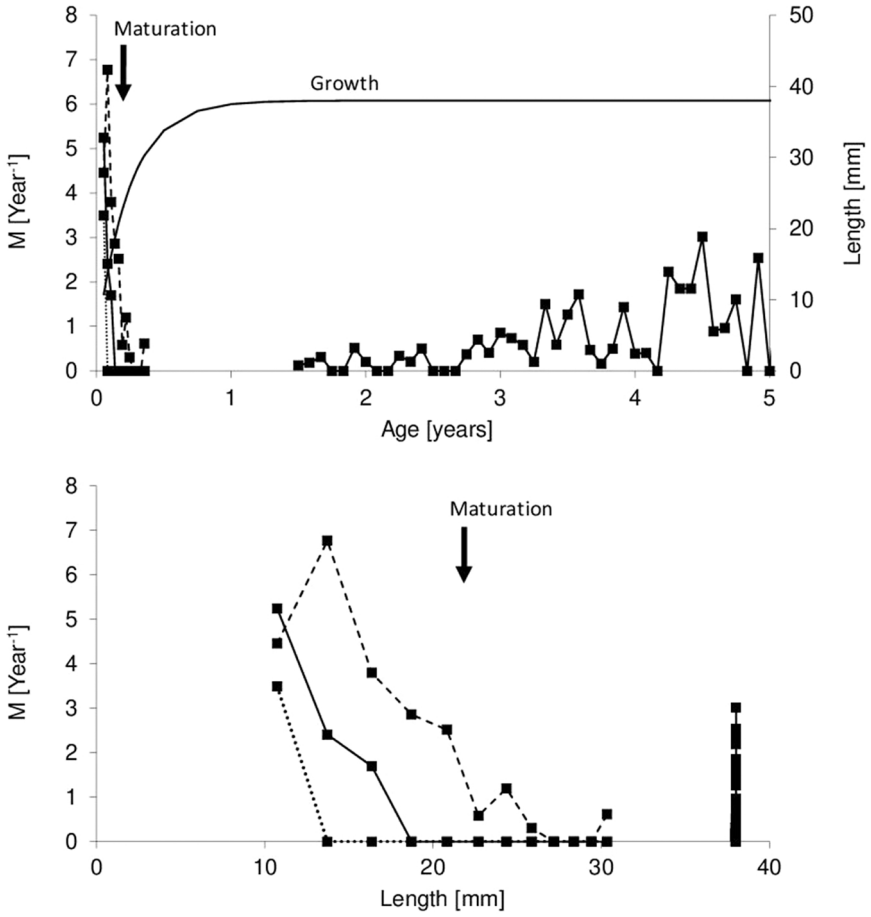

Fig. 1. Lifetime natural mortality patterns of zebrafish *Danio rerio* maintained in laboratory environments. The mortality schedules for juveniles and younger adults reflect three different stocking densities (dotted line: low, solid line: medium, dashed line: high). Data from Gerhard et al. (2002) and (Hazlerigg et al., 2012).

density-dependence occurs in M, given its influence on mortality patterns and fundamental importance to fish population dynamics and fisheries management (Rose et al., 2001; Lorenzen, 2008; Rindorf et al., 2022).

Lifetime mortality patters are strongly shaped by evolution. Evolutionary theory suggests that natural selection should act intensely on processes in the pre-reproductive and early reproductive periods but have little effect on processes in the late and post-reproductive period (Carnes et al., 1996). Evolutionary considerations may fall short of offering the elusive 'universal law of mortality', but they provide important insights into the mortality puzzle. The signature of evolution is likely to be most clearly visible in patterns of intrinsic mortality which may be revealed in captive laboratory populations when extrinsic causes of mortality are minimized (Carnes et al., 2006).

2.2. Biology and ecology of mortality in fish populations

I review the biological and ecological processes affecting natural mortality in fish populations in three steps, considering processes that underlie the decline of mortality rates with size and/or age in juveniles and adults, processes that may cause mortality to increase in older fish and finally, processes that may lead to density-dependence in M. The declining phase of the lifetime mortality pattern is shaped by both extrinsic and intrinsic causes of mortality. Predation pressure is commonly viewed as a dominant extrinsic cause of mortality in fish populations. Predation pressure is also inherently size-dependent, due to combined effects of predator size structure and gape limitation (Andersen and Ursin, 1977; Anderson, 1988; Magnusson, 1995). Exposure to extremes of abiotic conditions is another extrinsic cause of mortality that may decline with size and age due to prevailing habitat use, with smaller and younger individuals tending to use pelagic or shallow water habitats subject to greater abiotic variation than the habitats often used by larger and older individuals (Harden-Jones, 1968; Lucas and Baras, 2001). Competitive pressures may also be highest in early juvenile stages (Werner, 1986). Like the extrinsic causes, intrinsic causes of mortality may also show a decline in intensity with size and age. As larvae and juveniles grow and develop, they increase reserves and physiological resilience, ability to move away from unsuitable conditions, predator avoidance skills, and disease resistance/immunity (Fuiman and Magurran, 1994; Sogard, 1997). From an evolutionary perspective, high and size-dependent mortality rates suffered by larval and early juvenile fishes are expected to select for high growth rates, subject to tradeoffs such as increased mortality risk associated with high rates of food acquisition (Arendt, 1997; Sogard, 1997; Billerbeck et al., 2001). Even if extrinsic causes of mortality are of major importance during the declining M phase, the mortality trajectory will nonetheless bear a strong signature of intrinsic biological processes and the way these have been shaped by evolution.

The increasing or senescent phase of the lifetime mortality pattern is thought to be governed predominantly by intrinsic causes of mortality, in particular costs of reproduction and senescence. Cost of reproduction is a bioenergetic cost (diversion of energy from somatic growth and other functions to reproduction) and may also include a direct mortality cost (e.g. when fish aggregated in spawning locations are subject to increased predation mortality). The significance of the cost of reproduction as a cause of mortality appears to be variable among fish populations and is fundamentally contested (Hutchings, 1994; Kuparinen et al., 2012; Trippel et al., 2014). Senescence is a decline in individual biological function with age and is thought to result from multiple underlying mechanisms including oxidative damage, accumulation of alleles whose deleterious expression during old age falls beyond the effective reach of natural selection, or accumulation of somatic mutations (Carnes et al., 1996; Cagan et al., 2022; Purchase et al., 2022). Senescence may affect reproductive performance (reproductive senescence) or mortality (actuarial senescence). Rapid and severe actuarial senescence is evident in semelparous fishes (Hendry et al., 2004; Carlson et al., 2007; Christiansen et al., 2008). Among iteroparous fishes, actuarial senescence has been reported in both laboratory and wild populations but does not appear to be very commonly observed (Beverton and Holt, 1959; Craig, 1985; Gerhard et al., 2002). Indeed, aging researchers have turned to fishes as study subjects in part for this very reason (Woodhead, 1998). The rarity of documented senescence among wild iteroparous fishes may be in part due to observational issues including truncated age distributions in heavily fished populations, confounding of natural mortality with gear selectivity, and the fact that old individuals are bound to be rare even under unfished conditions (He et al., 2011; Woodhead, 1998; Xia and Møller, 2022). However, delayed senescence is expected to evolve in fish populations that exhibit indeterminate growth and capacity for substantial, continued increase in fecundity with age because mortality remains subject to intense selection (Purchase et al., 2022; Reznick et al., 2002). A low extrinsic mortality rate may further reduce and delay senescence, while environmental stress may increase the expression of senescence (Mangel, 2008). Empirical evidence from meta-analyses suggests that on average, natural mortality rates in fish populations continue to decline with size (and age) after maturity (Charnov et al., 2013; Lorenzen et al., 2022). On average, therefore, actuarial senescence appears to be delayed to the extent that it does not stop or reverse the pattern of declining M observed in meta-analyses of mortality-size relationships.

Density-dependence in mortality is principally driven by competition for resources and/or density-dependent predation (Rose et al., 2001; Lorenzen, 2005). As experiments with laboratory populations show, density-dependent mortality has a strong intrinsic basis (Hazlerigg et al., 2012). Density-dependent mortality is largely confined to early juvenile stages in fish populations (Myers and Cadigan, 1993; Andersen et al., 2017; Lorenzen and Camp, 2019).

2.3. Intrinsic mortality in fishes: insights from captive fish populations

Separating extrinsic and intrinsic influences on lifetime mortality patterns in fishes has proved near impossible in the wild. However, captive populations in laboratory and aquaculture settings can shed light on patterns of intrinsic mortality. Aquaria and aquaculture systems are designed to minimize extrinsic causes of mortality by providing optimal environmental conditions and resources and eliminating or at least reducing predation mortality and exposure to pathogens. Mortality patterns displayed under such conditions reflect primarily intrinsic causes (Carnes et al., 2006). The observed mortality patterns are unlikely to be fully representative of intrinsic mortality patterns expressed in the wild, however, because fishes respond to 'pampering' in aquaculture conditions by displaying life history responses known as domestication effects (Thorpe, 2004; Lorenzen et al., 2012), Nonetheless, captive fishes provide perhaps the most informative glimpse of intrinsic M patterns we have at our disposal.

Zebrafish (*Danio rerio*) populations have been extensively studied in laboratory environments during virtually all parts of their lifecycle (Hazlerigg et al., 2012; Gerhard et al., 2002). Laboratory-reared zebrafish show a clear 'bathtub' pattern of declining M with size and age through their juvenile and adult life, as well as clear signs of senescence very late in life ((Fig. 1). They also show evidence of density-dependent M in smaller juveniles (Hazlerigg et al., 2012). Zebrafish are considered annual species in the wild (Spence et al., 2008). This is consistent with an expected $M \approx 5\ year^{-1}$ for a wild fish of this size and growth pattern (Lorenzen, 1996; Lorenzen et al., 2022) and a corresponding maximum age $a_{max} \approx 1$ (Hoenig, 1983; Then et al., 2015). Therefore, the senescence evident from laboratory populations of zebrafish is delayed well past the lifespan of the species in the wild, with increases in mortality becoming evident only after about 2.5 years of age (Figure 1). It is unclear how domestication may have affected the expression of senescence in the laboratory populations because this is subject to two counteracting influences. Domestication tends to accelerate the life cycle (Thorpe, 2004; Lorenzen et al., 2012) which should also lead to earlier

senescence, whereas the low extrinsic mortality rates suffered by captive fishes should lead to the evolution of delayed senescence (Purchase et al., 2022; Reznick et al., 2002). This uncertainty notwithstanding, zebrafish in laboratory environments demonstrate that size/age and density-dependence in juvenile mortality have an intrinsic biological basis and that senescence ultimately occurs but may be delayed to the extent that it would not be noticeable in the wild.

Further insights can be gained from comparative analyses of the allometric patterns of M in wild fishes, cultured fishes, and cultured fishes released into the wild (Fig. 2). Fishes cultured in ponds and tanks/raceways show similar allometric scaling of M which is more steeply negative than that of M in wild fishes (Lorenzen, 1996). Therefore, intrinsic mortality may decline even more steeply with size and age than extrinsic mortality, which is often viewed implicitly as the main source of size- and age- dependence in M. Interestingly, cultured fishes stocked into natural ecosystems are subject to very high overall levels of mortality which nonetheless follow the allometric scaling of mortality among wild fishes (Lorenzen, 2000, 2006). Due to domestication effects, cultured fishes tend to show poor intrinsic anti-predator responses, habitat selection, feeding, etc. which may make them extraordinarily vulnerable to extrinsic causes of mortality (Lorenzen et al., 2012). The high mortality rates suffered by cultured fishes in the wild show just how important intrinsic factors are in shaping natural mortality patterns, even in natural ecosystems where extrinsic causes of mortality are thought to account for the majority of deaths.

2.4. Size- or age-dependent?

Lifetime M patterns reflect both size-based and age-based processes, though size-based processes appear to predominate quantitatively and underlie some powerful generalizations (Lorenzen et al., 2022). The regularities of size- and age- dependent mortality in fish populations are macro-ecological patterns revealed through long-term and large-scale comparative analyses and carry the signature of ecological as well as evolutionary processes. These are general patterns we expect and find 'on average' and at that level, size and age are completely linked through a growth function. Therefore, size- and age-based processes can be integrated through use of a growth function and the size and age-based representations of the resulting patterns are equivalent.

3. Mortality models and quantitative generalizations

3.1. Size-based allometric scaling models

The scaling of natural mortality with body size in fishes has been investigated at the population, species, and community level (Peterson and Wroblewski, 1984; McGurk, 1986; Lorenzen, 1996). Allometric scaling models for mortality M with length L or weight W are of the form:

$$M(L) = M_{Lr}\left(\frac{L}{L_r}\right)^{c} \quad (1a)$$

$$M(W) = M_{Wr}\left(\frac{W}{W_r}\right)^{b} \quad (1b)$$

Where M_{Lr} and M_{Wr} are the mortality rates at reference length L_r or weight W_r. The allometric exponents for length and weight are related by $c = \beta b$, where β is the exponent of the length-weight relationship $W = \alpha L^{\beta}$.

Studies focused on the population level have very consistently shown M to scale with length to the power of $c \approx -1$, i.e. to be length-inverse (McGurk, 1996; Lorenzen, 2000; Lorenzen et al., 2022). Lorenzen et al. (2022) showed strong evidence for this scaling at the population level by applying a joint-slope mixed effects model to the mortality-size data sets assembled by Lorenzen (1996) (which includes the McGurk, 1986 data) and Gislason et al. (2010), separately and combined. Lorenzen et al. (2022) also showed that the intercept of the mortality-length relationship is positively related to the population's maximum length growth rate and that on average, large-growing species are subject to higher mortality-at-length than smaller-growing ones but experience lower mortality in adulthood due to their large size (Fig. 3).

At the community level or when analyzing among-population variation, M often scales with similar or somewhat less negative exponents between $c = -0.81$ and $c = -1.02$ (McGurk, 1986, 1987; Lorenzen, 1996; McCoy and Gillooly, 2008). From a theoretical perspective, multiple authors have suggested a 'metabolic' scaling exponent for community-level M of $c = -0.75$ (Peterson and Wroblewski, 1984; Dickie et al., 1987; Andersen, 2019). The differences between population and community-level scaling are reconciled when considering that populations of larger-growing species on average tend to have higher intercepts of the mortality-length relationship (Gislason et al., 2008; Lorenzen et al., 2022). This in effect tends to generate less-negative

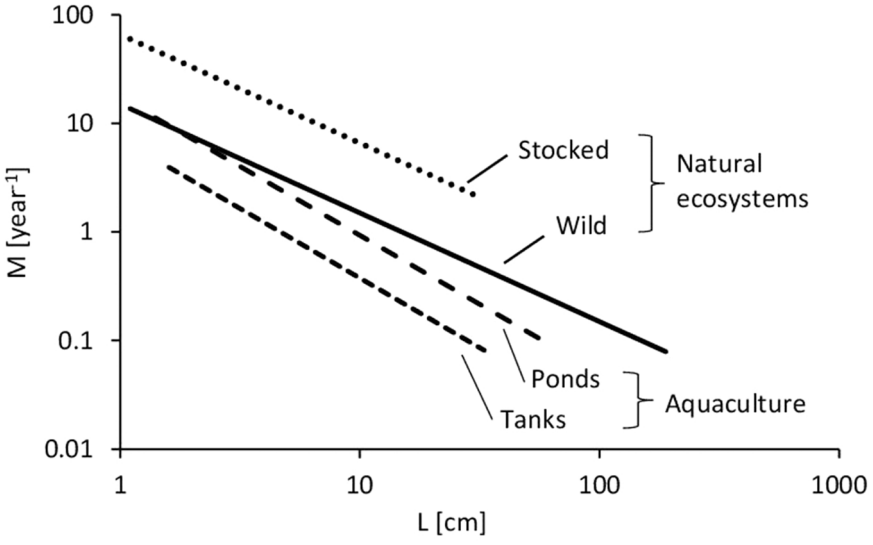

Fig. 2. Scaling of natural mortality with body length in natural ecosystems (wild fish and stocked hatchery-reared fish) and in aquaculture facilities (ponds/cages and tanks). Data from Lorenzen (1996, 2000) and Lorenzen et al. (2022).

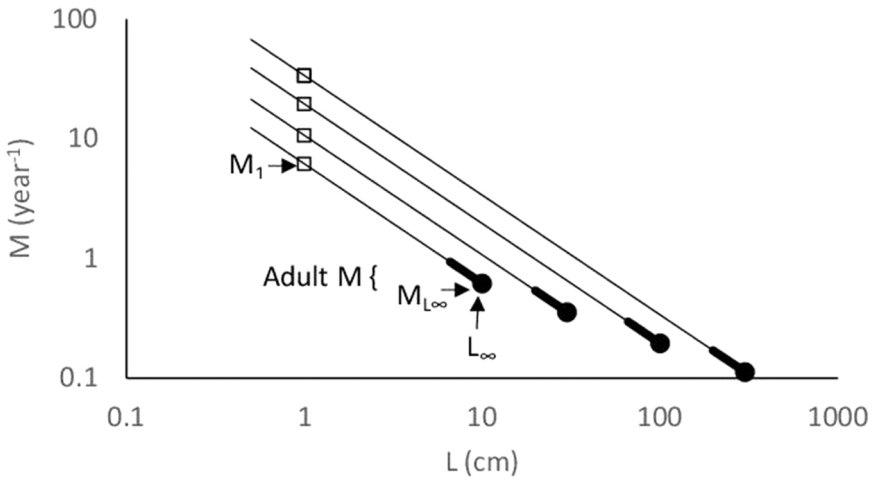

Fig. 3. Length-inverse natural mortality (M) models estimated for populations with different asymptotic lengths L_∞ (from Lorenzen et al., 2022). M_1 is the intercept at unit length (1 cm) while $M_{L\infty}$ is the intercept at L_∞. Thickened lines indicate the mortality pattern during the adult life phase (after maturity which is assumed to occur at 0.66 x L_∞).

scaling at the community level when populations with different maximum sizes are combined (Fig. 4).

Multiple regression models to predict M from body length and growth parameters have been developed by Gislason et al. (2010). Charnov et al. (2013) derived a theoretically inspired simplification of these models which structurally resembles an allometric scaling model with exponent c= −1.5 and intercept M_{Lr}=K at L_r =L_∞. However, the steep scaling exponent of c= −1.5 does not represent within-population scaling of M but rather an among-population pattern in (Gislason et al., 2010)'s comparative mortality data (Lorenzen et al., 2022),

From a fish population modeling and stock assessment perspective, the most important conclusions here are that population and community level scaling of M should be clearly distinguished and that within populations, M scales very consistently with length to the power of c ≈ −1. Some variation in scaling is likely to arise from variation in the data or differences in the estimation methods used (e.g. generalized linear regression vs. functional or non-parametric regression), and some variation may be systematic and process-based in nature (Pope et al., 2021). On the other hand, large deviations such as the community-level scaling c ≈ −0.75 or Charnov's c= −1.5 arise when population effects are disregarded and are unlikely to be representative of scaling within populations.

The most important use of size-based mortality models has been in the context of meta-analyses as described above, where they have led to powerful insights and generalizations. Size-based M models can also be directly incorporated into size-based population of multi-species models and stock assessment methods (Hall et al., 2006; Andersen, 2020).

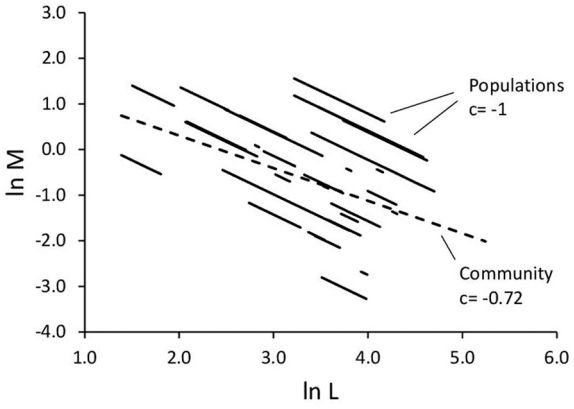

Fig. 4. Population vs. community (or ensemble of populations) level scaling of natural mortality with body length. Modified from Fig. 4 in Lorenzen et al. (2022), based on data from Gislason et al. (2010).

3.2. Age-based models

Age-based models can be developed through conversion of size-based models to an age-base using a growth function, or directly from age-based considerations. Some of the more commonly used age-based models are detailed in Table 1 and illustrated in Fig. 4 for an example stock, the U.S. South Atlantic greater amberjack (*Seriola dumerili*).

An age-based transformation of the allometric mortality model, most commonly using a von Bertalanffy growth function, is the age-based model most directly derived from the results of size-based meta-analyses. Used with allometric scaling estimates from Lorenzen (1996) or the isometric c= −1 (Lorenzen, 2000; Lorenzen et al., 2022) the model (often colloquially referred to as 'Lorenzen M′, Table 1) has found wide application in fisheries stock assessments in the Southeastern USA and in some tuna commissions (e.g. McKechnie et al., 2017; ICCAT, 2018, SEDAR, 2018), as well as in other contexts such as mark-recapture studies (Coggins et al., 2006; Lorenzen, 2006). In most applications, the scaling exponent is fixed while the intercept is either adjusted to align with a constant M estimate for the recruited stock or estimated directly within the assessment model. Often, an empirical estimator developed for constant M (e.g. Pauly, 1980; Hoenig, 1983; Then et al., 2015) is used to inform the intercept of the Lorenzen M. For example, Fig. 5 shows Lorenzen M curves for intercepts adjusted so that mean Lorenzen M in the mature population equals the constant M values predicted by the widely used Hoenig (1983) maximum-age-based estimator and the Then et al. (2015) growth-based estimator. This approach to using the Lorenzen M is consistent with empirical evidence for the generality of the scaling exponent and for patterns of variation in the intercept (Lorenzen et al., 2022). Variations on the theme of this model include a version that uses the scaling and intercept parameters derived theoretically by Peterson and Wroblewski (1984) and a set of bi-phasic mortality models that combine size-dependent juvenile with constant adult M components (Dureuil et al., 2021).

The 'Charnov M′ (Table 1) is an age-based transformation of the corresponding length-based Charnov et al. (2013) model. As outlined above, there are concerns that this model does not represent within-population scaling of M (Lorenzen et al., 2022), and these concerns are particularly relevant to the population modeling and stock assessment applications typically associated with the age-based transformation. In the example shown in Fig. 5, Charnov M is at a level

Table 1
Age-dependent natural mortality models (assuming von Bertalanffy growth where relevant). Here, a is age; a_0 = age at length L= 0; M(a) is the natural mortality rate at age; $M_{L\infty}$ is the natural mortality rate at asymptotic length L_∞; K is the von Bertalanffy growth rate; c is the allometric scaling of mortality with body length L (c=−1 for the length-inverse mortality model); Mτ is the asymptotic mortality rate and B is a coefficient for the Caddy model; M_d, M_c, and M_s, are the mortality coefficients and b_d and b_s are the exponents for the for the declining (d), constant (c) and senescent (s) components of the Siler model.

Name	Equation (s)	References
Lorenzen allometric and length-inverse	$M(a) = M_{L\infty} \quad (1 - e^{-K(a-a_0)})^c$	Lorenzen (1996, 2000);Lorenzen et al. (2022)
Charnov	$M(a) = K(1 - e^{-K(a-a_0)})^{-1.5}$	Charnov et al. (2013)
Chen & Watanabe	$M(a) = \begin{cases} \frac{K}{1 - e^{-K(a-a_0)}}, a \le a_M \\ \frac{K}{\gamma_0 + \gamma_1(a - a_M) + \gamma_2(a - a_M)^2}, a > a_M \end{cases}$ $\gamma_0 = 1 - e^{-K(a_M - a_0)}$ $\gamma_1 = Ke^{-K(a_M - a_0)}$ $\gamma_2 = -\frac{1}{2}K^2 e^{-K(a_M - a_M)}$ $a_M = \frac{1}{K} ln(1 - e^{Ka_0}) + a_0$	Chen and Watanabe (1989)
Caddy	$M(a) = M_\tau + \frac{B}{a}$	Caddy (1991)
Siler	$M(a) = M_d e^{-b_d a} + M_c + M_s e^{b_s a}$	Siler (1979)

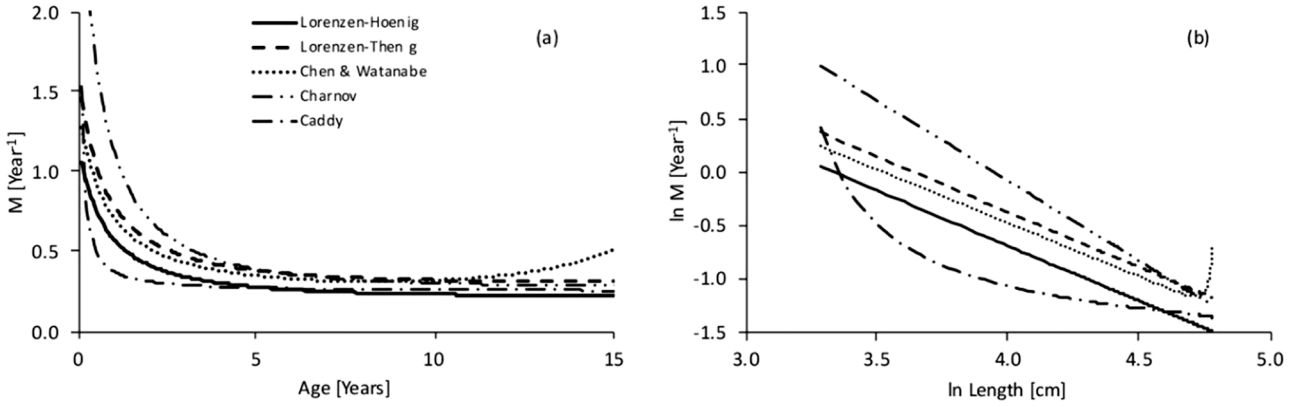

Fig. 5. Comparison of age-dependent natural mortality (M(a)) models for South Atlantic Greater Amberjack (*Seriola dumerili*): Lorenzen M scaled to the Hoenig t_{max} – based and Then growth-based M estimates for the mature population; Chen & Watanabe M; Charnov M, and Caddy M fitted to the Lorenzen-Hoenig M (see Table 1). Life history parameters: Von Bertalanffy growth K= 0.284 $year^{-1}$ and L_∞ = 120 cm, a_0 = −0.786 years and maximum age a_{max} = 20 years (SEDAR, 2020).

similar to the Then-adjusted Lorenzen M for old and large fish, but increases much more steeply towards younger and smaller fish.

The Chen and Watanabe (1989) model is a bi-phasic model divided into a pre-senescent phase where M declines inversely with length modeled by a von Bertalanffy growth function, and a senescent phase where mortality increases with age (Table 1). The Chen & Watanabe M is entirely predicated on the population's von Bertalanffy growth parameters in a way that reflects a mixture of theoretical concepts and mathematical convenience. It is consistent with certain observed patterns but is neither a fully theoretical nor a truly empirical model. The declining phase model is equivalent to the length-inverse Lorenzen M with intercept $M_{L\infty}$ = K and this phase continues until the 'end of the reproductive life span' a_M. Therefore, the length-inverse model is used for much of the lifespan, consistent with the empirical scaling result of Lorenzen et al. (2022) but more constrained in the intercept. The assumed onset of senescence in the Chen & Watanabe model is consistent with the idea of delayed senescence in iteroparous fishes, but there is no clear theoretical reason or empirical evidence for the particular senescence pattern assumed in the model. The Chen & Watanabe model for South Atlantic greater amberjack is illustrated in Fig. 5. In summary, the model describes key patterns that have since been broadly corroborated in empirical meta-analyses but is too constrained to fully capture empirical patterns, at least in its original parameterization. The model has seen practical application particularly in elasmobranch fisheries (Simpfendorfer et al., 2005).

The Caddy (1999) model was developed based on the idea of applying the reciprocal relationship between mortality rate and the mean age of survivors to a cohort over time. This results in a simple age-based mortality model with two parameters (Table 1). The model is fitted to M-at-age data and has been used mostly in the Mediterranean region (Abella et al., 1997; Caddy and Abella, 1999). A Caddy model fitted to the Lorenzen M(a) pattern (intercept adjusted to Hoenig M) is shown in Fig. 5. I this case, the Caddy model suggest a steep decline of M with age in early juveniles followed by a more or less constant M, a pattern substantially at odds with the empirically supported allometric scaling and length-inverse models.

The Siler model (Table 1) is a very general competing risks model comprised of additive terms for declining, constant and senescent (increasing) mortality components (Siler, 1979). With no fewer than five parameters, the model is very flexible and can approximate all of the age-based models discussed above and illustrated in Fig. 5. That makes the model useful for simulation studies such as in Punt et al. (2021) but limits its practical application to the most data-rich assessment situations. In the fisheries context, the model has been used mostly in marine mammal population dynamics (Barlow and Boveng, 1991).

Of the age-based mortality models reviewed here, all account for

declining M but only two (the Chen & Watanabe and Siler models) have senescence terms (Table 1; Fig. 5). The Lorenzen and Chen & Watanabe models are consistent with meta-analyses of within-population mortality-size relationships (Section 3.1), the Siler model can be configured to be, and the Charnov and Caddy models diverge. The senescence term hard-wired in the Chen & Watanabe model has no specific biological or empirical basis and the model is sometimes used without that term. Since senescence terms are rarely used in stock assessment applications, we lack reliable generalizations about the pattern of senescence in iteroparous fishes, other than that we expect it to be delayed and gradual (Section 2).

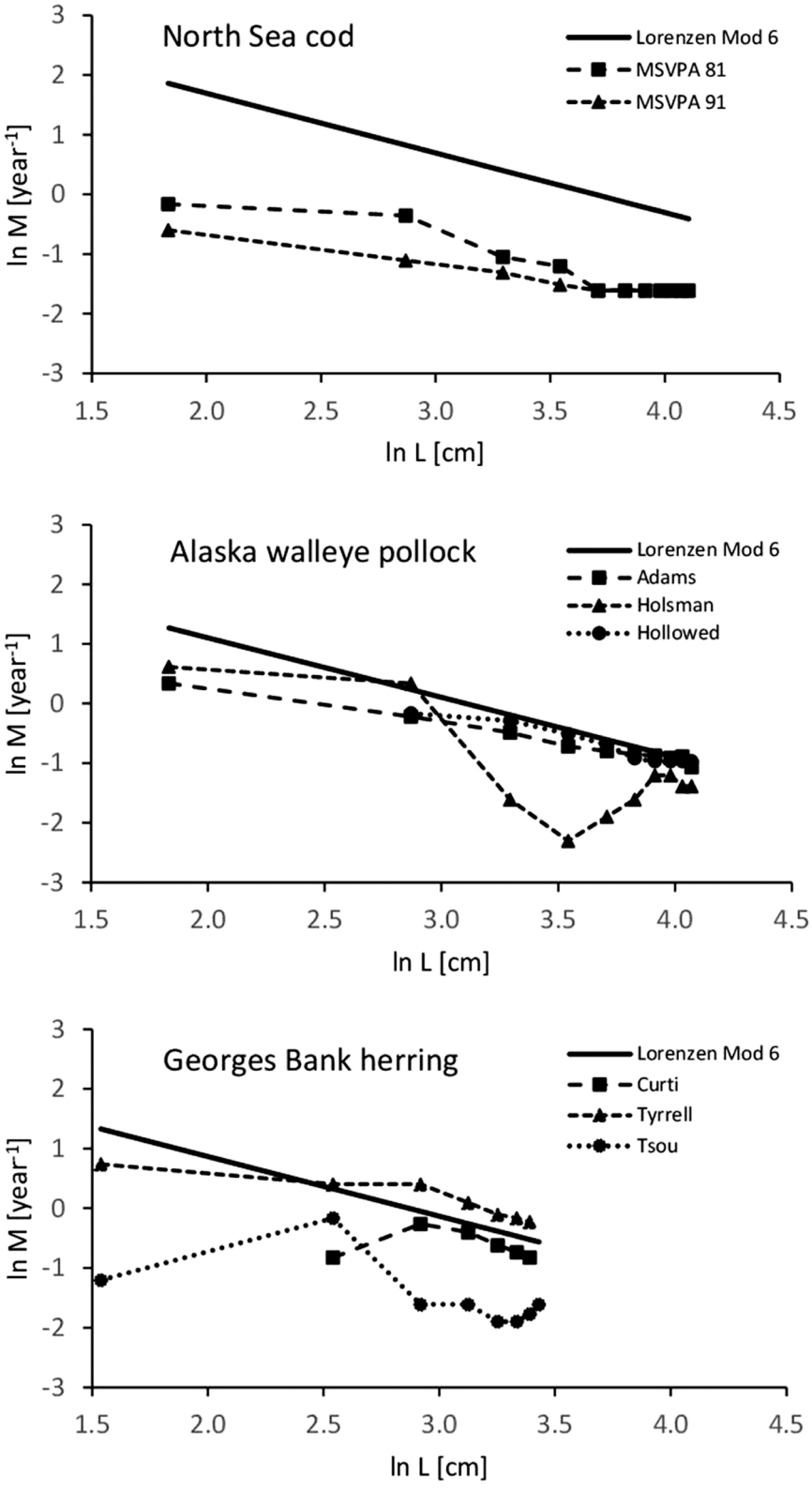

Fig. 6. Comparison of comparative empirical and predation/multispecies model-derived natural mortality M(L) in relation to body length for North Sea cod (*Gadus morhua*), Alaska walleye pollock (*Theragra chalcogramma*) and Georges Bank herring (*Clupea harengus*). Length-inverse (c=−1) Lorenzen M with $M_{L\infty}$ predicted from growth parameter K (Model 6 in Table 3, Lorenzen et al., 2022) for all species. MSVPA results in 1981 and 1991 for cod (Pope et al., 2021); Adams et al. (2022), Holsman et al. (2016), Hollowed et al. (2000) for pollock; (Curti et al., 2013), Tyrrell et al. (2008), Tsou and Collie (2001) for herring. The predation model M(L) estimates were derived by transforming M(a) estimates from the original studies using stock-specific growth models.

3.3. Multi-species or ecosystem models of predation mortality

In addition to size-or age-based models of closed mathematical form, mortality patterns can be derived from predation terms in multi-species or ecosystem models. Andersen and Ursin's (1977) multi-species model and the subsequently developed multi-species virtual population analysis (MSVPA) (Magnusson, 1995) established the practice of separating natural mortality into a predation component M2 and a residual or non-predation component M1. In applications, M1 is typically fixed a priori while M2 is calculated as the ratio of consumption by predators to biomass of the stock. In an age-structured framework where a is age:

$$M(a) = M1(a) + M2(a) = M1(a) + \frac{Consumtion\ by\ predators\ (a)}{Biomass\ (a)} \quad (2)$$

The predation mortality M2(a) and total natural mortalit M(a) derived from such models are typically variable and do not follow a simple functional form. Residual mortality M1(a) subsumes all causes of mortality not explicitly accounted for by predation terms (including any predators not explicitly modeled in M2(a) but is generally thought to represent non-predation mortality. It should be noted here that estimates of M2(a) are not independent of M1(a) because both affect biomass at age, the denominator of the M2(a) term (Eq. 2). In most cases, M1(a) is assumed to be constant at a level similar to that of the constant M used in single species assessment models (Pope, 1991; Pope at al, 2021), though a bathtub-shaped (Hall et al., 2006) or increasing (Holsman et al., 2016) functional form have also been used. Since M1(a) essentially represents the signature of intrinsic mortality, there are good reasons to reject the idea that it should be size-and age-independent (see Section 2 and Carnes et al., 2006). Compared to M2, the M1 term has received scant scientific attention even though it can substantially influence the overall mortality patterns generated by multi-species and ecosystem models.

Limited work has been done to compare and reconcile directly estimated and predation-model-derived lifetime mortality patterns. A comparison of total natural mortality estimates at length (M(L)=M1(L) +M2(L)) from various predation and multispecies models with the length-inverse Lorenzen M(L) pattern for the same stocks (derived from Model 6, Lorenzen et al., 2022) is shown in Fig. 6. The predation model M(L) estimates were derived by transforming M(a) estimates from the original studies using stock-specific growth models. This comparison reveals broad similarities and some important differences. In a broad sense, the length-inverse and predation mortality models show similar patterns, all are more similar to each other than to the traditional constant M. Beyond those similarities, the scaling of mortality estimates from predation/multispecies models is approximately length-inverse or slightly less negative for larger fish, but often substantially less negative for fish below about 15 cm length (2.7 on the ln L scale in Fig. 6). The overall level of M(L) from the predation and multispecies models here is similar to, or lower than the Lorenzen M(L) predictions. The length-inverse model therefore captures key features of the mortality patterns estimated from predation and multispecies models but predicts a steeper increase and overall higher level of M for small juveniles. One possible explanation for this pattern is that residual mortality M1 may be higher and more size dependent than assumed in the predation and multispecies models. Obviously, this idea is consistent with evidence for intrinsic M patterns (Section 2.3). The effect of adding different residual mortality M1(L) terms to the estimated predation mortality M2(L) pattern for Alaska walleye pollock from (Adams et al., 2022) is shown in Fig. 7. In this case, M2(L) scales with length approximately to the power of − 1, but adding a constant M1(L) term leads to a less negative scaling in M(L). However, adding a size-dependent M1(L) term (for example with the 'metabolic' scaling of c=−0.75 or an 'aquaculture' scaling of c=−1.25) (Figure 2) gives rise to an M(L) scaling more in line with the − 1 expected from comparative empirical studies (Lorenzen et al., 2022). Of course, the aim of this simple analysis is not to make the predictions match, but to understand why and how they differ. There are good reasons why population-specific estimates derived from predation modeling might differ from those predicted by comparative meta-analyses. At the same time, consistent differences found across many such comparisons may indicate underlying structural issues such as incompatible assumptions about residual mortality M1.

Predation mortality, multi-species and ecosystem models are increasingly used in in a stock assessment context in data-rich regions such as the North Atlantic (Pope et al., 2021; Curti et al., 2013) and the North Pacific (Hollowed et al., 2000; Adams et al., 2022). As shown here, such models are broadly consistent with the M scaling relationships derived from empirical meta-analyses (Section 3.2) but may benefit from better representation of intrinsic mortality in the M1 term.

3.4. Modeling density-dependence in early juvenile mortality

Density-dependence may affect M in early juveniles. Modeling density-dependence in M should not be a concern except in settings where the dynamics of juveniles smaller than 20% of the population's L_∞ must be explicitly modeled (Lorenzen and Camp, 2019). This may be the case when small juveniles are removed (e.g. by fishing or water abstractions) or added to the population (e.g. by stocking of hatchery-reared fish). Several approaches to this problem have been developed. Lorenzen (2005) proposed an approach to 'unpacking' the Beverton-Holt stock-recruitment relationship into multiple stanzas

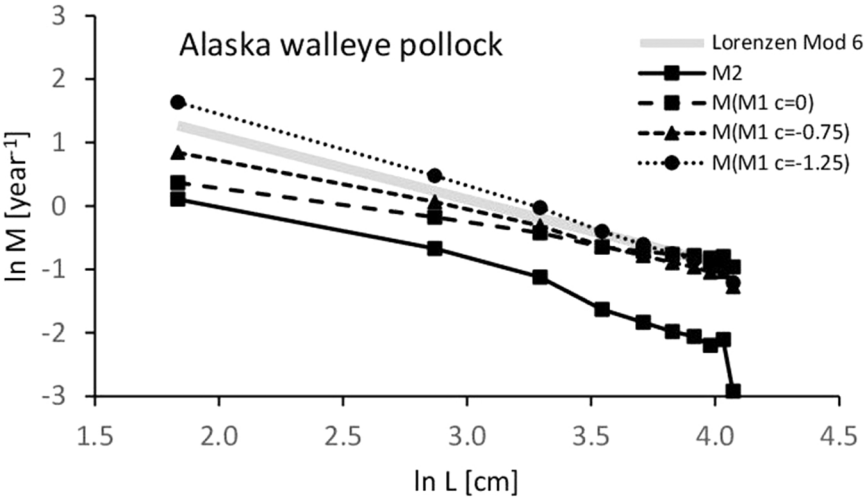

Fig. 7. Illustration of the effect of different assumptions about residual mortality M1(L) on total natural mortality M(L)=M1(L) +M2(L) of Alaska walleye pollock (*Theragra chalcogramma*). M2(L) and constant baseline M1(c=0, $M1_{L\infty}$=0.33) from Adams et al. (2022). The alternative size-dependent M1(L) models are M1(c=−0.75, $M1_{L\infty}$=0.2) and M1(c=−1.25, $M1_{L\infty}$=0.2). Also shown for comparison is the length-inverse (c=−1) Lorenzen M with $M_{L\infty}$ predicted from growth parameter K (Model 6 in Table 3, Lorenzen et al., 2022).

informed by the length-inverse mortality model. Camp et al. (2017) described a simplified version of this approach and Johnston et al. (2018) adapted it the Ricker stock-recruitment relationship. Powers (2014) developed a density-dependent M model as an alternative to the Lorenzen M, which it could partially substitute for at sizes/ages where density-dependence is strong. Further approaches focused on estimating juvenile density-dependence from young fish surveys or bycatch in shrimp trawls are given in Myers and Cadigan (1993); Gazey et al. (2008) and Forrest et al. (2013).

4. Synthesis

4.1. Emerging paradigm: generalized length-inverse mortality (GLIM)

Key strands of evidence from this review of biological considerations, empirical generalizations and theoretical models lead to a coherent paradigm for natural mortality in fishes that can be described as 'generalized length-inverse mortality' (GLIM). The core, overarching pattern of natural mortality in juvenile and adult fishes is one of scaling with the inverse of body length. This generalized form of this relationship allows moderate deviations from strictly length-inverse scaling ($c \approx -1$) and can account for senescence in older ages and for density-dependence in early juveniles when a need to explicitly consider these processes is indicated. Size- and age-dependent natural mortality patterns in fish populations are synthesized and the generalized length-inverse mortality (GLIM) paradigm illustrated in Fig. 8. The log-log plot typically associated with the study of mortality-size relationships (Fig. 8a) particularly illustrates the central length-inverse mortality relationship. In smaller juveniles (at lengths below about 20% of L_∞, Lorenzen and Camp, 2019), the relationship may become "braided" by density-dependence. Conversely, in large and old fish near L_∞, mortality may increase due to senescence. The same patterns are shown on linear scales against size (Fig. 8b) and age (Fig. 8c), the perspectives typically associated with stock assessment applications. It is useful here to note the changes in perspective associated with the different plots, for example that juvenile mortality occupies a large part of the length-based pattern in log space, whereas senescence could potentially occupy a substantial part of the linear age-based pattern.

The GLIM is strongly supported by empirical meta-analyses of mortality-size relationships (Section 3.2) and is broadly consistent with multi-species and ecosystem models of predation mortality (Section 3.3). It also builds on a long history of use of length-inverse M as a simple generalization in theoretical modeling studies (Beyer, 1989; Chen and Watanabe, 1989; Lorenzen, 2005).

The length-inverse scaling of M within populations ($c \approx -1$) is more steep than the scaling commonly observed at the community level or when analyzing M estimates assembled from multiple populations and species without accounting for population effects. This illustrates the import influence of factors intrinsic to the organism in shaping lifetime mortality patterns even when extrinsic causes such as predation account for the largest share of deaths. The mechanisms underlying the distinct within-population scaling are not well understood, but selection for fast growth and concomitant elevated mortality in juveniles combined with delayed senescence in (iteroparous) adults is likely to play a role. This hypothesis is further supported by the observation that intercepts of mortality-length relationships are positively correlated with maximum growth rate in among-population comparisons (Lorenzen et al., 2022).

The GLIM pattern differs in important respects from the 'bathtub' mortality curves observed in humans, which consistently show very rapid decline in early life, reach the lowest point prior to the onset of reproductive maturity, and then increase again (Carnes et al., 2006; Engelman et al., 2017). By contrast, the GLIM declines more gradually through the juvenile phase and continues to decline well into adulthood for most iteroparous fishes (Fig. 8). Even for semelparous fishes, the declining M phase is known to follow the gradual, length-inverse pattern (McGurk, 1996). The long and gradual decline phase in the GLIM likely

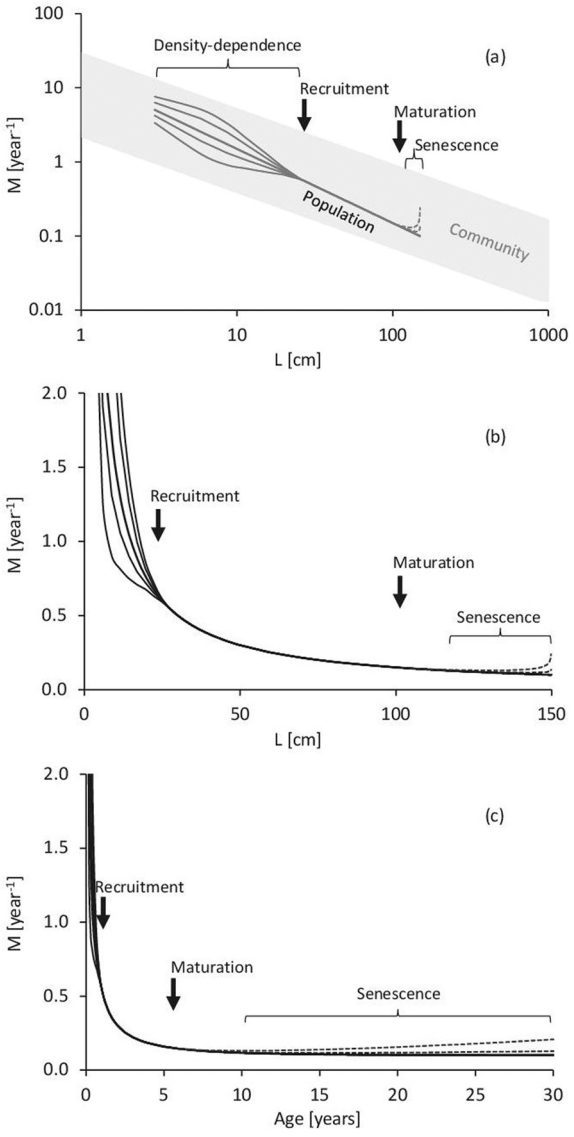

Fig. 8. Synthesis of size- and age-dependent natural mortality patterns in the life cycle of fishes, illustrating the generalized length-inverse mortality (GLIM) paradigm. (Length-inverse M base model representing a population with L_∞ = 150 cm, K= 0.2 year^{-1} and $M_{L\infty}$=0.1 year^{-1}).

reflects the universal size dependence in M associated with aquatic communities, modified by intrinsic M components that give rise to a distinctly more steep within-population scaling.

4.2. Operationalizing GLIM

The GLIM paradigm may be operationalized as a length-based or age-based model in closed functional form as part of a population model or stock assessment (Sections 3.1 and 3.2; Table 1). The approximately length-inverse functional form can be combined with different approaches for estimating the intercept including use of empirical predictors (already a commonly used approach), estimation within the stock assessment model, or mark-recapture studies (Section 3.2). Since it is rarely possible to estimate both the scaling and the intercept of the mortality-size relationship from data available for a particular stock, the robust generalization about scaling inherent in the GLIM is of great practical value.

The GLIM can also be operationalized through predation mortality modeling in a multi-species or ecosystem framework, which will yield

broadly similar results particularly with an appropriate residual M1(a) term (Section 3.3). Dynamic multi-species and ecosystem models of course account for changing species interactions over time and may yield different long-term predictions (Pope, 1991; Plagányi et al., 2022), but in a static or short-term application their mortality estimates are broadly compatible with GLIM (Pope, 1991; Tyrrell et al., 2011; Pope et al., 2021).

A senesce term can be added to the length-inverse GLIM 'core model' when significant senescence is observed or hypothesized. The Siler and Chen & Watanabe models (Section 3.2; Table 1) include senescence terms that can be adapted for use with the GLIM 'base model'. Unfortunately, we do not have reliable quantitative generalizations about the pattern of senescence in iteroparous fishes other than that we expect it to be delayed and gradual (Ahti et al., 2021; Purchase et al., 2022).

Density-dependence may need to be incorporated into the GLIM 'core model' if the dynamics of early juvenile stages are to be explicitly represented in an assessment model. This is likely to be necessary only when juveniles of lengths less than 20% of the population's L_∞ are to be explicitly modeled (Lorenzen and Camp, 2019). Section (3.4) outlines some approaches to modeling juvenile density-dependence.

The GLIM is likely to capture the most important size- and age-dependent features of natural mortality in most fish stocks, judging from the comparative meta-analyses and predation mortality models discussed above (Section 3). Moreover, in most cases, it will not be necessary to include senescence or density-dependence terms, but these issues should be considered in specifying the M model. Given the strength of empirical support for the GLIM, a compatible M model should form part of the 'base model' for most stock assessments or at least be included as a sensitivity run. Of course, as with any very general model, the GLIM will not be appropriate for every fish population or every modeling or assessment application. It will provide a robust starting point (certainly more realistic than the constant M axiom), but analysts should be prepared to diverge from GLIM when evidence points towards a different model or approach being more appropriate for the population or problem at hand.

4.3. Implications of GLIM for the theory of fishing and related applications

Compared to the theory of fishing based on the traditional constant M assumption, GLIM implies various incremental changes but no radical departure. GLIM tends to predict higher natural mortality overall in the recruited stock and higher absolute levels of recruitment (Pope, 1991; Tyrrell et al., 2011; Pope et al., 2021). It also implies that harvesting of juvenile stages has less impact on the stock and that the contribution of large and old fish to spawning biomass is greater than under the constant mortality axiom (Ahrens et al., 2020). Operationally, as mentioned above, the GLIM can be and has been effectively integrated with key concepts and methods developed using the constant M assumption. Since the GLIM has already been effectively adopted in many fisheries where size or age-dependent M or predation mortality models are being used, the M paradigm change is well under way. Adoption of size and age-dependent M has enabled the use of fisheries assessment models to address management questions considered outside the scope of models using constant M, including the assessment of bycatch of early juvenile fishes in shrimp trawls (Gazey et al., 2008; SEDAR, 2018), fisheries enhanced through releases of juvenile hatchery fish (Lorenzen, 2005; Hervas et al., 2010; Johnston et al., 2018), or restoration of juvenile habitat (Camp et al., 2020).

5. Consequences of mis-specifying the functional form of M in stock assessments

Accepting the GLIM as the best general representation of size- and age-dependent M, it is useful to consider the implications of mis-specifying size- or age-dependent M in population modeling and stock assessment applications. While many general ecological implications of size-dependence in natural mortality rates hold regardless of the precise scaling (Andersen, 2019), the purpose of modeling such relationships in stock assessment applications is to derive quantitative estimates of stock status and evaluate the effects of alternative management measures. In this case, the precise quantitative characterization of the size- and age-dependent M matters. Use of models that differ substantially from the GLIM is likely to lead to systematic biases in assessments.

Even moderate mis-specification of size- and age-dependent M can have significant quantitative consequences for assessments. For example, a stock assessment for South Atlantic greater amberjack shows that the fishery has been overfished for much of its recent history and only just recovered when using the (Hoenig-adjusted) Lorenzen M (Fig. 5) but suggest that the fishery is underexploited and can be expanded when using the Charnov M (SEDAR, 2020). This is not surprising given the substantial differences in juvenile natural mortality rates between the two models (Fig. 5). Another example is the bigeye tuna *Thunnus obesus* fisheries assessment for the Eastern Pacific Ocean (Punt et al., 2019). Expansion of a purse seine fishery for smaller and younger fish was associated with a concurrent, apparent recruitment regime shift estimated by the stock assessment model to account for the additional removals which did not appear to affect catch rates of older tuna in the longline fishery. The apparent regime shift disappeared when a traditionally used age-dependent mortality schedule was replaced the length-inverse Lorenzen M, which predicted higher juvenile natural mortality rates and thus reduced the impact of expanding purse seine catches on recruitment estimates. These two examples illustrate how even moderate differences between alternative size and age-dependent M models can have significant consequences.

The traditional constant mortality assumption in fisheries models is a mathematically convenient simplification, but is likely to mis-specify the underlying mortality pattern in most cases. Fortunately, this mis-specification often has only mild consequences (Thorson and Prager, 2011; Deroba and Schueller, 2013; Punt et al., 2021). Compared with a lifetime pattern of declining natural mortality, assuming constant mortality at an average level for the recruited stock leads to an overestimation of the impact of harvesting young fish and an underestimation of the impact of harvesting old fish. Switching to the GLIM therefore will allow for greater sustainable harvest of smaller/younger individuals but strengthen the case for protecting large/old individuals (Ahrens et al., 2020). The biases introduced by using a constant M are likely greatest in magnitude and can have wide-ranging effects on model performance when fish are harvested over a wide range of sizes including juveniles, when this harvesting is carried out by fleets of very different selectivity, or when attempting to estimate dome-shaped selectivity curves.

As discussed above, senescence is likely to be delayed and may be minimal for many iteroparous fish populations and is not commonly included in either the GLIM or the traditional constant M model. Moreover, age classes in which senescence may be expressed are likely to account for only a small fraction of abundance and spawning biomass. Therefore, accounting for actuarial senescence may not be a priority for routine stock assessment and fisheries modeling, but of course the mortality model will be mis-specified if the population does show substantial senescence. The broad consequences of this mis-specification are that the abundance of old and large fish and their spawning contribution would be overestimated (Ahti et al., 2021). In addition, there are very specific concerns in the stock assessment realm regarding the confounding of actuarial senescence with dome-shaped gear selectivity patterns when senescence is present but not modeled (Thompson, 1994; He et al., 2011).

Finally, not accounting for density-dependence in early juveniles when it is indeed present is a mis-specification of the M model or the recruitment assumption (Lorenzen and Camp, 2019). This violation of assessment model assumptions can lead to substantial bias in the estimation of stock size, stock status, and recovery potential (Forrest et al.,

2013).

6. Conclusions

Current biological understanding, empirical analyses, and theoretical models of natural mortality in fish populations can be integrated into a coherent, generalized length-inverse mortality (GLIM) paradigm. The GLIM, whether implemented in closed functional form or through multi-species modeling of predation and residual mortality, is already being operationalized in many fisheries assessments and should be more widely adopted as a standard for natural mortality modeling, effectively replacing the traditional 'constant M axiom'. While understanding and quantifying natural mortality in fish populations remains a complex and challenging problem (Pope et al., 1991; Plagányi et al., 2022), the GLIM paradigm synthesizes key patterns in a way that is informed by biology, empirically supported, and easily operationalized for stock assessments. Consequences of mis-specifying the functional form of natural mortality are moderate in many cases but can be quite substantial in others.

Further research is indicated in three areas. First, it would be highly desirable (if not necessarily possible) to establish predictors for where and when senescence effects are likely to be significant and need to be considered as modifications of the length-inverse model. Second, exploring and reconciling systematic differences between natural mortality patterns derived from multi-species and ecosystem models and those measured empirically in field populations is likely to yield insights into the modeling of residual mortality and improve mortality predictions overall. Third, further development and testing of density-dependent mortality models for early juveniles would provide a more reliable basis for explicit modeling of these life stages in cases where that is required.

CRediT authorship contribution statement

Kai Lorenzen: Conceptualization, Formal analysis, Writing – original draft, Writing – review & editing.

Declaration of Competing Interest

The author declares that he has no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability

Data will be made available on request.

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References

Abella, A.J., Caddy, J.F., Serena, F., 1997. Do natural mortality and availability decline with age? An alternative yield paradigm for juvenile fisheries, illustrated by the hake Merluccius merluccius fishery in the Mediterranean. Aquat. Living Resour. 10, 257–269.

Adams, G.D., Holsman, K.K., Barbeaux, S.J., Dorn, M.W., Ianelli, J.N., Spies, I., Steward, I.J., Punt, A.E., 2022. An ensemble approach to understand predation mortality for groundfish in the Gulf of Alaska. Fish. Res. 251, 106303.

Ahrens, R., Allen, M.S., Walters, C., Arlinghaus, R., 2020. Saving large fish through harvest slots outperforms the classical minimum-length limit when the aim is to achieve multiple harvest and catch-related fisheries objectives. Fish Fish. 21, 483–510.

Ahti, P.A., Uusi-Heikkilä, S., Marjomäki, T.J., Kuparinen, A., 2021. Age is not just a number–Mathematical model suggests senescence affects how fish populations respond to different fishing regimes. Ecol. Evol. 11, 13363–13378.

Andersen, K.H., 2019. Fish Ecology, Evolution and Exploitation: A New Theoretical Synthesis. Princeton Monographs in Population Biology 62. Princeton University Press, Princeton, N.J, p. 257.

Andersen, K.H., 2020. Size-based theory for fisheries advice. ICES J. Mar. Sci. 77, 2445–2455.

Andersen, K.H., Jacobsen, N.S., Jansen, T., Beyer, J.E., 2017. When in life does density dependence occur in fish populations? Fish Fish 18, 656–667.

Andersen, K.P., Ursin, E., 1977. A multispecies extension to the Beverton and Holt theory of fishing, with accounts of phosphorus circulation and primary production. Meddr. Danm. Fisk. -Og. Havunders. 7, 319–435.

Brodziak, J., Ianelli, J., Lorenzen, K., Methot, R. (Eds.), 2011. Estimating natural mortality in stock assessment applications. NOAA Tech. Memo. NMFS-F/SPO-119.

Anderson, J.T., 1988. A review of size dependent survival during pre-recruit stages of fishes in relation to recruitment. J. Northw. Atl. Fish. Sci. 8, 55–66.

Arendt, J.D., 1997. Adaptive intrinsic growth rates: an integration across taxa. Q. Rev. Biol. 72, 149–177.

Barlow, J., Boveng, P., 1991. Modeling age-specific mortality for marine mammal populations. Mar. Mammal. Sci. 7, 50–65.

Beverton, R.J., Hylen, A., Østvedt, O.J., Alvsvaag, J., Iles, T.C., 2004. Growth, maturation, and longevity of maturation cohorts of Norwegian spring-spawning herring. ICES J. Mar. Sci. 61, 165–175.

Berkeley, S.A., Hixon, M.A., Larson, R.J., Love, M.S., 2004. Fisheries sustainability via protection of age structure and spatial distribution of fish populations. Fisheries 29, 23–32.

Beverton, R.J.H., Holt, S.J., 1957. On the Dynamics of Exploited Fish Populations. Her Majesty's Stationery Office, London, UK, p. 553.

Beverton, R.J.H., Holt, S.J., 1959. A Review of the Lifespans and Mortality Rates of Fish in Nature, and Their Relation to Growth and Other Physiological Characteristics. In Ciba Foundation Symposium-The Lifespan of Animals (Colloquia on Ageing), Volume 5. John Wiley and Sons, Chichester, UK, pp. 142–180.

Beyer, J.E., 1989. Recruitment stability and survival - simple size-specific theory with examples from the early life dynamics of marine fish. Dana 7, 45–147.

Billerbeck, J.M., Lankford, T.E., Conover, D.O., 2001. Evolution of intrinsic growth and energy acquisition rates. I. Trade-offs with swimming performance in *Menidia menidia*. Evolution 55, 1863–1872.

Blanchard, J.L., Jennings, S., Law, R., Castle, M.D., McCloghrie, P., Rochet, M.J., Benoît, E., 2009. How does abundance scale with body size in coupled size-structured food webs? J. Anim. al Ecol. 78, 270–280.

Caddy, J.F., 1991. Death rates and time intervals: is there an alternative to the constant natural mortality axiom? Rev. Fish. Biol. Fish. 1, 109–138.

Caddy, J.F., Abella, A.J., 1999. Reconstructing reciprocal M vectors from length cohort analysis (LCA) of commercial size frequencies of hake, and fine mesh trawl surveys over the same grounds. Fish. Res. 41, 169–175.

Cagan, A., Baez-Ortega, A., Brzozowska, N., et al., 2022. Somatic mutation rates scale with lifespan across mammals. Nature 604, 517–524.

Camp, E.V., Larkin, S.L., Ahrens, R.N., Lorenzen, K., 2017. Trade-offs between socioeconomic and conservation management objectives in stock enhancement of marine recreational fisheries. Fish. Res. 186, 446–459.

Camp, E.V., Lorenzen, K., Taylor, M.D., 2020. Impacts of habitat repair on a spatially complex fishery. Estuar. Coast. Shelf Sci. 244, 106102.

Carlson, S.M., Hilborn, R., Hendry, A.P., Quinn, T.P., 2007. Predation by bears drives senescence in natural populations of salmon. PloS One 2, e1286.

Carnes, B.A., Olshansky, S.J., Grahn, D., 1996. Continuing the search for a law of mortality. Popul. Dev. Rev. 22, 231–264.

Carnes, B.A., Holden, L.R., Olshansky, S.J., Witten, M.T., Siegel, J.S., 2006. Mortality partitions and their relevance to research on senescence. Biogerontology 7, 183–198.

Charnov, E.L., Gislason, H., Pope, J.G., 2013. Evolutionary assembly rules for fish life histories. Fish Fish 14, 213–224.

Chen, S., Watanabe, S., 1989. Age dependence of natural mortality coefficient in fish population dynamics. Nippon Suisan Gakkaishi 55, 205–208.

Christiansen, J.S., Præbel, K., Siikavuopio, S.I., Carscadden, J.E., 2008. Facultative semelparity in capelin *Mallotus villosus* (Osmeridae)-an experimental test of a life history phenomenon in a sub-arctic fish. J. Exp. Mar. Biol. Ecol. 360, 47–55.

Coggins, L.G., Pine, W.E., Walters, C.J., Martell, S.J.D., 2006. Age-structured mark–recapture analysis: a virtual-population-analysis-based model for analyzing age-structured capture–recapture data. New Am. J. Fish. Manag. 26, 201–205.

Craig, J.F., 1985. Aging in fish. Can. J. Zool. 63, 1–8.

Curti, K.L., Collie, J.S., Legault, C.M., Link, J.S., 2013. Evaluating the performance of a multispecies statistical catch-at-age model. Can. J. Fish. Aquat. Sci. 70, 470–484.

Deroba, J.J., Schueller, A.M., 2013. Performance of stock assessments with misspecified age-and time-varying natural mortality. Fish. Res. 146, 27–40.

Dickie, L.M., Kerr, S.R., Boudreau, P.R., 1987. Size-dependent processes underlying regularities in ecosystem structure. Ecol. Monogr. 57, 233–250.

Dureuil, M., Aeberhard, W.H., Burnett, K.A., Hueter, R.E., Tyminski, J.P., Worm, B., 2021. Unified natural mortality estimation for teleosts and elasmobranchs. Mar. Ecol. Prog. Ser. 667, 113–129.

Engelman, M., Seplaki, C.L., Varadhan, R., 2017. A quiescent phase in human mortality? Exploring the ages of least vulnerability. Demography 54, 1097–1118.

Forrest, R.E., McAllister, M.K., Martell, S.J., Walters, C.J., 2013. Modelling the effects of density-dependent mortality in juvenile red snapper caught as bycatch in Gulf of Mexico shrimp fisheries: implications for management. Fish. Res. 146, 102–120.
Fuiman, L.A., Magurran, A.E., 1994. Development of predator defences in fishes. Rev. Fish. Biol. Fish. 4, 145–183.
Gazey, W.J., Gallaway, B.J., Cole, J.G., Fournier, D.A., 2008. Age composition, growth, and density-dependent mortality in juvenile red snapper estimated from observer data from the Gulf of Mexico penaeid shrimp fishery. N. Am. J. Fish. Manag. 28, 1828–1842.
Gerhard, G.S., Kauffman, E.J., Wang, X., Stewart, R., Moore, J.L., Kasales, C.J., Demidenko, E., Cheng, K.C., 2002. Life spans and senescent phenotypes in two strains of Zebrafish (*Danio rerio*). Exp. Gerontol. 37, 1055–1068.
Gislason, H., Pope, J.G., Rice, J.C., Daan, N., 2008. Coexistence in North Sea fish communities: implications for growth and natural mortality. ICES J. Mar. Sci. 65, 514–530.
Gislason, H., Daan, N., Rice, J.C., Pope, J.G., 2010. Size, growth, temperature and the natural mortality of marine fish. Fish Fish. 11, 149–158.
Haddon, M., 2011. Modelling and Quantitative Methods in Fisheries, second ed. CRC Press, Boca Raton.
Hall, S.J., Collie, J.S., Duplisea, D.E., Jennings, S., Bravington, M., Link, J., 2006. A length-based multispecies model for evaluating community responses to fishing. Can. J. Fish. Aquat. Sci. 63, 1344–1359.
Harden-Jones, F.R., 1968. Fish Migration. Arnold, London.
Hazlerigg, C.R.E., Lorenzen, K., Thorbek, P., Wheeler, J.R., Tyler, C.R., 2012. Density-dependent processes in the life history of fishes: evidence from laboratory populations of zebrafish *Danio rerio*. PLOS One 7 (5), e37550.
He, X., Ralston, S., MacCall, A.D., 2011. Interactions of age-dependent mortality and selectivity functions in age-based stock assessment models. Fish. Bull. 109, 198–216.
Hendry, A.P., Morbey, Y.E., Berg, O.K., Wenburg, J.K., 2004. Adaptive variation in senescence: reproductive lifespan in a wild salmon population. Proc. Roy. Soc. Lond. B 271, 259–266.
Hervas, S., Lorenzen, K., Shane, M.A., Drawbridge, M.A., 2010. Quantitative assessment of a white seabass (*Atractoscion nobilis*) stock enhancement program in California: post-release dispersal, growth and survival. Fish. Res. 105, 237–243.
Hilborn, R., Walters, C.J., 1992. Quantitative Fisheries Stock Assessment: Choice, Dynamics and Uncertainty. Chapman & Hall,, London, UK, p. 570.
Hoenig, J.M., 1983. Empirical use of longevity data to estimate mortality rates. Fish. Bull. 82, 898–903.
Hollowed, A.B., Ianelli, J.N., Livingston, P.A., 2000. Including predation mortality in stock assessments: a case study for Gulf of Alaska walleye pollock. ICES J. Mar. Sci. 57, 279–293.
Holsman, K.K., Ianelli, J., Aydin, K., Punt, A.E., Moffitt, E.A., 2016. A comparison of fisheries biological reference points estimated from temperature-specific multi-species and single-species climate-enhanced stock assessment models. Deep Sea Res. II 134, 360–378.
Hutchings, J.A., 1994. Age-and size-specific costs of reproduction within populations of brook trout, *Salvelinus fontinalis*. Oikos 70, 12–20.
ICCAT (International Commission for the Conservation of Atlantic Tunas) 2018. Report of the 2018 ICCAT Bigeye Tuna Stock Assessment Meeting. ICCAT, Madrid, Spain. ⟨https://www.iccat.int/Documents/SCRS/DetRep/BET_SA_ENG.pdf⟩.
Johnston, F.D., Allen, M.S., Beardmore, B., Riepe, C., Pagel, T., Hühn, D., Arlinghaus, R., 2018. How ecological processes shape the outcomes of stock enhancement and harvest regulations in recreational fisheries. Ecol. Appl. 28, 2033–2054.
Jørgensen, C., Holt, R.E., 2013. Natural mortality: its ecology, how it shapes fish life histories, and why it may be increased by fishing. J. Sea Res 75, 8–18.
Koopman, J.J., Wensink, M.J., Rozing, M.P., van Bodegom, D., Westendorp, R.G., 2015. Intrinsic and extrinsic mortality reunited. Exp. Gerontol. 67, 48–53.
Kuparinen, A., Hardie, D.C., Hutchings, J.A., 2012. Evolutionary and ecological feedbacks of the survival cost of reproduction. Evol. Appl. 5, 245–255.
Lorenzen, K., 1996. The relationship between body weight and natural mortality in fish: a comparison of natural ecosystems and aquaculture. J. Fish. Biol. 49, 627–647.
Lorenzen, K., 2000. Allometry of natural mortality as a basis for assessing optimal release size in fish stocking programmes. Can. J. Fish. Aquat. Sci. 57, 2374–2381.
Lorenzen, K., 2005. Population dynamics and potential of fisheries stock enhancement: practical theory for assessment and policy analysis. Philos. Trans. Roy. Soc. Lond. B 260, 171–189.
Lorenzen, K., 2006. Population management in fisheries enhancement: gaining key information from release experiments through use of a size-dependent mortality model. Fish. Res. 80, 19–27.
Lorenzen, K., 2008. Fish population regulation beyond 'stock and recruitment': the role of density-dependent growth in the recruited stock. Bull. Mar. Sci. 83, 181–196.
Lorenzen, K., Camp, E.V., 2019. Density-dependence in the life history of fishes: when is a fish recruited? Fish. Res. 217, 5–10.
Lorenzen, K., Beveridge, M.C.M., Mangel, M., 2012. Cultured fish: integrative biology and management of domestication and interactions with wild fish. Biol. Rev. 87, 639–660.
Lorenzen, K., Camp, E.V., Garlock, T.M., 2022. Natural mortality and body size in fish populations. Fish. Res. 252, 106327.
Lucas, M., Baras, E., 2001. Migration of freshwater fishes. Blackwell Science, London.
Magnusson, K.G., 1995. An overview of the multispecies VPA—theory and applications. Rev. Fish. Biol. Fish. 5, 195–212.
Mangel, M., 2008. Environment, damage and senescence: modelling the life-history consequences of variable stress and caloric intake. Funct. Ecol. 22, 422–430.
Mangel, M., Abrahams, M.V., 2001. Age and longevity in fish, with consideration of the ferox trout. Exp. Gerontol. 36, 765–790.
Mangel, M., Brodziak, J., DiNardo, G., 2010. Reproductive ecology and scientific inference of steepness: a fundamental metric of population dynamics and strategic fisheries management. Fish Fish. 11, 89–104.
Maunder, M.N., Piner, K.R., 2015. Contemporary fisheries stock assessment: many issues still remain. ICES J. Mar. Sci. 72, 7–18.
McCoy, M.W., Gillooly, J.F., 2008. Predicting natural mortality rates of plants and animals. Ecol. Let. 11, 710–716.
McGurk, M.D., 1986. Natural mortality of marine pelagic fish eggs and larvae: role of spatial patchiness. Mar. Ecol. Prog. Ser. 34, 227–242.
McGurk, M.D., 1987. Natural mortality and spatial patchiness: reply to Gulland. Mar. Ecol. Prog. Ser. 39, 201–206.
McGurk, M.D., 1996. Allometry of marine mortality of Pacific salmon. Fish. Bull. 41, 77–88.
McKechnie, S., Pilling, G., Hampton. J., 2017. Stock assessment of bigeye tuna in the western and central Pacific Ocean. Western and Central Pacific Fisheries Commission. WCPFC Document WCPFC-SC13–2017/SA-WP-05.
Methot, R.D., Wetzel, C.R., 2013. Stock synthesis: a biological and statistical framework for fish stock assessment and fishery management. Fish. Res. 142, 86–99.
Myers, R.A., Cadigan, N.G., 1993. Density-dependent juvenile mortality in marine demersal fish. Can. J. Fish. Aquat. Sci. 50, 1576–1590.
Pauly, D., 1980. On the interrelationships between natural mortality, growth parameters, and mean environmental temperature in 175 fish stocks. ICES J. Mar. Sci. 39, 175–192.
Peterson, I., Wroblewski, J.S., 1984. Mortality rate of fishes in the pelagic ecosystem. Can. J. Fish. Aquat. Sci. 41, 1117–1120.
Plagányi, É.E., Blamey, L.K., Rogers, J.G., Tulloch, V.J., 2022. Playing the detective: using multispecies approaches to estimate natural mortality rates. Fish. Res. 249, 106229.
Pope, J.G., 1991. The ICES Multispecies Assessment Working Group: evolution, insights and future problems. ICES Mar. Sci. Symp. 193, 22–33.
Pope, J.G., Gislason, H., Rice, J.C., Daan, N., 2021. Scrabbling around for understanding of natural mortality. Fish. Res. 240, 105952.
Powers, J.E., 2014. Age-specific natural mortality rates in stock assessments: size-based vs. density-dependent. ICES J. Mar. Sci. 71, 1629–1637.
Punt, A.E., Cadrin, S., Fu, D., Lorenzen, K., Methot, R., Piner, K., Walter, J., 2019. 2nd Review of the Stock Assessment of Bigeye Tuna in the Eastern Pacific Ocean. Interamerican Tropical Tuna Commission Special Report, WSBET-02.
Punt, A.E., Dunn, A., Elvarsson, B.Þ, Hampton, J., Hoyle, S.D., Maunder, M.N., Methot, R. D., Nielsen, A., 2020. Essential features of the next-generation integrated fisheries stock assessment package: a perspective. Fish. Res. 229, 105617.
Punt, A.E., Castillo-Jordán, C., Hamel, O.S., Cope, J.M., Maunder, M.N., Ianelli, J.N., 2021. Consequences of error in natural mortality and its estimation in stock assessment models. Fish. Res. 233, 105759.
Purchase, C.F., Rooke, A.C., Gaudry, M.J., Treberg, J.R., Mittell, E.A., Morrissey, M.B., Rennie, M.D., 2022. A synthesis of senescence predictions for indeterminate growth, and support from multiple tests in wild lake trout. Proc. Roy. Soc. B 289, 20212146.
Quinn, T.J., Deriso, R.B., 1999. Quantitative Fish Dynamics. Oxford University Press.
Reznick, D., Ghalambor, C., Nunney, L., 2002. The evolution of senescence in fish. Mech. Ageing Dev. 123, 773–789.
Rindorf, A., van Deurs, M., Howell, D., Andonegi, E., Berger, A., Bogstad, B., Cadigan, N., Elvarsson, B.P., Hintzen, N., Roland, M.S., Taylor, M., Trijoulet, V., van Kooten, T., Zhang, F., Collie, J., 2022. Strength and consistency of density dependence in marine fish productivity. Fish Fish 23, 812–828.
Rose, K.A., Cowan Jr., J.H., Winemiller, K.O., Myers, R.A., Hilborn, R., 2001. Compensatory density dependence in fish populations: importance, controversy, understanding and prognosis. Fish Fish. 2, 293–327.
SEDAR, Southeast Data, Assessment and Reviews, 2018. SEDAR 52 Stock Assessment Report: Gulf of Mexico Red Snapper. SEDAR, North Charleston, SC. ⟨https://sedarweb.org/docs/sar/S52_Final_SAR_v2.pdf⟩.
Southeast Data SEDAR Assessment and Reviews SEDAR 59 Stock Assessment Report: South Atlantic Greater Amberjack 2020 SEDAR North Charleston, SC.(https://sedarweb.org/docs/sar/SEDAR59_fullSAR_3.31.2020.pdf).
Siler, W., 1979. A competing-risk model for animal mortality. Ecol 60, 750–757.
Simpfendorfer, C.A., Bonfil, R., Latour, R.J., Musick, J.A., Bonfil, R., 2005. Mortality estimation. FAO Fish. Tech. Pap. 474, 127–142.
Sogard, S.M., 1997. Size-selective mortality in the juvenile stage of teleost fishes: a review. Bull. Mar. Sci. 60, 1129–1157.
Spence, R., Gerlach, G., Lawrence, C., Smith, C., 2008. The behaviour and ecology of the zebrafish, *Danio rerio*. Biol. Rev. 83, 13–34.
Then, A.Y., Hoenig, J.M., Hall, N.G., Hewitt, D.A., 2015. Evaluating the predictive performance of empirical estimators of natural mortality rate using information on over 200 fish species. ICES J. Mar. Sci. 72, 82–92.
Thompson, G.G., 1994. Confounding of gear selectivity and the natural mortality rate in cases where the former is a nonmonotone function of age. Can. J. Fish. Aquat. Sci. 51, 2654–2664.
Thorpe, J.E., 2004. Life history responses of fishes to culture. J. Fish. Biol. 65, 263–285.
Thorson, J.T., Prager, M.H., 2011. Better catch curves: incorporating age-specific natural mortality and logistic selectivity. Trans. Am. Fish. Soc. 140, 356–366.
Trippel, E.A., Butts, I.A., Babin, A., Neil, S.R., Feindel, N.J., Benfey, T.J., 2014. Effects of reproduction on growth and survival in Atlantic cod, *Gadus morhua*, assessed by comparison to triploids. J. Exp. Mar. Biol. Ecol. 451, 35–43.
Tsou, T.S., Collie, J.S., 2001. Estimating predation mortality in the Georges Bank fish community. Can. J. Fish. Aquat. Sci. 58, 908–922.
Tyrrell, M.C., Link, J.S., Moustahfid, H., Overholtz, W.J., 2008. Evaluating the effect of predation mortality on forage species population dynamics in the Northeast US

continental shelf ecosystem using multispecies virtual population analysis. ICES J. Mar. Sci. 65, 1689–1700.
Tyrrell, M.C., Link, J.S., Moustahfid, H., 2011. The importance of including predation in fish population models: implications for biological reference points. Fish. Res. 108, 1–8.
Vetter, E.F., 1988. Estimation of natural mortality in fish stocks: a review. Fish. Bull. 86, 25–43.
Walters, C.J., Martell, S.J., 2004. Fisheries Ecology and Management. Princeton University Press.
Werner, E.E., 1986. Species interactions in freshwater fish communities. In: Diamond, J., Case, T. (Eds.), In Community Ecology. Harper & Row, New York, pp. 344–358.
Woodhead, A.D., 1998. Aging, the fishy side: an appreciation of Alex Comfort's studies. Exp. Gerontol. 33, 39–51.
Xia, C., Møller, A.P., 2022. An explanation for negligible senescence in animals. Ecol. Evol. 12, e8970.