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Natural mortality: Its ecology, how it shapes fish life histories, and why it may be increased by fishing ☆

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ABSTRACT

A stronger focus on natural mortality may be required to better understand contemporary changes in fish life histories and behaviour and their responses to anthropogenic drivers. Firstly, natural mortality is the selection under which fish evolved in the first place, so a theoretical understanding of effects of natural mortality alone is needed. Secondly, due to trade-offs, most organismal functions can only be achieved at some cost in terms of survival. Several trade-offs might need to be analysed simultaneously with effects on natural mortality being a common currency. Thirdly, there is scattered evidence that natural mortality has been increasing, some would say dramatically, in some fished stocks, which begs explanations. Fourthly, natural mortality most often implies transfer of mass and energy from one species to another, and therefore has foodweb and ecosystem consequences. We therefore analyse a model for evolution of fish life histories and behaviour, where state-dependent energy-allocation and growth strategies are found by optimization. Natural mortality is split into five different components, each specified as the outcome of individual traits and ecological trade-offs: a fixed baseline mortality; size-dependent predation; risk-dependent growth strategy; a fixed mortality when sexually mature; and mortality increasing with reproductive investment. The analysis is repeated with and without fishing. Each component of natural mortality has consequences for optimal life history strategies. Beyond earlier models, we show i) how the two types of reproductive mortality sometimes have similar and sometimes contrasting effects on life history evolution, ii) how ecosystem properties such as food availability and predation levels have stronger effects on optimal strategies than changing other mortality components, and iii) how expected changes in risk-dependent growth strategies are highly variable depending on the type of mortality changed.

1. Introduction

Natural selection has moulded fish and their life histories through millions of years, and those individuals that best avoided natural mortality and were able to reproduce left the descendants we observe today. Natural mortality is not only an external force that shapes life histories and other traits through natural selection, but it is also the outcome of behavioural and life history strategies. In this paper we use evolutionary modelling to study how between-species variation in ecology and trade-offs has consequences for optimal life history strategies and the resulting rate of natural mortality.

In fisheries science, natural mortality is treated almost like an orphan, vividly illustrated by John Pope's cartoon of how 'M = ?' metamorphosed into a magical 'M = .2' now being applied ubiquitously (e.g., Jennings et al., 2001, p. 203). Admittedly, great efforts go into quantifying natural mortality and its large-scale patterns of variation across species (Gislason et al., 2010), but most theory has treated natural mortality as an externally set parameter, usually of M = .2. Some studies have included size-dependent mortality (Enberg et al., 2009; Jørgensen and Fiksen, 2006), a trade-off between survival and growth rate (Dunlop et al., 2009b; Enberg et al., 2009), or mortality costs of spawning (Hutchings, 2005, 2009). In this paper we expand the model by Jørgensen and Fiksen (2010) where several traits were linked to mortality through trade-offs.

To motivate our study, we give four reasons for why we believe an increased focus on natural mortality might be advantageous.

First, the exploitation of fish stocks is increasingly being recognized as a potential driver for contemporary evolution of life history traits (reviewed, e.g., by Allendorf et al., 2008; Dunlop et al., 2009a; Fenberg and Roy, 2008; Hard et al., 2008; Heino and Godø, 2002; Hutchings and Fraser, 2008; Jørgensen et al., 2007; Kuparinen and Merilä, 2007; Law, 1991, 2000). There is evidence that the rate of this evolution is orders of magnitude faster than one would expect from the fossil record (Darimont et al., 2009; Jørgensen et al., 2007;

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Sharpe and Hendry, 2009; see also Andersen and Brander, 2009). Trends on decadal timescales have been observed, particularly for age and size at maturation (Ricker, 1981; reaction norm studies reviewed by Dieckmann and Heino, 2007) but also for reproductive investment (Rijnsdorp et al., 2005; Yoneda and Wright, 2004). Because life history traits are changing so rapidly, one could expect consequences for natural mortality, but which? To address this, we present a model that links life history evolution to changes in natural mortality.

Second, behaviour is a flexible way for organisms to respond to changes in their environment. Fishing gear is often designed to exploit a species' behavioural tendencies, such as schooling, foraging behaviour, swimming, or habitat use, and is therefore likely to cause changes in behaviour over time as fish either evolve (slow) or learn (potentially much faster) countermeasures. It has for example been shown that gillnets preferentially harvest individuals with bold personality traits, probably because they have higher food requirements and are more active (Biro and Post, 2008). Angling works in similar ways (Philipp et al., 2009; Redpath et al., 2009). Behaviour is hard to observe, however, and time-series data from wild populations hardly exist. One crude way of studying behaviour in theoretical models is to be specific about likely trade-offs (Krebs and Davies, 1993; Lima, 2009; Lima and Dill, 1990). By including trade-offs that represent behaviour, the model in this paper can suggest likely broad-scale changes in behaviour.

The third reason natural mortality may warrant intensified focus is that some studies have begun noticing temporal trends towards increasing natural mortality. Natural mortality has been referred to as the most critical and important parameter, but also the most difficult to obtain (Pauly, 1980). Studies of temporal changes in natural mortality are few. Atlantic cod in the Gulf of St Lawrence were estimated to have a natural mortality of 0.1–0.2 year^{-1} in the 1980s, but by the 2000s it had risen to values as high as 0.6 year^{-1} (Swain, 2011; Swain and Chouinard, 2008). A similar estimate was suggested for winter skates (Swain et al., 2009) and white hake (Benoît et al., 2011). Such increases in natural mortality may be part of the explanation collapsed stocks are not recovering. The temporal increases in natural mortality are particularly worrisome because they follow a pattern expected by theory (Fig. 1). Using a model that assumed trade-offs between several life history traits and survival, Jørgensen and Fiksen (2010) predicted that natural mortality would increase with roughly half the fishing mortality once the fish stock has had sufficient time to reach a new evolutionary equilibrium. This estimate was robust to parameter variation over a wide range. Of course, other anthropogenic and natural drivers of change could also affect natural mortality.

A fourth reason for studying natural mortality is its trophic and food web implications. Natural mortality usually involves one species being eaten by another, and therefore describes flows of energy and mass through ecosystems. In the cod example above, there is ample evidence that predation from seals has increased numerically and is now a significant contributor to mortality (Benoît et al., 2011; Savenkoff et al., 2007). But which way does causality operate? Has the increasing seal abundance led to increased predation on cod? Or has the previously high fishing pressure led to changes in cod life history and behaviour, whereby higher exposure to seals became adaptive? The latter implies that changes in the cod may have contributed to the seal explosion.

For these four reasons we believe it is timely to focus more on understanding what natural mortality is, how its mechanisms vary between species, how it has shaped fish life histories, and how it can be changed by fishing and other anthropogenic and natural drivers. Below we describe a model with relationships that link natural mortality to life history traits, ecology, and behaviour.

For the model we have chosen parameters representative of flatfish. Flatfish are physiologically and ecologically adapted to benthic environments (Link et al., 2002), with cryptic morphology and a potentially low metabolic rate that allow them to bury or hide in open habitats in which one otherwise would be exposed and vulnerable. Several species are targeted by fisheries, and particularly trawling and dredging practices are known to alter the survivability of flatfish beyond those harvested (Link et al., 2002). As a group, flatfish are distributed worldwide and exhibit considerable interspecific variation in terms of their life histories (Roff, 1991). Although the parameters chosen in this paper best represent European plaice in the North Sea, the model describes general ecological relationships with trade-offs that involve survival, and are likely applicable to other flatfish species and harvested marine organisms in general.

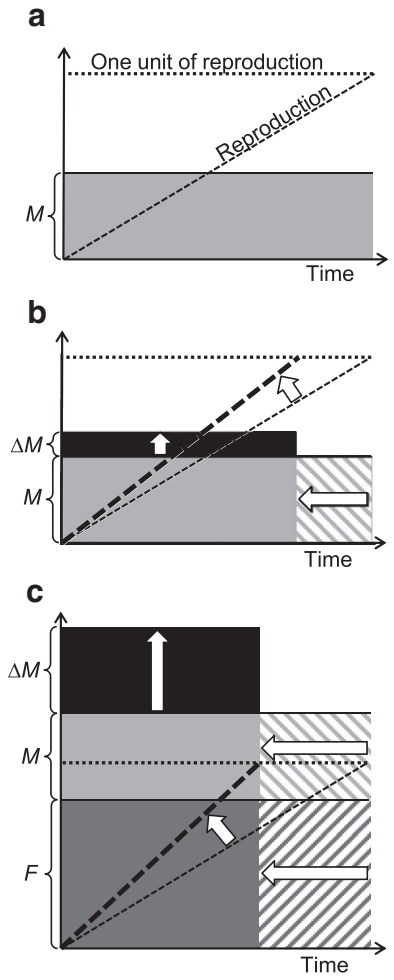

Fig. 1. Conceptual figure of how fishing mortality might lead to adaptations that involve increased natural mortality. a) Consider a mature fish, so that natural mortality M can be considered roughly constant over time, indicated by the horizontal black line. Because survival is given by the formula $S = e^{-Mt}$, survival is higher the smaller the grey area below the mortality curve is. Assume furthermore that this fish reproduces or builds up reserves designated to reproduction at a constant rate (dashed line), and that the total time shown here equals the time required to complete one unit of reproduction (for example, average reproduction in one year). b) Imagine now that this fish can change its strategy and speed up its rate of reproduction, but that this can only be achieved by taking higher risks which leads to an increase, ΔM, in natural mortality. This could for example be more risk-prone foraging behaviour, building larger gonads that make it harder to escape predators, down-regulating the immune system, or spending more time searching for high quality mates. Thus, the unit of reproduction can be achieved faster, and this might evolve as long as the area saved (hatched area) is larger than the area of new mortality accepted (black area). c) Add fishing mortality F. Accelerated reproduction will now shorten also the duration the fish risks being fishing, i.e. the dark hatched area is also saved. Consequently, a riskier strategy that involves even higher natural mortality might evolve as long as the black area of new accepted natural mortality is smaller than the areas of mortality saved (hatched).

2. Model description

The modelling approach in this paper uses a state-dependent energy-allocation model in which optimal fish life-history strategies are found by optimization. It is built on a previous model and explained only briefly here; for further details please refer to the full model description in Jørgensen and Fiksen (2010). Here, the formulation of mortality has been extended, particularly with regard to reproduction-related mortality that is split into several components. Natural mortality has been split into 5 components that reflect ecological processes, allowing for a comprehensive understanding of the forces that mould fish life histories. We have also chosen parameters to resemble a flatfish, European plaice (*Pleuronectes platessa*) in the North Sea.

A key point is that the model finds optimal values for the foraging strategy φ, and the energy allocation strategy α, for each combination of age and length (individual states). These strategies are then simulated to produce the results shown.

2.1. Growth, allocation, and reproduction

The model we use for growth is a simplified bioenergetics model, similar to the growth models by Lester et al. (2004) and Quince et al. (2008). It is assumed that the net energy intake R (kg year^{-1}; thus in mass equivalents) scales allometrically with body size as $R = HW^b$, where W (kg) is somatic body mass, b (dimensionless) the allometric scaling exponent, and H (kg^{1-b}year^{-1}) a foraging rate that is the outcome of a foraging strategy that we optimize (see below).

The available resources are allocated such that somatic body mass and gonads G (kg), respectively, grow as:

$$dW/dt = (1-\alpha)R, \text{and} \tag{1a}$$

$$dG/dt = \alpha R/v. \tag{1b}$$

Here v (dimensionless) is the ratio of the energetic cost of gonadic tissue relative to somatic tissue. Gonads are interpreted in a broad sense and do not only include the gonad itself but all energetic costs associated with reproduction, including mating behaviour and spawning migrations. Gonad mass can therefore be thought of as reproductive investment, measured in units equivalent to body mass. Spawning takes place at the end of each year, and we assume lifetime expected production of gonad mass is proportional to fitness. Body length (L, cm) is isometrically related to weight as $W = kL^3$.

2.2. Mortality

The focus of this paper is how a life history strategy is the outcome of natural selection operating on ecological relationships, in particular the trade-offs with survival. Natural mortality is therefore split into 5 categories (all with unit year^{-1}) that correspond to behavioural or morphological features of the organism. The rationale and equations are given in this section, and below we explain how we use optimization to find the best life history trajectory given a set of parameter values (Variables are summarized in Table 1 and parameter values given in Table 2). The model's sensitivity to these values was tested varying each parameter value and reporting on the effect on an optimal flatfish life history.

2.2.1. Size-dependent predation

There is considerable empirical evidence that mortality in aquatic and marine environments is size-dependent, where mortality rate declines with body size L (cm) as

$$M_{\text{predation}}(L) = cL^{-d} \tag{2}$$

Table 1
Variables used in a model for life history evolution in flatfish. Dimensionless variables are assigned '–' in the column for unit.

Variable	Description	Unit
α	Strategy variable: allocation of available resources towards reproduction	–
φ	Strategy variable: level of risk accepted in relation to foraging	–
L	Body length	cm
R	Net resources available for growth and reproduction (mass equivalents)	kg year^{-1}
W	Somatic body mass	kg
H	Food intake coefficient given level of accepted risk	kg^{1-b}year^{-1}
T	Time (continuous)	years
Z	Total mortality rate	year^{-1}
G	Mass of gonads	kg
Q	Gonado-somatic index	–

(Fig. 2a). A typical value of the scaling parameter is $d = 0.75$ (dimensionless; Andersen and Beyer, 2006; Gislason et al., 2010; McGurk, 1986; Peterson and Wroblewski, 1984). One of our central assumptions is that the scaling of predation mortality influences the size-dependent scaling of many of the other components of natural mortality (as was also assumed by Jørgensen and Fiksen, 2010). A fish that is vulnerable to predation will therefore be so also when it is foraging, reproducing, or performing other activities.

2.2.2. Mortality related to foraging

As for other animals, an adaptive growth strategy has likely evolved among fish (reviewed in Arendt, 1997; Enberg et al., 2012). Although higher growth can be achieved by reallocating resources from other functions, e.g. immunity, behaviour, or cognition, such reallocation would amount to increasing the net availability of resources in the same way that increasing foraging would and here we treat these effects as the same and denote the ensuing mortality as M_{growth}. At the behavioural level, the field of foraging ecology has long studied how optimal patch choice balances expected foraging returns against predation risk (Mangel and Clark, 1986; McNamara and Houston, 1986). Lima and Dill (1990) provide an insightful review, and here we use a general formulation whereby increased foraging leads to increased mortality M_{growth}. Several mechanisms can underlie such a relationship. For example: the safest foraging opportunities can be exploited first, and to achieve a higher foraging rate the fish might need to include more risky patches or unsafe time windows (Clark and Levy, 1988). The trade-off can also be physiological, as in Atlantic silversides where individuals with higher voluntary meal size (Billerbeck et al., 2000) are being eaten more often by

Table 2
Parameters used in a fish life history model, chosen to resemble plaice in the North Sea. Dimensionless parameters are assigned '–' in the column for unit.

Parameter	Description	Value	Unit
b	Exponent of energy intake function	0.70	–
$h_{\max}$	Asymptotic level of foraging rate, at infinite risk	6	kg^{1-b}year^{-1}
$h_{1/2}$	Risk level at which half the asymptotic foraging rate is achieved	1	–
k	Coefficient in length-weight relationship	$9.5 \cdot 10^{-3}$	kg cm^{-3}
q_{ref}	Gonadosomatic index at which $M_{\text{gonads}} = M_{\text{predation}}$	0.15	–
p	Exponent scaling cost of carrying gonads	2	–
u	Coefficient for mortality cost of participation in spawning	0.5	–
c	Coefficient for size-dependent predation	2.5	year^{-1}
d	Scaling for size-dependent predation	0.75	–
v	Cost of gonad tissue relative to somatic tissue	2	–

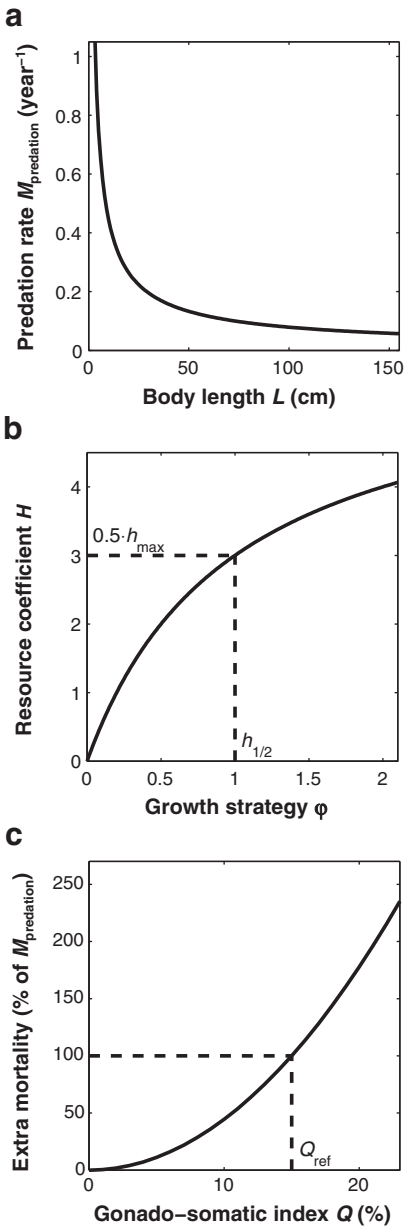

Fig. 2. Main assumptions of the model. a) We assume the same size-dependent scaling of mortality rate as found in many empirical meta-analyses and theoretical studies, where natural mortality is an allometric function of length scaling as $L^{-0.75}$. b) The fish can change the coefficient that determines resource acquisition rate by changing their growth strategy φ. The model is not specific on mechanism, but this may be done for example by more risky foraging behaviour or down-regulating immune function. Extra mortality is assumed proportional to φ and with the same size-dependent scaling as predation in panel a. c) Increased reproductive investment causes increased mortality, with the same scaling with size as predation in panel a. For reproductive investment we use the gonado-somatic index (GSI) Q as proxy, but this should be interpreted in the broad sense as also including behaviour, migrations, expression of secondary sexual characters etc.

predators (Lankford et al., 2001) because they are poorer swimmers (Billerbeck et al., 2001) due to the elevated oxygen requirements of higher digestion rates (Arnott et al., 2006). Such a relationship can be particularly relevant for flatfish, where respiration rates are depressed when buried but can be elevated by lying on top of sediments or even arching to get their heads above the layer affected by sediment metabolism (Gibson and Robb, 1992; Howell and Canario, 1987; Stoner and Ottmar, 2003). To reflect ecology where intake rate can be increased but patchiness and behavioural and physiological constraints may involve diminishing returns, we assume a saturation function for the relationship between the foraging strategy φ and the food intake coefficient H:

$$H = \frac{h_{max} \cdot \varphi}{h_{1/2} + \varphi} \tag{3}$$

(Fig. 2b). We then specify foraging-related mortality as proportional to φ:

$$M_{growth}(\varphi, L) = \varphi \cdot M_{predation}(L). \tag{4}$$

We assume the same size-dependence as predation mortality, with the rationale that the same predators are likely to be active during foraging as during other activities. There is thus a trade-off between net ingested resources and survival.

2.2.3. Reproduction

The effects of reproduction on survival were one of the first trade-offs that received attention in life history theory (Gadgil and Bossert, 1970; Williams, 1966). In early papers a general shape was often assumed, but empirical studies on fish now give us some insights into what this relationship may look like. Here we split the cost of reproduction into two components, related to the burden of carrying gonads and to costs of exposure and mate search at the spawning grounds.

2.2.3.1. Mortality at spawning grounds. Spawning behaviours include mate search, courtship, and mating, and each of these behaviours often require that the individual is relatively exposed to predators. Further, the possibility of reproduction may focus attention towards mates and mating opportunities and reduce the attention given to potential predators. We therefore include a cost of being at the spawning grounds in terms of increased mortality. We use the same scaling with body size as predation mortality, and introduce a coefficient u to determine the level:

$$M_{spawning}(L) = u \cdot M_{predation}(L). \tag{5}$$

2.2.3.2. Mortality due to carrying gonads. Fish need to fit their gonads in their body cavity, which makes them rounder and stiffer and in turn compromises swimming abilities. In general terms, the power required to overcome hydrostatic friction increases proportional to the cross-sectional area (Vogel, 1994). One could therefore expect that rounder fish have poorer swimming performance, which can compromise escape behaviour and thus increase risk of predation. Few studies have quantified this directly, but Ghalambor et al. (2004) found declining swimming performance throughout the roughly four-week gestation period in guppies, a livebearer where brood mass increases linearly over time. In livebearers the developing brood has, in addition to the effect on the mother's body shape, increasing oxygen demands that additionally limit the mother's performance.

For the purposes of this paper we assume that mortality related to carrying gonads, M_{gonads}, increases with reproductive investment as a function of the gonado-somatic index $Q = G/(W+G)$ (dimensionless), and that it follows the same size dependence as predation:

$$M_{gonads}(Q, L) = M_{predation}(L) \cdot (Q/q_{ref})^p, \tag{6}$$

Here q_{ref} is a reference value at which the mortality from reproduction equals the size-dependent predation component, i.e., at which $M_{gonads} = M_{predation}$. For $p > 1$, mortality rate accelerates with increasing reproduction (Fig. 2c).

2.2.4. Unavoidable mortality

The above list is likely not exhaustive, so we also add a baseline or unavoidable mortality M_{fixed}. This is a fixed mortality rate that affects

all individuals independent of their state or their actions, and it should be interpreted to include sources of mortality not accounted for by the more specific mechanisms listed above and that do not show size dependence. Examples include diseases, catastrophic environmental effects, meteors, and the like.

2.2.5. Fishing mortality

Finally, we add fishing mortality F. This could easily be extended to include gear selectivity and size dependence, but to make interpretation easier we use a fixed mortality rate that does not vary with individual size or state.

Total mortality rate (Z; year^{-1}) is then

$$Z(\varphi, Q, L) = M_{\text{predation}}(L) + M_{\text{foraging}}(\varphi, L) + M_{\text{gonads}}(Q, L) + M_{\text{spawning}}(L) + M_{\text{fixed}} + F. \quad (7)$$

Annual survival probability (S) in year t is thus found as $S = \exp(-\int_{T=t}^{t+1} Z(T)\mathrm{d}T)$.

2.3. Optimization and simulations

We use optimization by dynamic programming (Clark and Mangel, 2000; Houston and McNamara, 1999) to find optimal strategies for energy allocation and the growth strategy. The method works backwards from a maximum age of 30 years and finds the optimal response for each combination of the individual state variables age and length. Numerically, strategies are found for discrete values of the individual state variables age (in years) and length (in cm). Between year t and $t+1$, mortality rate is calculated in 24 finer temporal steps and total mortality rate summed. This numerical implementation gives results that are indistinguishable from a fully continuous approach. An advantage compared to alternative modelling methodologies is that strategies are flexible and free to vary depending on age and size, which allows strategies e.g. to vary depending on maturity status. The method only finds the global optimum. The fitness measure we use is expected reproductive value, i.e. the gonad production at age discounted by survival until that age, summed over the entire lifespan. The predicted life histories are evolutionary endpoints — the adaptations one could expect to observe given sufficient time for evolution to reach equilibrium, and given that the ecology described by the parameter set remains constant. Thereafter we simulate an individual following this optimal strategy (using interpolated values for lengths that fall between discrete values), leading to the trajectories of growth, survival, and reproduction shown in the results section. A fuller description of the method and equations for numerical implementation can be found in Jørgensen and Fiksen (2010).

2.4. Parameterization

We chose to model the European plaice as a representative species of flatfish, and the model only considers females. Parameters were then chosen so that predicted growth without fishing corresponded to a maximum observed length of 100 cm and weight of 7 kg (maximum length and weight recorded in http://www.fishbase.se). Weight-at-age data for plaice in the North Sea (ICES, 2011) were averaged over the period 1980–2010 and converted to lengths assuming a condition factor of $k = 0.0105$ (which assumes some gonad mass). A life history that fits well with these data was predicted as the evolutionary endpoint at a fishing mortality of $F = 0.13$ year^{-1}. The rationale for this choice is that our model is limited to finding evolutionary endpoints, so this value for F represents partial evolution to the higher and more variable fishing rates this stock has been exposed to in the recent past. The model contains no environmental stochasticity so some of the predictions appear as bang–bang strategies with abrupt transitions between a juvenile growth phase and an adult reproductive phase, but when constraints dominate there is often a more gradual shift, i.e. indeterminate growth.

For parameters of the functions that make up natural mortality we reasoned from ecological processes as follows (values are given in Table 2). We first assumed that the cryptic appearance and burying behaviour imply a low baseline mortality (M_{fixed}) in the absence of activities that expose the individual, and also a low predation rate (low value of c). However, because plaice live on sandy bottom where any activity may be visible, we assumed that foraging behaviour is costly as any movements will reveal the position of the individual and attract the attention of predators. Similarly, we assumed that reproductive investment is relatively costly as large gonads would make a bulky appearance that is harder to hide, and require more oxygen for metabolic needs which could imply larger movements of the gill operculum or lead the flatfish to bury less deep or even lie on top of the sediments to ensure efficient respiration. Higher metabolic needs could also lead to poorer swimming abilities and reduce chances of successful escape from predators. Because plaice eggs contain many lipid droplets we assume that the cost of producing gonad tissue was twice that of somatic tissue (Dawson and Grimm, 1980 measured this to be ~1.75, our value also includes costs of behaviour etc.). The exact values were found by tuning parameter values until the model's predictions for several phenotypic patterns fit observations: age and size at maturity, growth trajectory, and maximum length and weight in the absence of fishing, as well as gonado-somatic index and total natural mortality.

3. Results

By breaking down natural mortality into several ecological relationships and trade-offs, the model illustrates how contrasting selection pressures may interact and influence fish life history strategies in diverse ways. Within the model, mortality rate emerges as a result of adaptive processes associated with growth and reproduction. Here we first report the effects of fishing mortality to establish a life history that resembles that observed for plaice today (Fig. 3). We then vary parameter values for the individual components of natural mortality. In Fig. 4 we assume a population of plaice that has partly adapted to fishing, and show results for the evolutionary endpoint at $F = 0.13$ year^{-1}. This is meant to reflect the current population of plaice, and predictions for how variation in natural mortality parameters might affect current populations. We then compare this with changes in life histories of varying the same parameters in an unfished stock ($F = 0$ year^{-1}; Fig. 5), corresponding to the pristine situation before fishing levels intensified.

The general expectation from theory is that increasing the general level of mortality will lead to earlier maturation (e.g., Law and Grey, 1989) and increased reproductive investment (e.g., Williams, 1966). When it comes to how growth will change, predictions vary and the evolutionary dynamics are poorly understood (Dunlop et al., 2009b; Enberg et al., 2012; Jørgensen and Fiksen, 2010; Miller, 1957). The model in this paper also varies parameters in several of the trade-offs that link survival to behavioural and life history traits — for these expectations are not clear-cut.

3.1. Baseline predictions and fishing mortality

Under increasing fishing mortality F, the key life history response was a reduction in size and age at maturation (Fig. 3). The model only considers females, and for these age of maturation was predicted to decline from 15 years in the total absence of fishing to 4 years at $F = 0.13$ year^{-1}, to 2 years at the highest fishing mortality investigated ($F = 0.4$ year^{-1}). Over the same increase in F, relative reproductive investment roughly doubled. These results are typical of the effect

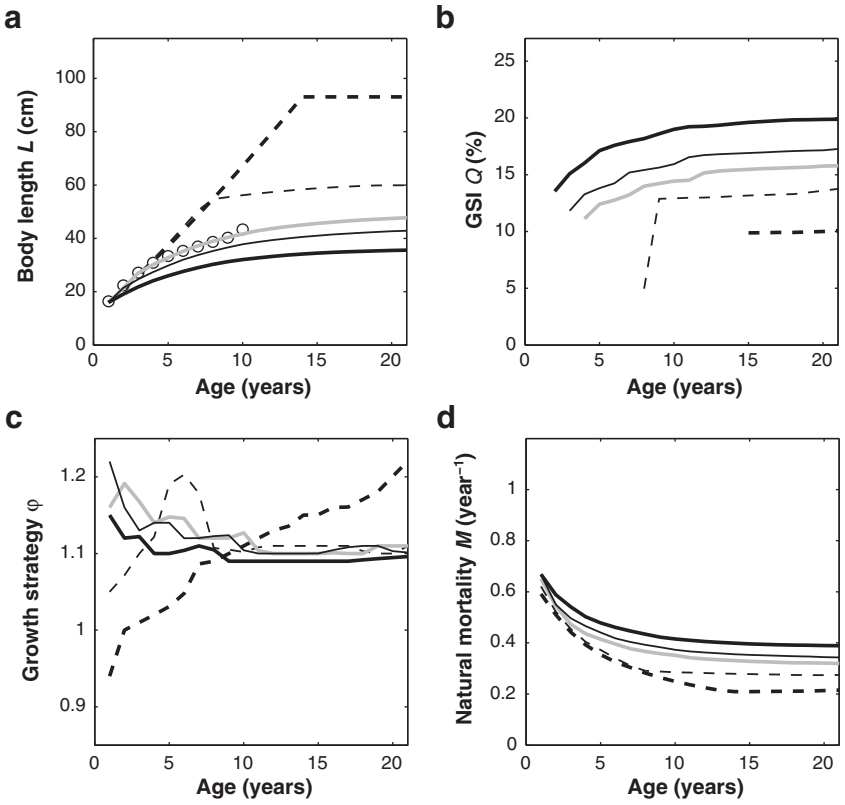

Fig. 3. Effects of fishing on life history traits in a flatfish, using parameters resembling plaice. a) Trajectories for length-at-age. Plaice may reach 7 kg and 100 cm according to fishbase, and this is the asymptotic size predicted in the absence of fishing (thick dashed line). At a fishing mortality of $F = 0.13$ year^{-1} (grey line), the model predicts a growth trajectory that is very similar to the average size-at-age reported by ICES for the period 1957–2010 (open circles). Growth trajectories level off the year prior to first reproduction, as resources are diverted towards gonads. At higher levels of fishing (thin solid, $F = 0.2$; thick solid, $F = 0.4$) the fish build larger gonads and become sexually mature earlier in life. The opposite responses are predicted at lower fishing mortalities (thin dashed, $F = 0.05$; thick dashed, $F = 0.0$). b) Predicted gonado-somatic index Q as percentage of total body mass. c) Predicted growth strategy φ. d) Effect of fishing on changes in natural mortality. In all panels, line style denotes fishing mortality as given in panel a.

of fishing upon life history strategies, whereby a reduction in age and size at maturation as well as an increase in reproductive effort are predicted by theory and observed empirically.

The model also predicted that as fishing mortality goes up, fish tend to take more risk to accelerate growth prior to maturation, despite increased mortality (Fig. 3c). After maturation, the level of risk depends on body size and is higher the larger the fish is, mainly because the cost of taking risks is lower as size-dependent predation is lower. The overall consequence of all these life history changes is that natural mortality goes up in the populations adapted to fishing, especially at older ages (Fig. 3d).

3.2. Changing size-independent mortality

The life history changes predicted by an increase in the basic level of size-independent mortality M_{fixed} follow standard life history theory: sexual maturity at smaller size and earlier age as well as increased reproductive investment. These changes were small in the life history adapted to fishing (Fig. 4a–d) but were qualitatively the same but of greater magnitude in the life history with no fishing (Fig. 5a–d). Note that because we assumed size-independent fishing, changing M_{fixed} is synonymous to changing F. Prior to maturation, a riskier growth strategy φ was also observed as a consequence of higher M_{fixed} (Figs. 4c, 5c). After maturation the growth strategy was very similar across different values of M_{fixed}, but as adult size is smaller at higher M_{fixed}, the same growth strategy will result in higher mortality because of the size-dependent scaling that affects also growth-related mortality (Eq. (4)). Changing M_{fixed} led to a general increase in natural mortality across all ages (Figs. 4d, 5d).

3.3. Changing size-dependent predation mortality

By increasing the coefficient c that determines the level of size-dependent predation (Figs. 4e–h, 5e–h), the model predicts a large response towards earlier maturation at smaller size. As increasing c increases the overall level of mortality, this is as expected from theory. The value of c also has a strong effect on the growth strategy, as the benefits that follow from having more resources incur a higher mortality rate when size-dependent predation is higher. Here it can also be seen, when c is low, that the growth strategy φ increases during the juvenile phase as predation declines, following from the size-dependent scaling of predation mortality (Figs. 4g, 5g). Because changing the value of c changes predation rates, the effect on natural mortality rates is strong as expected (Figs. 4h, 5h).

3.4. Changing food availability — the risk half-saturation

The risk half-saturation parameter $h_{1/2}$ can be described as referring to an ecological relationship, where lower values translate to an increase in food availability such that the same resources can be obtained at lower foraging risk. Consequently, safer behaviours are observed when $h_{1/2}$ takes lower values (Figs. 4k, 5k), the increased food availability means that growth can be faster, and the model predicts maturation later and at larger size as well as with a lower investment into reproduction (Figs. 4i–j, 5i–j). In terms of evolutionary fitness the model predicts that it is better to divert energy towards reproduction rather than growth when food availability is limited. The value of $h_{1/2}$ has consequences for the natural mortality rate across all ages (Figs. 4p, 5p).

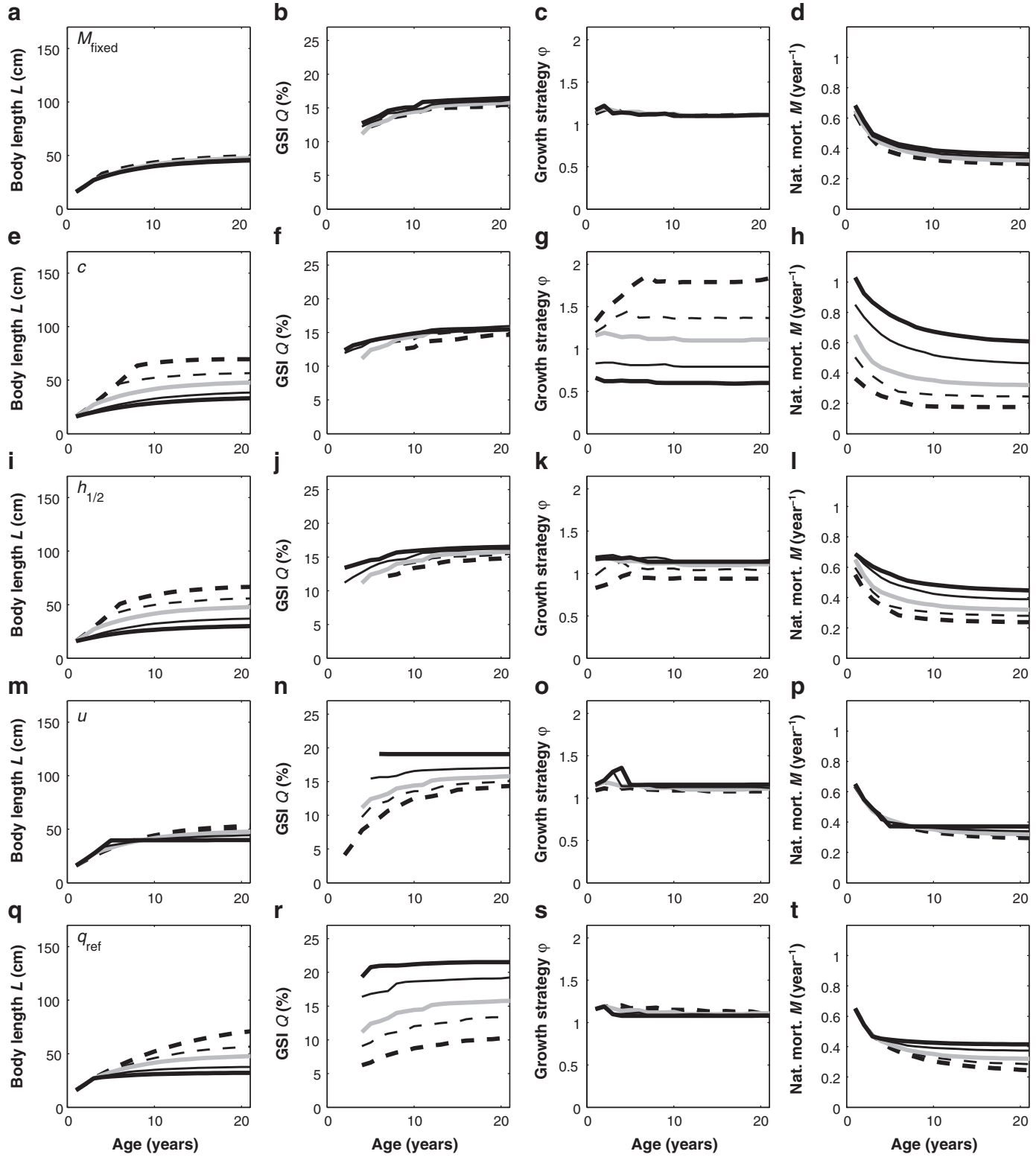

Fig. 4. Predicted lifetime trajectories in a fished flatfish population given variation in parameters of 5 components of natural mortality. a–d) fixed baseline mortality by varying M_{fixed}; e–h) size-dependent predation mortality by varying c; i–l) level of risk required for foraging by varying $h_{1/2}$; m–p) mortality cost of being at the spawning grounds by varying u; q–t) cost of carrying gonads by varying q_{ref} (note that for q_{ref}, higher values imply lower mortality). The grey line is a standard set of parameters set to resemble plaice and with a fishing mortality $F = 0.13$ year^{-1}. Line style denotes varying parameter values (in percent of standard value): thick dashed (50%); thin dashed (75%); grey (100%); thin solid (150%); thick solid (200%), except for M_{fixed} (panels a to d) which was varied more to make differences visible (0%, 50%, 100%, 200%, and 300%, respectively).

3.5. Changing mortality at the spawning grounds

The spawning activity itself may be linked to mortality, for example due to migrations, mate search, courtship, or spawning behaviour. In our model this is implemented as a fixed mortality that applies to all reproductive individuals but not to immatures, with the level given by the parameter u. When spawning becomes more costly in terms of mortality (higher u), maturation is delayed and takes place

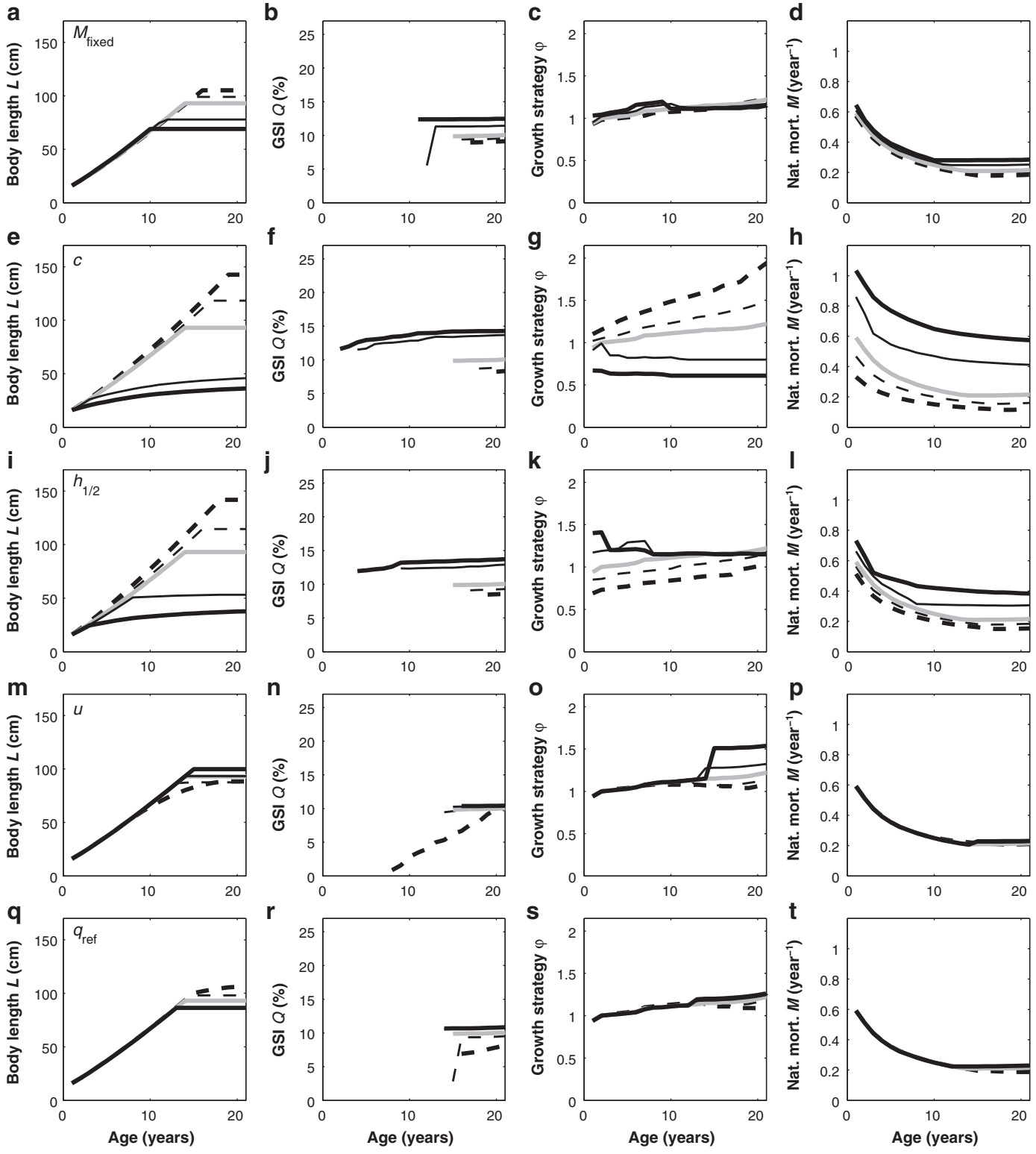

Fig. 5. Responses to changing natural mortality components in the absence of fishing. See caption of Fig. 4 for legend; the only difference is that $F = 0.0$ year^{-1}.

at larger size and reproductive investment goes up (Figs. 4m–n, 5m–n). As immatures the model predicts the same growth strategy regardless of u, but after maturation the growth strategy depends on size so that fish that mature larger are predicted to grow faster (Figs. 4o, 5o). The combined effect on natural mortality is small, but of larger magnitude for early-maturing life histories (compare Figs. 4p and 5p).

3.6. Mortality related to gonads

The final contribution to natural mortality we included was a trade-off between the intensity of reproductive investment and survival. We assumed an accelerating cost of reproductive investment, and varied the parameter q_{ref}, which is the broad-sense gonado-somatic index that incurs the same mortality as the size-dependent predation

component (Fig. 2c). This parameter only matters from age at first reproduction and onwards, but if the costs of reproductive investment become high then maturation itself can be delayed (e.g. Fig. 5q, r). A lower value for q_{ref} implies a higher mortality cost of reproductive investment, and leads to smaller gonads (Figs. 4r, 5r) and a higher larger body sizes post maturation because less resources are used for gonads (Figs. 4q, 5q). The model's prediction for the growth strategy depends on the fishing mortality: in the absence of fishing the larger-bodied fish (low q_{ref}) take less risk and prioritize survival until future spawning seasons (Fig. 5s), but when adapted to fishing this becomes a less viable option and more risk is accepted among adult fish (Fig. 4s). This is likely partly driven by the fishing mortality itself (as explained in Fig. 1) and partly because the adult size differences across parameter variation in q_{ref} are larger in the life histories adapted to fishing (compare Figs. 4q and 5q). Interestingly, as the mortality from having large gonads is increased, the total natural mortality declines, and most so in the population adapted to fishing.

4. Discussion

In this paper we use a model as a conceptual tool to investigate how fishing and changes in ecological relationships may influence the selective landscape on fish life histories and behaviour. Our main message is that natural mortality is not an externally imposed factor, but emerges from the behavioural and life history tactics of individuals, and that these strategies may be under selection or shaped by anthropogenic or ecological relationships that one can study and monitor over time.

4.1. Fishing-induced evolution of a broader set of traits

The field of fishing-induced evolution has, probably rightly, historically focused on age at maturation because early trends were detected, maturity status and size are quantifiable and relatively easy to observe, and there was available theory to explain the changes (reviewed e.g., by Dieckmann and Heino, 2007). But there is a need to considerably broaden the perspective on possibly evolving traits. In some cases data exist to suggest fishing-induced trends that likely have an evolutionary component: morphology, particularly condition, in salmonids (Ricker, 1981, 1995), lake whitefish (Handford et al., 1977), and common carp (Wohlfarth et al., 1975); increased fecundity in Atlantic cod (Yoneda and Wright, 2004), lake whitefish (Thomas et al., 2009), and perhaps European plaice (Rijnsdorp et al., 2005); growth in Pacific salmonids (Ricker, 1981, 1995), Atlantic cod (Swain et al., 2007) and lake whitefish (Thomas and Eckmann, 2007); and seasonal timing e.g. of salmon runs (Quinn et al., 2007). Beyond these, theory has suggested further traits that might evolve, e.g. migration distance (Jørgensen et al., 2008; Opdal, 2010), hermaphroditism (Sattar et al., 2008), and skipped spawning (Jørgensen et al., 2006). In this paper we follow Jørgensen and Fiksen (2010) and use general ecological relationships that can reflect multiple traits. For example, the growth strategy φ can represent foraging behaviour, meal size, digestive physiology, or internal trade-offs such as how resources are allocated between e.g. immune defence, integument or armoury on the one side versus available resources for growth on the other (Enberg et al., 2012). This makes the model more general as it indicates which groups of functions are sensitive to different types of mortality changes, but it also leaves a remaining challenge, often species-specific, of identifying exactly which traits are actually under current selection.

4.2. A diversified view on reproductive mortality

A particular contribution of the model in this paper is the partitioning of reproductive mortality into a fixed threshold mortality (parameter u; Figs. 4m–p, 5m–p), due to e.g. exposure and migrations, and a variable mortality relating to the actual reproductive investment (parameter q_{ref}; Figs. 4q–t, 5q–t). The threshold mortality has often been used in life history models for fish (e.g. Hutchings, 1999, 2005; Jørgensen and Fiksen, 2006), whereas a trade-off between the intensity of reproduction and survival has a long history in life history theory (Gadgil and Bossert, 1970; Williams, 1966).

Both an increase in the fixed spawning cost (higher u) and the gonad-specific cost (lower q_{ref}) cause later maturation, but they have the opposite effect on reproductive investment. A high threshold cost of reproduction will cause high gonad mass because it is not beneficial to accept the costs of spawning if the reproductive output is low. In contrast, a high cost of large gonads will tend to select for smaller gonads and rather spawning over many consecutive years.

There is also an interesting interaction with fishing (compare Figs. 4 and 5). In the pristine stock, increasing both types of reproductive mortality caused an increase in adult size. In the population adapted to fishing, in contrast, increasing the threshold cost of reproduction caused smaller adult size for a wide range of age-classes whereas increasing the cost of gonads consistently increased adult size. Understanding which ecological and physiological relationships and interactions cause reproductive mortality is therefore crucial to predicting species-specific responses to fishing or environmental change, and may be sensitive to parameter values of the involved trade-offs.

The differential response to types of reproductive mortality also has consequences for how one considers the potential evolutionary impact of a spawner fishery. Because increased fishing mortality on sexually mature fish (analogous to increasing u) leads to weak selection for later maturation these have been considered as having less evolutionary impact than fishing mortality targeting juvenile or all age-classes (first highlighted by Law and Grey, 1989). We extend this perspective by showing that not all reproductive mortalities act in the same way. For example, a seasonally short and concentrated fishery may affect individuals the same regardless of their reproductive investment (as u), whereas a prolonged fishery may target those fish that reproduce more intensely and stay longer at the spawning grounds (similar to q_{ref}). Different fishing gear may also be more u-like, for example purse seines that catch entire schools more or less regardless of individual variation, or more q_{ref}-like, for example gillnets that target round body shapes or trawls where poor swimming performance from large gonads can increase risk of capture.

It is worth contemplating how increased mortality of carrying gonads led, somewhat surprisingly, to quite a substantial reduction in predicted natural mortality in the population adapted to fishing (Fig. 4t). This happens because building smaller gonads and rather prioritizing repeated spawning becomes beneficial. This effect may be worth looking further into for its potential relevance for evolutionary optimal fisheries management.

4.3. The ecosystem embedding

There are also many other drivers beyond fishing that may cause temporal changes in the phenotypic traits of fish. Considerable attention has been devoted to disentangle effects of fishing from those of, e.g., temperature and nutrient loading (e.g. Grift et al., 2003; Opdal, 2010; Rijnsdorp and van Leeuwen, 1996; Swain et al., 2007; Thomas and Eckmann, 2007; Thomas et al., 2009). For the model in this paper, the level of size-dependent predation and the risk associated with foraging and growth can be viewed as having strong influences from ecosystem properties, and they were, interestingly, the parameters that led to the largest changes in predicted life histories.

Of particular relevance to flatfish is that benthic trawling, and particularly beam trawls or tickler chains that dig into or stir up the sediments, may make food available by destroying or uncovering

organisms or, over time, favouring benthic species with higher growth and production (Hiddink et al., 2008). In the terms of our model, more available food implies that same amount can be obtained at a lower foraging risk, which was studied by varying the parameter $h_{1/2}$ (Figs. 4i–l, 5i–l). Everything else being the same, our model predicts that increased food availability has a strong effect on optimal life histories (faster growth, larger adult size, later maturation, and lower GSI) as well as a relatively strong reduction in natural mortality across all life stages.

Changes in the abundance of predator communities may influence prey species. This is complicated by how predators may affect different life stages differentially, with consequences for co-existence (de Roos et al., 2008), or even be kept in check by what is normally considered their prey (Persson et al., 2007).

Our model suggests that a change in size-specific predation has large effects on all life history traits and mortality. Many of the relationships in our model propagate the effects of size-dependent predation, for example when more risky foraging or building larger gonads expose individuals more to predation. This suggests that, although extremely difficult, there is a need for strengthened focus on how changes in communities (e.g. Jennings et al., 1999) influence predation pressures (Gislason et al., 2008), which affect plasticity and evolution of life history and behaviour, which in turn feed back to community structure again. Some researchers have started this daunting challenge (e.g. Abrams, 2009; Abrams and Matsuda, 2005; Gårdmark et al., 2003).

4.4. Observed changes

Most traits are in a trade-off with survival. It can therefore be that the prediction of an increased natural mortality is robust and a signal that something is going on. Increased mortality could follow from earlier maturation at smaller size, which is the general expectation from fishing-induced evolution, but it could also follow from other learned or evolved trait changes.

There is evidence that natural mortality has gone up in some heavily harvested stocks, for example some Atlantic cod stocks off Canada (Shelton et al., 2006; Swain, 2011; Swain and Chouinard, 2008). The increase in grey seal abundance may be a significant contributor to this (Benoît et al., 2011; Savenkoff et al., 2007), but concomitant changes in life history parameters (Swain, 2011; Swain and Chouinard, 2008) may indicate a role for fishing-induced adaptations too (Hutchings, 2005; Jørgensen and Fiksen, 2010; Swain, 2011). Can it be that changes in cod behaviour and life history strategies now expose cod more to their seal predators, leading to more food for seals and increasing seal populations? If so, there could be a positive feedback loop where elevated mortality from fishing or seal predation causes higher seal predation. Such positive feedbacks could explain why cod populations have not recovered, despite relaxed harvesting. Comparing expected rates of fishing-induced evolution towards riskier strategies and reverse evolution in the absence of fishing may shed light on the lack of recovery in these stocks.

To identify the specific traits that evolve in specific populations is a particular and non-trivial challenge. In hindsight it might be that we scientists might be perceived as utterly naïve because we didn't recognize early on the really important traits that were undergoing evolutionary change. It can be timely to remind oneself of Leslie Orgel's second rule: Evolution is cleverer than you are. Thus, our imagination is likely preventing us from having the appropriate expectations of what is going on. This is particularly true for models, which need to assume traits and relationships, with the consequence that traits not assumed to be evolvable in a model will, by definition, not be predicted to evolve. Maybe the model in this paper, with general classes of mechanisms that absorb the selection pressure instead of particular traits, can help broaden the perspective and motivate researchers with more specialist knowledge to think about how their focal traits or species might respond to the rapid changes in the seas.

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