Modelling fishing-induced adaptations and consequences for natural mortality

Christian Jørgensen and Øyvind Fiksen

Abstract: When trade-offs involving predation and mortality are perturbed by human activities, behaviour and life histories are expected to change, with consequences for natural mortality rates. We present a general life history model for fish in which three common relationships link natural mortality to life history traits and behaviour. First, survival increases with body size. Second, survival declines with growth rate due to risks involved with resource acquisition and allocation. Third, fish that invest heavily in reproduction suffer from decreased survival due to costly reproductive behaviour or morphology that makes escapes from predators less successful. The model predicts increased natural mortality rate as an adaptive response to harvesting. This extends previous models that have shown that harvesting may cause smaller body size, higher growth rates, and higher investment in reproduction. The predicted increase in natural mortality is roughly half the fishing mortality over a wide range of harvest levels and parameter combinations such that fishing two fish kills three after evolutionary adaptations have taken place.

Résumé : Lorsque les compromis entre la prédation et la mortalité sont perturbés par les activités humaines, on s'attend à ce que les comportements et les cycles biologiques changent, ce qui affecte les taux de mortalité naturelle. Nous présentons un modèle général de cycle biologique dans lequel trois relations courantes relient la mortalité naturelle aux traits du cycle biologique et au comportement. D'abord, la survie augmente en fonction de la taille corporelle. Ensuite, la survie diminue en fonction du taux de croissance à cause des risques reliés à l'acquisition et l'allocation des ressources. Enfin, les poissons qui investissent beaucoup dans la reproduction ont une survie réduite à cause des coûts associés aux comportements reproducteurs ou à la morphologie qui réduisent le succès de l'évitement des prédateurs. Le modèle prédit un taux de mortalité naturelle accru comme réaction adaptative à la récolte. Cela élargit les modèles antérieurs qui ont montré que la récolte peut produire des tailles corporelles réduites, des taux de croissance plus élevés et un investissement accru dans la reproduction. L'accroissement prédit de la mortalité naturelle correspond en gros à la moitié de la mortalité due à la pêche sur un large éventail d'intensités de récoltes et de combinaisons de paramètres, de telle sorte que la pêche de deux poissons en élimine trois une fois que les adaptations évolutives ont eu lieu.

[Traduit par la Rédaction]

Introduction

Current fishing levels have been suggested to induce evolutionary changes in fish stocks on decadal time scales (Law and Grey 1989; Rijnsdorp 1993; Jørgensen et al. 2007). A reduction in age and size at maturation has been the most widely studied response to increased fishing mortality, partly because suitable data exist (Dieckmann and Heino 2007), but changes in growth rate (Ricker 1981) and reproductive investment (Yoneda and Wright 2004) have also been documented. In this paper, we turn the focus to natural mortality rates. Many of the trade-offs that shape behaviour and life histories involve predation risk. When fish behaviour and life histories change, it is likely that natural mortality rates might change too. Here we investigate the potential consequences of fishing-induced adaptations for natural mortality rates in a model with three general relationships that link survival with life history traits and behaviour.

The first relationship describes how predation typically declines with size in marine systems (Peterson and Wroblewski 1984), scaling with weight as $W^{-0.25}$ or length as $L^{-0.75}$. The probability of encountering a predator large enough to engulf a prey decreases with size. Theoretical approaches that combine principles of mass balance, the allometric scaling of metabolic rate, and mechanics of predation lead to emergent size spectra where predation scales as $W^{-0.25}$ (Sheldon et al. 1972; McGurk 1986; Andersen and Beyer 2006). When maturing at a smaller size, fish spend more of their life under higher levels of size-dependent predation.

Second, foraging often involves exposure to risk when searching for food (Lima and Dill 1990). Schooling species trade between foraging and safety through their position within or distance from the group (Pitcher and Parrish 1993). Fish are mainly visual feeders, and seeing well normally implies increased risk of being seen. Pelagic organ-

Received 9 September 2009. Accepted 23 April 2010. Published on the NRC Research Press Web site at cjfas.nrc.ca on 11 June 2010. J21401

Paper handled by Associate Editor Eric Taylor.

C. Jørgensen[1,2] and Ø. Fiksen. Department of Biology, University of Bergen, Box 7803, N-5020 Bergen, Norway.

[1]Corresponding author (e-mail: christian.jorgensen@bio.uib.no).
[2]Present address: Uni Research, Box 7810, N-5020 Bergen, Norway.

isms can easily modulate risk exposure by vertical migration from well-lit surface layers to dark, deep waters, thus trading foraging success against survival (Clark and Levy 1988). Similar trade-offs between growth and survival can also arise through physiological mechanisms. For example, Atlantic silversides (*Menidia menidia*) that ingest larger meals are poorer swimmers and experience higher rates of predation (Billerbeck et al. 2001; Lankford et al. 2001). The reason is that they use more of their oxygen budget for digestion and therefore have less aerobic scope available for escaping predators (Arnott et al. 2006). This cost accelerates with growth rate (Munch and Conover 2004). Growth can also be enhanced by allocating less resources to processes such as immune defence (Lochmiller and Deerenberg 2000), with negative implications for survival. These diverse growth-related costs all ultimately compromise survivorship.

The third relationship is a trade-off between reproductive investment and survival. Reproduction often involves display behaviour that may attract predator attention (Endler 1987), mate search that may lead to more frequent predator encounters (Kiørboe 2008), and development of large gonads that may reduce swimming performance (Ghalambor et al. 2004).

Natural selection is acting on these relationships simultaneously so that foraging behaviour, growth strategies, sexual maturation, and reproductive investment evolve in concert. Because harvesting essentially is just another form of predation, fishing is expected to cause evolutionary changes in the optimal life histories. Because all the trade-offs described above involve mortality and predation, we expect there to be consequences also for natural mortality. In other words, should fish take higher risks in their foraging behaviour, growth strategy, or reproductive effort now that their longevity is greatly reduced by fishing? Our aim is to predict the expected change in natural mortality rates using life history theory to model fishing-induced evolution.

Materials and methods

We use a relatively simple state-dependent life history model in which optimal life histories are found by optimization. The central mechanisms are energy acquisition and energy allocation, with corresponding decision variables being the risk-taking of the growth strategy (trading off growth versus survival) and energy allocation between somatic growth and reproduction (trading off growth versus reproduction and reproduction versus survival). In the following description, capital letters such as W (weight) and L (length) denote functions, whereas lowercase letters such as $w(a)$ and $l(a)$ denote specific values that these functions take, here at the particular age a.

Life history assumptions

We assume that available resources (R, in equivalents of body mass (grams·year^{-1})) increase with somatic body mass (W, grams) as

$$R = h \cdot W^b \quad (1)$$

where b (dimensionless) is an allometric scaling exponent. The parameter h adjusts net available resources up and down depending on the individual's growth strategy (φ, dimensionless). High φ may represent more risky foraging behaviour (Lima and Dill 1990), elevated digestion at the cost of the ability to escape predators (Billerbeck et al. 2001; Lankford et al. 2001), or reduced allocation to, e.g., immune defence (Lochmiller and Deerenberg 2000). In particular, we assume that h increases asymptotically up to h_{max}:

$$h = \frac{h_{max} \cdot \varphi}{h_{1/2} + \varphi} \quad (2)$$

Here $h_{1/2}$ (dimensionless) is the half-saturation constant. This is analogous to a Holling type II functional response, but where higher risk acceptance of φ leads to higher growth rates through the growth coefficient h in eq. 1. It is reasonable to parameterise increased resource availability in the environment as a lower value for the half-saturation constant $h_{1/2}$ so that a higher growth could be achieved at lower levels of risk acceptance. Following that interpretation, the asymptote h_{max} would be the physiological maximum growth rate with ad libitum food and when no trade-offs that involve predation or mortality would constrain growth rates. The φ is modelled as state-dependent, so that the optimal φ is found independently for each age a (years) and body length l (cm), i.e., $\varphi(a, l)$.

A second state-dependent strategy, also depending on age a and length l, describes resource allocation $\alpha(a, l)$, which divides R between somatic growth and gonads (G, grams):

$$dW/dt = (1 - \alpha) \cdot R \quad (3)$$

$$dG/dt = \alpha \cdot R \quad (4)$$

Length (L, cm) is isometrically related to weight, $W = kL^3$. Gonads are interpreted in a broad sense to include, e.g., mating behaviour and spawning migrations. Growth, mortality, and the accumulation of gonads were modelled as continuous processes, whereas spawning takes place once per year (at the year's end). Conceptually, our approach falls within the class of energy allocation models (e.g., Roff 1983; Kozlowski 1991), and we separate between irreversible structural mass and reversible reserves and gonads as done also by, e.g., de Roos and Persson (2001) and Jones et al. (2002). The growth model is similar to that developed by Lester et al. (2004) and Quince et al. (2008), except that we use an exponent b that deviates from 2/3.

Mortality is split into five categories (all with unit year^{-1}). (*i*) There is a fixed base level of size-independent mortality, M_{fixed}. (*ii*) Size-dependent predation (Peterson and Wroblewski 1984; Andersen and Beyer 2006) declines with body size as $M_{predation}(L) = cL^{-d}$. (*iii*) The φ leads to increased growth-related mortality M_{growth}. Having high φ leads to increased predation mortality: there is thus a trade-off between net ingested resources and survival. We assume that this mortality follows the same size dependence as predation, $M_{growth}(\varphi, L) = \varphi M_{predation}(L)$. (*iv*) Reproduction-related mortality $M_{reproduction}$ increases with reproductive investment and follows the same size dependence as predation. We model this as $M_{reproduction}(Q, L) = M_{predation}(L) \cdot (Q/q_{ref})^p$, where q_{ref} is a reference value at which the mortality from reproduction equals the size-dependent predation component, i.e., at which $M_{reproduction} = M_{predation}$. For $p > 1$, mortality rate accelerates with increasing reproduction as, e.g., observed in

guppies (Ghalambor et al. 2004). (*v*) Finally, we added fishing mortality (F), which may depend on body length. Total mortality rate (Z, year^{-1}) is then $Z(\varphi, Q, L) = M_{\text{fixed}} + M_{\text{predation}}(L) + M_{\text{growth}}(\varphi, L) + M_{\text{reproduction}}(Q, L) + F$. Annual survival probability (S) is thus found as

$$S(A) = \exp\left(-\int_{t=A}^{A+1} Z\,\mathrm{d}t\right) \quad (5)$$

Optimization and implementation

As fitness measure, we used R_0, defined as the expected lifetime production of gonads, i.e., the sum of G over all ages discounted by survival probability until that age. We used the optimization technique of dynamic programming (Houston and McNamara 1999; Clark and Mangel 2000) to find optimal trajectories of growth, survival, and reproduction. This is a process-based approach working from mechanisms to population-level patterns, in contrast to Bayesian and other statistical approaches that identify patterns suggestive of mechanisms at work beginning with data. In our case, these trajectories are specified by the state-dependent strategies $\varphi(a, l)$ and $\alpha(a, l)$ for all combinations of ages a (in steps of 1 year) and lengths l (in steps of 1 cm) that maximize R_0. Maximum age ($a_{\max}$) was set to 30 years after which all individuals die, and maximum length ($l_{\max}$) was set to 200 cm ($a_{\max}$ and $l_{\max}$ are technical limits in the algorithm and should be well beyond the ages and sizes regularly observed to avoid boundary effects).

Equations 3, 4, and 5 allow us to relate body mass at the start of the year to body mass, gonad mass, and survival at the end of the year. The continuous time integrals were solved numerically by dividing each year into J discrete intervals for which size, gonads, and mortality were updated, while the strategies $\alpha(a, l)$ and $\varphi(a, l)$ remained fixed within each year. In the results shown here, we used $J = 24$ (two time steps per month), at which the strategies and individual trajectories were indistinguishable from a (computationally slow) test simulation of $J = 1000$ (full discrete time equations are given in Appendix A). In the current formulation of the model, we did not include seasonal cycles.

Given $w(a)$ and the strategies $\alpha(a, l)$ and $\varphi(a, l)$, we find $w(a + 1)$ from which length at the next age is found as

$$l' = \left(\frac{w(a+1)}{k}\right)^{1/3} \quad (6)$$

As optimization criterion for fitness, we define $v(a, l)$ as the expected future production of gonads until $a_{\max}$ of an individual of length l at age a. By assuming that individuals have no future fitness at $a_{\max}$, the dynamic programming equation shows how reproductive value ($v(a, l)$) at age a and length l can be found through recursive iterations:

$$v(a, l) = \max_{\alpha,\varphi}\{s(a)[g(a+1) + v(a+1, l')]\} \quad (7)$$

Because we use optimization, the predicted life histories are evolutionary end points (examples of optimal strategies are shown in Supplemental Fig. S1[3]). The rest of the results shown are from simulations of an individual following the optimal strategies over its lifetime (initialised at 16 cm and 1 year). In the results, we focus on traits that could be quantified in empirical studies; rather than the hidden process variable α, we therefore present reproductive investment as the gonado-somatic index $Q = G/(W + G)$ (dimensionless).

Parameterisation

We manually tuned the parameters for mortality so that total natural mortality without fishing was 0.2 at $l = 100$ cm for an immature fish and with $M_{\text{predation}}$, M_{growth}, and $M_{\text{reproduction}}$ being of equal magnitude. Resulting parameter values were $b = 0.7$, $d = 0.75$, $k = 0.01$, $c = 2$, $h_{\max} = 5.5$, $h_{1/2} = 0.10 \cdot h_{\max}$, $q_{\text{ref}} = 0.25$, $p = 2$, and $M_{\text{fixed}} = 0.067$. The model's sensitivity to these values was tested varying each parameter value and reporting the effect on the main prediction: the increase in natural mortality as a function of the imposed harvesting mortality. We also tested how a minimum landing size in the fishery or constraints on maximum reproductive investment affected the model's predictions. The sensitivity analysis is shown briefly herein (further details can be found in Supplemental Fig. S2[3]).

Results

The model predicts that natural mortality rates increase with increased harvesting. This result extends the standard expectation for life history responses to fishing, namely, that fish will mature earlier and at smaller sizes (e.g., Law and Grey 1989). Natural mortality increases because life histories evolve towards smaller size, more risky foraging, and elevated reproduction (Figs. 1, 2).

Mortality increases most after maturation

The age-specific response suggests that the effect of these adaptations is strongest from maturation onwards (Fig. 3) for two reasons. First, higher reproductive investment slows growth and fish therefore spend the remainder of their life at a smaller size with higher size-dependent predation rates. Second, intensified reproduction mean larger gonads, which incur reduced survival. As maturation age declines, these effects begin earlier in life.

Fishing two fish kills three

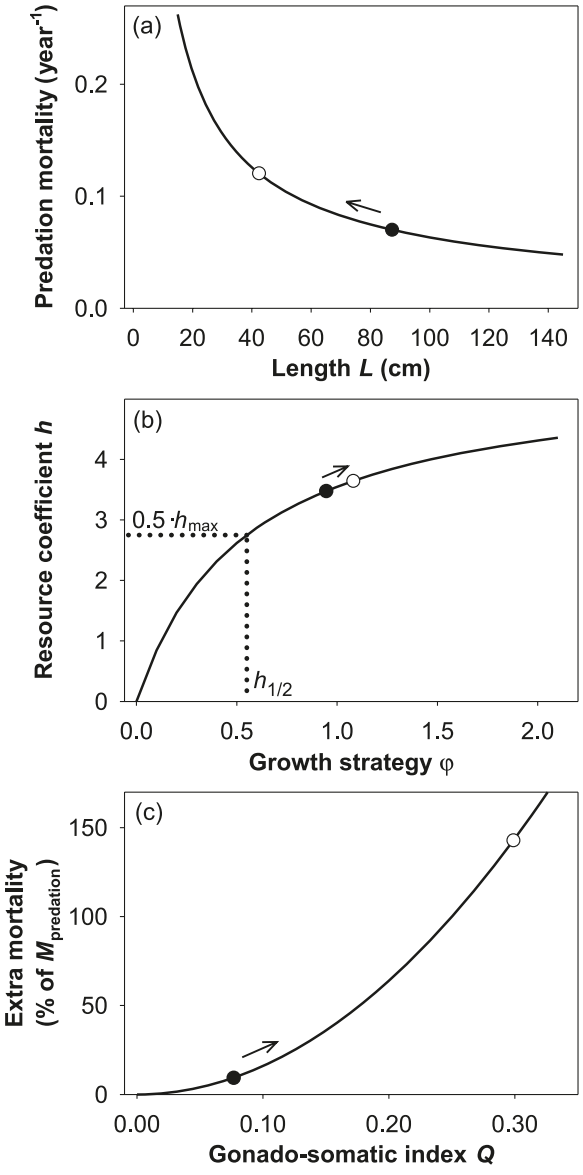
Natural mortality rates increase almost proportionally with harvest rates up to 0.5 year^{-1} and from then on somewhat slower than proportionally (Fig. 4*a*). The extra mortality rate is approximately half the harvest rate. Fishing two fish thus kills three, once life histories and behaviour have adapted to the new harvest regime.

Trends in productivity may mask adaptation

Fishing reduces population size and may weaken density-dependent competition for food. If food abundance increases (in the model by lowering the value of $h_{1/2}$), fish may grow faster and accept less risk through the mechanisms described above, with the overall effect of reducing natural mortality (Fig. 4*b*). However, optimal strategies still accept more natural mortality as harvest rates go up (moving along each line

[3] Supplementary data for this article are available on the Journal web site (http://cjfas.nrc.ca).

Fig. 1. Central assumptions of a life history model that quantifies fisheries-induced effects on natural mortality rates. (*a*) Size-dependent predation mortality declines with size, $M_{predation} \propto W^{-0.25} \propto L^{-0.75}$. Symbols denote size at 12 years for the optimal strategy under no fishing (solid circle) and when harvest rate is 0.5 year^{-1} (open circle). (*b*) The assumed trade-off between growth strategy (φ) and the coefficient h of the net resource availability function. The values for the half-saturation constant ($h_{1/2}$) and half the asymptotic level ($0.5 \cdot h_{max}$) are indicated with dotted lines. Symbols (as in *a*) denote the optimal growth strategy of a 40 cm fish. (*c*) The relationship between extra predation mortality and reproductive investment, quantified as the gonado-somatic index (Q, gonad mass divided by total mass). Symbols (as in *a*) illustrate optimal strategy for a 12-year-old fish.



towards the right). Thus, although natural mortality may fluctuate depending on density dependence and environmental conditions, life history adaptations to fishing will make it fluctuate at a higher level.

A gradual increase in food abundance may reduce natural mortality rate as there is less need for risky foraging and the abundant food may allow larger body sizes. Thus, higher food levels (a move from the broken to the continuous to the dotted lines in Fig. 4*b*) could mask concurrent evolution towards higher natural mortality rates (moving from left to right on each line in Fig. 4*b*). However, if food level fell to original levels, the new adaptations towards riskier behaviour and smaller size would imply rising natural mortality rates. Furthermore, evolutionary changes in behaviour and life histories are likely to have slower dynamics than changes in food abundance. The evolved higher natural mortality rates may therefore prevail for some time even if fishing pressure were reduced. This effect is consistent with the surprisingly slow recovery of many fish stocks that have collapsed from overfishing (Hutchings 2000).

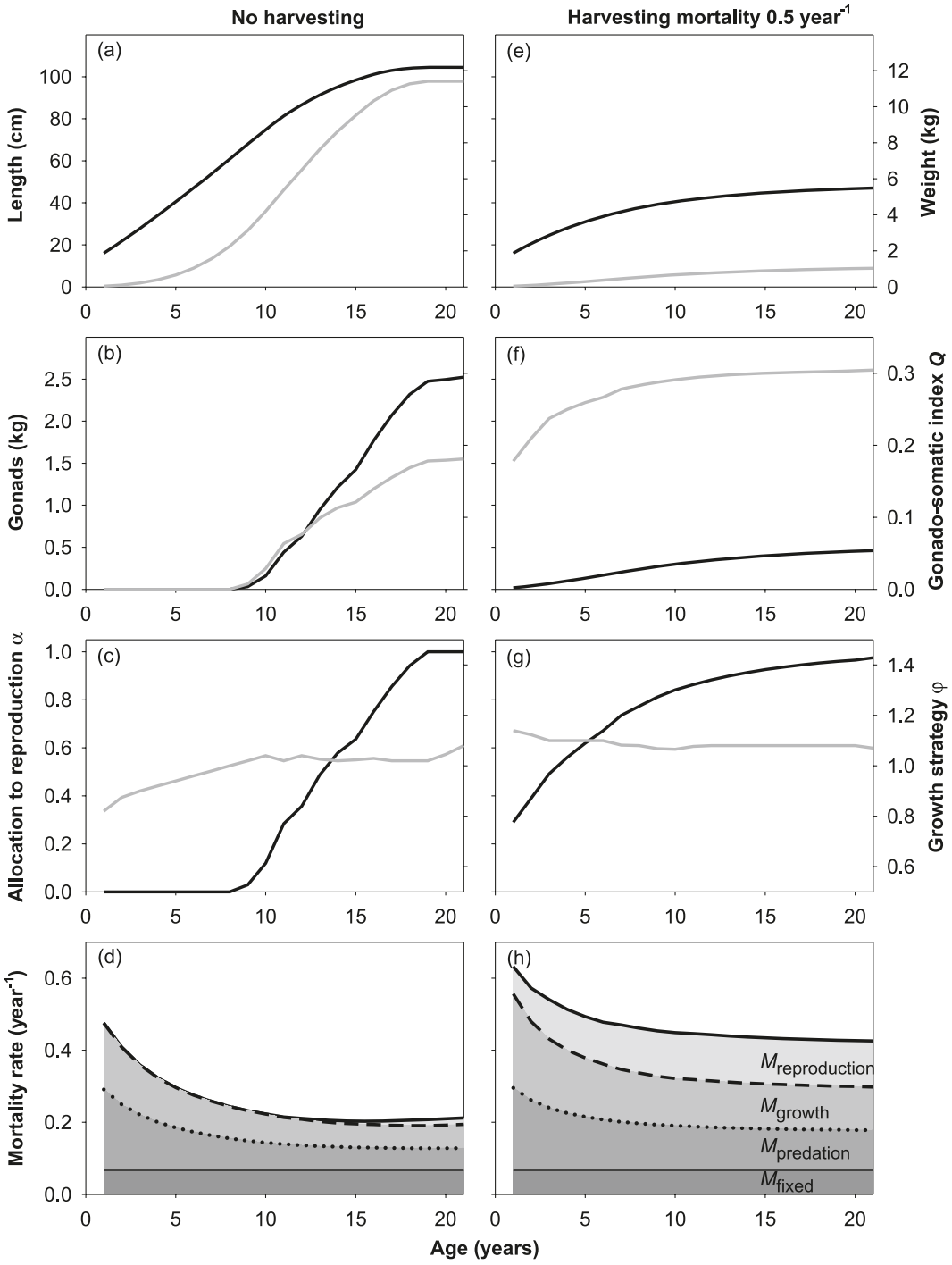
Sensitivity analysis

The prediction of increased natural mortality is robust to variation in parameter values (Fig. 4*c* and Supplementary data[3]). We checked sensitivity by letting central parameters vary from the standard configuration. Although this may alter life history traits such as age, maturation, and growth rates considerably, the response in terms of increased natural mortality rates was much less variable. Almost all trade-offs in the model, and many in nature, involve mortality risk either directly or indirectly. Under fishing, the key life history response is to increase current reproduction (while still alive) at the expense of future reproduction (which may never be realised). Although this can happen through many routes, e.g., increased acquisition, reduced size at maturation, or increased reproductive investment, the common consequence is that survival is sacrificed.

The model is most sensitive to variation in the parameter p, which describes how quickly mortality increases as reproductive investment increases. Especially, the model predicts dramatic consequences for natural mortality if even a small reproductive investment would inflict mortality costs ($p = 1$; Fig. 4*c*). Mechanisms that scale reproduction mortality approximately linearly with reproductive investment likely include mate search, if more intense reproduction requires more matings, and prolonged presence at the spawning grounds, for example, in batch spawners that need to mature batches of eggs successively (Kjesbu et al. 1996). The parameter value of $p = 2$ in the standard scenario leads to less dramatic predictions and takes into account that several survival costs are initially low but accelerate with the intensity of reproduction. Examples of such costs include hydrodynamic drag of bulky body shapes and reduced swimming performance due to competing metabolic needs (Ghalambor et al. 2004; Munch and Conover 2004).

Other parameters to which the model is sensitive are as follows. (*i*) If survival costs only kick in when gonads are large, the model predicts that natural mortality will increase less ($p = 3$). (*ii*) If there is a minimum size limit in the fishery, life histories may evolve to spend more time in size windows of lower mortality. We modelled fisheries selectivity as $Y(L) = [1 + \exp(-u(L - L_{50}))]^{-1}$, with $u = 0.1$ (steepness) and $L_{50} = 40$ (cm) being the length at which there is 50% probability of being harvested; total fishing mortality for a given length is then $F(L) = F_{max} \cdot Y(L)$, where F_{max} is the fishing mortality at sizes where the selectivity curve $Y(L)$ approaches 1 (further minimum size limits are reported

Fig. 2. Individual trajectories of (*a* and *e*) growth in terms of length (bold line, left axis) and weight (shaded line, right axis), (*b* and *f*) reproduction quantified as gonad weight (bold line, left axis) and gonad-somatic index (shaded line, right axis), (*c* and *g*) optimized strategy for allocation (bold line, left axis) and growth strategy (shaded line, right axis), and (*d* and *h*) acquired mortality found through forward simulation of state-dependent life history strategies. The left column (*a–d*) is optimized for no harvest and the right column (*e–h*) represents a harvest rate of 0.5 year^{-1}.

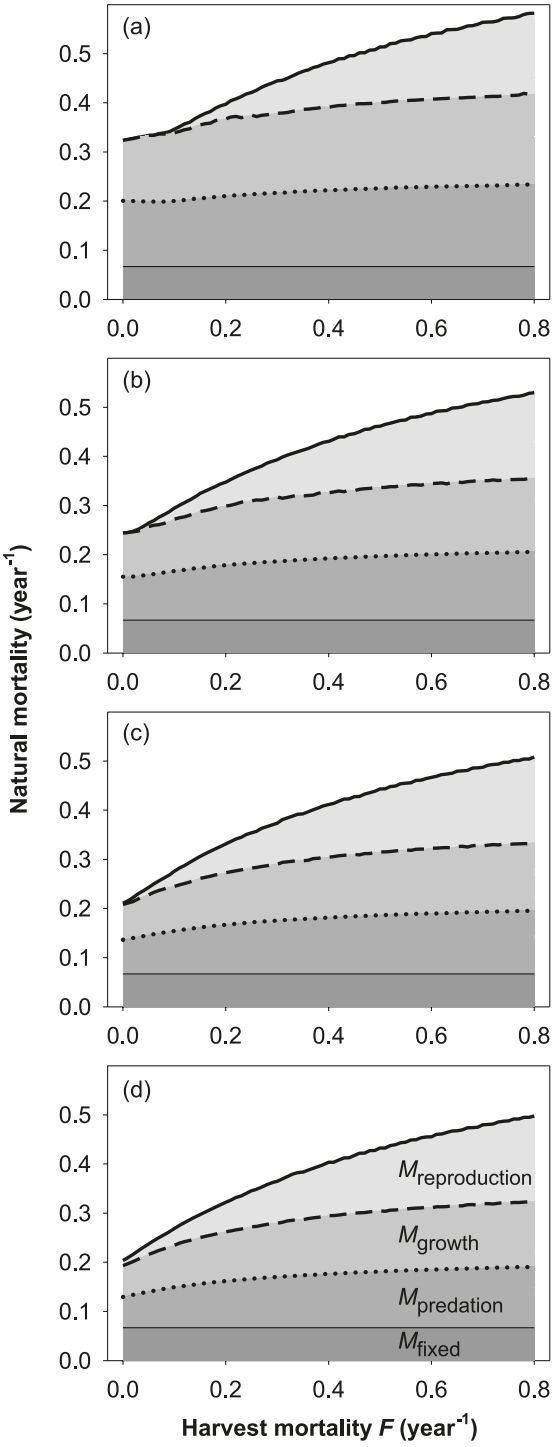


in Supplemental Fig. S2 (*h*), available online[3]). (*iii*) If the magnitude of adaptations are constrained by imposing limits in the model, then the extra natural mortality may reach a ceiling. We imposed a constraint so that the gonado-somatic index (Q) cannot exceed a value of 0.25, which led the extra natural mortality to level out at around 0.1 from $F = 0.2$ onwards.

Discussion

Over the last century, industrial fishing has (*i*) changed the marine community composition towards smaller-sized species at lower trophic levels (Pauly et al. 1998), (*ii*) shifted population distributions towards younger and smaller fish, and (*iii*) caused evolutionary change in life his-

Fig. 3. Age-dependent life history responses to harvesting mortality. The four components of natural mortality rate are shown for ages (*a*) 4, (*b*) 8, (*c*) 12, and (*d*) 16 years for optimal life history strategies under increasing harvesting pressure F (x axis).



tory traits towards earlier maturation at smaller sizes (reviewed in, e.g., Dieckmann and Heino 2007; Jørgensen et al. 2007; Allendorf et al. 2008). In this paper, we focus on the evolutionary implications and used a model to predict that natural mortality will increase as a consequence of earlier maturation at smaller size, higher risk acceptance in foraging behaviour and growth strategies, and intensified reproductive effort.

The changes predicted by our model are rather dramatic. With no harvesting, it predicts a fish life history that matures at age 9 with a gonado-somatic index of 10%–20% and a length at age 12 of 90 cm. In contrast, the predicted life history adapted to a harvesting rate of 0.5 year^{-1} matures at age 1, the gonado-somatic index exceeds 25% already at age 5, and the fish is only 40 cm when it is 12 years old. This difference is comparable with that between a cod and a herring, and it might be unlikely that an evolutionary change of this magnitude will take place in the foreseeable future. To interpret the model, it may therefore help to think of the trade-offs that we assumed as representative of classes of mechanisms. A general problem with models is that they allow for only a few evolving traits, whereas in real species, all traits can evolve simultaneously. In a model, the total selection pressure thus needs to be absorbed by these few traits, whereas there are more degrees of freedom for evolution in the wild. For example, we assumed that increased investment in reproduction would lead to a higher gonado-somatic index, with increased mortality through predation as a consequence. Translated to the wild, this does not necessarily imply that fish will have larger gonads — there are several ways in which fish can achieve higher reproductive investment. For example, the fish may invest more in spawning by prolonging the time used for spawning activity, spend more time and energy migrating between feeding and spawning areas, or engage more in display and mating behaviour. These kinds of changes can all have similar implications for predation rates as our assumed trade-off. An alternative mechanism that links reproduction and survival could be starvation after severe depletion of energy stores due to spawning. In general, starvation tolerance increases with size, which would require a different formulation for the trade-off between reproduction and survival than what we have implemented.

Survival depends on the mortality level (the rate) and the duration of exposure to that mortality. When external mortality (fishing) goes up, adaptations that shorten exposure to that mortality will increase survival more than before, and that extra benefit can be achieved through several trade-offs through which fish accept higher mortality rates to reproduce more earlier in life.

Assumption: predation declines with size

One of our key assumptions is that predation mortality declines with body length according to an allometric scaling relationship of the type $M \propto L^{-d}$. Based on comparisons across species covering a wide size range (Brown et al. 2004), we used a value of $d = 0.75$. However, these types of scaling relationships between species are not necessarily good descriptors of the scaling relationship within a species, which is what we model (Tilman et al. 2004). When we therefore tested for the sensitivity to this central parameter, the model's predicted increase in natural mortality rate was more severe for higher values of d. Even with $d = 0$, however, the model predicted that natural mortality would increase by roughly 25% of the fishing mortality. The value $d = 0$ means that predation does not depend on size, which describes the unlikely situation that a 2 m tuna has the same

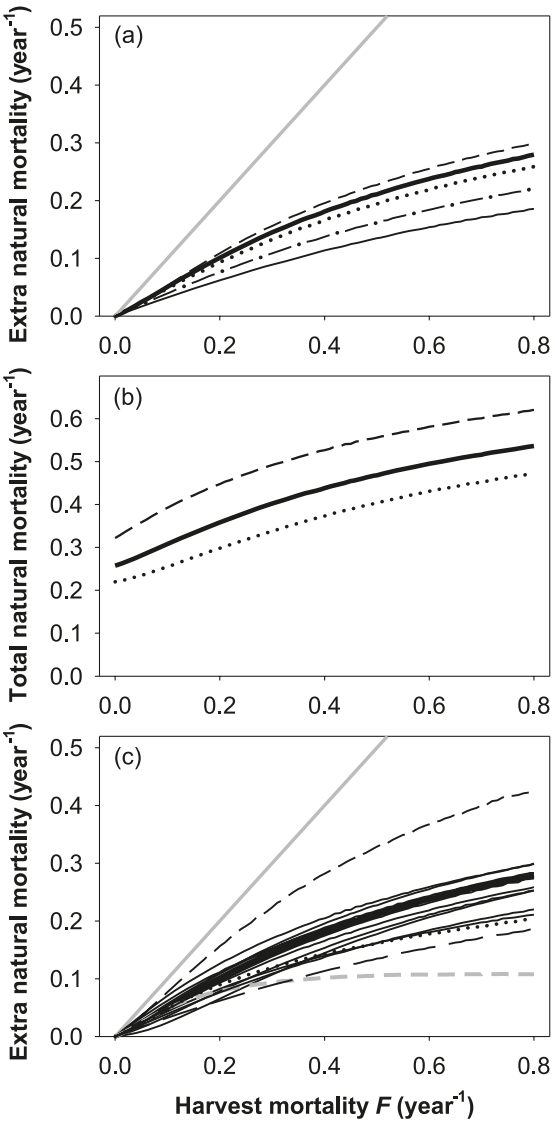


Fig. 4. Total effect on natural mortality in relation to harvest rate, averaged over the first 20 years of life. (*a*) The thick continuous line uses the standard assumption that $d = 0.75$ in the allometric scaling of predation mortality with size, $M \propto L^{-d}$. Sensitivities to this central parameter are shown with the broken ($d = 0.9$), dotted ($d = 0.6$), dash–dotted ($d = 0.3$), and thin lines ($d = 0$); natural mortality was kept constant for a 60 cm fish. The shaded line denotes 1:1. (*b*) Total natural mortality (fishing mortality is excluded) when varying the half-saturation constant $h_{1/2}$ of the growth strategy – resource intake relationship from Fig. 1*b*. The continuous line is for standard parameters. The dotted line has an initial slope that is twice as steep ($h_{1/2} = 0.05 \cdot h_{max}$), representing a situation with abundant and accessible food. Optimal strategies still have higher natural mortality when there is harvest but less than in the standard situation. For the broken line, the initial slope is shallower, representing food shortage ($h_{1/2} = 0.20 \cdot h_{max}$). Increasing food will reduce mortality from broken to continuous to dotted lines. (*c*) Sensitivity analysis (more detailed figures are shown in the online supporting information[3]). The extra natural mortality that results from standard parameters is shown in thick continuous line. In many cases, predictions do not change notably when parameters are varied; this can be seen as the cluster of thin continuous lines around the main prediction (from top to bottom as their effect is at $F = 0.80$: $h_{1/2} = 1.1$; $d = 0.90$, $h_{max} = 4.5$, $q_{ref} = 0.35$, [thick line with standard parameters], $q_{ref} = 0.15$, $b = 0.65$, $h_{max} = 6.5$, $d = 0.60$, $h_{1/2} = 0.28$, $b = 0.75$, $d = 0.3$, $p = 3$). Notable exceptions, discussed in the text, are as follows: $p = 1$ (short-dashed line); a minimum size limit in the fishery of 40 cm (dotted line); $d = 0$ (long-dashed line); and a constraint so that gonads the gonado-somatic index Q cannot exceed a value of 0.25 (shaded broken line).

probability of being eaten per unit time as a 10 mm tuna larvae. However, fish larvae generally have predation rates in the range of 0.05 to 0.5 day^{-1}, or 18 to 180 year^{-1} (McGurk 1986), compared with the standard assumption of adult mortality rate of around 0.2 year^{-1} generally used in fisheries science (Jennings et al. 2001, p. 203). Thus, some sort of decline in predation mortality seems to be supported by data. The exact value of d changes our model's quantitative predictions, but the qualitative pattern is persistent, namely increased natural mortality rate as life histories adapt to fishing.

Example: Canadian cod stocks

The potential implications of evolutionary changes in natural mortality rate can be illustrated with an example from the literature. Many Canadian stocks of Atlantic cod (*Gadus morhua*) were heavily harvested prior to their collapse throughout the 1980s. A fishing moratorium was implemented in the early 1990s, although some fishing continued (Shelton et al. 2006). Despite these severe management restrictions, the stocks have failed to recover. Using survey data from the southern Gulf of St Lawrence stock, Swain and Chouinard (2008) found a temporal trend towards higher natural mortality, with the estimate for recent years being 0.6 year^{-1}, three times higher than the presumed value prior to 1980. Swain and Chouinard (2008) write that "causes of the apparent increase in M are unknown." Applying the insights from our model, however, it seems that several of the mechanisms that lead to higher natural mortality in our model have been observed in the Canadian cod stocks. First, the driver of change in our model is fishing pressure, and fishing mortality rate on these stocks have typically been in the range of 0.3 to 1.0 year^{-1} for decades and, furthermore, increasing in the years leading up to the moratorium (Myers et al. 1997). Second, size and age at maturation have declined in most of the stocks, presumably driven by fishing-induced evolution (Olsen et al. 2005). In our model, high fishing mortality made it optimal to mature early while still alive. Third, size at age has declined for postmature fish, presumably as a response to fishing, at least in the southern Gulf of St. Lawrence stock (Sinclair et al. 2002; Swain and Chouinard 2008). This is again consistent with the model's predictions: early maturation redirects resources towards reproduction and will therefore decelerate growth so that, for all ages after maturation, the fish are of smaller body size. Increasing reproductive investment would have similar effects in reducing size at age of postmature fish (Heino et al. 2008). Fourth, recruitment per unit biomass is higher in recent years than prior to the 1970s (Swain and Chouinard 2008). This is also in accordance with our model, which predicted that reproductive investment would go up.

Thus, all these patterns are consistent with the fishing-induced effects modelled in our analysis, where riskier energy acquisition, earlier maturation, and elevated reproductive investment all bring about higher natural mortality as an adaptive response to cope with increased fishing mortality. It is important to emphasise that there are other nonexclusive explanations for most of these patterns. Many elements of these Canadian ecosystems have changed, and both size-selective fishing and density dependence have been variable in the Gulf of St. Lawrence stock in particular (Sinclair et al. 2002; Swain et al. 2007, 2008). The general lack of recovery of cod in the area has been attributed to, e.g., discarding of young fish (Myers et al. 1997) and regime shifts (Petrie et al. 2009). The increased recruitment in recent years may be due to released density dependence when the spawning population is small, as is commonly observed in many fish stocks, but there has also been a general increase of small fish species in this area that might reflect reduced abundance of large predators (Benoît and Swain 2008). The timing of events may also suggest a role for other factors: for southern Gulf of St. Lawrence cod, maturation age decreased in the 1960s and 1970s, before the decline in size at age, which took place in the 1970s and 1980s. It could still be that maturation age changed first but that an effect of decelerated individual growth only occurred later, in response to an increase in reproductive investment. However, evidence suggests that fisheries size selectivity and density-dependent growth processes have also contributed (Sinclair et al. 2002; Swain et al. 2007).

Our model provides new hypotheses consistent with observed changes in these cod stocks, but the change may also be explained by several alternative ecological mechanisms. It will be interesting to see from future analyses the role that the various hypotheses have played relative to each other. Often these explanations do not exclude each other, and probably ecological and evolutionary processes have worked in concert to produce the final outcome.

Also, we are not aware of any studies that document increased risk-taking in foraging behaviour or increased susceptibility to disease or infection for these stocks. Some indirect evidence could be interpreted in that direction, but the inferences that can be sustained are weak for the moment. For example, Myers et al. (1997) compared output from virtual population analysis with survey data and found that young fish were dying faster than could be predicted. They homed in on discarding as a potential cause, but our model suggests that the increased mortality could come from increased risk-taking in foraging behaviour or by down-regulating, e.g., immune defence. Because these fish were young and mostly immature, it is unlikely that the cause lies with maturation or reproduction.

Implications of increased mortality

In many ways, natural mortality is a loss term that removes fish from the population so that it cannot be harvested (fishermen's perspective) or its existence appreciated (conservationist's perspective). In most cases, natural mortality implies predation. The predator that benefits may be a "dead end" (jellyfish) or a marine mammal that is protected. If the model's predictions are only partly right, adaptations caused by fishing are likely to make mortality greater for the fish resources we value the most.

Quantitatively, the model's predictions were robust to parameter variation. The model predicted that natural mortality would increase by roughly half the fishing mortality. Fishing two fish would thus cause the death of a third one, once adaptations in life history traits and behaviour have taken place.

Perhaps harvest can be done differently to minimize or even reverse the evolutionary consequences (e.g., by modifying the gear's size selectivity; Law 2007; Jørgensen et al. 2009). Or perhaps one cannot harvest without causing such detrimental side effects. Regardless of the long-term consequences, a systemic increase in natural mortality necessitates increased awareness among resource managers to avoid short-term catastrophes. If an increase in natural mortality goes unnoticed in fisheries surveys, stock assessments could systematically overestimate stock size or the stock's recruitment. A dire consequence could be catch quotas that are persistently set too high with an imminent risk of stock collapse.

Earlier models

Links between life history traits and natural mortality have been considered also in earlier models, but those studies have not quantified the consequences of harvest-induced adaptations for natural mortality rates. In a model for Atlantic cod, Hutchings (2005) assumed that postreproductive mortality was higher the earlier the fish became sexually mature. He concluded that a lower maturation age is more likely to cause negative population growth, partly as a consequence of the increased postmature mortality rate. Trade-offs between natural mortality and growth, maturation, or reproduction have also been incorporated in several individual-based models for fish (e.g., Dunlop et al. 2007, 2009*a*; Enberg et al. 2009), but predictions for natural mortality rates have, to our knowledge, not been reported.

Ecosystem perspectives

In a wider ecological perspective, harvesting may have cascading effects for entire ecosystems, where not only predation rates, but also productivity and food availability, might change (Jackson et al. 2001), with potential feedback effects on traits that evolved in the first place. In a whole-lake experiment, harvesting of the prey (charr) relaxed its density-dependent regulation, with the consequence that its predator (trout) was released from stunting and subsequently recovered (Persson et al. 2007). Likewise, harvesting a predator or a prey can bring about evolutionary trait changes in the other (Gårdmark et al. 2003; Matsuda and Abrams 2004; Abrams and Matsuda 2005).

Because harvesting has removed many of the large fish predators from the world's oceans (Pauly et al. 1998), it could be argued that this would lead to reduced natural mortality on their prey. The picture is complicated by the fact that top predators other than fish have shown variable responses. For example, while some marine mammal species have declined, others are prospering and their impact as predators has increased (Clapham et al. 1999; Bowen et al. 2003). In relation to the cod example, grey seals have multiplied in the Northwest Atlantic, and although they may keep

cod populations down, it seems that the reduced predation from cod on many smaller fish species is now compensated for by increased predation from seals and whales (Savenkoff et al. 2007).

How fast may species change?

The method we used, optimization, allows us to find expected evolutionary end points when little is known about heritabilities or rates of change for the manifold mechanisms that relate life history traits to mortality. How fast traits will change in natural populations depends on the type of trait. Traits that are responding directly to environmental or social cues such as behaviour often respond quickest (Lima and Dill 1990; Creel and Christianson 2008). Other traits are contingent on developmental history or are phenotypically plastic and expressed according to the experienced environment; their dynamics is slower but may still change within a life time. Examples of such traits are morphology and physiology, where alternative morphs or acclimatisation may lead to different phenotypes in different environments (Morita and Fukuwaka 2006). Finally, other traits may require genetic evolution over several generations. The rate of fishing-induced evolution has not yet been quantified directly as temporal changes in gene frequencies, so in the meanwhile, we need to rely on indirect evidence from observations on phenotypes (Dunlop et al. 2009*b*). Several forms of fishing-induced changes have taken place on decadal time scales or faster (Ricker 1981; Sharpe and Hendry 2009; see also quantification of evolutionary rates in online appendix to Jørgensen et al. 2007), but especially for observational data from the wild, there might be unknown factors and phenotypic plasticity that contribute to phenotypic trends. When accounting for known environmental trends, the rate of evolution is still fast, often taking place within decades (Grift et al. 2003; Kraak 2007; Swain et al. 2007). Other studies have suggested that evolution may be slower, either using theoretical arguments (Brown et al. 2008; Andersen and Brander 2009) or by arguing that some of the change could be explained by processes that have been omitted from the analysis (many of the arguments were presented in the theme section by Marshall and Browman 2007). Models that treat evolution explicitly suggest rates that are comparable with observed changes (Enberg et al. 2009). At present, it is difficult to conclude how fast fishing-induced evolutionary changes may be. Until alternative environmental factors are put forward that can explain the observed phenotypic trends in many fish stocks, in our view, the most parsimonious explanation is that the similar effect in widespread stocks is at least partly caused by fishing-induced adaptations.

In some cases, life history traits and behaviour are correlated and interact with the fishing activity, which can affect the strength of selection and therefore the rate of evolution. For example, angling removed a bold and fast-growing domesticated strain more rapidly than the control in a whole-lake experiment (Biro and Post 2008). In that experiment, the strongest selection took place on the first day of fishing. In addition to a direct effect on the phenotype composition in the population, it would also have a more delayed and persistent effect as the genes of the survivors are passed on to future generations.

Although many years have now passed since fishing pressure on the Canadian cod stocks was greatly reduced, the absence of fishing mortality does not necessarily imply that reverse evolution back to the original trait distributions may be fast (Law and Grey 1989). In an evolutionary individual-based model that incorporated several of the same trade-offs that we used, reverse evolution of age at maturation, growth rate, and reproductive investment was slower by a factor of 10 or more compared with the fishing-induced evolutionary change (Enberg et al. 2009). Whereas fast-reproducing strategies (early maturation, fast growth, high reproductive investment) have a great advantage when fishing mortality is high, they are nearly as good as the more slowly reproducing strategies when mortality is low, which leads to a characteristic asymmetry in evolutionary rates (Law and Grey 1989).

Predation rarely leaves an observable trace, and time series of fisheries and survey data that could have been used to detect trends over time are typically burdened by temporal variability. It is understandably tempting to focus on the more easily quantifiable measures such as size, age, and other characteristics of the fish that are alive and can be caught in fisheries or surveys. Because morphology, behaviour, physiology, life history strategies, and intra- and interspecific interactions together determine natural mortality, a cross-disciplinary effort is needed to build an understanding of the underlying mechanisms and ecological consequences. A large-scale increase in natural mortality rates induced by fishing will have consequences for fish population dynamics and fisheries productivity and raises challenges for fisheries management and policies for sustainable harvesting. Apart from the Canadian cod, a systematic increase in natural mortality has to our knowledge not been documented. Despite the difficulties in quantifying natural mortality, one would expect that an increase of the magnitude that our model predicts would not go unnoticed, as it could lead to a persistent lack of fit in population and assessment models in regular use today.

Acknowledgements

We thank Dag L. Aksnes, Katja Enberg, and Marc Mangel for comments and Doug Swain and an anonymous researcher for insightful and constructive reviews. Part of the research and writing was conducted while C.J. was visiting the Mangel lab at the University of California Santa Cruz, funded by the Research Council of Norway.

References

Abrams, P.A., and Matsuda, H. 2005. The effect of adaptive change in the prey on the dynamics of an exploited predator population. Can. J. Fish. Aquat. Sci. **62**(4): 758–766. doi:10.1139/f05-051.

Allendorf, F.W., England, P.R., Luikart, G., Ritchie, P.A., and Ryman, N. 2008. Genetic effects of harvest on wild animal populations. Trends Ecol. Evol. **23**(6): 327–337. doi:10.1016/j.tree.2008.02.008. PMID:18439706.

Andersen, K.H., and Beyer, J.E. 2006. Asymptotic size determines species abundance in the marine size spectrum. Am. Nat. **168**(1): 54–61. doi:10.1086/504849.

Andersen, K.H., and Brander, K. 2009. Expected rate of fisheries-

induced evolution is slow. Proc. Natl. Acad. Sci. U.S.A. **106**(28): 11657–11660. doi:10.1073/pnas.0901690106. PMID: 19564596.

Arnott, S.A., Chiba, S., and Conover, D.O. 2006. Evolution of intrinsic growth rate: metabolic costs drive trade-offs between growth and swimming performance in *Menidia menidia*. Evolution, **60**(6): 1269–1278. PMID:16892976.

Benoît, H.P., and Swain, D.P. 2008. Impacts of environmental change and direct and indirect harvesting effects on the dynamics of a marine fish community. Can. J. Fish. Aquat. Sci. **65**(10): 2088–2104. doi:10.1139/F08-112.

Billerbeck, J.M., Lankford, T.E., Jr., and Conover, D.O. 2001. Evolution of intrinsic growth and energy acquisition rates. I. Trade-offs with swimming performance in *Menidia menidia*. Evolution, **55**(9): 1863–1872. PMID:11681741.

Biro, P.A., and Post, J.R. 2008. Rapid depletion of genotypes with fast growth and bold personality traits from harvested fish populations. Proc. Natl. Acad. Sci. U.S.A. **105**(8): 2919–2922. doi:10.1073/pnas.0708159105. PMID:18299567.

Bowen, W.D., McMillan, J., and Mohn, R. 2003. Sustained exponential population growth of grey seals at Sable Island, Nova Scotia. ICES J. Mar. Sci. **60**(6): 1265–1274. doi:10.1016/S1054-3139(03)00147-4.

Brown, J.H., Gillooly, J.F., Allen, A.P., Savage, V.M., and West, G.B. 2004. Toward a metabolic theory of ecology. Ecology, **85**(7): 1771–1789. doi:10.1890/03-9000.

Brown, C.J., Hobday, A.J., Ziegler, P.E., and Welsford, D.C. 2008. Darwinian fisheries science needs to consider realistic fishing pressures over evolutionary time scales. Mar. Ecol. Prog. Ser. **369**: 257–266. doi:10.3354/meps07601.

Clapham, P.J., Young, S.B., and Brownell, R.L. 1999. Baleen whales: conservation issues and the status of the most endangered populations. Mammal Rev. **29**(1): 37–62. doi:10.1046/j.1365-2907.1999.00035.x.

Clark, C.W., and Levy, D.A. 1988. Diel vertical migrations by juvenile sockeye salmon and the antipredation window. Am. Nat. **131**(2): 271–290. doi:10.1086/284789.

Clark, C.W., and Mangel, M. 2000. Dynamic state variable models in ecology. Oxford University Press, New York.

Creel, S., and Christianson, D. 2008. Relationships between direct predation and risk effects. Trends Ecol. Evol. **23**(4): 194–201. doi:10.1016/j.tree.2007.12.004. PMID:18308423.

de Roos, A.M., and Persson, L. 2001. Physiologically structured models — from versatile technique to ecological theory. Oikos, **94**(1): 51–71. doi:10.1034/j.1600-0706.2001.11313.x.

Dieckmann, U., and Heino, M. 2007. Probabilistic maturation reaction norms: their history, strengths, and limitations. Mar. Ecol. Prog. Ser. **335**: 253–269. doi:10.3354/meps335253.

Dunlop, E.S., Shuter, B.J., and Dieckmann, U. 2007. Demographic and evolutionary consequences of selective mortality: predictions from an eco-genetic model for smallmouth bass. Trans. Am. Fish. Soc. **136**(3): 749–765. doi:10.1577/T06-126.1.

Dunlop, E.S., Baskett, M.L., Heino, M., and Dieckmann, U. 2009*a*. Propensity of marine reserves to reduce the evolutionary effects of fishing in a migratory species. Evol. Appl. **2**(3): 371–393. doi:10.1111/j.1752-4571.2009.00089.x.

Dunlop, E.S., Enberg, K., Jørgensen, C., and Heino, M. 2009*b*. Toward Darwinian fisheries management. Evol. Appl. **2**(3): 245–259. doi:10.1111/j.1752-4571.2009.00087.x.

Enberg, K., Jørgensen, C., Dunlop, E.S., Heino, M., and Dieckmann, U. 2009. Implications of fisheries-induced evolution for stock rebuilding and recovery. Evol. Appl. **2**(3): 394–414. doi:10.1111/j.1752-4571.2009.00077.x.

Endler, J.A. 1987. Predation, light intensity and courtship behavior in *Poecilia reticulata* (Pisces, Poeciliidae). Anim. Behav. **35**(5): 1376–1385. doi:10.1016/S0003-3472(87)80010-6.

Gårdmark, A., Dieckmann, U., and Lundberg, P. 2003. Life-history evolution in harvested populations: the role of natural predation. Evol. Ecol. Res. **5**: 239–257.

Ghalambor, C.K., Reznick, D.N., and Walker, J.A. 2004. Constraints on adaptive evolution: the functional trade-off between reproduction and fast-start swimming performance in the Trinidadian guppy (*Poecilia reticulata*). Am. Nat. **164**(1): 38–50. doi:10.1086/421412. PMID:15266369.

Grift, R.E., Rijnsdorp, A.D., Barot, S., Heino, M., and Dieckmann, U. 2003. Fisheries-induced trends in reaction norms for maturation in North Sea plaice. Mar. Ecol. Prog. Ser. **257**: 247–257. doi:10.3354/meps257247.

Heino, M., Baulier, L., Boukal, D.S., Dunlop, E.S., Eliassen, S., Enberg, K., Jørgensen, C., and Varpe, Ø. 2008. Evolution of growth in Gulf of St. Lawrence cod? Proc. R. Soc. B, **275**: 1111–1112. doi:10.1098/rspb.2007.1429.

Houston, A.I., and McNamara, J.M. 1999. Models of adaptive behaviour: an approach based on state. Cambridge University Press, Cambridge, UK.

Hutchings, J.A. 2000. Collapse and recovery of marine fishes. Nature (London), **406**(6798): 882–885. doi:10.1038/35022565. PMID:10972288.

Hutchings, J.A. 2005. Life history consequences of overexploitation to population recovery in Northwest Atlantic cod (*Gadus morhua*). Can. J. Fish. Aquat. Sci. **62**(4): 824–832. doi:10.1139/f05-081.

Jackson, J.B.C., Kirby, M.X., Berger, W.H., Bjorndal, K.A., Botsford, L.W., Bourque, B.J., Bradbury, R.H., Cooke, R., Erlandson, J., Estes, J.A., Hughes, T.P., Kidwell, S., Lange, C.B., Lenihan, H.S., Pandolfi, J.M., Peterson, C.H., Steneck, R.S., Tegner, M.J., and Warner, R.R. 2001. Historical overfishing and the recent collapse of coastal ecosystems. Science (Washington, D.C.), **293**(5530): 629–637. doi:10.1126/science.1059199. PMID:11474098.

Jennings, S., Kaiser, M.J., and Reynolds, J.D. 2001. Marine fisheries ecology. Blackwell Publishing, Malden, Massachusetts.

Jones, W., Gurney, W.S.C., Speirs, D.C., Bacon, P.J., and Youngson, A.F. 2002. Seasonal patterns of growth, expenditure and assimilation in juvenile Atlantic salmon. J. Anim. Ecol. **71**(6): 916–924. doi:10.1046/j.1365-2656.2002.00679.x.

Jørgensen, C., Enberg, K., Dunlop, E.S., Arlinghaus, R., Boukal, D.S., Brander, K., Ernande, B., Gårdmark, A., Johnston, F., Matsumura, S., Pardoe, H., Raab, K., Silva, A., Vainikka, A., Dieckmann, U., Heino, M., and Rijnsdorp, A.D. 2007. Ecology: managing evolving fish stocks. Science (Washington, D.C.), **318**(5854): 1247–1248. doi:10.1126/science.1148089. PMID: 18033868.

Jørgensen, C., Ernande, B., and Fiksen, Ø. 2009. Size-selective fishing gear and life history evolution in the Northeast Arctic cod. Evol. Appl. **2**(3): 356–370. doi:10.1111/j.1752-4571.2009.00075.x.

Kiørboe, T. 2008. Optimal swimming strategies in mate-searching pelagic copepods. Oecologia (Berl.), **155**(1): 179–192. doi:10.1007/s00442-007-0893-x.

Kjesbu, O.S., Solemdal, P., Bratland, P., and Fonn, M. 1996. Variation in annual egg production in individual captive Atlantic cod (*Gadus morhua*). Can. J. Fish. Aquat. Sci. **53**(3): 610–620. doi:10.1139/cjfas-53-3-610.

Kozlowski, J. 1991. Optimal energy allocation models — an alternative to the concepts of reproductive effort and cost of reproduction. Acta Oecol. **12**: 11–33.

Kraak, S.B.M. 2007. Does the probabilistic maturation reaction

norm approach disentangle phenotypic plasticity from genetic change? Mar. Ecol. Prog. Ser. **335**: 295–300. doi:10.3354/meps335295.

Lankford, T.E., Jr., Billerbeck, J.M., and Conover, D.O. 2001. Evolution of intrinsic growth and energy acquisition rates. II. Trade-offs with vulnerability to predation in *Menidia menidia*. Evolution, **55**(9): 1873–1881. doi:10.1111/j.0014-3820.2001.tb00836.x. PMID:11681742.

Law, R. 2007. Fisheries-induced evolution: present status and future directions. Mar. Ecol. Prog. Ser. **335**: 271–277. doi:10.3354/meps335271.

Law, R., and Grey, D.R. 1989. Evolution of yields from populations with age-specific cropping. Evol. Ecol. **3**(4): 343–359. doi:10.1007/BF02285264.

Lester, N.P., Shuter, B.J., and Abrams, P.A. 2004. Interpreting the von Bertalanffy model of somatic growth in fishes: the cost of reproduction. Proc. R. Soc. Lond. B Biol. Sci. **271**(1548): 1625–1631. doi:10.1098/rspb.2004.2778.

Lima, S.L., and Dill, L.M. 1990. Behavioral decisions made under the risk of predation — a review and prospectus. Can. J. Zool. **68**(4): 619–640. doi:10.1139/z90-092.

Lochmiller, R.L., and Deerenberg, C. 2000. Trade-offs in evolutionary immunology: just what is the cost of immunity? Oikos, **88**(1): 87–98. doi:10.1034/j.1600-0706.2000.880110.x.

Marshall, C.T., and Browman, H.I. 2007. Introduction to theme section on disentangling the causes of maturation trends in exploited fish populations. Mar. Ecol. Prog. Ser. **335**: 249–251.

Matsuda, H., and Abrams, P.A. 2004. Effects of predator–prey interactions and adaptive change on sustainable yield. Can. J. Fish. Aquat. Sci. **61**(2): 175–184. doi:10.1139/f03-147.

McGurk, M.D. 1986. Natural mortality of marine pelagic fish eggs and larvae — role of spatial patchiness. Mar. Ecol. Prog. Ser. **34**: 227–242. doi:10.3354/meps034227.

Morita, K., and Fukuwaka, M.A. 2006. Does size matter most? The effect of growth history on probabilistic reaction norm for salmon maturation. Evolution, **60**(7): 1516–1521. PMID:16929668.

Munch, S.B., and Conover, D.O. 2004. Nonlinear growth cost in *Menidia menidia*: theory and empirical evidence. Evolution, **58**(3): 661–664. PMID:15119450.

Myers, R.A., Hutchings, J.A., and Barrowman, N.J. 1997. Why do fish stocks collapse? The example of cod in Atlantic Canada. Ecol. Appl. **7**(1): 91–106. doi:10.1890/1051-0761(1997)007[0091:WDFSCT]2.0.CO;2.

Olsen, E.M., Lilly, G.R., Heino, M., Morgan, M.J., Brattey, J., and Dieckmann, U. 2005. Assessing changes in age and size at maturation in collapsing populations of Atlantic cod (*Gadus morhua*). Can. J. Fish. Aquat. Sci. **62**(4): 811–823. doi:10.1139/f05-065.

Pauly, D., Christensen, V., V, Dalsgaard, J., Froese, R., and Torres, F., Jr. 1998. Fishing down marine food webs. Science (Washington, D.C.), **279**(5352): 860–863. doi:10.1126/science.279.5352.860. PMID:9452385.

Persson, L., Amundsen, P.-A., de Roos, A.M., Klemetsen, A., Knudsen, R., and Primicerio, R. 2007. Culling prey promotes predator recovery — alternative states in a whole-lake experiment. Science (Washington, D.C.), **316**(5832): 1743–1746. doi:10.1126/science.1141412. PMID:17588929.

Peterson, I., and Wroblewski, J.S. 1984. Mortality rates of fishes in the pelagic ecosystem. Can. J. Fish. Aquat. Sci. **41**(7): 1117–1120. doi:10.1139/f84-131.

Petrie, B., Frank, K.T., Shackell, N.L., and Leggett, W.C. 2009. Structure and stability in exploited marine fish communities: quantifying critical transitions. Fish. Oceanogr. **18**(2): 83–101. doi:10.1111/j.1365-2419.2009.00500.x.

Pitcher, T.J., and Parrish, J.K. 1993. Functions of shoaling behaviour in teleosts. *In* Behaviour of teleost fishes. *Edited by* T.J. Pitcher. Chapman & Hall, London, UK. pp. 363–439.

Quince, C., Abrams, P.A., Shuter, B.J., and Lester, N.P. 2008. Biphasic growth in fish I: theoretical foundations. J. Theor. Biol. **254**(2): 197–206. doi:10.1016/j.jtbi.2008.05.029. PMID:18606423.

Ricker, W.E. 1981. Changes in the average size and average age of Pacific salmon. Can. J. Fish. Aquat. Sci. **38**(12): 1636–1656. doi:10.1139/f81-213.

Rijnsdorp, A.D. 1993. Fisheries as a large-scale experiment on life-history evolution — disentangling phenotypic and genetic effects in changes in maturation and reproduction of North Sea plaice, *Pleuronectes platessa* L. Oecologia (Berl.), **96**(3): 391–401. doi:10.1007/BF00317510.

Roff, D.A. 1983. An allocation model of growth and reproduction in fish. Can. J. Fish. Aquat. Sci. **40**(9): 1395–1404. doi:10.1139/f83-161.

Savenkoff, C., Castonguay, M., Chabot, D., Hammill, M.O., Bourdages, H., and Morissette, L. 2007. Changes in the northern Gulf of St. Lawrence ecosystem estimated by inverse modelling: evidence of a fishery-induced regime shift? Estuar. Coast. Shelf Sci. **73**(3–4): 711–724. doi:10.1016/j.ecss.2007.03.011.

Sharpe, D.M.T., and Hendry, A.P. 2009. Life history change in commercially exploited fish stocks: an analysis of trends across studies. Evol. Appl. **2**(3): 260–275. doi:10.1111/j.1752-4571.2009.00080.x.

Sheldon, R.W., Prakash, A., and Sutcliffe, W.H., Jr. 1972. The size distribution of particles in the ocean. Limnol. Oceanogr. **17**: 327–340.

Shelton, P.A., Sinclair, A.F., Chouinard, G.A., Mohn, R., and Duplisea, D.E. 2006. Fishing under low productivity conditions is further delaying recovery of Northwest Atlantic cod (*Gadus morhua*). Can. J. Fish. Aquat. Sci. **63**(2): 235–238. doi:10.1139/f05-253.

Sinclair, A.F., Swain, D.P., and Hanson, J.M. 2002. Disentangling the effects of size-selective mortality, density, and temperature on length-at-age. Can. J. Fish. Aquat. Sci. **59**(2): 372–382. doi:10.1139/f02-014.

Swain, D.P., and Chouinard, G.A. 2008. Predicted extirpation of the dominant demersal fish in a large marine ecosystem: Atlantic cod (*Gadus morhua*) in the southern Gulf of St. Lawrence. Can. J. Fish. Aquat. Sci. **65**(11): 2315–2319. doi:10.1139/F08-175.

Swain, D.P., Sinclair, A.F., and Hanson, J.M. 2007. Evolutionary response to size-selective mortality in an exploited fish population. Proc. R. Soc. Lond. B Biol. Sci. **274**(1613): 1015–1022. doi:10.1098/rspb.2006.0275.

Swain, D.P., Sinclair, A.F., and Hanson, J.M. 2008. Evolution of growth in Gulf of St. Lawrence cod: reply to Heino *et al*. Proc. R. Soc. Lond. B Biol. Sci. **275**(1639): 1113–1115. doi:10.1098/rspb.2007.1727.

Tilman, D., HilleRisLambers, J., Harpole, S., Dybzinski, R., Fargione, J., Clark, C., and Lehman, C. 2004. Does metabolic theory apply to community ecology? It's a matter of scale. Ecology, **85**: 1797–1799.

Yoneda, M., and Wright, P.J. 2004. Temporal and spatial variation in reproductive investment of Atlantic cod *Gadus morhua* in the northern North Sea and Scottish west coast. Mar. Ecol. Prog. Ser. **276**: 237–248. doi:10.3354/meps276237.

Appendix A. Discrete time approximations of continuous time functions

The continuous time integrals were solved numerically by subdividing each year into J shorter steps. Knowing the weight at a given time $a + j/J$, weight in the next of these smaller steps, $a + (j+1)/J$, can be found as

$$w(a + (j + 1)/J) = w(a + j/J) + [1 - \alpha(a, l(a))] \cdot h \cdot w^b(a + j/J)$$

By doing this for every small time step j between a and $a + 1$, the discrete time approximation of the continuous time integral is

$$w(a + 1) = w(a) + [1 - \alpha(a, l(a))]\, h \int_{t=a}^{a+1} w^b(t)\, \mathrm{d}t \approx w(a) + [1 - \alpha(a, l(a))]\, h \frac{1}{J} \sum_{j=1}^{J} w^b(a + j/J)$$

Gonad size at the end of the year, $g(a + 1)$, is found in an analogous way:

$$g(a + 1) = \alpha(a, l(a)) h \frac{1}{J} \sum_{j=1}^{J} w^b(a + j/J)$$

Mortality consists of four components. In the equation below, size-independent mortality is M_{fixed}. Predation mortality depends on size, which changes throughout the year so it needs to be updated and summed over the shorter time steps j. The summation is thus predation mortality $cl^{-d}(a + j/J)$, which in addition to being a separate component of mortality is multiplied by $\varphi(a, l(a))$ to get the growth-related mortality and $[q(a + j/J)/q_{\text{ref}}]^p$ to get the reproduction-related mortality. The final component is fishing mortality, which is specified by the harvest rate F and may include a size-selectivity function $Y(L)$ that depends on size:

$$z(a) = M_{\text{fixed}} + \frac{1}{J} \sum_{j=1}^{J} \left[cw^{-d}(a + j/J) \left(1 + \varphi(a, l(a)) + \left(\frac{q(a + j/J)}{q_{\text{ref}}} \right)^p \right) + F \cdot y(l(a + j/J)) \right]$$

Having found the total mortality rate over the year ($z(a)$), annual survival probability ($s(a)$) is equal to $\exp(-z(a))$.

Supporting Information

Fishing-induced adaptations and natural mortality

Christian Jørgensen and Øyvind Fiksen

Contents:

Figure S1. Optimal allocation and growth strategies.

Figure S2. Sensitivity to varying parameters values.

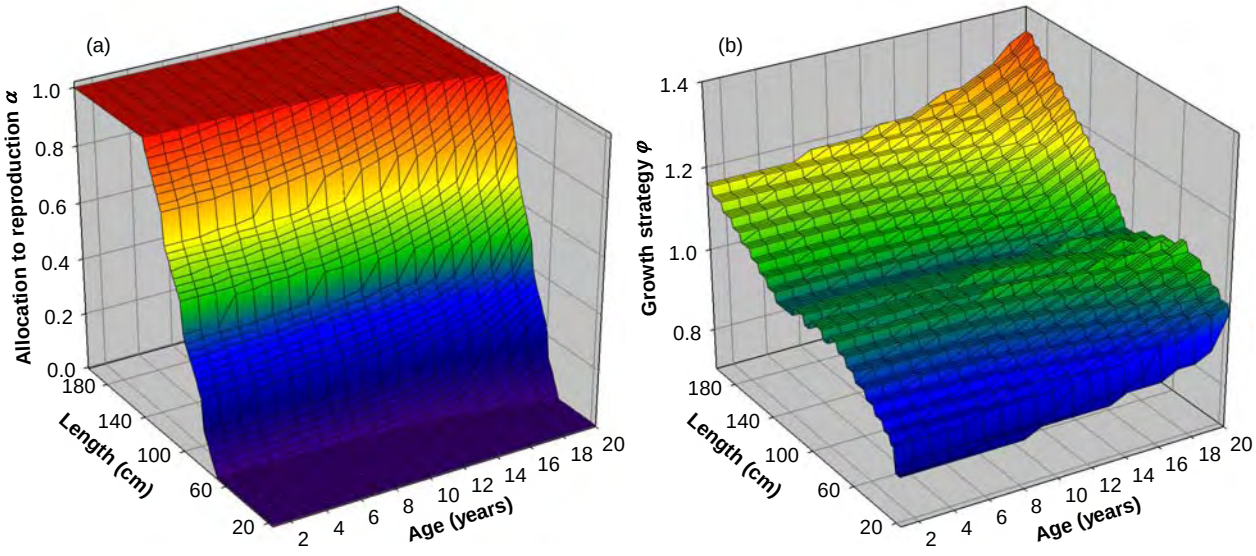


Figure S1. (a) Optimal allocation to reproduction α and (b) the growth strategy φ to illustrate the state-dependent strategies found by dynamic programming. The increased reproductive investment and growth strategy for high ages is a terminal effect of the maximum age of 30 years. All the results presented in the main article and below are for up to 20 years only, where these effects are minimal. Parameters as in the main paper and no harvesting.

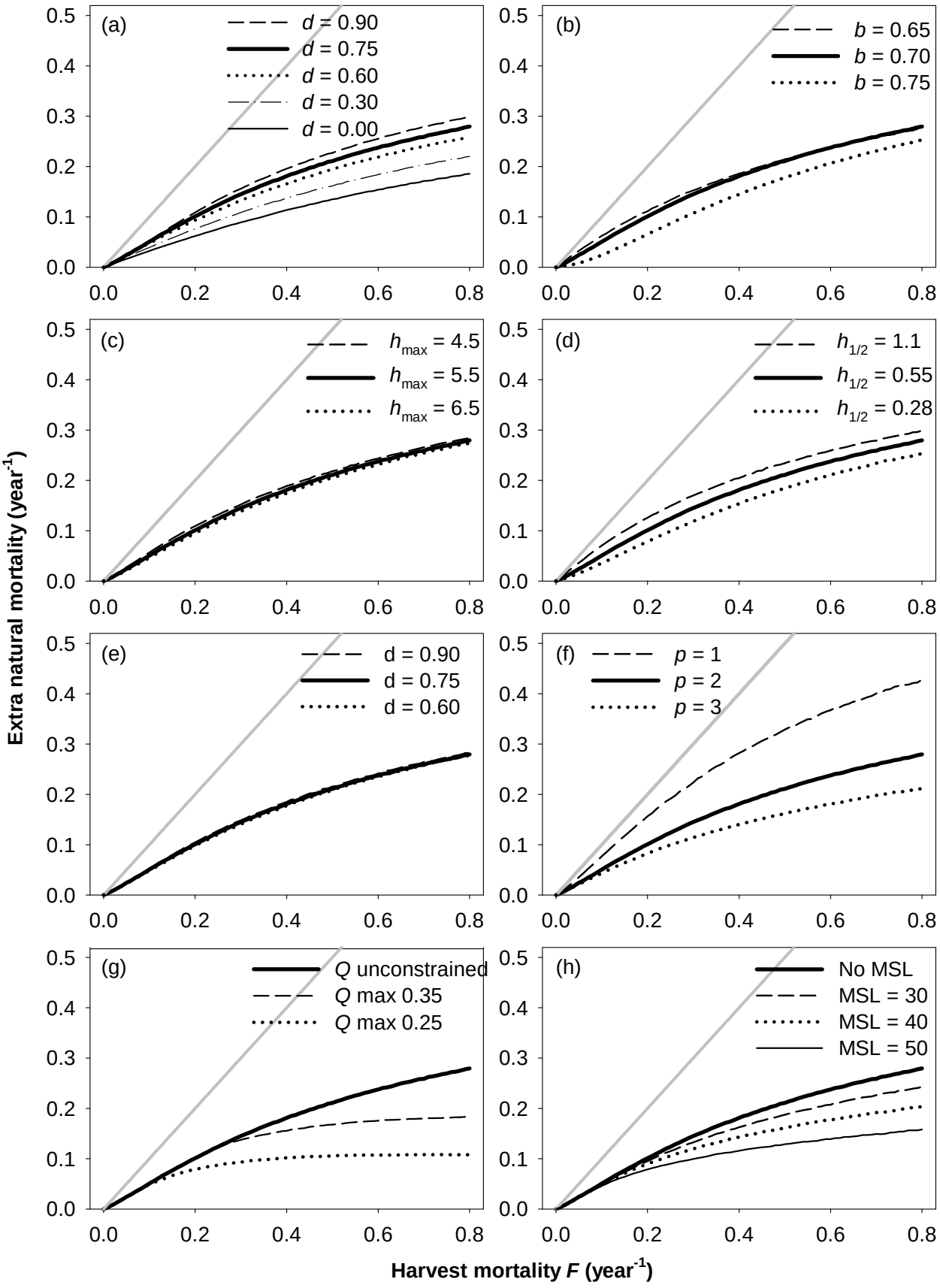

(a)
d = 0.90
d = 0.75
d = 0.60
d = 0.30
d = 0.00
(b)
b = 0.65
b = 0.70
b = 0.75
(c)
h_max = 4.5
h_max = 5.5
h_max = 6.5
(d)
h_1/2 = 1.1
h_1/2 = 0.55
h_1/2 = 0.28
(e)
d = 0.90
d = 0.75
d = 0.60
(f)
p = 1
p = 2
p = 3
(g)
Q unconstrained
Q max 0.35
Q max 0.25
(h)
No MSL
MSL = 30
MSL = 40
MSL = 50
Extra natural mortality (year-1)
Harvest mortality F (year-1)
0.0
0.1
0.2
0.3
0.4
0.5
0.6
0.8

Figure S2 (previous page). The thick solid line is the standard scenario and it is repeated in each panel to make comparison easier (parameters are given in the legend to Figure 1 of the main paper). The remaining panels are sensitivity to varying (a) the exponent d that scales predation with size; (b) the intake exponent b; (c) the asymptote h_{max} of the Holling type II equation relating growth strategy φ to resource intake; (d) the half-saturation constant $h_{1/2}$ of the same Holling function; (e) the gonado-somatic index q_{ref} at which reproduction mortality equals predation mortality $M_{predation}$; (f) the exponent p relating gonado-somatic index to reproduction mortality; (g) the effect of including a constraint formulated as a max value of reproductive investment Q; and (h) the effect of a strictly enforced minimum size limit in the fishery, where the harvest mortality is $F(L)=F_{max}\cdot Y(L)$, where F_{max} is maximum harvest mortality and fisheries selectivity is modelled as $Y(L)=[1+\exp(-u(L-L_{50}))]^{-1}$ with u=0.1 (steepness) and L_{50} (cm) being the length at which there is a 50% probability of being harvested.