Vol. 335: 249–251, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published April 16

THEME SECTION

Disentangling the causes of maturation trends in exploited fish populations

Idea and coordination: **C. Tara Marshall, Howard I. Browman**

CONTENTS

Introduction

C. Tara Marshall[1,*], Howard I. Browman[2]

[1]School of Biological Sciences, University of Aberdeen, Tillydrone Avenue, Aberdeen AB24 2TZ, UK
[2]Institute of Marine Research, Austevoll Research Station, 5392 Storebø, Norway

Evolutionary theory predicts that selective harvesting will shift the frequency of genes for heritable traits towards those that express well-adapted phenotypes (Law 2007, this Theme Section). Applying this logic, commercial fisheries that select on the basis of size could reasonably be expected to favor the genotype for maturation at smaller sizes (and/or younger ages), given that rapidly maturing individuals will be more likely to reproduce prior to capture. This form of fisheries-induced evolutionary response is a parsimonious explanation for the observed long-term trend towards earlier maturation that has been observed in several commercially exploited fish stocks (Rijnsdorp 1993, Trippel 1995). Unfortunately, it is not possible to test this hypothesis directly, because the genes associated with maturation have not been isolated.

Probabilistic maturation reaction norms (PMRNs) (Heino et al. 2002) have been proposed as a statistical tool for helping to disentangle genetic from plastic effects on maturation (reviewed by Dieckmann & Heino 2007, this Theme Section). For a given cohort, the PMRN describes the maturation probability of individuals that have survived to reach a given age and size. A midpoint curve, illustrating the age and size at which the maturation probability is 50%, is often used to summarise the PMRN. A shift in cohort-specific PMRNs over time is interpreted as being consistent with (although not direct evidence of) a genetic change at the population level. For example, a shift through

*Email: c.t.marshall@abdn.ac.uk

time and across cohorts towards midpoints located at smaller sizes and younger ages agrees with the expected life history response to high rates of selective exploitation.

On the basis of directional shifts in PMRNs fit to historical data, a number of studies have concluded that fisheries-induced evolution in maturation rates has occurred (see Dieckmann & Heino 2007). For example, PMRNs were used to infer that a fisheries-induced evolutionary response in the maturation rates of the Northern cod stock was rapid and preceded stock collapse (Olsen et al. 2004). Fisheries-induced changes to the genetic structure of a population alter the genetic diversity in life-history traits (Conover & Munch 2002, Kenchington et al. 2003). Such structural changes to the population are regarded as rapidly induced, but slow to reverse (Law & Grey 1989, Barot et al. 2004). Further, since the individual growth rate of many fishes slows following maturation, a decrease in the size at which individuals mature could result in reduced yields (Law & Grey 1989). For these reasons, fisheries managers are increasingly being challenged to adopt so-called Darwinian fisheries approaches to management (Conover 2000, Kenchington et al. 2003, Conover et al. 2005).

Despite the intuitive appeal of fisheries-induced evolution, the PMRN-based evidence is unavoidably circumstantial, due to the lack of unambiguous genetic evidence. Further, the majority of PMRNs published to date employ observations from fisheries databases to estimate the probability of a given cohort becoming mature as a function of age and length. While these databases are potentially long enough to detect evolutionary responses, they contain information on a limited suite of biological variables (typically age, length, weight, sex and stage of maturation) measured at discrete times. These databases do not contain information which would allow the sequence of hormonal and morphological changes that precede maturation (see Thorpe 2007, Wright 2007; both in this Theme Section) to be resolved in detail. Inferences made using the PMRN approach are therefore based on the assumption that age and length at the point at which individuals become mature accurately reflect the physiological status of immature individuals at discrete time points when they are 'deciding' whether to proceed with, or delay, maturation. These developmental decisions are influenced by environmental conditions (Aubin-Horth et al. 2006) and by the magnitude of stored energy reserves in relation to anticipated energy expenditures (Thorpe et al. 1998). The assumption that age and length at the point of maturation are an accurate representation of the physiological processes that have occurred throughout the immature stage is difficult to test for wild stocks, although rate-based PMRNs have been developed for organisms studied in captivity (Van Dooren et al. 2005). If this assumption is invalid, then it is possible that the environmental effects on decisions regarding maturation have not been accurately controlled for by PMRNs.

The goal of this Theme Section is to present some of the latest information on physiological processes leading to maturation in fishes and to assess the strengths and weaknesses of the PMRN approach in light of this information. Contributions were sought from individuals with experience in experimental and/or field research in fish growth and maturation, as well as experts in evolutionary aspects of maturation. Their remit was to discuss questions such as

- Is the PMRN approach suitable for diagnosing genetic changes in maturation rates, and if not, what method would be suitable?
- Is the current evidence for fisheries-induced evolution conclusive, and if not, why not?
- Is there contradictory evidence?
- Have environmental effects on maturation been adequately accounted for?
- Can the physiological basis for maturation be captured by maturation reaction norms?
- How can maturation reaction norms be made more realistic?
- What type of studies would enhance our understanding of genotypic control of maturation?

The preceding questions indicate that the Theme Section was initially conceived as an outlet for viewpoints other than those of proponents of the PMRN approach — we felt that the viewpoint of the latter group was well-represented by a number of recent publications. Once the roster of contributors was confirmed, Ulf Dieckmann and Mikko Heino — the preeminent developers of the PMRN approach to fisheries data — were informed of the Theme Section and provided with a list of contributors and titles. They were invited to contribute and generously agreed to prepare a historical review that summarized their own perspective. We are most grateful to Ulf and Mikko for accepting our invitation, and for providing such a thorough and scholarly article. It will do much to clarify understanding of the PMRN approach.

Although a minority of authors exchanged drafts of their manuscripts, this was not coordinated. Therefore, the Theme Section should not be viewed as an interactive debate on the PMRN approach. The Theme Section was not confined entirely to the PMRN approach: the contribution by Law (2007) highlights the importance of having older age-classes in the population. When read together, the contributions to this Theme Section will surely impress upon readers that disentangling the multiple causes of maturation trends in

fish populations is an inherently complex task. Consequently, it is not surprising to find a range of perspectives on what factors are of principal importance. Nonetheless, there was a degree of convergent thinking amongst the contributors regarding the proximate cues for maturation that are of critical importance. Criticisms of the PMRN approach to analyzing maturation trends centered on the possibility that proximate cues for maturation were not adequately accounted for. The contribution by Dieckmann & Heino (2007) is distinctive in that it does not cover the physiology of maturation, but instead concentrates on a wide range of evolutionary and ecological effects on maturation. Their contribution makes clear that the PMRN approach to analyzing trends in maturation arose from a consideration of the ultimate causes of maturation, rather than proximate cues. These 2 contrasting viewpoints can be considered as yet another variant of the debate over the relative importance of bottom-up (environment impacting maturation through physiological effects) and top-down (fisheries as selective predation impacting maturation through genetic effects) regulation of life histories. Reconciling the 2 viewpoints, such that inter- and intra-annual variation in the physiological status of immature individuals is eliminated as a potentially confounding element in the reaction norm analysis, would strengthen conclusions regarding both the time-scale and magnitude of the fisheries-induced evolutionary response in maturation rates.

There are several encouraging signs that the large gap between studies investigating proximate and ultimate sources of variation in maturation rates is being bridged. For example, 3-dimensional PMRNs have been developed to account for the effect that condition (as represented by a morphometric index) has on maturation of North Sea plaice (Grift et al. 2007). Morita & Fukuwaka (2007, this Theme Section), Kraak (2007, this Theme Section) and Marshall & McAdam (2007, this Theme Section) fit PMRNs to data, indicating the willingness of biologists to evaluate the strengths and weaknesses of PMRNs from a critical but open-minded viewpoint. These recent studies demonstrate that a combination of approaches, integrating biology, physiology and ecology with analytical techniques and theory, will be required to disentangle the sources of variation in the maturation rates of exploited fish populations.

Acknowledgements. We thank the contributors for their patient and timely efforts to comply with our editorial suggestions. We thank U. Dieckmann and M. Heino for comments on an earlier draft. H.I.B.'s research, and his editorial activity, are supported by the Institute of Marine Research, Norway, the Research Council of Norway, and the Inter-Research Science Center.

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Vol. 335: 253–269, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published April 16

OPEN ACCESS

REVIEW

Probabilistic maturation reaction norms: their history, strengths, and limitations

Ulf Dieckmann[1,*], Mikko Heino[2,1,3]

[1]Evolution and Ecology Program, International Institute for Applied Systems Analysis, Schlossplatz 1, 2361 Laxenburg, Austria
[2]Institute of Marine Research, PO Box 1870 Nordnes, 5817 Bergen, Norway
[3]Department of Biology, University of Bergen, PO Box 7800, 5020 Bergen, Norway

ABSTRACT: Probabilistic maturation reaction norms (PMRNs) are emerging as a flexible and general tool for characterizing phenotypic plasticity in maturation schedules. Describing an organism's probability of maturing as a function of its age and size, PMRNs offer several beneficial features: (1) PMRNs overcome systematic biases that previously marred the estimation of deterministic maturation reaction norms for populations with probabilistic growth and maturation; (2) PMRNs remove the effects of varying mortality rates and average juvenile somatic growth rates from descriptions of maturation schedules; (3) PMRNs are defined at the level of individuals and can thus be treated as phenotypes when applying methods of quantitative genetics; (4) PMRNs serve as indispensable ingredients in process-based dynamical models of a population's age and size structure; and (5) PMRNs are readily extended to include effects on maturation of individual or environmental factors other than age and size. Owing to this combination of features, PMRNs allow many effects of phenotypic plasticity to be stripped away from the description of maturation schedules, so that residual trends are suggestive of genetic adaptation in maturation schedules. Here we review the historical developments that led to the introduction of PMRNs and address frequently asked questions about their interpretation, utility, and application.

KEY WORDS: Maturation · Reaction norm · Phenotypic plasticity · Genetic adaptation · Fisheries-induced evolution · Life-history transition · Energy allocation · Micro- and macroenvironment

INTRODUCTION

Maturation is the most important life-history transition in plants and animals. Whereas some characteristics of organisms (including their length, weight, and condition) change gradually during their lifetime, and others (including age, as well as length in many species) even change monotonically, maturation involves a transition between 2 qualitatively different states, turning juveniles into adults. Understanding the proximate and ultimate determinants of maturation is one of organismal biology's central challenges. As must be expected, there are ecological and evolutionary determinants of maturation. The ecological determinants operate through phenotypic plasticity: the realized maturation schedules of individual organisms thus depend on the environmental conditions they are, or have been, experiencing. In contrast, the evolutionary determinants operate through genetic adaptation: the maturation schedules of populations of organisms thus depend on the selection regimes they have experienced.

Integrating the ecological and evolutionary dimensions of maturation into a synthetic framework for research has not been easy. Studies of phenotypically plastic maturation have not usually addressed evolutionary questions. Conversely, studies of genetic adaptation in the maturational process have not often

*Email: dieckmann@iiasa.ac.at

accounted for phenotypic plasticity. Partly, this simply reflects the general difficulty in evolutionary ecology of bridging the gap between the timescales required for studying ecological and evolutionary change. Because of the disparate durations involved, ecological change is mostly directly observable in empirical analyses, whereas understanding evolutionary change often requires more complex, indirect, and/or theory-based analyses. Experimental protocols, statistical methods, comparative approaches, and theoretical models jointly accounting for phenotypic plasticity and genetic adaptation in maturation schedules thus remained beyond the research community's reach for a long time.

Here we discuss an approach, based on so-called probabilistic maturation reaction norms (PMRNs), that we believe offers the potential for overcoming these difficulties. The roots of this approach extend more than 2 decades into the past. First, we summarize background information on plastic and genetic maturation adaptation (see below); the subsequent section offers a historical overview, revisiting the salient scientific developments and reflecting on their significance in a broader context. The reaction-norm approach to maturation is also currently stimulating interesting discussions. In the third section below, we address the main questions and potential misunderstandings we have encountered in our dialogues with colleagues.

PLASTIC AND GENETIC MATURATION ADAPTATIONS

Variability in maturation is ubiquitous among species and individuals. This is just as expected, since maturation schedules are based on energy allocation strategies; such strategies are flexible, even within a given morphological *bauplan* or physiological setup. Moreover, differential energy allocations have substantial fitness implications, and optimal energy allocations change with environmental conditions. In fact, the flexibility of maturation schedules is so pronounced that researchers have taken to borrowing terminology from behavioral studies, and figuratively speak of a 'maturation decision' or 'decision to mature': After prioritizing energy allocation to somatic growth as a juvenile, when should an organism become adult and start investing in reproduction?

The balance between the fitness benefits of early maturation (increased survival to first reproduction and decreased generation time) and late maturation (increased body size and enhanced effective fecundity) involves several classical life-history tradeoffs. These include the tradeoffs between (1) current reproduction and survival, (2) current reproduction and growth, and (3) current reproduction and condition (Stearns 1992). Maturation schedules adapt to environmental conditions under the constraints imposed by these tradeoffs, and therefore are far from arbitrary.

To understand maturation responses to environmental conditions, it is helpful to distinguish between 3 aspects of the environment: (1) The ancestral environment, characterized by environmental conditions that remain stable over generations and to which a population has had time to adapt through natural selection; (2) the macroenvironment (Gavrilets & Scheiner 1993, Gavrilets & Hastings 1994, Hartl & Clark 1997), characterized by environmental conditions with predictable phenotypic effects collectively experienced by groups of individuals; (3) the microenvironment, characterized by environmental conditions that are specific to individuals. Along this gradient of environmental predictability, maturation responses are determined by different means: responses to the ancestral environment come about through genetic adaptation, responses to the macroenvironment are based on phenotypic plasticity, while responses to the microenvironment are typically regarded as developmental noise.

After genetic adaptation to the ancestral environment has run its course, the maturation schedule of a population in a habitat imposing, for example, low juvenile mortality will differ markedly from that of a population typically experiencing high juvenile mortality. As a result of phenotypic plasticity in response to the macroenvironment, maturation decisions will be affected by the environmental conditions individuals encounter. In terms of mechanism, the latter occurs for 3 related, yet separate, reasons. (1) Current environmental conditions constrain current energy allocations; for example, when energy intake is low, necessary allocations to maintenance will leave little room for allocations to growth or reproduction. (2) In the presence of positive temporal autocorrelations, the recent past offers valuable clues for anticipating environmental conditions in the immediate future; for instance, one month with good food supply is likely to be followed by another such month. (3) Past environmental conditions are bound to leave their mark on an individual, in terms of states such as length, weight, and/or condition, subsequently implying different optimal allocation strategies. For illustration, consider the benefits of allocating energy to survival, growth, condition, and current reproduction, which will trade off differently for a well fed specimen compared to an individual with poor body condition.

The considerations above make it evident that genetic and plastic adaptations in maturation schedules are inexorably intertwined. Whenever the relative roles of these 2 types of adaptation are not already known, the comprehensive understanding of maturation schedules will require an integrative approach.

HISTORICAL OVERVIEW

Flexible responses and temporal trends in the maturation schedules of fishes have been described in many empirical studies. The references mentioned below are but a small sample.

Maturation trends

Alm (1959) was perhaps the first fisheries scientist to systematically examine plasticity in maturation in a major species of fish. Specifically, Alm investigated how size at maturation in Eurasian perch *Perca fluviatilis* varied with the somatic growth rates that individuals experienced during their juvenile period. Based on his experimental analyses, Alm suggested that size at maturation in this species was small for fast-growing individuals, larger for individuals experiencing intermediate juvenile growth rates, and smaller again for slow-growing individuals. The last relationship is particularly relevant for species exhibiting stunting: with a low expectation of future growth, maturing at an otherwise suboptimal small body size happens to be the best option available.

Early studies indicating that fishing can induce reductions in the mean length or age at maturation of target species include the following: Rollefsen (1938, 1953), Hansen (1949), Powles (1958), Garrod (1967), Hylen & Dragesund (1973), Oosthuizen & Daan (1974), Borisov (1978), Ponomarenko et al. (1980), and Hylen & Rorvik (1983) on Atlantic cod *Gadus morhua*; Pitt (1975) on American plaice *Hippoglossoides platessoides*; Schaffer & Elson (1975) on Atlantic salmon *Salmo salar*; Handford et al. (1977) on lake whitefish *Coregonus clupeaformis*; Tikhonov (1977) on yellowfin sole *Limanda espersa*; Templeman et al. (1978) and Templeman & Bishop (1979) on haddock *Melanogrammus aeglefinus*; and Ricker (1981) on Pacific salmon *Oncorhynchus* spp.

In a comprehensive analysis of Atlantic groundfish stocks in the southern Gulf of St. Lawrence and on the Scotian Shelf, Beacham (1983a,b,c,d,e,f) reported significant declines (30 to 50%) in mean age and length at maturation during extensive commercial exploitation in the 1960s and 1970s. The species analyzed were Atlantic cod *Gadus morhua*, haddock *Melanogrammus aeglefinus*, American plaice or long rough dab *Hippoglossoides platessoides*, yellowtail flounder *Limanda ferruginea*, witch flounder or grey sole *Glyptocephalus cynoglossus*, and greater argentine *Argentina silus*.

Beacham (1987, p. 152) proposed that researchers

> may have difficulty accepting that size and age at maturity of groundfish is a dynamic character that responds to changes in population size or fishery selection.

Following up on earlier suggestions by Borisov (1978), Beacham (1987, p. 150) also explicitly highlighted the potential for fisheries-induced evolution in maturation schedules:

> To the extent that size and age at maturity are genetically determined, fish which mature at smaller sizes or younger ages have a selective advantage during intensive fisheries. Genotypes that reproduce before being fully recruited to the fishery can have a selective advantage over those that mature at larger sizes or older ages.

He judiciously concluded that

> one unresolved issue is whether the changes in size or age at maturity are a result of a density-dependent response to decreased stock size or a result of genetic change within a population.

Based on theoretical predictions of mortality-induced maturation evolution (Law 1979), and encouraged by matching experimental results in the water flea *Daphnia magna* (Edley & Law 1988), Law & Grey (1989) suggested that the determination of maximal sustainable fisheries yields should account for fisheries-induced evolution. Thirty years earlier, a review by Miller (1957) had still concluded that there was little evidence for any genetic adaptation caused by fishing. Popular articles by Sutherland (1990) and Law (1991) informed a wider audience about the perils of fisheries-induced evolution in general, and of fisheries-induced maturation evolution in particular. Similar caveats had already been raised by Nelson & Soulé (1987) and were further explored at the scale of an edited volume by Stokes et al. (1993) and in the form of reviews by Smith (1994) and Sheridan (1995). These accounts made it unmistakable that understanding fisheries-induced maturation trends was no longer a challenge of fundamental biological interest alone, but was also begging ponderous questions of considerable socio-economic consequence for fisheries science and management. This conclusion has lost no force until today.

Reporting on the longest yet documented trends in fish maturation, Jørgensen (1990) (see also Godø 2000) showed that in Northeast Arctic cod *Gadus morhua* both age at maturation and size at maturation had been declining consistently since the 1920s. A similar long-term decline, between the periods 1904–1911 and 1960–1990, was demonstrated for North Sea plaice *Pleuronectes platessa* by Rijnsdorp (1989, 1993a,b). Publication of these seminal investigations let it appear increasingly unlikely that observed maturation trends could be interpreted as mere phenotypically plastic responses to decadal environmental fluctuations.

An overview by Trippel (1995) helped to draw attention to the ubiquity of maturation trends in marine fish populations: all around the globe, exploited fish were maturing at smaller size and younger age. Trippel summarized the 2 alternative hypotheses for explaining fisheries-induced maturation trends that had

shaped the earlier discussions. The first hypothesis was based on phenotypic plasticity: fishing reduces stock biomass and density, and thus allows the remaining fish to grow faster and mature earlier. Such effects of fishing have traditionally been referred to as compensatory responses. The second hypothesis was based on genetic adaptation: fishing favors genotypes maturing at younger ages and smaller sizes. Such an explanation prominently implicates fisheries-induced evolution. Trippel (1995, p. 767) drew the conclusion that

> despite arguments and evidence that evolution might be occurring in exploited fish populations, fishery managers have not considered it seriously.

This was so even though, according to his evaluation,

> shifts in maturity are most likely to be a consequence of a mix of factors having both compensatory and genetic origins

(Trippel 1995, p. 766–767). Trippel also explained that studies of fisheries-induced maturation evolution would need to account for the fact that

> genetic influence on age at maturity acts in the form of a capability with rather wide latitude (i.e. phenotypic plasticity) and not by strict age-specific inheritance of age at maturity from parent to progeny

(Trippel 1995, p. 766).

Strategies for disentangling phenotypic plasticity and genetic adaptation

With the recognition thus becoming inescapable that neither phenotypic plasticity nor genetic adaptation could be left out of empirical or theoretical analyses of fisheries-induced maturation trends, appropriate methods for first respecting and then disentangling phenotypic plasticity and genetic adaptation had to be found. Over the past 2 decades, 4 qualitatively different strategies — respectively based on experimental manipulation, comparative studies, countergradient or countertrend variation, and reaction norm analysis — have been put forward. We outline these alternative approaches in turn below.

The first strategy operates through controlled experimental manipulation. The best example is provided by a remarkable line of research extending over the past 25 years, through which Reznick and colleagues investigated life-history variation in the Trinidadian guppy *Poecilia reticulata* (see Reznick & Ghalambor 2005 for a brief overview). Combining an experimental approach in the field and laboratory with comparative studies, Reznick and colleagues demonstrated that maturation schedules in the small, fast-growing, and short-lived guppy systematically varied with predation-induced mortality (Reznick & Endler 1982), that these differences were partly genetic (Reznick 1982, Reznick et al. 1996) and partly plastic (Reznick 1990), and that considerable maturation evolution could occur in the course of just 7 to 18 generations (Reznick et al. 1990, 1997). Phenotypic plasticity in maturation schedules of Trinidadian guppies was also found to go beyond the simple pattern that would result if size at maturation were fixed and age at maturation merely varied inversely with juvenile growth rate (Reznick 1990).

A second strategy is based on comparative studies. After documenting systematic differences in maturation between different local populations as a first step, hypotheses about the underlying causes are established in a second step by identifying factors that significantly covary with maturation across populations. As a third step, additional information must be used to evaluate the extent to which putative local adaptations to the identified factors are genetically based. This third step can involve the following: (1) common-garden rearing, as in a study by Goto (1993) on maturation plasticity in river sculpin *Cottus hangiongensis*; (2) successive exclusion of alternative explanations relying on phenotypic plasticity, as in a study by Johnson & Belk (1999) on maturation evolution in Utah chub *Gila atraria*; or (3) testing how well the observed differences agree with theoretical predictions based on life-history evolution, as in a study by Hutchings (1993) on maturation evolution in brook trout *Salvelinus fontinalis*.

A third strategy seeks to identify cases in which phenotypic variation caused by phenotypic plasticity and genetic adaptation point in opposite directions. It is in this vein that Trippel (1995, p. 766) suggested that

> evidence contrary to the compensatory theory may assist in building support for the effects of inheritance on age at maturity.

In other words, if opposite directions are predicted for phenotypic changes based on plastic and genetic effects, and 1 direction is observed, then it does not qualitatively matter that both effects may be acting jointly, since the dominating effect is likely to exist. For example, if age at maturation is observed to decrease while temperatures fall and stock densities rise, an explanation based on genetic adaptation will be strengthened, since the alternative explanation based on phenotypic plasticity predicts the reverse trend. Such phenotypic patterns in time can aptly be characterized as countertrend variation (CnTV), in analogy to phenotypic patterns in space described as countergradient variation (CnGV). According to Conover & Schultz (1995), the latter are said to occur when genotypes are distributed across an environmental gradient in nature such that genetic influences more than counteract environmental influences on a trait. Evidence suggestive of fisheries-induced evolution and strengthened by the observation of countertrend vari-

ation has been put forward, for example, by Polovina (1989; in the Hawaiian spiny lobster *Panulirus marginatus*: size at onset of egg production declined even though population density decreased) and by Olsen et al. (2005; in northern cod *Gadus morhua*: age at maturation declined even though growth rates decreased).

Circumstances under which one of the 3 approaches outlined so far can be applied are rather restrictive, since they require, respectively, experimental treatment (not an option for most marine species), spatial replication (problematic for marine stocks that intermingle or differ in too many environmental factors all at once), or countertrend variation (dependent on lucky coincidence and becoming increasingly unlikely as the number of considered environmental factors is increased). It is for these reasons that reliable conclusions about fisheries-induced maturation evolution remained stalled for decades.

Maturation reaction norms

Known as reaction-norm analysis, a fourth strategy for disentangling phenotypic plasticity and genetic adaptation in maturation schedules eventually helped to overcome this deadlock. Stearns (1983), Reznick (1990), and Rijnsdorp (1993a,b) put forward the same idea: reaction norms of fish maturation had to be considered to account for phenotypic plasticity, and these reaction norms had to be tested for temporal trends to evaluate whether or not genetic adaptations in the maturation schedules of fish were occurring. This approach naturally resulted from reaction norms having become appreciated, since the 1960s, as the most suitable descriptors of phenotypic plasticity (Sarkar 1999). The only open question was: How are maturation reaction norms to be defined?

A partial answer was provided in the work of Stearns and colleagues, who introduced maturation reaction norms to fish demography in order to describe how age and size at maturation were affected by growth rates (Stearns 1983, Stearns & Crandall 1984, Stearns & Koella 1986). This approach gave rise to bivariate reaction norms (Schlichting & Pigliucci 1998, p. 58): 1 environmental variable, average juvenile somatic growth rate, affects 2 phenotypic variables, age at maturation and size at maturation. While these dependences could alternatively be expressed by 2 univariate reaction norms — one describing age at maturation as a function of growth rate, the other describing size at maturation as a function of growth rate — the elegance of Stearns' construction derives from using the 2 phenotypic variables, size and age at maturation, as the axes of a single 2-dimensional diagram, capturing the environmental factor, average somatic growth rate during the juvenile period, by their ratio. For any observed combination of size and age at maturation, the corresponding average juvenile somatic growth rate is given by the slope of the line connecting this combination to the diagram's origin. An additional benefit was that growth trajectories could readily be superimposed on these diagrams, so that the age and size at maturation resulting for a given juvenile growth trajectory and maturation reaction norm was easily predicted by the curves' intersection. Stearns (1983, p. 73) rightly argued that the reaction norms thus defined constitute 'the minimal definition of the [maturation] phenotype necessary to understand genetic change'.

A limitation of the maturation reaction norm concept as introduced by Stearns and colleagues was that it assumed maturation to occur deterministically along the maturation reaction norm. In other words, for a given maturation reaction norm and a given juvenile growth trajectory, maturation was predicted to occur exactly at their intersection, resulting in a precise combination of age and size, without variation. This implied that an individual's probability of maturing had to jump from 0 to 1 abruptly when its growth trajectory crossed the maturation reaction norm curve. The same notion of deterministic maturation reaction norm was discussed by Reznick (1990, 1993) in the context of guppy maturation, and continues to be important in contemporary theoretical studies (e.g. Berrigan & Koella 1994, Day & Rowe 2002, Ernande et al. 2004, Burd et al. 2006). There have also been many other life-history models in which either the phenotypic age at maturation (e.g. Heino 1998) or the phenotypic size at maturation (e.g. Gårdmark & Dieckmann 2006) evolves; the latter 2 approaches are equivalent if there is no variation in growth. As was already pointed out by Stearns (1983), assuming the phenotypic age or size at maturation to be fixed is equivalent to assuming either vertical or horizontal deterministic maturation reaction norms. In natural populations, however, maturation ages and sizes tend to exhibit considerable phenotypic and genotypic variation even along given growth trajectories. The simple reason is that an individual's age and size do not usually suffice for predicting its maturation status with certainty. And even when other or additional macroenvironmental factors are considered, variation in microenvironmental factors will continue to imply maturation uncertainty.

Defining PMRNs

It is thus clear that maturation processes in most organisms will have to be described probabilistically. This conclusion was indeed foreshadowed in the context of metamorphosis research by Wilbur & Collins

(1973), who suggested describing an individual's probability of metamorphosing as a function of its weight and recent weight increase. Likewise, an individual's probability of maturing can be described as a function of its size and average juvenile somatic growth rate, or equivalently, as a function of its size and age. An early, still only semi-quantitative, response to this challenge was offered by Policansky (1983).

First attempts at defining probabilistic maturation reaction norms (PMRNs) took another route and were based on estimating the fraction of mature individuals in a population as a function of their age and size. This approach appears to have been considered independently, as suggested by the absence of any cross-citations, by Perrin & Rubin (1990), Rijnsdorp (1993a), Haugen (2000), and Morita & Morita (2002). Defining PMRNs in terms of the probability of being mature was certainly tempting, since it naturally extended the maturity-ogive concept, widely used in stock assessments, from 1 dimension (fraction mature as a function of either age *or* size) to 2 dimensions (fraction mature as a function of age *and* size). Statistical methods for estimating maturity ogives were thus already well established. Unfortunately, these attempts suffered from 2 main drawbacks. (1) Maturity ogives are properties of populations that have no counterpart at the level of individuals, and therefore cannot be interpreted as phenotypes for which evolution could induce genetic adaptations. (2) Even more importantly, maturity ogives are not only describing the maturation process itself, but also vary with the growth and mortality of individuals. Accordingly, maturity ogives change when conditions for growth and/or mortality are altered. This means, in particular, that maturity ogives cannot help to discriminate between genetic adaptations in maturation schedules and phenotypically plastic maturation changes resulting from variations in growth conditions.

The PMRNs introduced by Heino et al. (2002a) helped to overcome these problems. Instead of describing an individual's probability of being mature as a function of its age and size, PMRNs describe an individual's probability of becoming mature (typically, during 1 season) as a function of its age and size. PMRNs account for the inherent stochasticity of the maturation process by allowing an individual's probability of maturing to increase from 0 to 1 continuously, instead of jumping from 0 to 1 abruptly along a deterministic reaction norm. This approach offers a number of advantages. (1) The estimation of PMRNs overcomes systematic biases that result when deterministic maturation reaction norms are estimated for populations with probabilistic growth and probabilistic maturation. (2) PMRNs remove the effects of varying mortality rates and/or average juvenile somatic growth rates from descriptions of maturation schedules. (3) PMRNs are defined at the individual level. They can thus be treated as phenotypes when applying methods of quantitative genetics, so that population-level polymorphisms in maturation schedules can be analyzed correctly. (4) PMRNs serve as indispensable ingredients in process-based dynamical models of a population's age and size structure.

Estimating PMRNs

Several statistical techniques have been introduced to estimate PMRNs. When age- and size-specific densities of immature and newly matured individuals are known, PMRNs can be estimated as described by Heino et al. (2002a). In particular, in semelparous fish all mature individuals are newly matured, so that the estimation method of Heino et al. (2002a) can be applied directly. When densities of immature and mature individuals are known, but newly matured individuals (first-time spawners) cannot be distinguished from those that already matured earlier (repeat spawners), PMRNs can be estimated using auxiliary information on growth trajectories, as described by Barot et al. (2004a) and Heino et al. (2007). When newly matured individuals can be distinguished, but densities of only mature individuals are known, PMRNs can be estimated using auxiliary information on the average shape of growth trajectories and age-based maturity ogives, as described by Heino et al. (2002b). Finally, when individual ontogenetic trajectories can be observed in full, which requires repeated non-lethal observations and thus is impractical for field studies of marine fish, PMRNs can be estimated using methods analogous to survival analysis as described by Van Dooren et al. (2005).

Applying PMRNs

To date, PMRNs have been estimated for more than a dozen fish stocks. Relevant empirical studies include the following: Heino et al. (2002c), Barot et al. (2004b), and Olsen et al. (2004, 2005) on various populations of Atlantic cod *Gadus morhua*; Grift et al. (2003) on plaice *Pleuronectes platessa*; Engelhard & Heino (2004a,b) on Atlantic herring *Clupea harengus*; Barot et al. (2005) on 3 populations of American plaice *Hippoglossoides platessoides*; Dunlop et al. (2005) on smallmouth bass *Micropterus dolomieu*; and Haugen & Vøllestad (2007) on grayling *Thymallus thymallus*. In all of these studies growth-related phenotypic plasticity could be stripped from the description of maturation schedules, and in almost all of them long-term trends in maturation schedules were documented. That maturation trends remain ubiquitous, even when growth-related phenotypic plasticity is controlled for, rules out compensatory growth as

a sufficient explanation of these trends and also makes it unlikely that idiosyncratic, region- or stock-specific mechanisms could fully explain these widespread trends. The hypothesis of fisheries-induced evolution thereby gains strength as the most parsimonious explanation of maturation trends observed worldwide.

Age and size alone are almost never sufficient for predicting maturation probabilities with certainty. Therefore, the forecasting of these probabilities can potentially be improved by accounting for additional factors. In particular, body condition has been shown to affect the maturation process (e.g. Bernardo 1993, Metcalfe 1998, Marteinsdottir & Begg 2002). Moreover, even though size is often conveniently measured in terms of body length, it could alternatively be measured in terms of body weight. Three questions thus naturally emerge. Should length or weight be preferred as a measure of body size in PMRNs? What improvements in the predictive power of PMRNs may be gained by accounting for body condition? Can the trends observed in the PMRNs of many species be explained in terms of phenotypic plasticity based on body condition, so that these trends vanish once the effects of body condition on maturation probabilities are accounted for?

These questions have been addressed in a study by Grift et al. (2007) on maturation in North Sea plaice *Pleuronectes platessa*. It was shown that PMRNs based on age and weight provide slightly more accurate approximations of maturation probabilities than PMRNs based on age and length, but also that weight-based PMRNs imply much wider spreads of maturation probabilities than length-based PMRNs. Since weights exhibit much stronger fluctuations in the course of a season than lengths, weights will usually have to be normalized to a particular date during the season, a requirement that introduces additional assumptions and uncertainties into the estimation of maturation probabilities. Grift et al. (2007) thus suggested that, on balance, length-based PMRNs may be preferred.

The study by Grift et al. (2007) is also the first to report 3-dimensional PMRNs, alternatively based on age, length, and condition, or on age, weight, and condition. On this basis, Grift et al. (2007) demonstrated that — at least for North Sea plaice, which offers one of the best datasets internationally available for assessing changes in the maturation schedules of exploited marine fishes — incorporating body condition into the estimation of maturity probabilities increased the explained deviance only slightly, from 44–48% to 51–52%. The resultant 3-dimensional PMRNs not only showed how, at given size and age, maturation probability increased with condition, but also exposed that this impact of condition diminished with age.

Finally, the results reported by Grift et al. (2007) revealed several interesting temporal trends. (1) Clear residual trends towards maturation at younger ages and smaller sizes remained even after removing plastic effects on maturation captured by age, length, weight, and condition. (2) The probabilistic spread of both length- and weight-based PMRNs was found to have shrunk significantly over time, which means that maturation in North Sea plaice has become more deterministic over the course of the last half century. (3) Age and condition, as opposed to size, are nowadays affecting the maturation probabilities of North Sea plaice less than they did decades ago.

Mollet et al. (2006) estimated 3-dimensional PMRNs for North Sea sole *Solea solea*, and extended the analysis to include the effects of ambient temperature. This extension is interesting, since temperature might have a direct impact on maturation, over and above its indirect impact on maturation through its effect on growth. Again, the question was whether the observed long-term trends in sole maturation towards younger ages and smaller sizes would vanish once phenotypic plasticity based on body condition or on the temperature experienced prior to maturation was accounted for. This turned out not to be the case. Mollet et al. (2006) thus concluded that their results provided additional support for fisheries-induced maturation evolution, as more explanatory variables were accounted for. In a similar vein, Baulier et al. (2006) took the analysis of condition effects further by evaluating body condition in terms of the hepatosomatic index, which is based on liver weight, rather than in terms of Fulton's condition index, which is based on total weight. Using this physiologically more accurate measure in estimating 3-dimensional PMRNs based on age, length, and condition, Baulier et al. (2006) found that a long-term trend towards earlier maturation, consistent with fisheries-induced maturation evolution, remained for all analyzed populations (NAFO divisions 2J3KL, 3Ps, and 3NO) of Atlantic cod *Gadus morhua*, even after the effects of hepatosomatic index were accounted for.

PITFALLS OF UNDERSTANDING

In the following section we address some frequently asked questions about PMRNs. In our experience, the issues touched on by these questions may sometimes result in pitfalls of understanding for scientists not yet familiar with this approach.

Is the term 'reaction norm' appropriate?

Norms of reaction describe how environmental factors influence phenotypic expression (Woltereck 1909, Schmalhausen 1949, Bradshaw 1965, Schlichting

1986). Reaction norms will usually be shaped by the ancestral environment of a population, endowing individuals with a flexible response to the variable macroenvironmental conditions they encounter and thereby contributing to the maximization of their lifetime reproductive success (Schlichting & Pigliucci 1998).

The preceding section has documented that maturation reaction norms were proposed — by Stearns (1983), Stearns & Crandall (1984), Stearns & Koella (1986), Reznick (1990), Perrin & Rubin (1990), and Rijnsdorp (1993a) — for the explicit purpose of capturing the salient, but of course not all, aspects of phenotypic plasticity in maturation schedules. The discussion above hopefully has also shown why reaction norms should indeed be regarded as the most suitable conceptual tools for achieving this purpose. A growing acceptance of this conclusion in the context of fish maturation can be regarded as no more than a postscript to developments that permeated evolutionary ecology decades ago (Sarkar 1999).

We find that several uncertainties lead to concerns about using reaction norms for capturing maturation plasticity.

(1) Since reaction norms were not commonly used in fisheries science until recently, researchers in this field are bound to display healthy skepticism towards an initially unfamiliar concept. Naturally, this effect is expected to wear off with time.

(2) Maturation reaction norms are never expected to account for all phenotypic plasticity in maturation. In that regard, they are no different to reaction norms in other areas of evolutionary ecology. Norms of reaction quantify phenotypic responses to variation in one or more specific macroenvironmental factors, and always operate under the 'ceteris paribus' assumption that all other environmental factors are equal. Consequently, the deterministic part of PMRNs (characterized by the overall shape of isoprobability contours) captures the plastic maturation response to the considered macroenvironmental factor(s), while their probabilistic part (characterized by the spread of isoprobability contours) accounts for the noise imposed by the microenvironment in conjunction with macroenvironmental factors that are unaccounted for.

(3) It seems that some misunderstandings easily arise from the bivariate nature of deterministic maturation reactions norms. As explained above, such reaction norms describe how age and size at maturation (a bivariate phenotypic character) depend on average juvenile somatic growth rates (a univariate environmental factor). That the environmental factor is not displayed on 1 axis of the reaction-norm diagram, as would be expected for univariate reaction norms, is sometimes seen as confusing, even though bivariate reactions norms are part of the standard repertoire of plasticity research (Schlichting & Pigliucci 1998, p. 58). For probabilistic maturation reaction norms, the correct interpretation is even more subtle: environmental variation is measured in terms of growth trajectories (a multivariate factor), while phenotypic variation is measured in terms of maturation probabilities along those growth trajectories (a multivariate character). The resultant dependence is then usually displayed by showing maturation probability as a function of size at age, thus averaging over all growth trajectories that may lead to any particular size at age.

(4) One might be tempted to stipulate that phenotypic plasticity should always be expressed in response to obvious indicators of environmental variation, such as ambient temperature or nutrient level. Despite the intuitive appeal of this, the notion of macroenvironment established in quantitative genetics (Gavrilets & Scheiner 1993, Gavrilets & Hastings 1994, Hartl & Clark 1997) is considerably more encompassing: a macroenvironment can be defined in terms of the ontogenetic change it engenders. In general, there are many ways to characterize a macroenvironment. In the present context, a particularly relevant option is to characterize the macroenvironment in terms of the growth trajectory it engenders. All individuals exhibiting that growth trajectory will then have experienced the same macroenvironment, and vice versa. This notion is more grounded in biological reality than may be evident at first sight. In particular, the growth of fishes is affected by a great many environmental factors; these include the densities of all relevant types of prey, the densities of conspecific and heterospecific competitors, ambient temperature, and environmental factors affecting growth by requiring more or less energy investment into other functions. Moreover, these factors are bound to vary with time within and between seasons. Separately accounting for all of their effects therefore is an overwhelming task. Instead, it is much more practical to let the organisms themselves do the integration over time and environmental effects, resulting in environmental variation being characterized by readily observable changes of individual states, such as size at age.

(5) One may wonder whether reaction norms ought to be defined for genotypes or populations. In a review of one of the few textbooks devoted to phenotypic plasticity (Pigliucci 2001), Fuller (2003, p. 387) highlighted this common source of confusion:

> Pigliucci initially defines 'plasticity' as the property of a genotype […] However, many of the studies he cites as examples of plasticity […] measure plasticity not on genotypes but families.

Of course, this is just as expected: when quantifying phenotypic plasticity in the wild, clonal lineages or inbred lines are not typically available. It is therefore

unavoidable that almost all reaction norms measured in the field must be interpreted as population-level weighted averages of the underlying genotype-level reaction norms, with genotype frequencies serving as the weights. Sarkar & Fuller (2003, p. 106) have suggested referring to genotype-level reaction norms as 'standard norms of reaction', while using the term 'generalized norms of reaction' for those norms of reaction that quantify 'plasticity at the level of populations or subspecies within a species, species within a genus, or taxa at higher levels.' While any such terminology has yet to become commonly established, highlighting the conceptual difference between genotype-level and population-level reaction norms is highly recommendable. In particular, plasticity researchers must be aware of this distinction when pursuing challenges like developing the quantitative genetics of reaction norms (Gomulkiewicz & Kirkpatrick 1992, Gavrilets & Scheiner 1993, Heino et al. 2007) or when comparing population-level reaction norms with genotype-level reaction norms, where the latter are available.

Weighing up these considerations, it seems as though there is good scientific reason for continuing to use the term 'maturation reaction norm' in line with traditions already established a quarter of a century ago. Ultimately, however, the question of how broadly or narrowly one would wish to define reaction norms is a matter of semantics. The term 'maturation schedule' may actually offer a useful alternative for referring to PMRNs whenever one seeks to avoid technical terminology or where connoting the traditions of plasticity research is not intended.

What happens to PMRN analyses when growth rates are under genetic control?

There is every reason to believe that the growth trajectories of many organisms, including those of fishes, are not only affected by environmental conditions but also possess a heritable component (e.g. Conover & Munch 2002, Birkeland & Dayton 2005; also many breeding programs in aquaculture are squarely based on the heritability of growth-related variation). So, since growth is not a purely environmental factor, may we still examine phenotypic plasticity in response to growth variation? Of course we may, as long as growth has an environmental component — and it always will have, owing to its critical dependence on energy intake. Some subtleties, however, have to be understood before the implications of this conclusion can be appreciated in full.

A first, and temptingly simple, approach to addressing the issue is to point out that, especially in the wild, environmental variation in growth usually exceeds genetic variation in growth by far. On this basis, one may then argue that growth is mainly environmentally determined, so that effects implied by any partial genetic determination of growth can be treated as small and inconsequential. This view closes the argument quickly and may even be essentially correct for marine fishes living in highly unpredictable environments.

At a second level of understanding, one will appreciate that as long as growth and PMRNs are genetically independent, genetic variation in growth will have much the same effect as environmental variation and only influence how a PMRN is sampled. If environmental variation in growth is small while there are, for example, 2 main growth genotypes in a population — one implying slower growth and the other faster growth — a bimodal distribution of growth trajectories may result. This, however, has no bearing on the estimation or interpretation of PMRNs, since PMRNs describe the probability of becoming mature, conditional on having reached a certain age and size. Therefore, a potential bimodality in the distribution of growth trajectories, or any other effect of growth genetics on them, does not affect the conditional probability of maturing that is captured by PMRNs.

To reach a third level of understanding, we have to consider cases in which growth trajectories not only possess a large genetic variance but also exhibit a large genetic covariance with PMRNs (with both quantities being measured relative to the total phenotypic variance in growth). In these cases, an individual's growth trajectory not only determines its size at age but also affects, through genetic correlations, its genotype-level PMRN. Just as before, however, the population-level PMRN results from averaging genotype-level PMRNs over the many alternative growth trajectories that lead to a given size at age. While this averaging becomes more complicated, the interpretation of the population-level PMRN remains unaffected.

Genetic dependence between growth and maturation will of course affect the evolution of maturation schedules: with such covariation, selection responses in maturation genetically imply growth evolution, just as well as selection responses in growth genetically imply maturation evolution. Such correlated selection responses at the level of quantitative traits are analogous to the phenomenon of 'genetic hitchhiking,' well known at the level of genes (Maynard Smith & Haigh 1974). Nevertheless, such dependences have no bearing on the use of PMRNs for detecting evolutionary changes in maturation schedules.

The salient difference between cases with and without genetically imposed growth–maturation covariation is the causal interpretation of genetic adaptations in PMRNs. When such covariation is strong, one cannot read-

ily determine whether evolving maturation schedules are under direct selection, or indirectly respond to selection on growth. In reality, selection will likely affect both traits simultaneously, so that the observed evolutionary response is a compromise constrained by whatever genetic growth–maturation covariation exists.

We can thus conclude that the estimation and interpretation of population-level PMRNs are immune to the existence of heritable variation in growth, and even to the existence of heritable covariation between growth and maturation. Also, the interpretation of changes in population-level PMRNs is unaffected by heritable growth variation, and is affected by heritable growth–maturation covariation only if this covariation is strong. The interplay between phenotypic plasticity and genetic adaptation in both growth and maturation is an exciting topic of life-history research, but does not affect how PMRNs can and should be used.

Are maturation ogives and PMRNs interchangeable?

PMRNs superficially resemble maturity ogives: both have something to do with maturity and can be expressed as probabilities that depend on age and size. However, while PMRNs describe the probability of becoming mature, maturity ogives describe the probability of being mature. At first sight, this subtle difference may appear inconsequential and may even lead to the idea that PMRNs and maturity ogives could be used interchangeably. This is wrong. Rather, PMRNs and maturity ogives are fundamentally different; both have important uses, but one cannot be used in lieu of the other.

Maturity (described by maturity ogives) is to maturation (described by PMRNs) what size is to growth: growth and maturation are processes that lead to changes in individual states, in this case in size and maturity, respectively. Assessing an individual's growth process by measuring its size is as good or bad as trying to assess the maturation process by measuring maturity; it is bad, since both size and maturity are poor measures of the underlying processes if used directly, but good, since through proper transformations they can reveal critical information about the underlying processes. For growth, this is very obvious: one cannot replace growth rate with size, but if an individual's size at 2 ages is known, simple subtraction and division by the corresponding time interval yields an estimate of the average growth rate during that time interval. For maturation, an analogous calculation scheme for transforming from ogives to PMRNs exists; however, this turns out to be a bit more complicated, and in general requires additional information on growth and mortality (Barot et al. 2004a).

Maturity ogives are useful for describing how maturity in a population depends on the age and/or size of individuals. However, for describing the process of maturation, maturity ogives are not directly helpful because maturation is but one of the 3 processes determining the dependence of maturity on age and size, the other 2 processes being growth and mortality. By contrast, a PMRN describes the maturation process alone. The main confounding effects of growth and mortality are removed because a PMRN describes the probability of maturing conditional on reaching a certain age and size, with growth and mortality determining the probability of reaching — i.e. growing to and surviving until — that age and size. PMRNs can thus be viewed as fundamental life-history characteristics describing the maturation process. In contrast, maturity ogives are compound demographic characteristics describing the joint outcome of the 3 processes of maturation, growth, and mortality.

Is it a problem that PMRNs may contain midpoints that lie outside the observed size range?

Full description of a PMRN involves describing the probability of maturing for all relevant combinations of age and size. This gives rise to reaction norm surfaces in diagrams that use age, size, and maturation probability as axes. Parts of these surfaces are then sampled by the relevant growth trajectories. Since such surface plots are difficult to read quantitatively, it has become customary to illustrate the shape of a PMRN surface through percentile curves in contour plots that use age and size as axes. The simplest illustration is to show only the midpoints, i.e. the sizes for each age at which the probability of maturing equals 50%. For some ages, this midpoint may easily lie outside the range of sizes observed at that age. This is typical, particularly for the youngest and oldest ages for which a PRMN is estimated. At these ages, the population's average probabilities of maturing are close to 0 or 1, respectively, so that unrealistically large or small sizes would be required for individuals to experience a maturation probability of 50%. Is this a problem? Conceptually not, since it merely signals that percentile curves for other maturation probabilities, closer to the population's average for the considered age, should be used to show the PMRN's shape at that age. Simply drawing a midpoint curve at 50% maturation probability does not imply that its entirety would be sampled by actual growth trajectories.

Two conclusions can be drawn from this brief discussion. (1) PMRN midpoints that are extrapolated from the observed size range are bound to be more prone to estimation errors than PMRN midpoints that are inter-

polated. As in regression analyses, estimations are least uncertain where data are most abundant. Midpoints for early and late maturation thus typically exhibit larger confidence intervals than those for intermediate ages. (2) The shape of PMRNs should best be documented by showing multiple isoprobability contours. The extent to which individual contours are shown across ages can then be limited to the range spanned by observed growth trajectories. This will often result in drawing percentile curves for 10 or 25% maturation probabilities at young ages, for a wider range of maturation probabilities including 50% at intermediate ages, and for 75 and 90% maturation probabilities at old ages. Indeed, showing a PMRN's isoprobability contours for the size ranges observed across ages will give the best possible impression of the actual maturation dynamics of a specific population. If these contours are narrowly spaced, maturation is close to deterministic. Widely spread isoprobability contours, on the other hand, signal that the maturation schedule is only weakly influenced by age and size. This may occur when there is significant micro- or macroenvironmental variation that is unaccounted for.

Does the PMRN approach assume that age and length are the only determinants of maturation?

By definition, PMRNs for age and size use age and size for describing systematic variations in maturation probability. Of course this does not imply perfect determination of maturation by age and size alone. In fact, the very reason that a probabilistic approach is called for is that age and size almost always are incomplete determinants of maturation. Not only are age and size far removed from the proximate triggers of maturation (Bernardo 1993), but also better predictions of maturation probability can often be obtained by taking into account an individual's energetic state, as well as its energy acquisition prospects (Metcalfe 1998, Thorpe et al. 1998, Morita & Fukuwaka 2006). In temporally autocorrelated environments, the latter can even be predicted from recent somatic growth rates. Also, directly accounting for environmental variables may improve predictions. For example, temperature affects many physiological processes, including growth, and may also have direct effects on maturation, at the very least by providing seasonal cues (Thorpe et al. 1998, Huber & Bengtson 1999).

Experience has shown that age and size together do provide useful information for describing which individuals are more likely to mature. Size alone is relevant, since physiological and ecological constraints probably impose some limits on how large an individual has to become before it can successfully mature and reproduce (Roff 1991, Metcalfe 1998). It is less clear whether age has any significance of its own. In organisms experiencing either senesce or repeated (even periodic) habitat destruction, a direct age effect must be expected, since mortality rates are then age-dependent. In general, however, it is the combination of age and size that gives information about the average somatic growth rate an individual has experienced in its past, which serves as an important indication of how well that individual has been faring.

Most PMRNs estimated to date have used age and length as the only explanatory variables. However, the concept of PMRN is more general, and nothing prevents the inclusion of other explanatory variables, either in addition to or in place of age and length. The role of other explanatory variables in the maturation of female North Sea plaice *Pleuronectes platessa* has been explored by Grift et al. (2007), who considered weight and condition (measured by Fulton's condition index) in addition to age and length. As expected, accounting for variation in body condition explains more of the variation in maturation probability than is possible based on age and length alone. However, the gain is small, and the extra demands on the dataset and its analysis are not negligible. Related studies by Baulier et al. (2006) and Mollet et al. (2006) on Northeast Arctic cod *Gadus morhua* and North Sea sole *Solea solea*, respectively, have already been summarized in the preceding section. Also, Morita & Fukuwaka (2006) have shown how using information on past growth increments in chum salmon *Oncorhynchus keta* can strengthen the prediction of maturation probabilities relative to using only total length at age.

In practice, the choice of explanatory variables is primarily a question of data availability: it is this availability rather than the PMRN method itself that imposes limitations. Wherever feasible, the inclusion of additional explanatory variables in the estimation of PMRNs is to be encouraged. Once enough PMRN studies have been carried out for different sets of explanatory variables, conclusions may perhaps be drawn about an optimal general set. At the present stage of our knowledge, however, using age and length together appears to offer the best practical compromise between predictive power and wide applicability.

Does the PMRN approach claim to disentangle all phenotypic plasticity from genetic changes?

Maturation reaction norms in general, and PMRNs in particular, help to disentangle the effects of phenotypic plasticity and genetic adaptation on maturation (Stearns 1983, Stearns & Crandall 1984, Stearns &

Koella 1986, Rijnsdorp 1993a, Heino et al. 2002b). Of course this does not imply perfect disentanglement, and it has never been claimed that all plastic effects are thus accounted for. What PMRN based on age and length can reliably remove from the description of maturation schedules are the plastic effects of varying average juvenile somatic growth rates. In many wild populations, including those of marine fishes, these effects are likely to be the dominant source of phenotypic plasticity in observed combinations of age and size at maturation.

The possibility of unaccounted for plastic effects has been explicitly acknowledged in several publications employing the PMRN method (e.g. Heino et al. 2002b, Olsen et al. 2004). In particular, such effects reveal themselves as noise in PMRNs. Microenvironmental factors that are unaccounted for lead to probabilistic spread in an estimated PMRN. Macroenvironmental factors that are unaccounted for contribute to this spread, but also to interannual variation in estimated PMRNs. Indeed, time series of PMRNs estimated for adjacent cohorts tend to be noisy. This variability arises from 2 independent sources: observation error and plastic effects not captured by the explanatory variables considered. As observation errors reflect both imperfect sampling and the fact that PMRNs usually cannot be estimated without making simplifying assumptions, it is difficult to tease apart how much of the noise in PMRN estimates can be attributed to either of these 2 sources. However, there can be no doubt that residual plastic effects remain. If potential explanatory variables other than age and length have been measured, one can include these in the PMRN estimation, thereby in theory accounting for more plastic effects. In practice, however, the incorporation of additional explanatory variables, such as weight or condition, is not necessarily warranted, since it may add much more observation error and reliance on simplifying assumptions (Grift et al. 2007).

Some further words of caution may be in order here. In particular, it is tempting to try to learn more about extra plastic effects on maturation by testing for correlations between PMRNs and additional explanatory variables. This approach was applied, for example, by Kraak (2007, this Theme Section) to test for a direct effect of temperature on maturation in North Sea plaice *Pleuronectes platessa*. As in any other application of time-series analysis, this approach will be acceptable only if there is enough variation in both time series and if long-term trends are absent. The trouble is that fisheries science has been struggling for decades with interpreting long-term maturation trends. Demonstrating that such trends correlate with trends in a potential environmental factor is statistically vacuous: trended time series are always correlated. The simple exercise of plotting PMRN midpoints against year — typically showing a declining trend — reminds us that fallacious inferences about causation may easily be drawn based on correlation alone. We thus suggest that incorporating environmental factors into the estimation of PMRNs, in addition to cohort or year class, is the only statistically sound approach to the identification and disentanglement of additional plastic effects on maturation.

Even though parallel trends in PMRNs and environmental factors will never be sufficient to establish a causal relationship, they are always necessary for such a relationship to hold. In other words, environmental factors not exhibiting trends matching those observed in a stock's PMRN are unsuitable for explaining such trends. This may seem obvious. However, a study of maturation trends in chum salmon *Oncorhynchus keta* by Morita & Fukuwaka (2006, p. 1516) that used 3 growth increments for describing juvenile growth history (as opposed to characterizing it by a single average juvenile growth rate) concluded as follows:

> The most recent growth condition was the most important factor affecting whether a fish matured during the subsequent breeding season. Because individuals of similar body size and same age can have different growth histories, the relationship between body size and maturation probability could be plastically modified by growth history. This may violate an assumption required to infer evolution, namely that size-related maturation trends in probabilistic reaction norms are immune to growth history.

These authors are entirely correct in emphasizing that long-term trends in juvenile growth trajectories not yet captured by average juvenile growth rate may confound age- and size-based PMRN estimates. The same confounding effects could very well arise from long-term trends in all the many other factors that, to a greater or lesser extent, are known to affect maturation. What Morita & Fukuwaka (2006) have not demonstrated, however, is the actual existence of long-term trends in juvenile growth trajectories that go beyond changes in average juvenile growth rate. If such a demonstration were possible, the salient extra explanatory variable(s) should, and could, be included in the PMRN estimation. If not, conclusions based on long-term PMRN trends simply remain unaffected.

Can PMRN analyses conclusively prove fisheries-induced evolution?

The answer to this question must of course be negative. The reason is that this response should be given to all questions of the type: Can observational phenotypic data conclusively prove evolution to be driven by a specific factor? Conclusively proving fisheries-induced

evolution would require 2 logically independent conclusions to jointly hold. One needs to address whether the observed change (1) is evolutionary and thus genetic, and (2) has been caused by fishing rather than by another selective force.

In principle, genetic change can be identified either through direct observation or indirectly through the observation of phenotypic change while ruling out relevant environmental change. The first option must be based on collecting and analyzing molecular genetic data. This would require identifying loci strongly affecting maturation, and showing that allele frequencies at these loci have changed in accordance with expectations based on past selection pressures. In addition to the technical question of how the necessary historical tissue samples may be obtained, the major challenge here is to identify the relevant loci and alleles. Genes specifically affecting life-history traits such as maturation in fish remain largely unknown, so proving genetic change in maturation in this manner is not feasible at present. Nevertheless, molecular ecology is developing rapidly, and we may not need to wait for too long before this approach becomes practical.

The second, indirect, option for identifying genetic change is based on common-garden experiments. If individuals of known common ancestry show phenotypic differences despite having been raised under identical conditions for more than 1 generation (to exclude parental effects), then these differences will have to be acknowledged as being of genetic origin. Moreover, if the observed phenotypic differences are larger than those expected from genetic drift alone, they may be interpreted as the result of adaptive evolution undergone since the common ancestry. This approach suffers from 3 drawbacks, and thus also largely remains impractical for demonstrating the genetic nature of temporal maturation trends in marine fish populations. (1) It requires certainty that the populations under investigation are of recent common ancestry. Without some lucky coincidence, this is usually not possible. (2) For common-garden experiments to be capable of controlling for all environmental variation they will often have to depart from strictly natural conditions, so that any observed phenotypic differences will not correspond closely to what is, or has been, going on in the wild. (3) Most importantly, the common-garden approach requires living specimens of the putatively genetically-differentiated populations. This means that when temporal trends are to be investigated, specimens from the ancestral population must not only be alive at present, but must also be 'unevolved' in the sense of not having been exposed to any salient selection pressures in the meantime (e.g. through being kept in captivity). Because of these challenges, common-garden experiments are much more likely to be practical for populations separated in space rather than in time. For example, Koskinen et al. (2002) were able to show rapid evolution in populations of grayling *Thymallus thymallus* by comparing life-history traits in separate contemporary populations stocked from the same source population about a century ago (see Stockwell et al. 2003 for more examples of evolution following introductions).

When it comes to establishing fishing as the cause of a particular life-history change, it has to be recognized that the inadvertent, natural experiments of exposing a specific stock to fishing offer the weakest possible setting — unreplicated and uncontrolled — for demonstrating a causal relationship. However, the credibility of fisheries-induced selection as the most likely causal factor of observed maturation trends may be strengthened in a number of ways.

(1) Alternative hypotheses can be evaluated independently, using the best available knowledge of factors affecting maturation and of changes in the environment. Observations of countertrend variation, as defined in the preceding section, are particularly valuable for this purpose. For example, the tendency of northern cod *Gadus morhua* to mature at younger ages increased during a period of high fishing intensity and adverse growth conditions (Olsen et al. 2004). This change in maturation is in accordance with expectations based on mortality-induced life-history evolution — toward early maturation when juvenile and adult mortality is high (e.g. Roff 1984, Law & Grey 1989, Heino 1998) — whereas the plastic effects of slow growth would have been expected to result in just the opposite change. Thus, among these 2 hypotheses, the first is clearly more credible. Naturally, an unlimited number of alternative hypotheses always exists, although all but a few of these will be implausible *a priori*.

(2) While replication in the strict sense is not feasible, fish stocks are often exposed to a similar 'treatment' in terms of increased fishing mortality. A large number of studies from different geographical areas are showing comparable trends in PMRNs (Heino et al. 2002c, Grift et al. 2003, 2007, Barot et al. 2004b, 2005, Olsen et al. 2004, 2005, Engelhard & Heino 2004a,b, Dunlop et al. 2005, 2007b, Baulier et al. 2006, Mollet et al. 2006, Haugen & Vøllestad 2007). The few outliers in this common pattern can be explained by population-specific characteristics of fisheries regimes or reproductive systems (Engelhard & Heino 2004a, Dunlop et al. 2007b). Such a ubiquitous pattern is suggestive of a common explanation, for which fisheries-induced evolution is a strong contender.

(3) Numerical models can be constructed that incorporate fishing pressures and other selective forces to systematically investigate the extent to which life-

history trends observed in nature are captured in these models. So called eco-genetic models account for salient genetic and ecological detail (Dunlop et al. 2007a,b) and are thus emerging as particularly suitable tools for carrying out such exercises (a related approach was used by Baskett et al. 2005). For example, an eco-genetic model of Northeast Arctic cod *Gadus morhua* shows that the model-predicted rate of PMRN evolution is compatible with observations (Godø et al. 2007). Such findings significantly increase the credibility of fishing as the driver of observed maturation trends, and of life-history evolution as the underlying mechanism.

PERSPECTIVES

To date, the estimation of probabilistic maturation reaction norms (PMRNs) has focused on observational field data from exploited fish stocks. To further our understanding of maturation processes in general, and of the properties of PMRNs in particular, we suggest 2 lines of future experimental research.

(1) Experiments should be implemented to estimate PMRNs under controlled environmental conditions and without the need for invoking simplifying assumptions in the estimation method. Specifically, the phenotypically plastic effects of different environmental factors — including ambient temperature, as well as different temporal sequences of good and bad growth conditions — can thus be systematically explored. These experiments will improve understanding of how low-dimensional PMRNs are affected by unaccounted for sources of phenotypic plasticity.

(2) Breeding experiments should be carried out to make progress with the quantitative genetics of PMRNs. These experiments will provide information on the heritability of PMRNs, as well as about the genetic covariances within PMRNs, and between PMRNs and other heritable life-history traits.

The improved understanding resulting from these 2 lines of experimental research will greatly aid the interpretation of field data and further the accurate modeling of fisheries-induced evolution.

The most exciting application of PMRNs is their contribution to the disentanglement of fisheries-induced evolution and phenotypic plasticity in maturation schedules. Until recently, it has been considered parsimonious to explain these trends as merely plastic effects, usually in response to conditions for accelerated growth resulting from diminished stock size. Age- and size-based PMRNs estimated for more than a dozen fish stocks have captured this main effect of growth-related phenotypic plasticity and have shown that this effect alone cannot explain the observed maturation trends. To overcome this explanation gap, it still has to be evaluated whether there might be other common trends in the environment of fish stocks showing long-term maturation trends that could reasonably be expected to cause plastic responses in maturation schedules. Until such environmental trends are recognized, the most parsimonious explanation of widely observed continual trends in the PMRNs of exploited stocks is fisheries-induced evolution. This is not a proof of fisheries-induced evolution, but a shift in the burden of proof in line with the precautionary approach.

Young et al. (2006) have highlighted the benefits for fisheries management from understanding causal connections between fish physiology and fish life history. Understanding the underpinning of phenotypic plasticity in maturation schedules through the estimation of PMRNs squarely falls into this remit. More generally, fisheries management in the era of the ecosystem approach will increasingly have to rely on stock assessment tools and modeling frameworks that do justice to the age and size structure of fish populations. PMRNs can be seen as useful, if not indispensable, tools for such modern approaches to stock assessment and modeling.

It should be appreciated that, just like maturation, other life-history transitions — including metamorphosis, smolting, and sex change — are also characterized by, often irreversible, adjustments of an individual's morphology or physiology. The same applies to developmental transitions like vitellogenesis, as well as to ontogenetic transitions in meristic or morphometric characters (e.g. Neuman & Able 2002). While these other life-history transitions may not be as widely taxonomically relevant as maturation, the probabilistic reaction norm approach as discussed in this review will be just as useful in describing and understanding these transitions (e.g. Wilbur & Collins 1973) as it is in the case of maturation.

The study of evolutionary consequences of fishing can look back on a history of more than 1 century (Rutter 1903, p. 134). However, much of the pioneering empirical work on maturation trends in exploited fish stocks remained inconclusive, so that simple theoretical models and experiments on short-lived species dominated the development of the field. Since evidence from the wild was largely lacking, research on fisheries-induced evolution defined the agenda of a few evolutionary ecologists, but made little impact on the larger community of fisheries scientists and managers. There is no doubt that one of the main reasons for this stagnation was the difficulty of disentangling environmental and genetic influences in phenotypic traits, including maturation. By facilitating the detection of fisheries-induced evolution in maturation schedules, the advent of PMRNs has provided the

momentum needed for bringing this important topic to the attention of a wider audience. A new generation of fisheries scientists and managers will need tools such as PMRNs for coping with the threats of further undesired fisheries-induced evolution.

Acknowledgements. We gratefully acknowledge financial support from the European Marie Curie Research Training Network FishACE (Fisheries-induced Adaptive Changes in Exploited Stocks), funded through the European Community's Sixth Framework Programme (Contract MRTN-CT-2004-005578). M.H. also acknowledges support from the Norwegian Research Council (Project 173417/S40).

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Editorial responsibility: Howard Browman (Associate Editor-in-Chief), Storebø, Norway

Submitted: December 12, 2006; Accepted: February 13, 2007
Proofs received from author(s): March 24, 2007

Vol. 335: 271–277, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published April 16

Fisheries-induced evolution: present status and future directions

Richard Law*

Biology Department, University of York, York YO10 5YW, UK

ABSTRACT: This essay comments on recent research on Darwinian fisheries science and on the future development of this field. From a practical point of view, the key question is: how fast are evolutionary changes caused by fishing happening? To answer this question, there is a need to understand intensities of selection generated by fishing, heritabilities and genetic correlations of the traits under selection, and whether the rates of change in traits predicted from this information are consistent with the changes observed. Although there is little doubt about the existence of phenotypic change in life-history traits of exploited fish stocks, there are few direct estimates of selection differentials caused by fishing. Results that are available, together with the relatively low heritabilities of life-history traits, suggest that the evolution caused by fishing occurs at a modest rate, and is likely to need a decadal time scale to be clearly observable. Given the pressing need for attention to fisheries in the short term, measures to control the longer-term evolutionary impact of fishing are most likely to be adopted if they also help to meet short-term objectives of management. With this in mind, the essay mounts a defence of large, old fish, the presence of which would be beneficial to stocks in the short term, and the conservation of which would set in motion selection for improved growth in the longer term.

KEY WORDS: Darwinian fisheries science · Evolution · Fisheries · Heritability · Life history · Maturation reaction norm · Selection differential · Selection response

INTRODUCTION

Over the last few years there has been a major increase in interest in Darwinian fisheries science, a term given to the study of evolution in fish stocks generated by fisheries (Conover 2000). This short essay comments on some of the progress in this field, and the debates that this research is leading to. These debates are mostly to do with the interpretation of probabilistic maturation reaction norms, which describe the probability of maturing as a function of age and size (Heino et al. 2002).

The essay also considers future management measures that might help to deal with fisheries-induced evolution, arguing that such measures are most likely to be adopted if they also support short-term objectives of management. Given the circumstances, the essay mounts an unashamed defence of big, old fish, on the grounds that both the short- and long-term health of fish stocks could benefit from giving them more protection against exploitation.

PREDICTED RATES OF CHANGE IN LIFE-HISTORY TRAITS

The key question from a practical perspective is: how quickly can fishing activities cause evolution in fish stocks? Fisheries-induced evolution is relevant to fisheries management if it happens on a time scale of years, but is of less immediate consequence if it happens on a time scale of hundreds of years. It is the quantitative rate (rather than the qualitative existence) of change that is most important to know about for practical purposes.

The life-history traits under selection are typically quantitative, determined both by environmental con-

*Email: RL1@york.ac.uk

ditions and by many genes with small effects. The standard modelling framework with which to address evolutionary questions of this kind is provided by quantitative genetics (Falconer & Mackay 1996). Quantitative genetics provides a simple recurrence relation to describe the selection response R, the change in the mean value of a quantitative trait from one generation to the next, in the short term, under directional selection:

$$R = h^2 S$$

Here S is the selection differential, the difference between the mean value of the trait before and after selection within a generation, and h^2 is the heritability of the trait, which is measured as the additive genetic variance divided by the total phenotypic variance (Falconer & Mackay 1996). We can therefore examine the heritabilities and the fisheries-induced selection differentials to gain some preliminary understanding of the rate of evolution.

Heritabilities

An organism's life-history broadly comprises the age- and size-dependent schedule of growth, survivorship and fecundity. There is no suggestion that the traits that make up the life history have to represent the coordinated expression of a set of genes controlling particular developmental pathways (Thorpe 2007, this Theme Section). It is enough that there exists additive genetic variance in the traits on which selection can operate.

Heritabilities are low in life-history traits in comparison with traits less closely coupled to fitness (Mousseau & Roff 1987). Compilations from the aquaculture industry suggest that values in the range 0.2 to 0.3 are often appropriate (Law 2000). Research conducted in the last few years supports this view; for instance, using full-sib families (within populations) reared in tanks, Gjerde et al. (2004) reported a heritability for weight of cod *Gadus morhua* at age 200 d of 0.3. Heritabilities could be smaller in the wild where environmental variation might well be greater; however, information is much harder to obtain in these circumstances. Nevertheless, a study into the potential for sea ranching of Atlantic salmon *Salmo salar* obtained a heritability of 0.36 for body weight after 1 winter at sea, similar to that found in experimental farms (Jónasson et al. 1997). In a similar way, Funk et al. (2005) were able to estimate heritabilities and genetic correlations of morphological and life-history traits of pink salmon *Oncorhynchus gorbuscha* on their return from the sea to spawn. Heritabilities of length were approximately 0.4; however, those of egg number and weight — the traits most closely related to fitness — were not significantly different from zero. Surprisingly, estimates of heritability in the wild in general are not radically different from those obtained under more controlled conditions (Weigensberg & Roff 1996).

Further evidence for the existence of genetic variation in the wild comes from differences in growth of cod sampled from different locations in the Northwest Atlantic when reared in a common environment (Purchase & Brown 2001). The same applied in a comparison of Northeast Arctic and Norwegian coastal cod (Svåsand et al. 1996). Imsland & Jonsdottir (2002) were cautious in attributing these differences to genetic causes because of the possible carryover of environmental effects from the wild into the common environment, and because the sample sizes were quite small; nevertheless, they were confident that there was a genetic component to growth in turbot *Psetta maxima* and halibut *Hippoglossus hippoglossus*. Also indicative of genetic variation in the wild is the existence of counter-gradient variation, where genes for faster growth compensate for the lower temperature and slower growth in populations at higher latitudes. Counter-gradient variation has been documented in several fish species, including populations of Atlantic silverside *Menidia menidia* reared in a common environment (Conover & Present 1990, Conover et al. 2005).

Selection differentials

A priori one might expect selection differentials caused by fishing to be substantial in view of the fact that fishing mortality rates are large and that fishing gear is often selective. Estimates of selection differentials generated by fishing are rare; however, early work on cod in part of the North Sea from 1984 to 1990 suggested that length-at-age was reduced by approximately 1 cm through selective fishing during the period when a cohort was entering the fishery (Law & Rowell 1993).

An extensive study on cod in the Gulf of St. Lawrence estimated selection differentials by back-calculating earlier length-at-age of surviving individuals from records of size retained in their otoliths (Sinclair et al. 2002a). They obtained selection differentials for the most part in the range −4 to +4 cm. This study is of special interest because there was a change from a selection differential in favour of individuals with greater length-at-age in the 1970s to one favouring those with lower length-at-age in the 1980s and early 1990s. Fishing mortality on this stock was strongly dome-shaped, being greatest on fish of intermediate body size (Sinclair 1998), probably because

fishing was mostly at water depths at which these fish were most abundant; large cod tend to be at greater depths (Swain 1993). The positive selection differential in the 1970s is consistent with this dome-shaped fishing mortality: individuals growing rapidly through the sizes most vulnerable to fishing would be favoured by virtue of their shorter period of vulnerability. However, abundance of the stock increased in the late 1970s, correlated with an overall reduction in the growth of individuals (Sinclair et al. 2002b), and it is suggested that slower growth, delaying entry into the fishery, then became more advantageous. This study illustrates how fishing coupled with spatial separation of size classes and environmental change can have quite subtle effects on selection.

Selection differentials of this magnitude, together with long generation times and low heritabilities of life-history traits, suggest that selection responses — while important — are likely to be quite modest on a year-to-year basis, and more likely to be detected from one decade to another than from one year to the next.

OBSERVED RATES OF CHANGE IN LIFE-HISTORY TRAITS

Evidence of temporal change in life-history traits of fish stocks was reviewed by Law (2000), and continues to accumulate. For instance, Hutchings (2005) documented a decline in age at maturation in 2 stocks of cod in the Northwest Atlantic: one stock that extends from Labrador southeast to the northern part of the Grand Banks, and another from the eastern Scotian Shelf. These results are consistent with those of earlier studies (Trippel et al. 1997). A study on reproductive investment by North Sea plaice *Pleuronectes platessa* over a period that began in the 1960s did not strongly suggest a change owing to fishing; however, there was a possibility of some change since the late 1940s (Rijnsdorp et al. 2005). Changes in maturation reaction norms are covered separately below.

Although there is much evidence for the existence of changes in life-history traits of exploited fish stocks, it is less clear whether the observed rates of change in life histories are quantitatively consistent with the likely heritabilities and selection differentials generated by fishing. To date, I am only aware of 1 study that brings together both the selection differential estimated from the catch data on a stock and a time series showing phenotypic change in a life-history trait. This is a study on Icelandic haddock *Melanogrammus aeglefinus*, motivated by a continuing downward trend in length-at-age of this stock, not easily accounted for by environmental factors (Thordarson 2005). This research has yet to be published, and shows a close match between the rate of phenotypic change and the selection differential, under a reasonable assumption about the heritability.

The difficulty of obtaining quantitative agreement between observed and predicted rates of change is evident in size at maturation of the Baltic cod (K. Andersen pers. comm.). A quantitative-genetic model suggested a decline in size at maturation by about 40 g per generation, a rate that has not been observed during the period of heavy exploitation since the 1950s. He suggested that a number of biological features absent from the model, such as greater quality of eggs from older fish, might help to reduce the discrepancy. An interesting feature of this analysis is that selection on size at maturation was disruptive: individuals with intermediate sizes at maturation had lower fitness than those with maturation at small or large sizes. Disruptive selection of this kind was also a property of a model of a size-structured, resource-consumer population under size-dependent harvesting (de Roos et al. 2006). A feature of this study was that fisheries-induced selection could cause an evolutionary regime shift such that the evolution of maturation at small sizes under harvesting would then be difficult to reverse.

Overall, there is little doubt about the existence of phenotypic change in the life-history traits of exploited fish stocks. However, the quantitative analysis of whether rates of change are consistent with likely heritabilities and selection differentials caused by fishing, allowing for change in the environment, is a critical issue needing more research.

THE MATURATION REACTION NORM AS AN INDICATOR OF GENETIC CHANGE

Much of the recent work on fisheries-induced evolution has centred on probabilistic reaction norms for maturation (Heino et al. 2002). This reaction norm describes the probability of maturing as a function of age and size, and is often depicted as a sequence of points in an age-size space along which the probability of maturing is 0.5. It is important to bear in mind that the maturation reaction norm is just one of a number of traits potentially liable to evolve through the operation of fisheries. Other traits include, for instance, growth (Thordason 2005), reproductive effort (Rijnsdorp et al. 2005) and morphology (Hamon et al. 2000). Changes in traits such as these may also be important for the health of wild fish stocks.

One reason why there is so much current research on maturation reaction norms is that, although individual growth itself is highly variable and contingent on the environment, the combinations of size and age at

which maturation occurs could be relatively insensitive to environment-induced variability. With this in mind, change over time in the reaction norm is interpreted as a signal of evolutionary (i.e. genetic) change, in contrast to variation in growth within a single reaction norm that is taken to reflect effects of the environment (Barot et al. 2004, Olsen et al. 2005). This idea is of much interest, but appears to be an assumption rather than an empirically verified observation in the context of marine fish stocks.

In fact, there is evidence that the environment can have substantial effects on maturation reaction norms based on size and age. This is evident for instance in the Northeast Arctic cod, in which age at maturation has been decreasing since the 1940s (Jørgensen 1990). Marshall & McAdam (2007, this Theme Section) show striking evidence of a shift in the feeding conditions for cod in the Barents Sea in the 1980s. Before 1980, the cod had lower weight-at-age at age 7 to 10 yr than after 1984, and a smaller proportion were mature in the earlier period. Accompanying this shift was a rather abrupt change in the length at 50% maturation of 6 yr old cod. This suggests that the maturation reaction norm was directly affected by a change in environmental conditions. Major environmental changes have been taking place in the Barents Sea, including increasing water temperatures since about 1980, and large fluctuations in the availability of capelin *Mallotus villosus* and herring *Clupea harengus*, which form an important part of the diet of this cod stock (Hjermann et al. 2007, D. Ø. Hjermann pers. comm., C. Marshall pers. comm.). However, the exact environmental cause of the change in maturation is not yet known.

The abrupt change in maturation of Northeast Arctic cod in the 1980s does not account for the longer-term decline in age at maturation over the 20th century, for which the most parsimonious explanation is evolution caused by development of a fishery on the feeding grounds early in the century. Although it has not been easy to achieve a close match between the rapid observed decline and that predicted by quantitative genetic models (Jørgensen 2005), results of recent models get quite close to the decline observed in the stock (C. Jørgensen pers. comm.).

A second piece of evidence for a direct effect of the environment on the reaction norm is a finding that maturation in chum salmon *Oncorhynchus keta* is better predicted by recent growth history of an individual than by its body size (Morita & Fukuwaka 2006). This is important because the maturation reaction norm then depends on prevailing features of the environment, such as availability of food, as opposed to being determined by a combination of age and size alone.

Third is a study by Olsen et al. (2004) that interpreted change in length at 50% maturity of cod aged 5 and 6 yr in the Northwest Atlantic as indicative of rapid evolution. An interesting feature of the results is that the direction of change in the time series altered within about 3 yr of the 1992 fishing moratorium, i.e. in cohorts in place before the moratorium was imposed. This change was consistent across the 3 areas documented and seems unlikely to be a consequence of noise (Fig. 3 in Olsen et al. 2004; see particularly the 5 yr old cod). A change so soon after the moratorium is evidence that pre-existing cohorts were responding in some way to the new environment, rather than evidence of an inherited genetic response. This is because information about the selection response becomes available only when the first cohorts born after the moratorium are reaching maturity 5 or 6 yr later. Such a change might reflect a phenotypic adjustment to the new environmental conditions without selection, indicating an environmental influence on this component of the reaction norm. In addition, such a change might reflect a selection differential generated by the new environmental conditions. In this latter case, it is interesting that in 2 of the 3 areas, change in cohorts born after the moratorium was small compared with change in cohorts in place at the time of the moratorium (Fig. 3 in Olsen et al. 2004). The lack of continuing change would suggest that the heritability of the trait was low. In either case, the post-moratorium time series suggest a substantial environmental influence on the component of maturation reaction norm investigated.

Apart from the evidence that the environment affects the maturation reaction norm, the evidence also suggests that there remains a substantial component of genetic, as well as environmental, variation in size-at-age in the wild. The best evidence comes from the studies on Atlantic salmon and pink salmon mentioned earlier (Jónasson et al. 1997, Funk et al. 2005). Other evidence is more circumstantial because it is based on heritability estimates on growth in experimental conditions. However, bearing in mind the findings of Weigensberg & Roff (1996), and the existence of counter-gradient variation (Conover & Present 1990, Conover et al. 2005), it would be surprising if genetic variation in size-at-age was not a common feature of fish populations in the wild.

On the basis of the evidence currently available, it therefore seems that the use of maturation reaction norms to disentangle genotypic and environmental effects does not yet have a strong empirical foundation. Some part of the variation in the maturation reaction norm is likely to be genetic, and some part of the changing maturation in fish stocks over time is likely to represent evolution. But, as with other life-history traits considered in the context of fisheries-induced evolution, there remains uncertainty about the relative strength of environmental and genetic signals.

It may be best to think of a life history as a set of correlated traits with potentially some genetic component to the correlations. The correlations matter because the effects of fisheries-induced selection then depend in part on them: when selection is applied to one trait, changes may occur in other correlated traits. For instance, Munch et al. (2005) found in Atlantic silverside that a size-selective harvest at age 190 d caused a detectable selection response for length at earlier ages, down to 90 d (see also Kirkpatrick 1993); however, correlated genetic changes for early life-history traits affecting recruitment were not evident. Genetic correlations also matter because fishing can be selective in various ways and probably acts on more than 1 trait at a time. As Hard (2004) noted in a quantitative-genetic study of the Chinook salmon *Oncorhynchus tshawytscha*, selection on correlated traits has the potential to be mutually reinforcing, so that the overall response is greater than would be expected from analysis of single traits in isolation. In practice, genetic correlations are hard to estimate, but some clues may be obtained from the phenotypic correlations of traits (Cheverud 1988, Roff 1995). For example, Funk et al. (2005) found a positive relationship between genetic and phenotypic correlations of traits in pink salmon; however, they suggested caution in adopting this approach because the relationship was sometimes lost.

Gaining knowledge of fisheries-induced evolution is not an easy task, and recent research on maturation reaction norms is an interesting attempt to disentangle the effects of genetic and environmental processes in genetically correlated life-history traits. However, it is important for biologists who work on fisheries to bear in mind that, while there can be no doubt that fishing causes genetic change in life histories, the rates of these changes remain uncertain.

TO THE FUTURE: IN DEFENCE OF BIG FISH

Decision making in management of fisheries tends to give greatest weight to the short term because the state of fish stocks and marine ecosystems a few years into the future is the most pressing priority. Fisheries-induced evolution is more subtle and gradual, but also of great importance if we are to leave healthy fish stocks for the future. However, the management focus on the short term suggests that longer-term Darwinian goals are most likely to be achieved if they are based on measures that also help to meet short-term goals. This leads to the question: are there management measures that would help in the short term, and that might also help to reverse longer-term evolutionary changes if these are thought to be deleterious to the stocks themselves or to the objectives of management?

Arguably, a key measure is to protect large, old fish within populations. This is not to question the importance of protecting small, young fish, currently an important driver of contemporary fisheries management and enshrined, for instance, as a major factor for the conservation of fish stocks in the Common Fisheries Policy of the European Union (Anon 2002). However, in our concern to protect young fish, we have been less concerned about the fate of larger, older fish within populations. Large, old fish do matter in both the short- and the long term for the reasons that follow.

In the short term of population dynamics, concentrating just on the protection of small, young fish leads to truncation of the size and age distributions (e.g. Ottersen et al. 2006). This makes fisheries increasingly dependent on young, inexperienced spawners that are less fecund and produce eggs and larvae of lower quality (Trippel 1995, Berkeley et al. 2004, Birkeland & Dayton 2006). This is deleterious for stock renewal; for instance, a low age diversity, corresponding to a large proportion of young individuals, is correlated with poor recruitment in Icelandic cod (Marteinsdottir & Thorarinsson 1998; see also Secor 2000). More generally, the truncation of the age structure caused by fishing removes a buffer against years of poor recruitment and exacerbates fluctuations in year-class strength. Hsieh et al. (2006) documented the temporal variability in larval population densities of 29 species, some exploited and some unexploited, over a 50 yr period. Fluctuations were greater in the exploited species than in the unexploited ones, a phenomenon that they attributed to truncation of age distributions owing to exploitation.

In the longer (evolutionary) term, failure to protect large, old fish may generate selection pressures that are deleterious to growth of fish. Individuals that pass more time before reaching a size at which they become vulnerable to fishing leave more descendants, all other things being equal. Evidence to support this comes from the selection differential for reduced length-at-age observed in Gulf of St. Lawrence cod in the 1980s and 1990s, when there seems to have been little opportunity for growing beyond a catchable size (Sinclair 2002a,b). Also, individuals that mature when smaller run less risk of being caught before maturation and leave more offspring, all other things being equal (caveats to this reasoning are discussed below).

In sum, there are benefits in both the short and longer term of maintaining large, old individuals within populations. As a management objective, this ought to be relatively uncontroversial: large fish have become so rare as a result of current fishing practices that the loss in yield arising from their protection should be relatively small. This is with the caveats that some reduction in fishing mortality at smaller sizes could be needed if a build-up of large fish was to be

achieved rapidly, and that large fish sometimes have intrinsically greater market value. It is important to achieve a reduction in fishing mortality on large fish without increasing fishing mortality on smaller fish, which would obviously be highly detrimental to fish stocks. Also, an awareness that large fish are potential predators of smaller fish is needed, and there could be circumstances in which this is deleterious to the yield.

In practice, protection of both of small and large fish requires patterns of fishing mortality that are greatest in fish of intermediate body size (dome-shaped fishing mortality). Such fishing mortality patterns are already in place in sports fisheries, such as slot-limit sturgeon fisheries in Washington State, and in commercial trap fisheries, such as the Maine lobster fishery. Some fishing gears, such as traps, gill nets and long lines, can be quite size selective in any event (Myhre 1969, Hamley 1975). Other gears can be made more size selective, for instance by using excluder devices for large fish on trawl nets, and by using maximum trawl times and speeds to make it easier for large fish to escape. Compromises are always going to be needed in multispecies fisheries. Marine protected areas (MPAs) for fish could help substantially in reducing selective effects of fisheries. A detailed model-based analysis of MPAs found that they could be as effective as other management tools at protecting stocks against fisheries-induced selection for early maturation (Baskett et al. 2005). To the extent that fish of different life stages live in different places, MPAs may be targeted at some life stages in preference to others, although the effects of such targeting could be counter-intuitive. For instance, it seems possible that protecting fish on spawning grounds, important in short-term management, could favour life histories in which fish mature and move to the protection of the spawning grounds at an earlier age.

There are several caveats to bear in mind. Blanket recommendations are dangerous, and measures for protecting large fish are best developed on a stock-by-stock basis to ensure that they are appropriate in context. It is also dangerous to develop management policies before the core science is available; for instance, dome-shaped fishing mortality could generate disruptive rather than directional selection. However, such points need to be balanced against an awareness that (1) many fish stocks no doubt already experience fisheries-induced selection, limited though our understanding of this is, (2) the resulting evolution may be hard to reverse, and (3) the precautionary principle places responsibility on us to leave our resources in a state that can be utilised as fully by our descendants as by ourselves. The full evolutionary picture is, of course, a good deal more complicated than given in this short commentary, depending on the balance between natural mortality and fishing mortality, the rate of growth and the size-dependence of egg production, the genetic architecture of the life history, the selection differentials generated by fishing, and the population dynamics.

CONCLUSIONS

The current interest in fisheries-induced evolution draws attention to this important, but previously neglected, subject. At the same time, it needs to be understood that the rates of genetic change are likely to be modest on a year-to-year basis. Given all the circumstances, it seems sensible to work towards measures that will help to resolve current short-term problems, and that will simultaneously generate selection regimes likely to help in the longer term. Taking greater care of larger, older fish seems especially important in this regard. This essay therefore joins the growing voice of opinion that argues that these fish should be given more protection than they receive at present (e.g. Trippel 1995, Conover & Munch 2002, Birkeland & Dayton 2005).

Acknowledgements. Thanks to D. Ø. Hjermann, S. Jennings, C. Jørgensen, C. T. Marshall, J. D. Reynolds, D. P. Swain and G. Thordason for helpful discussions during the preparation of this essay, and to the University of Calgary where this work was completed.

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Editorial responsibility: Howard Browman, (Associate Editor-in-Chief), Storebø, Norway

Submitted: August 3, 2006; Accepted: December 13, 2006
Proofs received from author(s): March 13, 2007

Vol. 335: 279–283, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published April 16

Understanding the maturation process for field investigations of fisheries-induced evolution

P. J. Wright*

FRS Marine Laboratory, PO Box 101 Victoria Road, Aberdeen AB11 9DB, UK

ABSTRACT: The probabilistic maturation reaction norm approach has been widely heralded as an empirical approach to distinguish between the effects of genetic selection and phenotypic plasticity on maturation probability. However, applications of this approach have considered maturation state in relation to fish size long after the period when fish make the 'decision' to mature. Evidence, mostly from salmonids, indicates that maturation is controlled by successive hormonal inhibition linked to energy state during critical periods of the year. Thus, any genetic selection cannot be acting upon the size of fish late in the reproductive cycle, but rather during the timing of these critical periods. Indeed, experimental studies demonstrate that size attained by late stages of gametogenesis need not necessarily be a good predictor of the probability of maturing. Therefore, changes in energy status around the time of maturation decisions represent an unknown and possibly significant source of variability in the reaction norm midpoints. Clearly, there is a need to apply physiologically realistic models of maturation probability — as have been developed for Atlantic salmon — to other fish species. Future investigation of fisheries-induced evolution may also benefit from examining historical changes in fecundity and by comparing current reproductive investment in fish from heavily and lightly exploited populations that are held under common-garden conditions.

KEY WORDS: Maturation · Fecundity · Fisheries-induced evolution · Critical periods

INTRODUCTION

Long-term shifts in life-history traits of fish, particularly a trend towards decreasing size and age at maturity, have been widely reported in heavily exploited fish stocks (Law 2000). Genetic selection generated by fishing has long been considered an important contributory factor to these changes (Ricker 1981, Trippel 1995, Law 2000). Controlled selection experiments have confirmed this potential for harvest-induced genetic change in life-history traits (Conover & Munch 2002). However, expressed life-history traits will be influenced not only by genetics but also by environmental variation through phenotypic plasticity. Distinguishing between the effects of genetic selection and phenotypic plasticity in wild fish populations is, therefore, a major challenge. Owing to the potential long-term impact of fisheries-induced evolution on the yield of fish stocks (Conover & Munch 2002), it is essential that we evaluate the relative contributions of genetic and environmentally induced variation on the observed phenotypic changes (Rochet et al. 2000).

The probabilistic maturation reaction norm (PMRN) approach was developed to provide a method for distinguishing the effects of genetic variation and growth-induced phenotypic plasticity on maturation. This method models the probability of maturing within a cohort based on the proportion of immature and mature individuals at a given size and age (Heino et al. 2002). By accounting for size at age, the probability of maturing is independent of variations in growth and survival that confound maturity-size relationships. Changes in the reaction norm mid-point have been interpreted as evolutionary shifts in maturity at size (Barot et al. 2004, Olsen et al. 2004). However, the PMRN approach has 2 important limitations. First, whilst the probabilistic treatment acknowledges that maturation cannot be fully accounted for by length and age alone, these are the only 2 parameters considered in the majority of published studies. Although this

*Email: p.wright@marlab.ac.uk

problem may be overcome with additional information on the energetic status of the fish, such as an index of condition, there is a second and more fundamental problem. The developmental 'decision' to spawn takes place long before spawning actually happens, yet applications of this method have been based on measurements taken on fish in advanced stages of reproductive development (Grift et al. 2003, Barot et al. 2004). Thus, the relationship between maturity and size used within the formulation of PMRNs reflects the outcome of the fish's continued gonadal development rather than its state when the initial maturation decision was made. To fully appreciate the consequence of this assumption, it is important to understand the proximate influences on maturation.

THE MATURATION PROCESS

There is now substantial evidence that maturation is not dependent on size thresholds or growth per se but rather is sensitive to an animal's growth and energetic status at particular times of year. The importance of time of year is evident from the ability to shift or even inhibit maturation by means of photoperiod manipulation (e.g. Shimizu et al. 1994, Bromage et al. 2001, Norberg et al. 2004). Photoperiod appears to alter the timing of the period when the physiological threshold must be exceeded for maturation to continue (Bromage et al. 2001). As Thorpe (2007, this Theme Section) describes for salmonids, maturation is controlled by successive inhibition through lipid-regulated switches during critical periods of the year (see also Silverstein et al. 1997). Whilst the physiological changes in the pituitary-gonad axis associated with these critical periods are still under investigation, it does appear that the insulin-like Growth Factor 1 signals the growth and nutritional status (Campbell et al. 2003, 2006), thus stimulating the release of hormones such as follicle-stimulating hormone and sex steroids (Campbell et al. 2003, 2006, Gen et al. 2003) involved with early gametogenesis.

Evidence that size attained by the late stages of gametogenesis is not necessarily a good predictor of the probability of maturing comes from laboratory experiments that followed the growth of size-matched groups of juveniles of the same age through to adulthood. Fig. 1 provides 2 such examples following fish growth from primary to secondary phases of oogenesis. The appearance of cortical alveoli vesicles containing yolk proteins generally indicates that oocytes will continue developing through to the secondary phase (true vitellogenesis) and subsequent spawning at the next breeding season. In these experiments, maturity-related differences in final length are clearly not related to fish size by the cortical alveoli phase of oocyte development. Fish that go on to mature in such experiments are characterised by high somatic growth and condition prior to the secondary phases of gametogenesis (Imsland et al. 1997, Yoneda & Wright 2005). Thus, size differences between mature and immature fish by the spawning season reflect an initially higher somatic growth rate in those that matured, followed by depressed somatic growth associated with the energy allocation to the secondary phase of gametogenesis (Yoneda & Wright 2005).

Whilst the PMRN assumes that a shift in the midpoint of the reaction norm can reflect a genetic effect, it is clear from the previous sections that any genetic selection cannot be acting on the size of fish late in the reproductive cycle. Thorpe (1986) proposed that genetic control is likely to act via the lipid/energy sensitive switches involved in the initial maturation decisions. Support for active inhibition of maturation has come from differences in the expression of genes involved in growth and reproduction between early maturing male, immature female and immature male Atlantic salmon *Salmo salar* (Aubin-Horth et al. 2005a,b). Therefore, if there has been a genetic change in maturation tendency, this would be expected to have acted on the threshold switches for maturation. Whilst the PMRN approach considers annual growth, it does not account for the effect of growth or lipid stores at the time of developmental decisions. Therefore, for the PMRN approach to have relevance to maturation decisions, it is necessary to demonstrate that the measurements used — i.e. maturity at pre-spawning length and annual growth increments — are correlated with proximate thresholds for maturation. This would require a close correlation between final length and lipid accumulated during the period when maturation decisions are made in wild fish.

There is often a close correlation among fish size, growth and primary lipid stores. For example, in salmonids, visceral fat level is often correlated with length (Simpson 1992), whilst in gadoids relative liver weight is generally related to somatic growth rate (Jobling 1988). As such, energy-dependent thresholds may covary with pre-spawning length and the annual growth increment measurements used for PMRNs. Nevertheless, mature fish tend to have a much higher liver energy than immature fish for a given size (Eliassen & Vahl 1982), and there can be large interannual variations in the relationship between liver size and fish size (Yaragina & Marshall 2000). Consequently, individual differences in lipid accumulation and storage around the time of maturation decisions could introduce a significant source of variation in the size at which fish initially 'decide' to mature. Measurement of the effects of this variation made just prior to

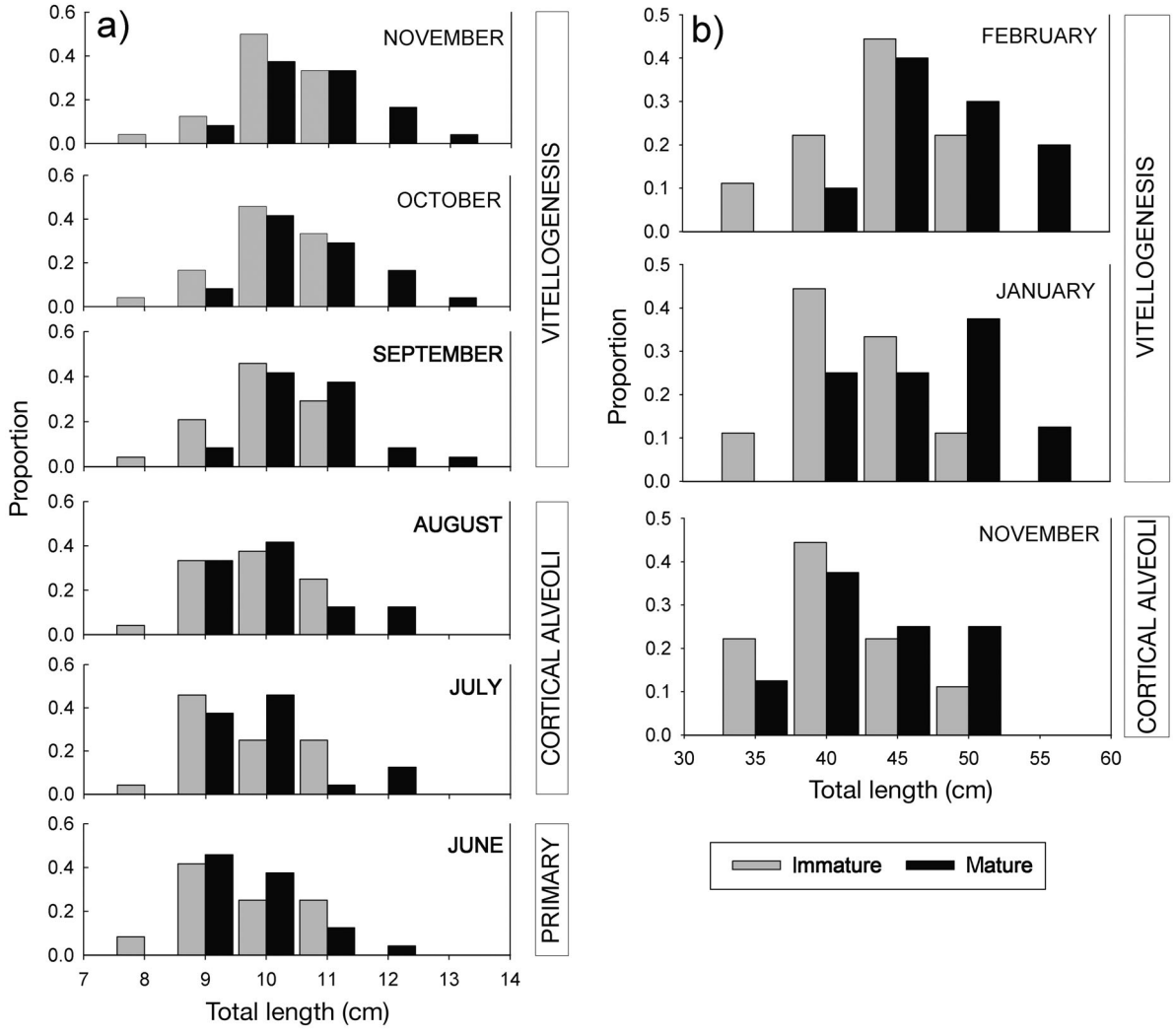

Fig. 1. Changes in proportional length composition of a group of (a) *Ammodytes marinus* (P. Boulcott & P. Wright unpubl. data) and (b) *Gadus morhua* (Yoneda & Wright 2005) reared under identical laboratory conditions. Data presented separately for mature and immature fish, based on assessment of gonad stage at the end of experiments. Periods during which primary, cortical alveoli and vitellogenic oocytes appear are indicated based on sub-samples taken throughout experiments

spawning suggests that lipid energy can account for an additional but comparatively small amount of the variation in maturity relative to size (Marteinsdottir & Begg 2002, Morgan 2004). However, because these studies were conducted in the late phases of gametogenesis, they may have little relevance to the effect of lipid energy at the time of maturation decisions. Therefore, changes in energy status around the time of maturation decisions represent an unknown and possibly significant source of variability in the reaction norm midpoints. If such variability exhibited a long term trend, then it might explain some of the apparent decline in reaction norm midpoints reported in some studies.

The ability to calculate PMRNs using just 2 parameters that are readily available for many fish stocks has made this a popular approach in the search for fisheries-induced evolution. However, the focus on the outcome size at age, well after maturation decisions have been made, is a serious source of uncertainty in the PMRN approach. A better understanding of the timing of maturation decisions may help identify more appropriate sample data sources and times for collection. If information on energetic status around the time of the maturation decisions was available for some stocks, then it may be possible to develop a more physiologically realistic model of maturation probability, as proposed by Day & Rowe (2002) and developed for Atlantic salmon (Thorpe et al. 1998). Owing to our limited knowledge of the maturation process for most species, it is clearly not possible to assess how changes to the timing of measurements of size or energetic status would influence estimates of reaction norm mid-

points. Changes in reaction norm midpoints of a similar magnitude to a fish's annual growth, such as that reported for Atlantic cod *Gadus morhua* from Georges Bank (Barot et al. 2004), certainly would suggest a substantial change in growth and therefore the state-dependent threshold for maturation. However, other explanations need to be explored, as suggested by the study of Marshall & McAdam (2007, this Theme Section) on liver energy variation in Northeast Arctic cod.

CHANGES IN REPRODUCTIVE INVESTMENT

In addition to the focus on maturation changes, future investigation of fisheries-induced evolution may benefit from examining other aspects of reproductive investment. In terms of life-time reproductive output, an individual maturing at a smaller size would have to invest more heavily in egg production with age if it is to compensate for the initial size effect on fecundity. Ultimately, this may lead to selection for higher fecundity at maturity and an increase in the slope of the fecundity-size relationship (Rochet et al. 2000). Evidence that increases in relative fecundity and lower body condition have occurred in heavily exploited stocks has come from studies of Atlantic cod (Yoneda & Wright 2004, Lambert et al. 2005) and haddock *Melanogrammus aeglefinus* (Wright 2005). However, as with maturation, disentangling environment and genetic effects on fecundity is difficult. It may be possible to determine the nature and magnitude of changes necessary to differentiate between a phenotypic and genetically determined effect on fecundity-size relationship in the field, based on knowledge of the proximal influences on fecundity gained from laboratory studies. For example, laboratory studies have demonstrated that the fecundity of first-time spawning Atlantic cod is closely correlated with body weight, regardless of the growth conditions that a fish is subjected to (Kjesbu & Holm 1994, Karlsen et al. 1995, Yoneda & Wright 2005, Skjæraasen et al. 2006). In contrast, the fecundity of repeat spawning Atlantic cod can be highly modified by feeding conditions prior to spawning (Kjesbu et al. 1991). Consequently, from this knowledge it would seem likely that temporal changes in fecundity at weight in first-time spawners is more indicative of a genetic change in reproductive investment than changes in the fecundity of repeat spawners.

Further evidence for the potential for fisheries-induced evolution to occur may come from a comparison of reproductive investment between heavily and lightly exploited populations. There is increasing evidence of population-specific differences in life-history traits from field investigations, including maturity and fecundity at size (Marteinsdottir & Begg 2002, Olsen et al. 2004, Yoneda & Wright 2004). Whilst much of the variability may be environmentally induced, common-environment experiments indicate the existence of genetically determined differences in some traits. For example, apparent genetic differences in some growth-linked parameters have been found between and within Atlantic silverside *Menidia menidia* (Conover et al. 2005) and Atlantic cod stocks (Purchase & Brown 2001, Salvanes et al. 2004). It may therefore be useful to contrast the state-dependent thresholds for maturation decisions among populations and then relate any differences to exploitation history.

Acknowledgements. Comments on an earlier draft of the essay by Drs. F. Neat and C. T. Marshall are greatly appreciated. This work was supported by Scottish Executive Project MF 07a.

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Editorial responsibility: Howard Browman (Associate Editor-in-Chief), Storebø, Norway

Submitted: August 3, 2006; Accepted: December 8, 2006
Proofs received from author(s): March 2, 2007

Vol. 335: 285–288, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published April 16

Maturation responses of salmonids to changing developmental opportunities

John E. Thorpe*

University of Glasgow, Institute of Biomedical and Life Sciences, Glasgow G12 8QQ, UK

ABSTRACT: Maturation is the allocation of energy to growth and differentiation of germinal tissue to the ultimate production of gametes. In Atlantic salmon *Salmo salar*, maturation begins in the egg soon after fertilisation and continues intermittently until the individual is capable of spawning. Completion of the process depends on exceeding genetically determined biochemical thresholds (lipid status) in critical seasons (through responsiveness to photoperiod cues). Hence, maturation is regulated by inhibition, and age and size at maturity depend on physiological efficiency (genetic endowment) and developmental opportunity (environmental context). This interaction of genetic diversity and developmental flexibility leads to multiple maturation trajectories (up to 32 in steelhead trout *Oncorhychus mykiss*) and wide variation in age and size at spawning. Severe depletion of a Kamchatka sockeye salmon *Oncorhychus nerka* population through 50 yr of oceanic fishing resulted in increasing proportions maturing rapidly at small size before emigration from freshwater. In the absence of such a fishery, genetic evidence suggests that stabilising selection would ultimately restore the anadromous, slower-maturing pattern as the predominant life style to such a stock.

KEY WORDS: Maturation · Salmon · Control by inhibition · Fishing effects · Stabilising selection

MATURATION

Maturation is a process of allocating energy during development to the growth and differentiation of germinal tissue into gonads and to the ultimate production of ripe gametes. In teleosts, it starts within hours or days after fertilisation of the egg, and in the Atlantic salmon *Salmo salar* L. for example, gonadal tissue begins to appear during the early embryo stage (Adams & Thorpe 1989). Thereafter, maturation proceeds intermittently over a period of years until the fish is finally capable of spawning. 'First maturity', as an event for the population biologist, identifies the culmination of the process rather than the process itself. Age-at-first-maturity and size-at-first-maturity are misleading measures, because they appear to suggest that steps towards this state of reproductive ripeness have not taken place until a specific age or size is reached at which spawning can occur. Indeed, both age and size as thresholds are concepts that are probably undetectable to a fish — how does a fish know how old or how large it is (Thorpe 1986)? The fish's developmental decisions are likely to be based on proximate cues, both internal and external, largely independent of size and age.

CONDITIONAL RESPONSES

If it was not already evident from the wild, the physiological flexibility of the maturation process has been demonstrated abundantly in aquaculture, where maturity has been accelerated or delayed both environmentally and genetically, at ages, sizes and seasons spanning and exceeding those recorded in the wild, to suit the needs of the cultivator. The opportunistic element in this was expressed well by Policansky (1983), who noted that '…under stable conditions with abundant food, [fishes] should grow rapidly and mature as soon as they are developmentally able to do so'.

*Email: johnethorpe@btinternet.com

Typically, fish populations respond to heavy exploitation with a reduction in both the age and the size at which the average individual completes sexual maturity (e.g. Ricker 1981). The rapidly reduced density of older, larger fish is presumed to enhance opportunities for development of younger, smaller individuals. Increased developmental rate is associated with earlier maturation at smaller size, and continued removal by the fishery of older, larger fish provides the opportunity for natural selection to favour earlier maturity (Thorpe 1993). There is also evidence of environmentally induced changes independent of fisheries in the rates and sizes at which fish achieve reproductive success in the wild (Bigler et al. 1996, Pyper & Peterman 1999), in particular, decreases in size and increases in age associated with increases in abundance.

ENVIRONMENTAL CONTEXT OF MATURATION

Currently, much effort is being expended on exploring variation in maturation rates, using the maturation reaction norm (the age-specific body size at which 50% of individuals become mature) and attempting to partition its variance into environmental and genetic components. However, the utility of this approach is questionable, since it is largely a statistical exercise on ultimate data that does not take into account the proximate physiological processes involved. Maturation is a cyclic process. In Atlantic salmon, completion of maturation with the production of ripe gametes within the first annual cycle is physiologically possible but infrequent, being limited apparently by lipid resources in the spring (Rowe & Thorpe 1990, Rowe et al. 1991, Simpson 1992, Arndt 2000). If lipid reserves in the spring are insufficient, further gonadal investment is arrested until the anniversary of fertilisation in the autumn. Then, given adequate lipid reserves, investment in gonadal tissue will start again (Thorpe 1994a). Provided that lipid stores remain sufficiently high throughout the winter, and can be replenished during a period of rapid growth in the spring, maturation will be maintained and the individual will be fully mature by the following November. If lipid stores are depleted over the winter to a level where they cannot be replenished in the spring, further gonadal investment is inhibited and maturation is postponed for another year. This response has been exploited in aquaculture to postpone maturation and increase somatic growth (Thorpe et al. 1990, Reimers et al. 1993). This control of maturation by successive inhibition through lipid-regulated switches continues until such time as the appropriate lipid thresholds are achieved in the critical seasons of autumn and spring, when progress to the complete development of ripe gonads and gametes can be completed.

Such a physiological process, played out in a range of environments, allows for a wide range of ages and sizes at final maturity and repeated ripenings in later years (Thorpe et al. 1998, Rikardsen et al. 2004). For example, there may be up to 32 distinguishable developmental trajectories to completion of maturity in 1 generation of steelhead trout *Oncorhynchus mykiss* Walbaum (Thorpe 1998). The fish will mature if the rate of acquisition of surplus energy during a critical period exceeds a genetically determined threshold. By this concept of maturity, temporal and spatial differences in adult sizes and ages depend on the physiological efficiency (i.e. genetic endowment) and developmental opportunity (environmental context) taken by individuals.

PHENOTYPIC AND GENOTYPIC CONSEQUENCES OF FISHING

The maturation process and the impact of fisheries upon it can be illustrated among Pacific salmonids. Typically, juveniles develop at first in fresh water and then emigrate, with a major growth phase at sea prior to returning to spawn in fresh water after 1 or more years. Heavy exploitation at sea leads to earlier maturity, presumably at first through reduced density and correspondingly improved developmental opportunity for the survivors. From a 50 yr study of a population of sockeye salmon *Oncorhynchus nerka* Walbaum in Lake Dal'neye, Kamchatka, Krogius (1979) and Varnavskaya & Varnavsky (1988) showed that the number of spawners returning from the sea decreased progressively from 62 000 per annum in the 1930s to only 4500 by the 1980s, as oceanic exploitation of maturing adults increased. The same pattern was recorded in several other Kamchatka sockeye populations over this period. With the decrease in returning fish, so the progeny populations in Lake Dal'neye decreased also. However, as sockeye density in the lake decreased, so zooplankton availability increased and with it individual development rate of juvenile sockeye here and in neighbouring lakes (Nikulin 1970). Progressively higher proportions of male sockeye matured rapidly in fresh water at small sizes as 'residents' (10 or more times smaller than sea-run adults), while their slower developing siblings emigrated to sea, providing evidence of association of improved developmental opportunity with early maturation. This tactic is seen more frequently in male salmonids, for whom maturation is energetically less expensive than it is for females. However, in Lake Dal'neye, eventually even female sockeye were maturing early at small sizes as

residents, so that by the mid-1980s residents accounted for 91.8% of the spawning population of males and 43.2% of the females. Hence, the maturation reaction norm that would have been associated with the typical anadromous behaviour of the species would have been representative no longer, because there were now not 1 but 2 sets of size distributions from which the norms would have been derived.

Beside this dramatic phenotypic change, Krogius (1979) also noted evidence of genetic change in the Lake Dal'neye stock. Altukhov & Varnavskaya (1983) and Altukhov & Salmenkova (1991) examined a number of sockeye stocks in Kamchatka and found greatest variation among them to be at the *Pgm* locus. Within the Lake Dal'neye population, heterozygosity at the *Pgm* locus in spawning resident males was 0.62 compared with 0.30 and 0.31 in 2 seasonal groups of older larger males returning from the ocean. Similar biases towards high heterozygosities were recorded from spawning resident males in Lakes Nachikinskoye and Blizhneye compared with larger, older sea-run males. Altukhov & Salmenkova (1991) concluded that the fishery was selecting heavily against the larger, less heterozygous males and in favour of the more heterozygous residents.

From independent experiments with pink salmon *Oncorhynchus gorbuscha* Walbaum with progeny sets from 8 different combinations of allozymes at 4 loci, Altukhov et al. (1991) showed that while high heterozygosity was associated with high developmental rate, the progeny of parents of average heterozygosity survived better than did those of high or low. They concluded that directional selection driving such populations towards a preponderance of smaller, earlier maturing, but highly heterozygous types would threaten population stability through reduced vitality. Hence, such populations might decline at a rate faster than could be attributed directly to fishing alone.

CONTROL OF MATURATION BY INHIBITION

Recent research by Aubin-Horth et al. (2005) revealed brain gene expression in Atlantic salmon that provides independent support for the physiological model of control of maturation by inhibition. The brain gene expression profiles suggested that delayed maturation and sea migration of immatures, the 'default' life cycle, resulted from an active inhibition of maturation, so that the emigrant juveniles represented the fish that had failed to complete maturity in that year (as was pointed out earlier, on independent physiological grounds, by Thorpe [1994b]). Since the overwhelming number of populations of Atlantic salmon are anadromous, and residency is numerically subsidiary to anadromy within those populations, this inhibition of maturation may be a genetic stabilising mechanism, favouring average rather than high or low heterozygosity (Altukhov 2006), and inhibiting Atlantic and other salmon populations from evolving toward residency. This would be a further reason for caution in focussing exploitation on the slower-developing, larger members of salmon populations, to avoid disturbance of the stabilising influence of genetic diversity.

Empirically, it is clear that salmonids are capable of completing maturation in their first year, but that most individuals are inhibited from doing so. N. Aubin-Horth's work promises to reveal more about the genetic components of this inhibition. Some external and internal conditions promoting inhibition are understood, but the physiological mechanisms responsible for seasonal arrest and subsequent restoration of investment of energy into gonads are not. Until the endocrine patterns involved are unravelled, it will not be possible to disentangle environmental and genetic components of variation in maturation of these fishes.

Acknowledgements. I thank C. T. Marshall and H. Browman for inviting me to contribute to this Theme Section.

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Editorial responsibility: Howard Browman, (Associate Editor-in-Chief), Storebø, Norway

Submitted: June 13, 2006; Accepted: December 8, 2006
Proofs received from author(s): February 28, 2007

Vol. 335: 289–294, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published April 16

Why age and size at maturity have changed in Pacific salmon

Kentaro Morita*, Masa-aki Fukuwaka

Hokkaido National Fisheries Research Institute, Fisheries Research Agency, 116 Katsurakoi, Kushiro 085-0802, Japan

ABSTRACT: Over the last few decades, the size at which Pacific salmon *Oncorhynchus* spp. attains maturity has decreased in many populations, whereas the age at maturity has increased. Both fisheries-induced evolution and environmentally-induced phenotypic plasticity could contribute to the changing age and size at maturity of Pacific salmon. We evaluated the potential for genetic changes in the maturation schedule of Japanese chum salmon using the probabilistic maturation reaction norm (PMRN) method. We found that the recent decrease in size at maturity, and increase in age at maturity, of Japanese chum salmon can be largely attributed to a phenotypic response to a reduced growth rate, but that fisheries-induced evolution should not be ruled out. Recent claims concerning fisheries-induced evolution of the maturation schedule are based on the decline in the age-specific body size at which the probability of maturing is 50%, a feature of PMRNs. However, the PMRN could change with changing environmental conditions. Therefore, a genetic change cannot be diagnosed only by the PMRN method.

KEY WORDS: Life history · Phenotypic plasticity · Conditional strategy · Thresholds

INTRODUCTION

Over the last few decades, the size at which Pacific salmon *Oncorhynchus* spp. attains maturity has decreased in many populations, whereas the age at maturity has increased (reviewed by Bigler et al. 1996). These patterns are well recognized in chum salmon *O. keta* (Ishida et al. 1993, Helle & Hoffman 1995, Kaev 1999), sockeye salmon *O. nerka* (Pyper et al. 1999, Holt & Peterman 2004) and pink salmon *O. gorbuscha* (Azumaya & Ishida 2000, Wertheimer et al. 2004). The size at maturity of endemic Asian masu salmon *O. masou* has also decreased (Tago 2002). In Japanese chum salmon, the average size at maturity of each age has decreased by ~5 cm, and the average age at maturity has increased by ~0.5 yr since the 1960s (Fig. 1a,b).

Genetic change associated with commercial fishing has been suggested as being a contributory factor to the observed decline in size at maturity (Ricker 1981, 1995). Ricker (1981, 1995) suggested that the gillnet fishery targeting maturing pink salmon removed large-sized fish at maturity that had grown fast, because almost all pink salmon mature at the same age, i.e. 2 yr. From 1952 to 1992, many Pacific salmon were caught in the high seas far from their spawning sites by the Japanese fishery using gillnets (Fig. 1c, Morita et al. 2006), which are highly size-selective (Ishida 1969). In addition, losses from gillnetting in the high seas, i.e. mortality caused by high-sea fishing with gillnets but not included in catch statistics, is substantial at ~25 to 50% (Ricker 1976). In such a situation, a fish that matured at a smaller size than the fishable size could take refuge from the high-seas fishery. Therefore, artificial selection toward a small size at maturity could occur for Pacific salmon. In contrast, Welch & Morris (1994) suggested that artificial selection for slower-growing fish cannot be the primary cause of the long-term decline in the size of pink salmon observed since the 1950s because their size at maturity in the early 1930s was similar to what it is today. Additionally, although it may explain the decline in size at maturity, it does not seem to address the increase in age at maturity (Healey 1986).

*Email: moritak@affrc.go.jp

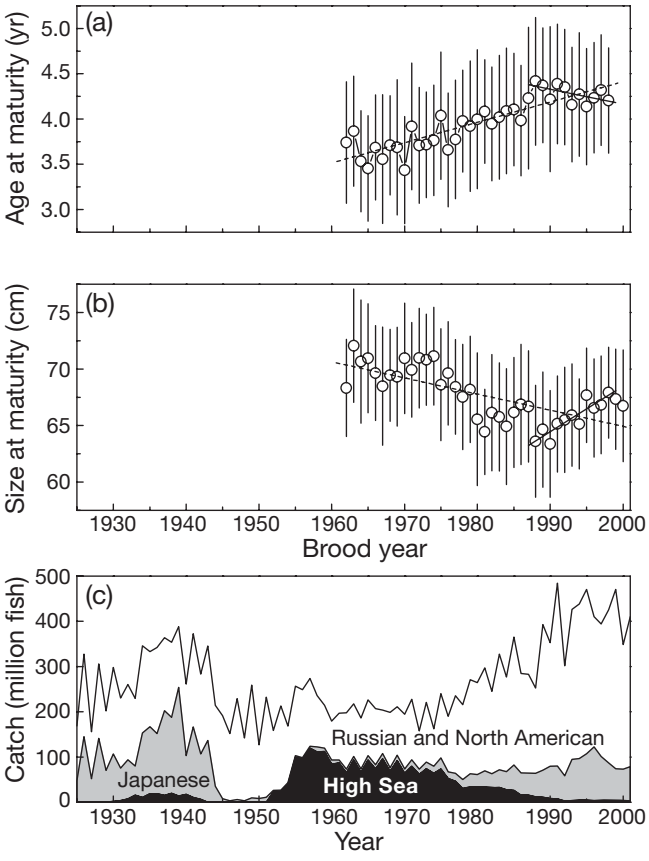

Fig. 1. (a) Long-term changes in average age at maturity of chum salmon *Oncorhynchus keta*: dashed line, 1962 to 1998, r = 0.87, p < 0.001; solid line, 1988 to 1998, r = –0.57, p = 0.07. (b) Long-term changes in average size at maturity of Age 4 yr chum salmon: dashed line, 1962 to 2000, r = –0.70, p < 0.001; solid line, 1988 to 1998, r = 0.90, p < 0.001. Biological data are averages from 3 major rivers in northern Japan (Ishikari, Tokachi and Nishibetsu) monitored by the National Salmon Resources Center, Fisheries Research Agency. Error bars are SD. (c) Catch trends of Pacific salmon *Oncorhynchus* spp. showing the Japanese, Russian and North American fisheries and Japanese high-seas fishery (Eggers et al. 2003)

The genetic changes associated with fishing become known as fisheries-induced evolution (Conover et al. 2005, Reznick & Ghalambor 2005). High fishing mortality and size-selective fishing are two of the most important causes of fisheries-induced evolution. Theoretical studies showed that selection for early maturation (i.e. decreased age and size at maturity) is more likely to occur, but selection for delayed maturation is also possible depending on the pattern of artificial selection (Law & Grey 1989, Heino 1998, Ernande et al. 2004). Fisheries-induced evolution has been suggested to account for some of the observed trends in age and size at maturity (e.g. North Sea plaice *Pleuronectes platessa*, Grift et al. 2003; Atlantic cod *Gadus morhua*, Olsen et al. 2004, 2005).

This essay comprises (1) a brief literature review of the environmental causes of the decline in size at maturity of Pacific salmon, (2) an evaluation of the potential for genetic changes in the maturation schedule of Japanese chum salmon, using the probabilistic maturation reaction norm (PMRN) method, and (3) a critical review of the application of the PMRN method to the study of fisheries-induced evolution.

ENVIRONMENTAL CAUSES OF THE DECLINE IN SIZE AT MATURITY

Environmentally-induced phenotypic plasticity contributes to the changing age and size at maturity of Pacific salmon (Ishida et al. 1993, 1995, Welch & Morris 1994, Cox & Hinch 1997, Pyper & Peterman 1999, Wertheimer et al. 2004). Most studies suggest relationships among temperature, population size, and body size at maturity. For example, the growth of Pacific salmon is density-dependent, and the number of individuals of this genus more than doubled during the last quarter of the 20th century. This increase in population size may have led to a decrease in per-capita food availability and, thus, a decrease in the size at maturity (Bigler et al. 1996). Ishida et al. (1993, 1995) found that increases in abundance and decreases in sea-surface temperature were significantly associated with the reduced body length of chum salmon in the North Pacific. Interestingly, these trends appear to have reversed during the 1988 to 1998 brood years (Fig. 1; Kaeriyama & Katsuyama 2001, Eggers et al. 2003, Kaev & Romasenko 2003), and size at maturity has been increasing with decreasing population size since the late 1990s (Kaeriyama & Katsuyama 2001). Pyper & Peterman (1999) reported that increases in abundance and sea-surface temperature were significantly associated with reduced adult body length in sockeye salmon in British Columbia and Alaska. However, the evidence supporting environmentally-induced phenotypic plasticity is based on correlation, and not causation.

ASSESSING FISHERIES-INDUCED EVOLUTION USING THE PMRN METHOD

Recent claims concerning fisheries-induced evolution of the maturation schedule are based on the decline in the age-specific body size at which the probability of maturing is 50%, a feature of PMRNs (e.g. Grift et al. 2003, Olsen et al. 2004, 2005). The PMRN method is based on the logistic regression:

$$\mathrm{logit}(p) = \log\left(\frac{p}{1-p}\right) = c_{0,a} + c_{1,a}l$$

where p is the probability of maturing, l is body size, $c_{0,a}$ is a constant for each age a, and $c_{1,a}$ is a coefficient for l for each age. Assuming that a fitted logistic regression is a cumulative distribution function, the body size at 50% probability of maturing indicates an average size threshold for maturity that can be calculated as $-c_{0,a} \times c_{1,a}^{-1}$, with a variance of $\pi^2/3c_{1,a}^2$ (Metcalf et al. 2003). Because it is usually difficult to identify newly matured fish (i.e. first-time spawning fish) for iteroparous species, a statistical method to estimate PMRNs when age at first maturity is unknown has been proposed (Grift et al. 2003, Barot et al. 2004). Usually, the variance in the size threshold for maturity is not negligible, indicating that genetic variance or factors other than body size affect maturation.

Morita et al. (2005) estimated the PMRN using data on chum salmon ascending the Shari River, eastern Hokkaido, Japan, during the breeding season of 1992 to 1997 (Fig. 2). Because Pacific salmon are semelparous species, the maturity ogive is the same as the PMRN, i.e. we need not use the method of Barot et al. (2004). The probability of maturing increased with fork length, but the reaction norm midpoint at 50% maturation probability (i.e. average threshold size at maturity) decreased with increasing age (Fig. 2). Note that the observed average size at maturity is markedly different from the reaction norm midpoint at 50% maturation probability. The average size at maturity increased with age, but this does not mean that older fish need a larger body size to mature.

Morita et al. (2005) simulated the potential modification of average age and size at maturity of chum salmon in response to changing growth rate using a size-structured model with constant age- and size-specific maturation rates (i.e. time-invariant PMRNs, as in Fig. 2). The results suggested that the decrease in size at maturity and increase in age at maturity over the last 4 decades could result from a reduced growth rate alone, without any change in the PMRN (Fig. 3). However, to our knowledge, no studies have reported the long-term trends in the PMRN in Pacific salmon, because it is generally difficult to estimate size-frequency distribution of immature *Oncorhynchus* spp.

CAN PMRNs DIAGNOSE GENETIC CHANGES?

The PMRN method quantifies the maturation schedule after accounting for the variation in body size at a given age. Therefore, the PMRN may be insensitive to phenotypic plasticity in growth because it assumes that body size is sufficient to determine maturation at a given age. If the variation in the threshold size at maturity, $\sigma^2 = \pi^2/3c_{1,a}^2$, is attributable to genetic variance (i.e. the heritability of the threshold size at maturity is high), the method would be useful for assessing genetic changes in the maturation schedule. To understand genetic changes in the PMRN, knowledge of the heritability of the threshold size at maturity is critical (cf. Wesselingh & de Jong 1995, Wesselingh &

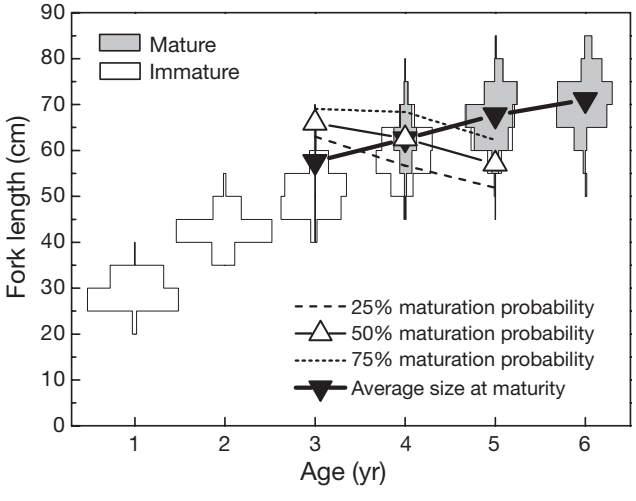

Fig. 2. *Oncorhynchus keta*. Probabilistic maturation reaction norm (PMRN) of chum salmon (Morita et al. 2005). Data include 3 cohorts, the 1989 to 1991 brood years, and pooled sexes. Left and right histograms for each age correspond to females and males, respectively. There is no significant difference in the PMRN between sexes; however, size at each age differed significantly between sexes. Details of data and their treatment are described elsewhere (Morita et al. 2005)

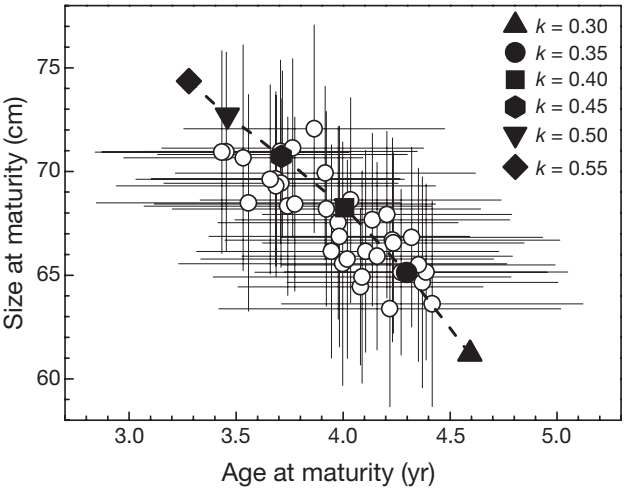

Fig. 3. *Oncorhynchus keta*. Simulated and observed covariation in average age at maturity and average size at maturity of Age 4 yr chum salmon. Filled symbols indicate simulated changes in response to changing growth rate k using a size-structured model (Morita et al. 2005) with an invariable probabilistic maturation reaction norm as in Fig. 2. Open circles are observed data as in Fig. 1a,b. Error bars are SD

Klinkhamer 1996). However, a comprehensive understanding of the sources of variation in the threshold size at maturity (genetic or environmental variance) is still lacking in fishes.

Spatial variation in threshold size may reflect variable environmental conditions provided by different habitats, or different genotypes adapting to different habitats. For example, the threshold size at maturity of male parr differs according to habitat in Atlantic salmon *Salmo salar* (Aubin-Horth & Dodson 2004, Baum et al. 2004, Aubin-Horth et al. 2006). Baum et al. (2004) and Aubin-Horth et al. (2006) showed that the threshold size at maturity decreased with altitude and distance from the mouth of the river, respectively. In addition to genotype, temperature, food availability, condition factors and growth history affect the maturation probability independent of body size at a given age (Bromage et al. 1992, Silverstein et al. 1998, Henderson & Morgan 2002, Dhillon & Fox 2004, Morgan 2004, Baum et al. 2005, Watanabe & Yatsu 2006, Morita & Fukuwaka 2006).

Morita et al. (2000) compared the maturation schedule of white-spotted charr *Salvelinus leucomaenis* in below-dam river sections accessible to the migrant form with above-dam river sections inaccessible to the migrant form. Because above-dam river sections are populated by only the resident form (i.e. precocious fish), it is hypothesized that above-dam fish will mature at earlier ages and smaller sizes. We reanalyzed these data (Morita et al. 2000, Morita & Yamamoto 2001) using a PMRN method (Fig. 4). Age 1+ yr males above the dam had a smaller threshold size at maturity than did those below. Because a significant difference was observed in PMRNs between above- and below-dam sections (likelihood ratio test $G^2_2 = 31.2$, $p < 0.001$), one may suppose that fish have evolved rapidly following isolation by dams. However, the PMRN for below-dam fish changed plastically when they were transplanted to an above-waterfall section about 16 mo before maturation ($G^2_2 = 36.3$, $p < 0.001$), and no significant difference between the sections was detected when fishes from both sections were transplanted to a common above-waterfall section ($G^2_2 = 0.425$, $p = 0.809$). Therefore, the observed difference in the PMRN for these populations could be largely attributable to differences in environmental conditions (Morita et al. 2000).

Morita & Fukuwaka (2006) examined the relationships among body size, previous growth history and maturation probability in chum salmon. Previous growth history was more closely linked to maturation probability than was body size, and the annual growth increment of the previous year was the most important factor affecting whether a fish matured during the subsequent season. This finding is consistent with endocrinological studies of salmonid maturation in which somatic growth during the fall and winter affected the onset of maturation in the next autumn (Campbell et al. 2006). Thorpe (1986) argued that body size is a measure of past performance; in contrast, growth rate is a measure of current performance and thus provides a better basis for developmental decisions than body size alone. Thus, the PMRN could change with changing growth patterns. Morita & Fukuwaka (2006) showed that the relationship between body size and maturation could probability be plastically modified by growth history because individuals of similar body size (at the same age) can have different growth histories.

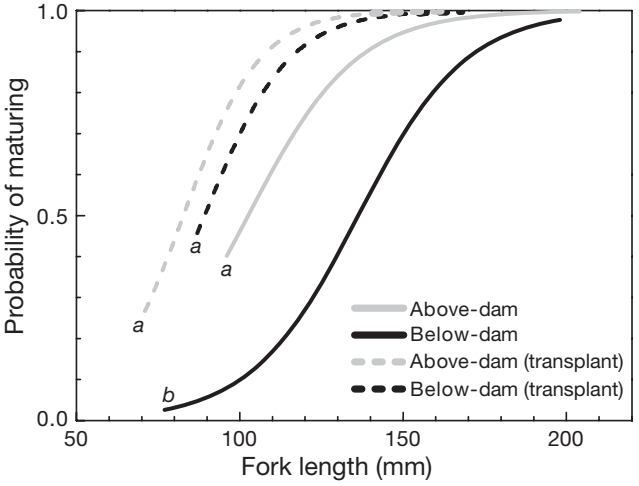

Fig. 4. *Salvelinus leucomaenis*. Fitted logistic regression of the probability of parr maturation based on fork length of Age 1+ yr male white-spotted charr above and below dams (data from Morita et al. 2000), and for fishes from each section transplanted to a common above-waterfall section about 16 mo before maturation (data from Morita & Yamamoto 2001). Regressions plotted for the range of fork lengths used. Different letters denote statistically significant differences based on likelihood-ratio tests

Therefore, genetic change cannot be diagnosed only by the PMRN method. When interpreting the trends of the PMRN, it is necessary to quantify how the age-specific body size at 50% probability of maturing is altered in response to changing temperature and food availability before invoking evolution. Even when age-specific growth rates show no change between years, food availability may show an increasing trend coincident with increasing age-specific maturation rates (i.e. declining trends in age and size at maturity) because energy allocation toward somatic growth versus reproduction should decrease. It is still unclear how the PMRN is altered in response to changing environment; this merits further study (e.g. Grift et al. 2007).

CONCLUDING REMARKS

The age and size at maturity of fishes, including salmonids, are flexible and depend on a variety of environmental conditions. The PMRN may also covary with changing environments. While it is important to detect evidence of the effects of fisheries-induced evolution on age and size at maturity, it is equally important to address the relative contribution of fisheries-induced evolution and environmentally induced phenotypic plasticity to the observed changes in age and size at maturity. For example, the recent decrease in size at maturity and increase in age at maturity of Japanese chum salmon can be largely attributed to a phenotypic response to a reduced growth rate, but fisheries-induced evolution should not be ruled out.

Acknowledgements. We thank the National Salmon Resources Center for providing data. We also thank C. T. Marshall and H. Browman for improving an earlier draft of this essay. This research was supported by Grant-in-Aid No. 16780143 from the Ministry of Education, Culture, Sports, Science, and Technology of Japan.

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Editorial responsibility: Howard Browman (Associate Editor-in-Chief), Storebø, Norway

Submitted: April 5, 2006; Accepted: December 11, 2006
Proofs received from author(s): March 8, 2007

Vol. 335: 295–300, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published April 16

OPEN ACCESS

Does the probabilistic maturation reaction norm approach disentangle phenotypic plasticity from genetic change?

S. B. M. Kraak*

Wageningen IMARES (Institute for Marine Resources and Ecosystem Studies), Wageningen UR, PO Box 68, 1970 AB, IJmuiden, The Netherlands

ABSTRACT: Two-dimensional probabilistic maturation reaction norms (PMRNs) define the probability that individuals mature as a function of age and size. PMRNs have recently been used to derive empirical support for the hypothesis that high fishing mortality has induced genetic change towards earlier maturation in exploited populations. However, the 2-dimensional PMRN method does not, strictly speaking, disentangle phenotypic plasticity from genetic change in maturation. Instead, it disentangles the contribution of variation in growth and mortality from other sources of variation contributing to changes in maturation. After removing the contribution of variation in growth and mortality, any remaining change is not necessarily purely genetic. Environmental factors may exist that directly affect the propensity to mature at a certain size and age. An observed trend in the size-age PMRN may therefore be partly or fully explained by a co-incidental trend in an (environmental) factor. As pointed out by Grift et al. (2007; Mar Ecol Prog Ser 334:213–224), such co-dependence is captured conceptually by multi-dimensional PMRNs, as opposed to 2-dimensional (size-age) PMRNs, where the higher dimensions represent the relevant variables. Dimensions that may partly or fully explain the observed trends in size-age PMRNs, other than evolution, include temperature, polluting endocrine-disrupting chemicals, the social structure of the population, and body condition; changes in the last two may be fisheries-induced. To illustrate these views, I re-analysed data on North Sea plaice, and demonstrated that temperature explains part of the shift in the size-age PMRN but that a residual shift remains. The latter finding supports the hypothesis of genetic change.

KEY WORDS: Probabilistic maturation reaction norms · Fisheries-induced evolution · Phenotypic plasticity · Maturation process

INTRODUCTION

During the last few decades, a number of studies have drawn attention to fisheries-induced adaptive genetic shifts in life history traits (e.g. Borisov 1978, Law & Grey 1989, Rijnsdorp 1993, Law 2000, Conover & Munch 2002, Heino et al. 2002a, Ernande et al. 2004). One of the best studied phenomena is that high fishing mortality produces a selection pressure favouring maturation earlier in life owing to a reduction in life-span expectancy (e.g. Borisov 1978, Law & Grey 1989, Rijnsdorp 1993, Ernande et al. 2004). This idea is conceptually straightforward: if the mortality rate of adults is high, individuals that are genetically predisposed to early maturation will have a higher chance of reproducing and passing on their genes to the next generation than late-maturing individuals. Progress in this field of research was made due to the development of a statistical technique (Heino et al. 2002b) through which so-called probabilistic maturation reaction norms (PMRNs) are determined from population data on age, length and maturity status of individual fish. PMRNs define the probability that an individual matures as a function of its age and size (Fig. 1a). Application of the PMRN method provided support for the hypothesis that fisheries-induced evolutionary

*Email: sarah.kraak@wur.nl

changes in age and size at maturation have occurred in some important exploited fish populations on contemporary time scales, e.g. in Atlantic cod (Heino et al. 2002a, Barot et al. 2004, Olsen et al. 2004, 2005), North Sea plaice (Grift et al. 2003) and American plaice (Barot et al. 2005). Fisheries management bodies are being advised to consider these effects of fishing (Rijnsdorp 1993, Browman 2000, Law 2000, Pauly et al. 2002) because a shift towards earlier maturation will lead to smaller average adult size, thereby lowering yields. Because genetic changes may be irreversible or can only be reversed over a much longer time period than that in which they were brought about (Law & Grey 1989), the consequences are long-lasting.

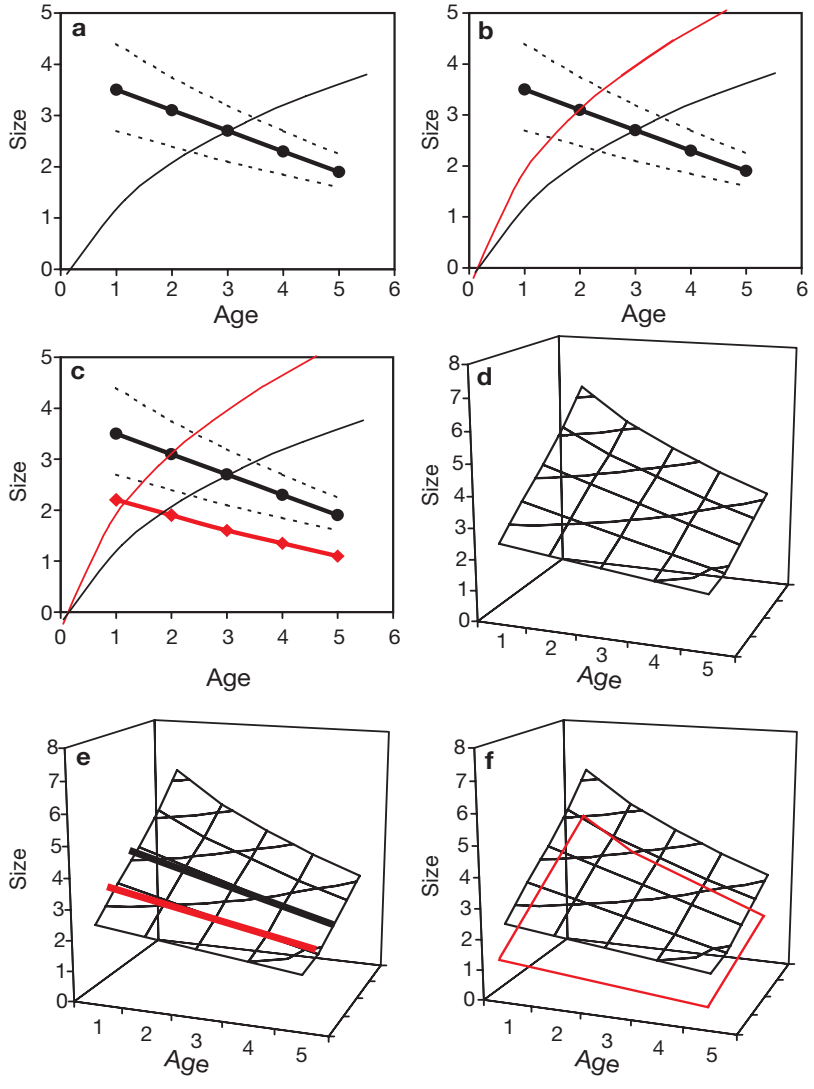

Fig. 1. (a–c) 2-dimensional probabilistic maturation reaction norms (PMRNs), defining the probability that an individual matures as a function of its age and size, and (d–f) 3-dimensional PMRNs, defining the probability that an individual matures as a function of its age, size and state along a 3rd dimension (e.g. condition or temperature). This is an illustration of the concept: no actual data were used. Straight solid black and red lines represent combinations of age and size at which the probability of maturing is 50%. Broken lines represent combinations of age and size at which the probability of maturing is 10 and 90%, respectively. Curved red and black lines represent average growth curves. Black and red planes represent combinations of age and size and state along the 3rd dimension at which the probability of maturing is 50%. Above and below these planes, planes can be envisaged as representing states at which the probability of maturing is for example 90 and 10%, respectively. (a) 2-dimensional PMRN, (b) 2-dimensional PMRN and increased growth rate (red curve), (c) shifted 2-dimensional PMRN (red straight line), (d) 3-dimensional PMRN, (e) 3-dimensional PMRN on which the size-age PMRN 'slid down' along the 3rd dimension (red straight line), (f) shifted 3-dimensional PMRN (red plane)

Let me describe the context in which the PMRN method was developed. Several decades ago it was found that, in some heavily fished populations, the age at maturation had declined (Trippel 1995). Three hypotheses were brought forward to link the shift to the high fishing mortality (e.g. Rijnsdorp 1993, Heino et al. 2002a, Grift et al. 2003, Barot et al. 2004, 2005). The first hypothetical cause is a demographic effect of high fishing mortality. In this case, the population simply became more dominated by younger and smaller individuals, while late-maturing fish were lost. According to the second hypothesis of phenotypic plasticity, the fish may have responded to the lower density of the heavily fished population and the associated greater food availability by growing faster. These faster-growing fish would simply reach the stage of maturation at an earlier age (Fig. 1b). The third hypothesis states that fish that were genetically predisposed to mature at a younger age and a smaller size were favoured by selection caused by high fishing mortality. These 3 hypotheses are not mutually exclusive. The method of PMRNs (Heino et al. 2002a,b,c) takes account of the first 2 effects, because it quantifies the probability of maturing conditional on a fish reaching a certain size at a certain age. This way, any effect that remains is likely to be a result of genetic change. Authors that used this method advertised it as a powerful approach that helps to disentangle the effect of phenotypic plasticity from the effect of genetic change (Fig. 1b,c; Heino et al. 2002a,c, Grift et al. 2003, Barot et al. 2004, 2005, Engelhard & Heino 2004, Olsen et al. 2005).

In this essay, I caution against drawing too strong an inference from the application of the PMRN method and question to what ex-

tent the method enables the disentanglement of phenotypic plasticity from genetic change. Even some authors who used the method previously expressed some caution in this regard (Heino et al. 2002a,c, Grift et al. 2003, Barot et al. 2004, 2005, Olsen et al. 2004, 2005). In the next section, I argue that environmental variables other than those that affect maturation through growth need to be taken into account. In the subsequent section this is illustrated through an example. The essay concludes with an answer to the questions raised in the title.

PHENOTYPIC PLASTICITY VS. GENETIC CHANGE

The PMRN method disentangles the contribution of variation in growth and survival from other sources of variation that contribute to changes in maturation (Olsen et al. 2005). This is not the same as disentangling phenotypic plasticity in maturation from genetic variation in maturation. Firstly, although variation in growth may have an important environmental component, it will often also have a genetic component (e.g. Conover & Munch 2002, Heino et al. 2002c). A population could hypothetically respond to fishing pressure genetically with faster growth (faster-growing fish would reach maturity earlier and thereby have a selective advantage) or slower growth (slower-growing fish delay exposure to capture; Sinclair et al. 2002a,b). More importantly however, it will not necessarily be the case that after removing the contribution of the variation in growth and mortality, any remaining change — observed as a shift in the PMRN in size-age space (Fig. 1c) — is purely genetic. Instead, environmental factors may exist that directly affect the propensity to mature at a certain size and age (Heino et al. 2002a,c, Grift et al. 2003, Barot et al. 2004, 2005, Olsen et al. 2004, 2005). Grift et al. (2007) thus introduced a multi-dimensional PMRN as opposed to a 2-dimensional (size-age) PMRN, where the higher dimensions represent the relevant environmental variables. An application of this approach (provided by Grift et al. 2007) used body condition as the third explanatory variable to characterize the probability of maturing. A 3-dimensional PMRN consists of a plane (Fig. 1d) instead of a line (Fig. 1a), which connects states in which fish with a given genetic predisposition would have a 50% probability of maturing.

A potential additional or alternative explanatory variable (Heino et al. 2002b,c, Olsen et al. 2004, 2005), which can be represented as an additional dimension, could be temperature. Temperature may have an effect on the probability of maturing by itself, independent of its effect via growth (Grift et al. 2003, Dembski et al. 2006). A fish that had grown at a certain rate, such that it had reached a certain size at a certain age, may have different probabilities of maturing depending on the temperature experienced. It remains to be shown whether globally increasing water temperatures have contributed to any of the published shifts in PMRNs (Fig. 1c,e).

Another candidate variable that has probably changed globally and more or less unidirectionally over the last few decades is the concentration of polluting chemicals in the water. Endocrine-disrupting chemicals (Zala & Penn 2004) may influence the tendency to mature (Devlin & Nagahama 2002). Social factors could also have an effect: in some fish species, the probability of maturing at a given size and age depends on the social structure and size composition of the local population (Fricke & Fricke 1977, Hobbs et al. 2004), but it is not known whether this is the case in those exploited species in which PMRNs have been investigated. The social structure of exploited fish populations may have changed as a result of fishing, e.g. through its effect on population density (Rowe & Hutchings 2003), on the size and age composition, and on the sex ratio in cases where fishing mortality affects the sexes differentially (Kell & Bromley 2004). The probability of maturing at a given length and age may also depend on body condition (Bromley 2000, Grift et al. 2007). Environmental changes (e.g. increased food availability resulting from fisheries-induced population decline) may have caused a trend in condition. If adopting the 2-dimensional view, one would perhaps infer genetic change (Fig. 1c), but in fact the hypothesis of a plastic response to environmental changes that affected condition would not have been excluded. The 2-dimensional PMRN may just have 'slid down' on the plane of the 3-dimensional PMRN in the direction of higher condition (Fig. 1e).

Hence, an observed shift in the PMRN in size-age space (Fig. 1c) is not conclusive evidence that a genetic change has taken place, or is the only cause, because the possibility of a phenotypically plastic response to environmental changes other than via growth (Fig. 1e,f) has not been excluded. It remains to be investigated whether trends in temperature, pollutants, social structure, condition or other variables coincided with any observed trend in the PMRN. An aim for researchers in this field could be to quantify the relative importance of candidate factors to the observed trend.

TEMPERATURE AND MATURATION IN NORTH SEA PLAICE

I report here a re-working of the data used by Grift et al. (2003). Grift et al. (2003) applied the 2-dimensional PMRN method to females of 41 cohorts (from 1955 to 1995) of North Sea plaice *Pleuronectes platessa* L., and

found that the PMRN for age and length at maturation had significantly shifted towards younger age and smaller length (Fig. 1c). They concluded that this long-term trend suggests a genetic response. They also observed short-term fluctuations superimposed on the long-term trend, and showed that these could be partly explained by short-term variations in temperature. For all ages, the probability of maturing at a given length increased significantly with the temperature experienced 2 and 3 yr prior to the year in which the maturity status of the fish was assessed. These lags were used because a previous analysis (Rijnsdorp 1993) had indicated that growth 2 and 3 yr earlier may affect maturation. Temperature itself exhibited a significant temporal trend (an average increase of 0.02° C per year, Fig. 2), but Grift et al. (2003) removed this trend because they wanted to analyse the short-term effect of temperature. However, in the context of the argument advocated in this essay, the trend seen in the 2-dimensional PMRN may be partly caused by this trend in temperature. The probability of maturing at a particular age and length may have increased as a result of increased temperature: the size-age PMRN may have 'slid down' on the plane of the 3-dimensional PMRN in the direction of higher temperature (Fig. 1e). In that case, not only the short-term fluctuations but also the long-term trend of the size-age PMRN may be partly explained by changes in water temperature. Only if a trend in the size-age PMRN remains after removal of the effect of temperature does the evidence support a genetic response (Fig. 1f).

I analysed the yearly PMRN midpoints (L_{p50}, the length at which the probability of maturing equals 50%) for each of the Ages 2 to 6 yr. I tested with simple regression whether the midpoints were correlated with

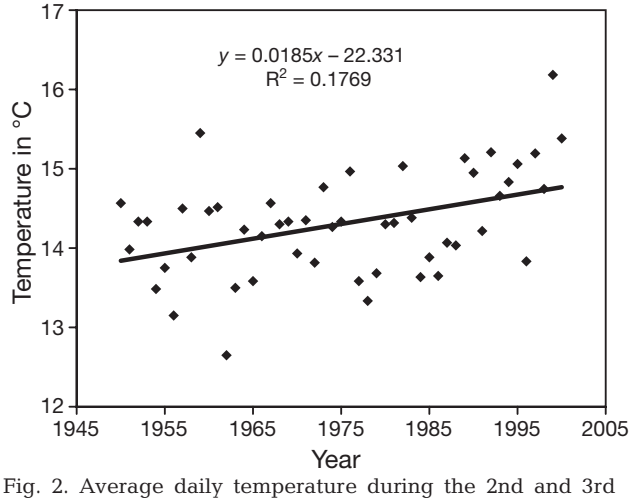

Fig. 2. Average daily temperature during the 2nd and 3rd quarter of the year measured at a fixed time of day at Den Helder (theNetherlands) after correction for the tidal phase; p = 0.002

the average temperature of the second quarter of the year before the maturity status of the fish was assessed (t_{yr-1}). North Sea plaice start their maturation process around July of the year before the spawning season, which extends from January to March (Rijnsdorp & Witthames 2005). I hypothesised that the temperature just prior to the onset of maturation may affect the probability of maturing. If the effect was significant, I removed this effect and tested whether the remaining variation (i.e. the residuals of the regression) was correlated with the average temperature of the 2nd and 3rd quarter 2 yr prior (t_{yr-2}). If that effect was significant, I also removed that effect and tested whether the remaining variation correlated with the 2nd and 3rd quarter temperature 3 yr prior (t_{yr-3}). If that effect too was significant, it was also removed and I tested whether the remaining variation correlated with the year of birth of the cohort. Similarly to Grift et al. (2003), I chose to test the effects of t_{yr-2} and t_{yr-3} because previous analysis (Rijnsdorp 1993) indicated that growth 2 and 3 yr earlier may affect maturation. The exact statistical treatment (e.g. in what order to remove effects, whether and how to deal with the autocorrelation of the annual temperatures, whether Bonferroni correction [Rothman 1990] is desirable) should depend on the hypothesised mechanisms of the influences of temperature at different times in development. I do not explicitly formulate candidate mechanisms here because this is outside the scope of this study; I merely want to emphasize the importance of exploring alternative explanations for shifts in PMRNs, with the analysis of the North Sea plaice data as an illustration.

Values of t_{yr-1} only correlated with the probability of maturing for Ages 3 and 6 yr (Table 1). An increase of 1°C corresponded to a decrease in the L_{p50} of 1.4 cm for Age 3 yr and 1.3 cm for Age 6 yr. For all ages except Age 6 yr, t_{yr-2} correlated significantly with the probability of maturing after removal of significant effects (Table 1). An increase of 1°C corresponded to decreases varying from 3.8 cm to 1.1 cm in the L_{p50}. For Ages 3 and 4 yr, an additional significant effect of t_{yr-3} was found (Table 1): the L_{p50} decreased by 1.6 and 1.0 cm, respectively, with an increase of 1°C. With these significant effects removed, the probability of maturing still significantly correlated negatively with cohort birth year for all ages. Hence, after taking the influence of temperature experienced 1, 2 and 3 yr earlier into account, there remained a time trend in the L_{p50}. For fish of Age 2 yr the L_{p50} decreased by 0.16 cm yr^{-1}, for Ages 3 and 4 yr by 0.09 cm yr^{-1}, and for Ages 5 and 6 yr by 0.10 cm yr^{-1} (Table 1).

Two conclusions can be drawn from this re-analysis. First, the trend in the PMRN for North Sea plaice as observed by Grift et al. (2003) can indeed be partly

Table 1. *Pleuronectes platessa*. Correlations of probabilistic maturatiom reaction norm (PMRN midpoints with t_{yr-1}, t_{yr-2}, t_{yr-3} and cohort after successive removals of significant effects (at $\alpha = 0.05$) (see section 'Temperature and maturation in North Sea plaice'). Slopes of significant regressions (at $\alpha = 0.05$) are given in **bold**; na: not applicable (Age 2 yr fish not yet born); p-values given in parentheses

Correlates	Age 2	Age 3	Age 4	Age 5	Age 6
t_{yr-1}	(0.2)	**−1.4** (0.02)	(0.16)	(0.08)	**−1.3** (0.02)
t_{yr-2}	**−3.8** (0.01)	**−1.8** (0.007)	**−1.4** (0.004)	**−1.1** (0.05)	(0.07)
t_{yr-3}	na	**−1.6** (0.008)	**−1.0** (0.02)	(0.12)	(0.13)
Cohort	**−0.16** (0.02)	**−0.09** (0.002)	**−0.09** (6×10^{-7})	**−0.10** (4×10^{-5})	**−0.10** (0.003)

explained by temperature changes over that time period (Fig. 1e). These results do not contradict the findings of Grift et al. (2003). These authors also demonstrated the effect of temperature; however, they did not test whether temperature changes can partly explain the trend in the 2-dimensional PMRN. My analysis of the effect of temperature is a preliminary exploration. Future work may address questions such as why the effect of t_{yr-1} is found only for Ages 3 and 6 yr, and by what mechanism is the probability to mature influenced by the temperature experienced immediately prior to the onset of maturation. Other questions remain as well, such as whether t_{yr-2} and t_{yr-3} affect the probability of maturing via their effect on growth during earlier development. Second, the effects of temperature are not sufficient to explain the observed trend in PMRN. A residual trend in time remains, and therefore the hypothesis that fisheries-induced genetic change has occurred still receives support (Fig. 1f). The support for this hypothesis increased through this analysis compared to basing it on the findings of Grift et al. (2003) alone, because some effects that might have been alternative explanations for the shifted PMRN have been removed.

Another illustration of how to deal with additional or alternative explanatory factors is provided by the study on North Sea plaice by Grift et al. (2007), where variation in body condition was taken into account. The fact that the 3-dimensional PMRN in size-age-condition space shifted significantly over time (Fig. 1f) lends further support to the genetic-change hypothesis than it received from the findings of Grift et al. (2003) alone.

CONCLUSION

Does the PMRN approach disentangle phenotypic plasticity from genetic change? No, total disentanglement cannot be reached, and yes, it certainly helps disentanglement. It is impossible to identify and quantify the effects of all the non-genetic candidate factors that may possibly influence maturation, such that any residual trends could be assumed to reflect only genetic change. However, on the positive side, the PMRN approach can make good progress along the never-ending road towards disentanglement, especially when more factors are considered. The continuing challenge remains to design and carry out studies aimed at obtaining ever-more insight into the complex causation of changes in maturation, but science will never reveal all the details. In my opinion, there lies great promise in the open-minded use of the multidimensional PMRN approach and variations thereof when tackling the challenge of disentangling causes of changes in maturation.

Acknowledgements. I thank C. T. Marshall for the invitation to contribute to this Theme Section. I thank A. D. Rijnsdorp and R. E. Grift for discussions and reviewing earlier versions of the manuscript. U. Dieckmann, M. Heino, B. Ernande, P. Bromley, M. Dickey-Collas, A. M. de Roos, F. Mollet, and B. Mundwiler are thanked for discussions and C. T. Marshall, H. Browman, and B. McAdam for their comments on the manuscript.

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Editorial responsibility: Howard Browman, (Associate Editor-in-Chief), Storebø, Norway

Submitted: February 2, 2006; Accepted: December 11, 2006
Proofs received from author(s): April 3, 2007

Vol. 335: 301–310, 2007 | MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser | Published April 16

OPEN ACCESS

Integrated perspectives on genetic and environmental effects on maturation can reduce potential for errors of inference

C. Tara Marshall*, Bruce J. McAdam

School of Biological Sciences, University of Aberdeen, Tillydrone Avenue, Aberdeen AB24 2TZ, UK

ABSTRACT: In exploited fish stocks, long-term trends towards earlier maturation have been interpreted as an evolutionary response to sustained, high fishing mortality. The evidence used to support this diagnosis consists of directional shifts in probabilistic maturation reaction norms (PMRNs) that are consistent with the expectation that high fishing mortality favours the genotype for early maturation. Most PMRNs describe the probability of becoming mature solely as a function of age and length. Because they do not fully account for several physiological aspects of maturation (including growth effects on maturation, critical time windows for maturation decisions and developmental thresholds for maturation), it is possible that the observed shifts in PMRNs that are currently being attributed to changes in genotype actually reflect environmental effects on maturation. In this study, a comparative approach was used to interrogate the historical database for Northeast Arctic cod *Gadus morhua* in relation to 2 contrasting but not mutually exclusive hypotheses: (1) that there is a significant effect of food availability on the probability of being mature, using condition as a proxy for food availability; and (2) that there has been a long-term shift in the PMRN for maturation in a direction that is consistent with a diagnosis of fisheries-induced evolution. The results show that the maturation trends in Northeast Arctic cod could be variously interpreted as showing a strong environmental effect, no genetic effect, or a strong genetic effect. If the scope of the analysis had been restricted to testing a single hypothesis related to either an environmental effect or a genetic effect, then the study could very easily have made a Type I error of inference. A more integrated view of maturation, incorporating key aspects of the physiological processes that culminate in maturation, is therefore required to avoid incorrect inferences about the underlying causes of earlier maturation.

KEY WORDS: Cod · *Gadus morhua* · Maturation · Probabilistic maturation reaction norms · Condition · Plasticity · Simulation · Fisheries-induced evolution

INTRODUCTION

In human populations, the age at puberty in females has declined steadily in several different countries (Parent et al. 2003). An association between increased adiposity and early maturation has been consistently shown (Kaplowitz et al. 2001). Other environmental factors that could have contributed to earlier maturation include increased exposure to pollutants (Denham et al. 2005), improved prenatal nutrition (Adair 2001) and higher levels of stress (Bellis et al. 2006). Because these factors are not mutually exclusive, it is proving difficult to unambiguously identify the causes (Herman-Giddens et al. 2004, Anderson & Must 2005). Identifying the factors that contribute to earlier maturation in exploited fish stocks is proving equally challenging. As is the case for human populations, environmental factors are important in determining when individual fish mature. For example, large reductions in stock size can increase per capita food availability, thereby enhancing individual growth and condition and leading to earlier maturation (Reznick 1993, Bigler et al. 1996, O'Brien 1999, Engelhard & Heino 2004). An additional complication for exploited fish stocks is that there is a plausible genetic explanation for trends towards earlier maturation. Because fishing mortality

*Email: c.t.marshall@abdn.ac.uk

is size-selective, the individuals who mature and reproduce prior to capture will make a disproportionately large contribution to the gene frequencies of subsequent generations (Law 2000). This phenomenon is considered to be an example of fisheries-induced evolution and it is of concern to managers because of the potential for substantial reductions in yield that could be difficult to reverse.

In recent years, fisheries-induced evolution has been reported in maturation rates of several commercial fish stocks (Grift et al. 2003, Barot et al. 2004a, 2005, Olsen et al. 2004, 2005). These diagnoses of fisheries-induced evolution have been made on the basis of directional shifts in probabilistic maturation reaction norms (PMRNs) that are consistent with a selective mortality favouring a genotype for earlier maturation. For a cohort (or group of cohorts), the PMRN describes the maturation probability at each age and length combination. If there is no change over time in cohort-specific PMRNs, then it is generally inferred that there has been no evolution; if the PMRN midpoints decline in time then fisheries-induced evolution is diagnosed. Obviously, this does not constitute prima facie evidence of a change in the gene frequencies for maturation rates. Rather, a diagnosis of evolution is made on the basis of changes in phenotype that are suggestive of genetic adaptation in maturation schedules. Many studies of fisheries-induced evolution acknowledge the inherently circumstantial nature of the evidence provided by directional shifts in the PMRN with the caveat that the evidence is consistent with but not unequivocal proof of evolution. It is also argued that, given the deleterious consequences that can potentially result from irreversible genetic change, strict application of the precautionary approach should draw the worst-case conclusion, i.e. that fisheries-induced evolution has occurred.

In any analysis, failing to consider the full range of plausible explanations for trends in maturation increases the likelihood that conclusions are confounded by factors that were not included. For example, studies that are primarily interested in genetic effects on maturation typically assume that environmental effects on maturation are accounted for by the PMRN and that any resulting directional shifts in the PMRN across cohorts reflect an evolutionary response. However, it is increasingly clear that many of the current generation of PMRN do not fully account for growth (Morita & Fukuwaka 2006) or condition effects, and have thus misrepresented environmental effects on maturation. It is possible that studies that have concluded that directional shifts in the PMRN are consistent with fisheries-induced evolution have made a Type I error of inference by rejecting the null hypothesis 'that there is no genetic effect' when it is correct. Similarly, studies that investigated time trends in maturation exclusively from the perspective of density- or environmentally-driven changes in food availability failed to consider the degree to which directional genetic selection could also be contributing to the observed long-term trends in maturation. If the environmental factor covaried with the intensity of genetic selection (e.g. high fishing mortality resulted in both improved feeding conditions and selection for early maturation), then the confounding of these 2 factors would increase the risk of a Type I error of inference.

Another approach would be to entertain several mutually exclusive explanations simultaneously, in comparable detail and using the same dataset. Such an approach is often used in geology where it is common for a single study to test 2 or more highly contrasting hypotheses (termed 'end-member hypotheses' in the geological literature) using a single, comprehensive suite of empirical observations (e.g. Lizarralde et al. 2004). All of the observations are evaluated as being either consistent or inconsistent with the highly contrasting hypotheses with the expectation that the observations would, on balance, provide stronger support for the more correct hypothesis. The success of the approach depends on having well-resolved databases that can be used to interrogate different facets of the complex phenomenon under scrutiny. Examples of this comparative approach to hypothesis testing are rarer in fisheries science but include McQuinn (1997), who undertook a comprehensive review of the literature pertaining to metapopulation structure of herring to determine whether there was greater support for the hypothesis that metapopulation structure was a plastic response to variable environmental conditions (the 'adopted migrant' hypothesis) compared with the hypothesis that the metapopulation structure of herring had a strong genetic basis that was partly maintained by persistent environmental features (the 'member-vagrant' hypothesis). An important advantage of this comparative approach is that it minimizes the possibility of selecting approaches and evidence that supports a particular hypothesis (Hilborn 2006).

Applying a similar approach, this essay will test 2 contrasting but not mutually exclusive hypotheses for interpreting the long-term trends in maturation that have been observed in the Northeast Arctic cod *Gadus morhua* stock (Jørgensen 1990, Marshall et al. 2006). The first analysis tests whether there is a significant effect of food availability on the probability of being mature, using condition as a proxy for food availability. Positive correlations between condition and the probability of being mature at a given age or length have been found for Icelandic cod (Marteinsdottir & Begg 2002), walleye (Henderson & Morgan 2002), American plaice (Morgan 2004) and sardine (Silva et al. 2006).

The second analysis uses the same database to test whether there have been long-term shifts in the PMRN of Northeast Arctic cod that could be interpreted as being consistent with a diagnosis of fisheries-induced evolution such as has been made for other cod stocks (Barot et al. 2004a, Olsen et al. 2004, 2005). The purpose of presenting both analyses, albeit briefly, is to illustrate both the limitations of having a narrow focus on complex phenomena such as maturation and the advantages of investigating maturation from a more integrated, physiological perspective.

VALIDITY OF THE COMPARISON

A direct comparison of the 2 analyses of maturity trends would be problematic if the maturity data underlying them differed. This is not the case for the 2 analyses presented here. The first analysis uses estimates of the probability of being mature at a given age (m_a) that are estimated annually by the International Council for the Exploration of the Sea Arctic Fisheries Working Group (ICES AFWG). The second analysis uses the same m_a values (aggregated by cohort rather than by year) in combination with the observed age-length keys (Marshall et al. 2004) for individual cohorts. The simulation-based approach (B. J. McAdam & C. T. Marshall unpubl. data) to estimating PMRNs was then applied using both the m_a and age/length keys for the cohort. The underlying maturity-at-age (m_a) data are the same in both analyses (obtained from the ICES AFWG), making for a like-with-like comparison. The most important distinction between the 2 analyses is in their principal dimensions: the first analysis is year-specific because environmental effects on maturation will be more strongly manifested on that dimension, whereas the second analysis is cohort-specific because genetic effects on maturation are only relevant for that dimension.

NORTHEAST ARCTIC COD

The Northeast Arctic cod stock has been fished by both Russia and Norway; consequently, both countries have well-resolved time series for many key biological variables. Since the end of WWII there has been a large increase in fishing mortality (Fig. 1a), which has resulted in the size composition of the spawning stock being shifted towards smaller sizes (Fig. 1b). Over the same time period the mean length at which 50% of the spawning stock are mature has decreased by approximately 40 cm (Fig. 1c); however, this trend could partly be an artefact of there being fewer large fish. A trend towards mature fish being heavier for their length is also evident (Fig. 1d). The direction of this trend suggests that changing abundance and a shift in size composition towards smaller fish has increased the per capita food consumption, resulting in higher condition. Higher condition is likely to result in earlier maturation (Henderson & Morgan 2002, Marteinsdottir & Begg 2002, Morgan 2004). Thus, a cursory analysis of the historical trends in the exploitation history and stock dynamics of Northeast Arctic cod suggests that there could be both genetic and plastic effects on maturation.

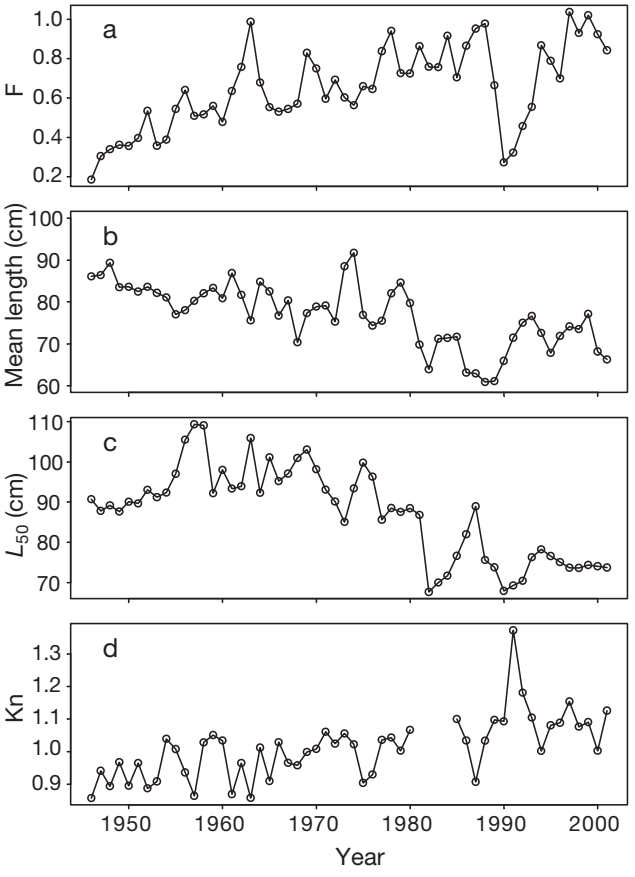

Fig. 1. *Gadus morhua*. Time trends in key population indicators for Northeast Arctic cod. (a) Average fishing mortality (F) of Ages 5 to 10 yr (from ICES Advisory Committee on Fishery Management 2002). (b) Mean length of the spawning stock (estimated from abundance-at-length data described by Marshall et al. 2006). (c) Length at 50% maturity (L_{50}) as estimated from length-based maturity data described by Marshall et al. (2006). (d) Relative condition index (Kn) estimated for the mean length at 50% maturity shown in (c). Kn was estimated as predicted weight at mean length at 50% maturity that year (estimated using the year-specific weight-length relationships in Table 3 of Marshall et al. 2004) divided by the weight at that length predicted using Eq. 10 in Marshall et al. (2006). Thus, Kn expresses weight at length in a given year as a proportion of the long-term (1946–2001) mean weight at that length. Estimates of Kn for 1980–1984 are missing owing to data quality problems described by Marshall et al. (2004)

Evidence of environmental effects on maturation of Northeast Arctic cod

Time series for both the m_a and weight-at-age (w_a) are updated annually by the ICES AFWG because they are used to estimate spawning stock biomass. It should be noted that Norwegian observations for the period 1980 to 1984 (inclusive) are unreliable and thus preclude accurate estimation of the m_a and w_a values for those years (Marshall et al. 2004). Bivariate plots of w_a versus m_a (both time series were obtained from ICES ACFM 2002) suggests that for the pre-1980 time period, there is a significant ($p < 0.05$) positive correlation between m_a and w_a for Ages 7 to 10 yr (Fig. 2). This correlation implies that in years when cod are heavy for their age, they are more likely to be mature. Interestingly, there is a distinct discontinuity between 2 time periods in the nature of the relationship. Observations pre-1980 show lower proportions to be mature and a positive relationship between m_a and w_a, whereas observations post-1984 show fish to have higher m_a but no overall relationship between m_a and w_a. The timing of the discontinuity is synchronous across these age classes; in other words, it affects several cohorts simultaneously. This suggests that the underlying cause of the discontinuity is non-genetic.

A difficulty with using w_a as a proxy for condition is that it combines length-at-age (l_a) with weight-at-length (w_l) into a single value (w_a). Consequently, a short, heavy fish could have the same value of w_a as a long, starved fish. To develop a more sensitive index of condition, age-based estimates of weight and maturity were converted to length-based equivalents, as is described by Marshall et al. (2006). Values of w_l were assumed here to be proxies for condition, which is a reasonable assumption given that the results that are reported here using w_l as a proxy for condition are consistent with the positive relationship between a more refined index of condition (liver weight for a given length). The lengths that were used here for estimating proportion mature-at-length (m_l) and w_l were 72.5, 82.5, 92.5 and 102.5 cm, which correspond to the midpoints of four 5 cm length classes (70–74.9 cm to 100–104.9 cm) that are representative of the length range over which the majority of Northeast Arctic cod are becoming mature. For the post-1984 period there is a significant ($p < 0.05$) positive correlation between m_l and w_l for all 4 of the lengths (Fig. 3). This indicates that in years when cod were heavier for their length, a higher proportion were mature. This result is consistent with the interpretation that the probability of being mature de-

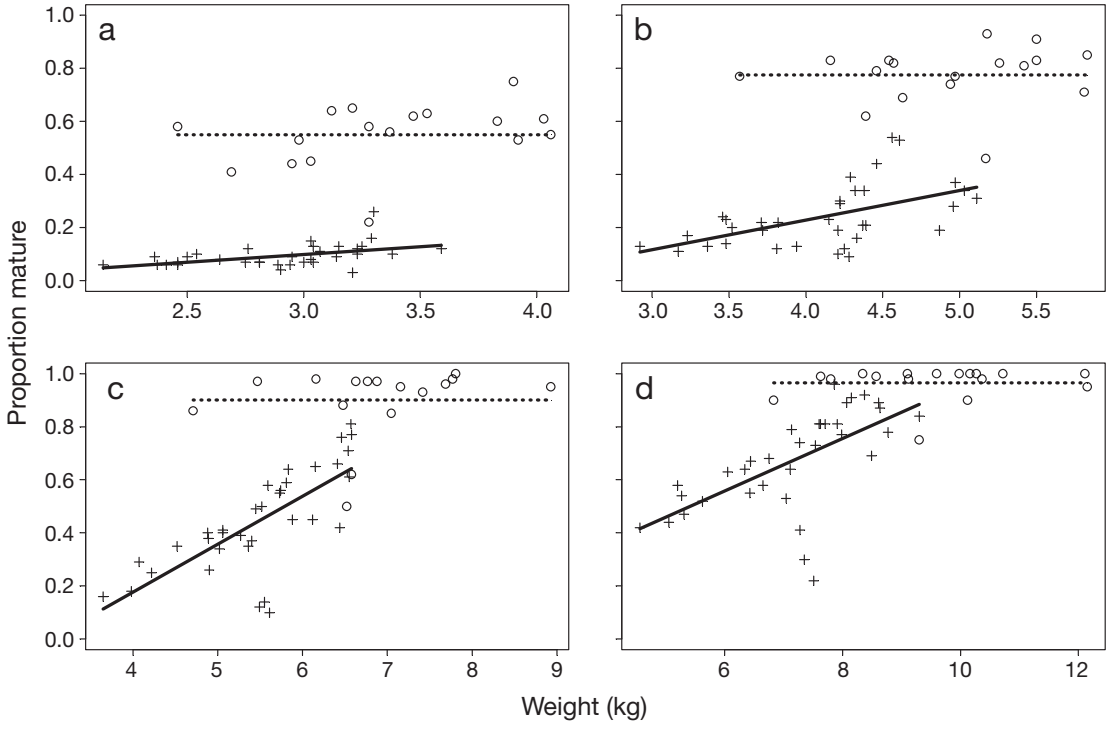

Fig. 2. *Gadus morhua*. Relationship between weight-at-age (w_a) and proportion mature-at-age (m_a) for different ages. Data are from ICES Advisory Committee on Fishery Management (2002). Circles denote data from 1984 to 2001 (inclusive); crosses denote data from 1946 to 1979 (inclusive). Solid line indicates significant ($p < 0.05$) linear relationship; dashed line indicates non-significant ($p > 0.05$) linear relationship. (a) Age 7, (b) Age 8, (c) Age 9, (d) Age 10 yr

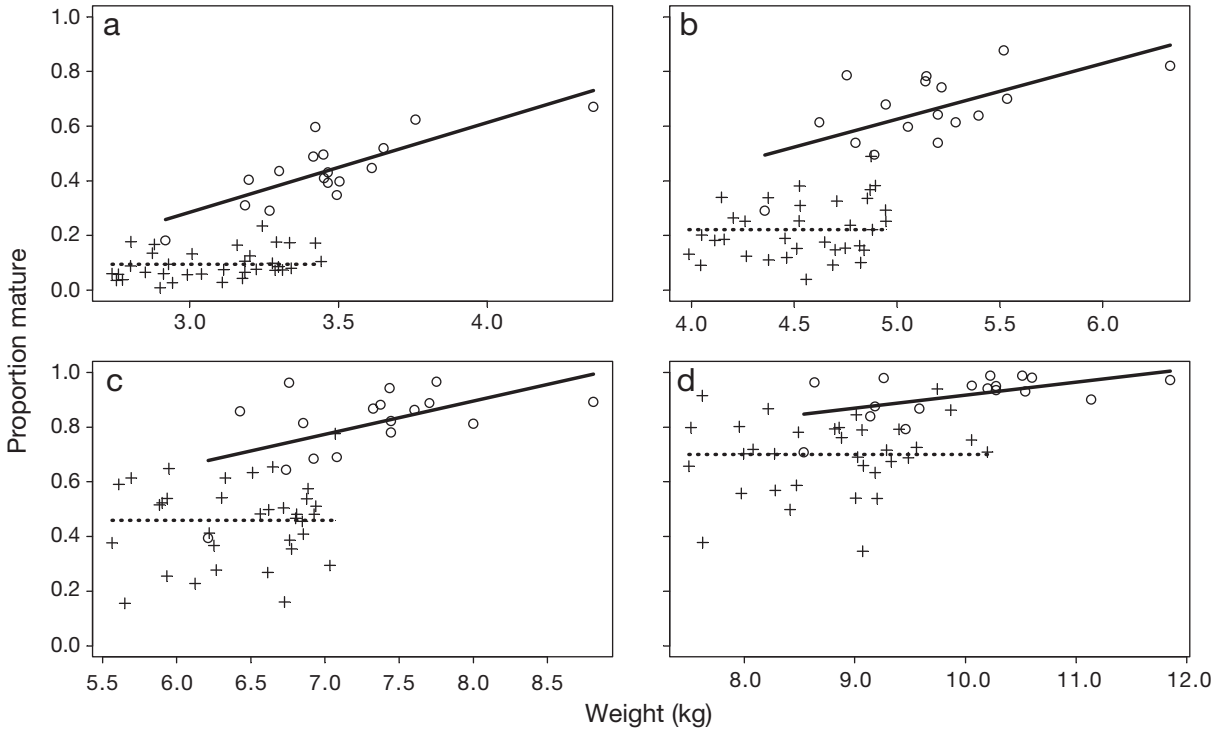

Fig. 3. *Gadus morhua*. Relationship between weight at length (w_l) and proportion mature-at-length (m_l) for different lengths. Data were converted from age-based values in Fig. 2 using the age/length keys described by Marshall et al. (2004). Lengths used for estimating both m_l and w_l were the midpoints of four 5 cm length classes. (a) 70–74.9 cm, (b) 80–84.9 cm, (c) 90–94.9 cm, (d) 100–104.9 cm. These length classes roughly approximate the age classes used in Fig. 2 (Ages 7 to 10 yr). Circles denote data from 1984 to 2001 (inclusive); crosses denote data from 1946 to 1979 (inclusive). Solid line indicates significant ($p < 0.05$) linear relationship; dashed line indicates non-significant ($p > 0.05$) linear relationship

pends on the amount of stored reserves. Therefore, factors such as population density and the environmental conditions that contribute to feeding success are impacting maturation rates in Northeast Arctic cod in this time period.

The discontinuity between the 2 time periods (1946 to 1979 and 1985 to 2001) that was evident in Fig. 2 is also evident in Fig. 3. Cod in the pre-1980 time period are generally leaner for their length compared with the post-1984 time period, which suggests that feeding conditions were poorer in the early time period, perhaps owing to decreased prey abundance and/or higher densities of cod resulting in increased competition. The positive relationship between m_l and w_l is evident only for the post-1984 period, possibly because cod span a larger gradient in w_l in this time period. The mechanisms responsible for generating a discontinuity in the maturation dynamics are beyond the scope of this study. However, other aspects of stock dynamics shifted abruptly around 1980, including the relationship between spawning stock biomass and recruitment (Marshall et al. 2006).

Evidence of fisheries-induced evolution in Northeast Arctic cod

The second hypothesis being tested here is that the phenotypic changes in maturation that were observed for Northeast Arctic cod (Fig. 1c) were the result of fisheries-induced evolution. Because Russia and Norway sample slow- and fast-growing portions of the stock, respectively, any analysis of changes in life history traits that are linked to growth (e.g. maturation) must use data from both countries. However, it is not possible to fit PMRN using individual-level data because historical data for individuals are not accessible for both Russia and Norway. Consequently, B. J. McAdam & C. T. Marshall (unpubl. data) have developed a simulation-based method that uses the age-length keys that are available for each year (Marshall et al. 2004) and the values of m_a to fit PMRNs for individual cohorts of Northeast Arctic cod stock over a long time period (1946 to 1989 cohorts). These 2 types of data (Fig. 4a) are readily available for many stocks, making the simulation-based method used here applicable to a wide range of stocks. The simulation-

based method of fitting a PMRN from a combination of age-length keys and m_a data was validated by comparing the PMRN fit using this method to published PMRNs estimated for North Sea plaice by Grift et al. (2003) using the method of Grift et al. (2003) and Barot et al. (2004b). On the basis of this validation exercise, the simulation-based PMRNs were judged to provide a description of long-term trends that was consistent with the approach of Barot et al. (2004b).

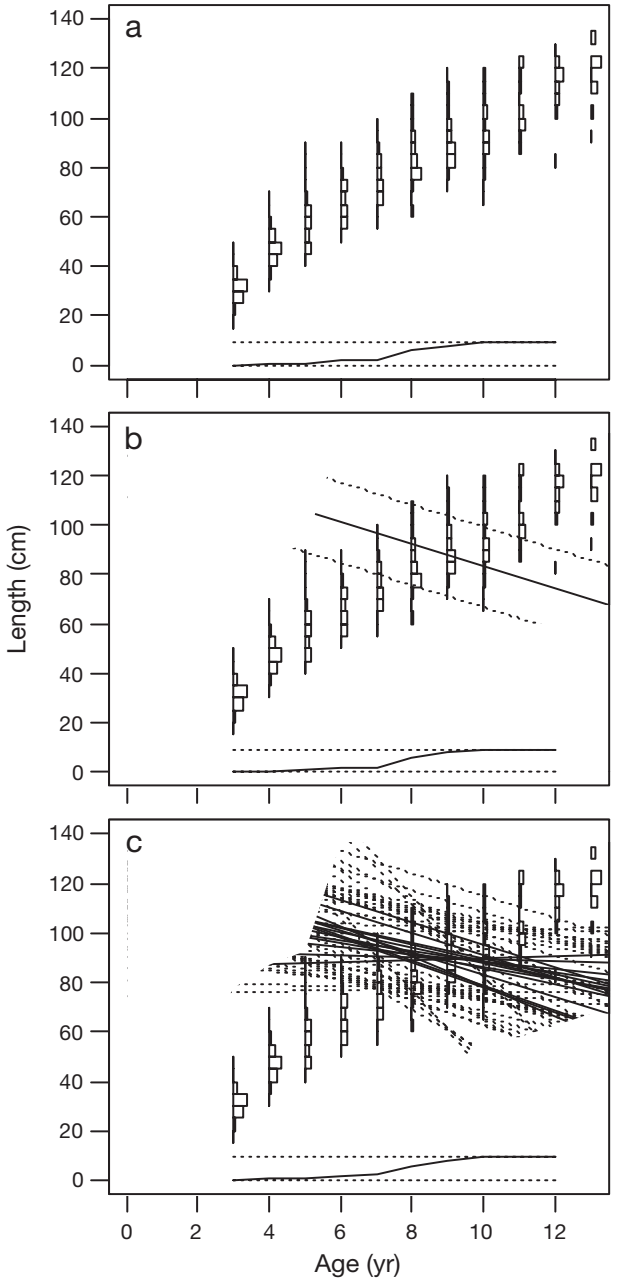

Fig. 4. *Gadus morhua*. Data for a single cohort (1980) and the fitted probabilistic maturation reaction norms (PMRNs). (a) Proportion of cod at each size class for each age shown as histograms. Cohort-specific values of the fraction mature-at-age (m_a) are shown beneath. These 2 data sources were used to fit the simulation-based PMRN shown in the subsequent 2 panels. (b) As above and with contour lines for the single best-fit PMRN. Solid line indicates 50% probability of maturation; dotted lines indicate 25% and 75% probabilities. (c) As above and with the subset of PMRNs that all have a 95% posterior probability

The PMRNs presented here for Northeast Arctic cod have the advantage of being fit using data representing the whole stock (see Marshall et al. [2004] for a description of how Russian and Norwegian age-length keys are combined; see ICES Advisory Committee on Fishery Management [2006] for a description of how Russian and Norwegian maturity data are combined to give m_a). Unlike other formulations for PMRNs (e.g. Barot et al. 2004b), the contours are constrained to be straight and parallel, as illustrated for the 1980 cohort in Fig. 4b. The linear PMRN models have 3 parameters: S_0, the intercept of the 50% probability of maturation contour with the length axis (i.e. the length that a hypothetical Age 0 yr fish would have to be to be mature); S', the slope of the contours; and W, the width between contours (i.e. the length difference between a fish with 25% probability of maturation and one with 50% probability). In the model fitting, a candidate PMRN is tested by repeatedly simulating the maturity status of fish and recording the fraction of simulations that match the actual observations of m_a for all ages in the cohort. The fraction of simulations that match observations is the probability of obtaining the observations, given that the PMRN model is correct: p(*observations*|*model*). After assessing a subset of possible models, Bayes' law can be used to calculate the relative probability of each model being correct, given the observations that were made, i.e. p(*model*|*observations*). Of the suite of possible PMRNs, one is identified as the most probable (Fig. 4b) on the basis of having the highest value of p(*model*|*observations*) relative to the other candidates. A subset of PMRNs (Fig. 4c) can be identified as having a 95% probability of explaining the observations in Fig. 4a.

Emulating the graphical approach used in Fig. 3 of Olsen et al. (2004) in their diagnosis of fisheries-induced evolution in Northern cod, the length for 50% maturation probability (L_{p50}) at Age 6 yr was plotted for the 1946 to 1989 cohorts (Fig. 5). Similar trends are observed in other age classes; however, Age 6 yr is presented here because it represents an age class for which the transition to maturity could be expected for a variable and occasionally large fraction of the stock (cf. Fig. 2). Over the full time period there has been a large decrease in L_{p50} (Fig. 5a), indicating that Age 6 yr cod are likely becoming mature at smaller lengths. The temporal trend in L_{p50} could be interpreted as being consistent with fisheries-induced evolution. Indeed, there are few qualitative differences between Fig. 5a and Fig. 3 of Olsen et al. (2004).

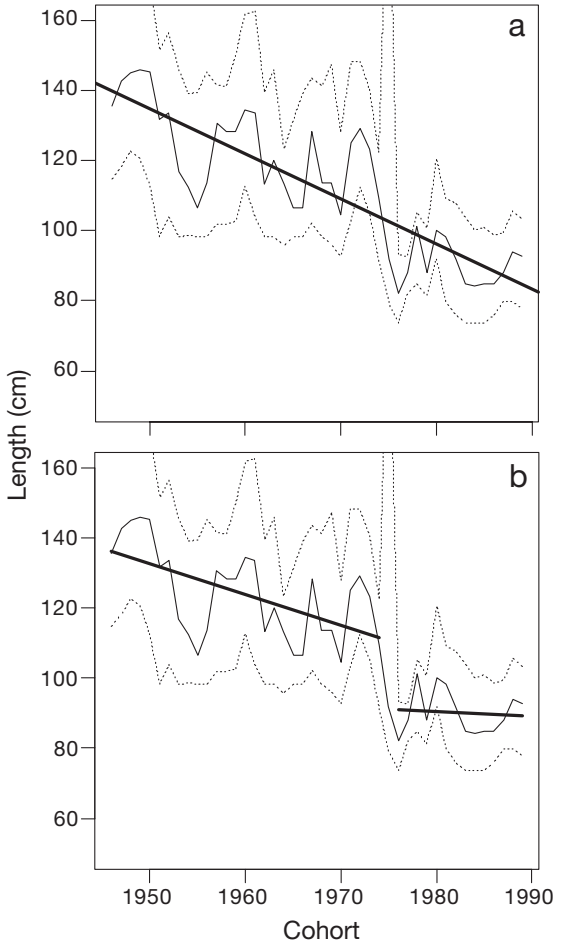

Fig. 5. *Gadus morhua*. Trend in the probabilistic maturation reaction norms (PMRNs) over time (1946 to 1989 cohorts shown). Solid line indicates length for 50% maturation probability (L_{p50}) at Age 6 yr for each cohort; dotted lines indicate 95% confidence interval for this value. (a) Bold line indicates linear trend in L_{p50} over time for the whole time series. (b) Time series for L_{p50} is divided into 2 parts: up to and including 1974, and 1976 and onwards. Bold line indicates linear trend in L_{p50} over the 2 time periods

Having undertaken the first analysis, there are several aspects of the PMRN analysis that are inconsistent with the tentative diagnosis of fisheries-induced evolution. Firstly, the rapidity of the changes in L_{p50} is striking. Specifically, there is a sudden shift to lower values in the 1975 and 1976 cohorts. This discontinuity is evident in a plot of S_0 versus the corresponding S' for all of the fitted PMRN for the 1973 to 1979 cohorts. The parameter space (i.e. the cluster of points that represent values of S_0 and S' of the fitted PMRN for a single cohort) for the 1973 and 1974 cohorts is non-overlapping with the parameter space for S_0 and S' for subsequent cohorts, indicating a sharp discontinuity in coefficients of the PMRNs between the 1974 and 1975 cohorts. Given that cod begin to mature around Age 5 and 6 yr, this abrupt shift corresponds to the discontinuity noted in the year-specific analysis (Fig. 2) around 1980. Secondly, the first analysis (Fig. 2) showed that the discontinuity happened simultaneously across age classes, suggesting that the cause is not genetic. Thirdly, the first analysis indicated that cod were more likely to be mature in years when they were heavy for their length (Fig. 3). Because condition of Northeast Arctic cod is dependent on the abundance of Barents Sea capelin (Yaragina & Marshall 2000), this is consistent with an environmental effect on maturation. Fig. 1d indicates that the condition of cod has increased over the full time period, and the post-1984 time period in particular is characterized by better condition. The effect of condition is not adequately represented in PMRNs that consider maturation as a function of only age and length. Length is an integrated measure of growth history but it is not a sensitive index of condition, either at the onset of maturation or in the critical periods in the run-up to maturation when the fish is 'deciding' whether to initiate maturation. Thus, by using length-based PMRNs to diagnose fisheries-induced evolution in Northeast Arctic cod, condition effects on maturation are being confounded with genetic effects.

Without the perspective provided by the first analysis, it would be possible to interpret the downward trend in L_{p50} (Fig. 5a) as being consistent with, though not direct proof of, fisheries-induced evolution. The null hypothesis of no directional shift in phenotype attributable to a genetic effect could have been rejected, with appropriate caveats, in favour of the alternative hypothesis (directional shift in phenotype is due to evolution). However, the additional perspective provided by the first analysis suggests that the long-term trend in maturation is distinctly discontinuous (Fig. 5b). This discontinuity contributes to the perception of a downward trend in L_{p50} (Fig. 5a). There is a downward trend in L_{p50} for the early time period, driven largely by high values in the post-WWII years, but no overall trend in L_{p50} for the more recent time period (Fig. 5b). The cause of the discontinuity is unknown but it is most likely non-genetic given that it was observed to be synchronous across age-classes.

This dual-pronged analysis has shown that part of the phenotypic variability in maturation that could be inferred by PMRNs to result from genetic change could instead be a consequence of the confounding influence of proximate cues, such as feeding and condition. The PMRN approach has recently been extended to 3 dimensions to account for variation in condition by including this as an additional dimension in the PMRN for plaice (Grift et al. 2007). However, it is also important to consider that the condition effect on maturation

is likely to occur well in advance of maturation itself (Morita & Fukuwaka 2006, Skjaeraasen et al. 2006). If the PMRNs are fit using observations for age, length and condition data that were measured at the point of maturation, then the true nature of the age, length and(or) condition effect on maturation could be obscured.

DEVELOPING MORE INTEGRATED PERSPECTIVES ON MATURATION

The comparative approach used here, which interrogated the same database in relation to 2 contrasting but not entirely mutually exclusive hypotheses, indicated that trends in maturation could variously be interpreted as showing a genetic effect (PMRNs show downward trend across cohorts; Fig. 5a), no genetic effect (PMRNs characterized by a pronounced discontinuity of non-genetic origin; Fig. 5b), or an environmental effect (positive relationship between condition and probability of being mature; Fig. 3). If the scope of the analysis had been restricted to testing for either a genetic or an environmental effect, then a Type I error of inference could very easily have been made. It is dangerous to interpret the temporal trends in PMRNs for the early cohorts (e.g. Fig. 5b) as evidence of genetic change when the model does not properly account for environmental effects on condition (Fig. 3).

Our results illustrate that disentangling phenotypic plasticity in maturation remains a challenge for fisheries science. A recent study into the potential for shifts in reaction norms for reproductive investment in plaice (Rijnsdorp et al. 2005, p. 841) similarly concluded that, 'although the weight loss during the spawning period is in agreement with the expected fisheries-induced change in reproductive investment, the alternative explanation of a phenotypic response to an environmental change cannot be excluded and is even more likely'. Such ambiguity is disconcerting for managers, who are increasingly being called upon to adopt Darwinian fisheries management strategies (Conover & Munch 2002). It is somewhat reassuring that the factors responsible for shifts in maturity schedules for humans are proving to be similarly elusive despite intensive research (Herman-Giddens et al. 2004, Anderson & Must 2005). Intriguingly, a recent longitudinal study tracking the body mass index of girls born in 1991 concluded that the most significant factor in predicting early puberty was the body mass index at age 36 months, a time point 'well before the onset of puberty' (Lee et al. 2007). The compatibility between this result for humans and the model of fish maturation proposed for salmonids by Thorpe et al. (1998) is striking. The attainment of sexual maturity must be regarded as the culmination of a sequence of physiological events and the precise nature of those events must be described before the genetic basis of maturation can be established with confidence.

The development of the PMRN approach (Heino et al. 2002) has greatly stimulated investigations into the genetic aspect of maturation dynamics. However, environmental effects on maturation have not as yet been adequately represented in this approach. The next generation of PMRNs should strive towards incorporating knowledge from applied areas of fisheries science. For example, the results of field- (Englehard & Heino 2004, Yoneda & Wright 2004) and laboratory-based (Rowe et al. 1991, Silverstein et al. 1998, Shearer & Swanson 2000) studies on maturation can be used to identify what proximate cues are important and how they should be parameterized. Accurate models of maturation clearly require a detailed knowledge of the environmental and endocrinological cues that trigger physiological responses in fat, blood hormones and gonad development (Okuzawa 2002, Campbell et al. 2006). Given that the decision to mature is made well in advance of maturation itself (Morita & Fukuwaka 2006, Skjaeraasen et al. 2006), PMRNs for maturation should be developed using data measured during the critical period preceding maturation. PMRNs could be made more realistic if maturation was dependent on attaining a critical size or physiological state (Thorpe et al. 1998, Day & Rowe 2002). The development of physiologically based reaction norms has been advocated for describing the developmental times of insects (Davidowitz & Nijhout 2004), and this line of research would be worth following as it could be a useful analogue for developing physiologically based reaction norms for fish maturation. Finally, the literature on trends in human maturation provides much relevant information regarding the heritability of maturation (Towne et al. 2005), gene polymorphisms associated with early maturation (Xita et al. 2005) and the inheritance pattern of maturation (de Vries et al. 2004).

CONCLUSIONS

Quantifying the degree to which selective harvesting changes the life-history traits of exploited species is a central task for ecology. It is both intuitive and logical that genetically based changes in life-history traits should be occurring in response to selectively applied mortality (Law & Grey 1989). Life-history traits in fish have sufficient heritability to evolve, and micro-evolutionary changes occur within a few generations in controlled experiments (Reznick et al. 1990, Conover & Munch 2002). While these arguments are appealing, they should not predispose researchers towards be-

lieving in fisheries-induced evolution to such an extent that the inherent limitations of the PMRN approach are downplayed (sensu Hilborn 2006). A more integrated view of maturation, incorporating key aspects of the physiological processes that culminate in maturation, can be developed by critically evaluating highly contrasting hypotheses using databases having a high degree of biological resolution. This will lead to more accurate inferences about the underlying causes of early maturation in exploited fish stocks.

Acknowledgements. Discussions with R. Law, Y. Lambert, L. O'Brien, J. Thorpe, P. Wright and N. A. Yaragina helped to develop a perspective on maturation in fish. An earlier draft was thoroughly reviewed by H. Browman and R. Law. Funding support was provided by the European Community (Contract No 022717 [SSP8]).

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Editorial responsibility: Howard Browman (Associate Editor-in-Chief), Storebø, Norway

Submitted: November 28, 2006; Accepted: January 17, 2007
Proofs received from author(s): March 12, 2007