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DETECTING FISHERIES-INDUCED LIFE-HISTORY EVOLUTION: AN OVERVIEW OF THE REACTION-NORM APPROACH

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ABSTRACT

Life-history theory unequivocally suggests that fishing acts as a powerful driver of life-history evolution in exploited fish populations. Because life-history traits are closely linked to the dynamics and productivity of fish populations, understanding and documenting the extent to which this expectation is borne out in reality is both scientifically and practically important. The primary empirical challenges are twofold: observing phenotypic change does not imply genetic change as life-history traits are phenotypically plastic, and fishing is but one potential driver of contemporary evolution. Here we focus on the first challenge by describing how to work toward disentangling genetic and plastic effects in the absence of genetic data. In particular, we explain how the consideration of maturation reaction norms helps to disentangle genetic and plastic changes in age and size at maturation. We first outline the logic and limitations of the maturation reaction-norm approach. We then review the most important statistical methods available for estimating maturation reaction norms from empirical data. For each of these methods, we discuss its domain of applicability together with its strengths and weaknesses.

Life-history theory suggests that much of the diversity in life-history characteristics can be attributed to alternative means of coping with different mortality patterns (Roff, 1992; Stearns, 1992). For fish, changes in mortality caused by industrial-scale fishing are drastic, in terms of both magnitude and pattern: mortality imposed by fishing can exceed natural mortality by several hundred percent and typically increases with body size, in stark contrast to mortality from natural predators, which declines with size (Peterson and Wroblewski, 1984; Sogard, 1997). Fishing must therefore have profoundly altered the fitness landscapes on which fish are evolving (Stokes et al., 1993; Law, 2000; Heino and Godø, 2002). The two most commonly voiced concerns resulting from this realization are that fishing may select for slower growth and for earlier maturation. Although the former suggestion remains theoretically poorly understood, many theoretical studies have addressed the evolution of the timing of sexual maturation—concluding that, for most types of fishing, increased fishing pressure favors maturation at younger age and smaller size (Law and Grey, 1989; Heino, 1998; Ernande et al., 2004; Gårdmark and Dieckmann, 2006).

For evolution to take place, selection must affect traits that are heritable. Aquaculture breeding programs show that life-history traits in fish are heritable to a degree similar to those in other taxa (Law, 2000; Friars and Smith, in press). In addition, experiments with fish and crustaceans have confirmed that harvesting can cause significant evolution within just a few generations (Silliman, 1975; Edley and Law, 1988; Conover and Munch, 2002; Reznick and Ghalambor, 2005). Fishing is therefore clearly causing life-history evolution in exploited stocks. Just how fast such evolution typically proceeds remains open to debate.

Establishing empirical evidence for or against fisheries-induced evolution in the wild is a challenging task. Already Nelson and Soulé (1987) have noted that changes in life-history traits do not suffice as evidence for genetic changes, because the envi-

ronment also influences life-history traits through phenotypic plasticity. Moreover, when life-history traits are estimated at the population level, they can be affected by changes in the demographic composition of a population. For example, the tendency of commercially exploited fish stocks to exhibit progressively earlier maturation over time suggests fisheries-induced evolution, but alternative explanations can often be entertained (Beacham, 1987; Nelson and Soulé, 1987; Reznick, 1993; Trippel, 1995). First, a mere demographic change toward dominance by younger fish causes a decrease in the average age at maturation, in analogy with Baranov's (1918) fishing-up effect. More importantly, however, the maturation process is very plastic, so earlier maturation could just reflect nongenetic responses to changes in the environment. In particular, earlier maturation is expected as a result of accelerated growth, which may occur as a compensatory response to relaxed resource competition when a stock is fished down. These mechanisms are therefore likely to have contributed to the documented trends in maturation age, but do they amount to sufficient explanations?

Building upon earlier work by Stearns (1983), Stearns and Crandall (1984), Stearns and Koella (1986), and Reznick (1993), the introduction of probabilistic maturation reaction norms (PMRNs; Heino et al., 2002b) has provided new momentum to the quest for detecting evolutionary maturation changes in the wild. Dieckmann and Heino (2007) offer a detailed overview of the history, strengths, and limitations of PMRNs. Here we focus on the practical aspects of reaction-norm analysis of fisheries-induced evolution. We set the stage with a brief overview of the general conditions for demonstrating fisheries-induced evolution, and then continue with an introduction to reaction norms in general. The main part of this overview is devoted to maturation reaction norms: a description of maturation reaction norms in general, an appraisal of the logic underlying the use of PMRNs in assessing fisheries-induced evolution, and a review of the various methods available for estimating PMRNs from empirical data. We conclude with a brief overview of the empirical studies that used PMRNs to suggest the occurrence of fisheries-induced evolution.

What Does Detecting Fisheries-Induced Evolution Require?

By definition, evolution implies a change over time, so the most obvious way of studying evolution in action is to follow populations over time. The other possibility is to use replication over space: if one compares contemporary populations with common ancestry, any thus observed genetic differences among them must have evolved. Hendry and Kinnison (1999) refer to these two alternative approaches as allochronic and synchronic design, respectively.

Conclusively proving that fisheries-induced evolution has taken place requires addressing two logically independent questions (Dieckmann and Heino, 2007). The first is about the nature of the change: is the change that is observed truly evolutionary, or does it result "only" from phenotypic plasticicity? A strict proof of evolutionary change can be achieved through molecular genetic analyses. The main limitation is that, at present, the genetic basis of phenotypic traits in fish is not yet sufficiently known. This situation will change in the future, although in many cases historic tissue samples suitable for allochronic analysis may simply not exist. Common-garden experiments are another option. The main drawback is that instances in which suitable populations with recent common ancestry are available for such experimental testing are relatively rare. In most situations, one must therefore rely on weaker ap-

proaches. One important option is to quantify and isolate the effects of confounding environmental effects through regression methods (Rijnsdorp, 1993a,b; Swain et al., 2007). This approach presumes that relevant environmental variables have been identified and recorded and, moreover, that the resultant data display sufficient contrast to allow robust isolation of the evolutionary and environmental components of phenotypic change. Another option is to focus on traits that are less affected—or "contaminated"—by confounding environmental influences. This focus can, at least in principle, be achieved by use of reaction norms as phenotypic traits. Why and how this use is possible will be explained in the next section.

The second question concerns the causes of the observed change: is fishing really among the drivers? Strictly speaking, answering this question would require experimentation. In contrast, studies based on fisheries data—without any controls or true replication—would seem to be particularly ill-positioned to demonstrate causal relationships, and yet the credibility of fisheries-induced selection as a driver of observed changes can be increased (Dieckmann and Heino, 2007). First, alternative hypotheses can be evaluated independently, with the best available knowledge about factors affecting the trait in question and about relevant changes in the environment. Second, although replication in the strict sense is not feasible, numerous fish stocks have been subjected to the same "treatment" of increased mortality. Third, one can construct dynamic models to examine which selective forces would explain the observed changes or, sometimes, the absence of change.

In summary, although reaching a strict proof of fisheries-induced evolution is virtually impossible, one can still hope that a specific interpretation of the observed change clearly emerges as the most credible or parsimonious candidate. Naturally, documenting contemporary evolution will remain exciting even if fishing is not its main driver, and even a better understanding of plastic responses can produce practically and scientifically important insights.

Reaction Norms and Fisheries-Induced Evolution

Reaction norms describe how a single genotype, or group of genotypes, gives rise to different phenotypes in different environments. To the extent that reaction norms are genetically determined traits, their changes are evolutionary. Environmental variability is the prerequisite for observing reaction norms, rather than a mere nuisance that adds noise to observations. If these "ideal" reaction norms, with all their environmental dependencies, could be observed, the problem of detecting evolution with phenotypic data would be solved.

In reality, however, two major challenges arise. First, reaction norms are not directly observable at the individual level—they are not part of the expressed, or "visible," phenotype. A single individual can only be exposed to one particular set of environmental conditions over its lifetime, so it cannot sample the whole reaction norm. Reaction norms are therefore typically measured at the population level. The second challenge is that, in practice, measured reaction norms cannot be interpreted a priori as being purely genetically determined. Because a change in a reaction norm can always be caused by changes in those aspects of the environment that are not considered, the utility of reaction norms for detecting evolution depends on the degree to which the key environmental effects have indeed been included as explanatory variables.

Measuring reaction norms of wild populations is challenging. One would need to observe, simultaneously, the phenotypes of individuals together with the environmental conditions they have been exposed to. As the latter may include both present and past conditions, measuring reaction norms that include dependencies on all the salient environmental dimensions will often be impossible. Reaction norms are therefore not a panacea when phenotypic data are used for documenting evolution, with one major exception. Reaction norms for age and size at life-history transitions, such as maturation, are free from some key problems that generally complicate reaction-norm estimation in the wild. These reaction norms are the focus of the rest of this article.

Reaction Norms for Age and Size at Maturation

The term "reaction norm for age and size at maturation" was introduced by Stearns and Koella (1986). A similar construct was considered in earlier studies (Stearns, 1983; Stearns and Crandall, 1984), but the reaction-norm terminology was not used there. Below, we review this classic maturation-reaction-norm concept before explaining its modern extensions. For a detailed description of historical developments, see Dieckmann and Heino (2007).

Classic Maturation Reaction Norms.—Traditionally, a reaction norm for age and size at maturation ("maturation reaction norm" for short) is defined as the curve that describes the combinations of age and size at which maturation occurs (Stearns and Koella, 1986; Roff, 1992; Stearns, 1992): whenever an individual's growth trajectory hits the reaction norm, maturation occurs with certainty (Fig. 1). Maturation events of individuals following different growth trajectories will then highlight a pattern of covariation in age and size at maturation, thus making the reaction norm visible. Therefore, the maturation reaction norms of genetically polymorphic populations in the wild are only observable as population-level characteristics.

Reaction norms for age and size at maturation are usually presented in two-dimensional diagrams featuring age and size on the diagram's two coordinate axes (Fig. 1). Therefore, in contrast to typical reaction-norm diagrams, environmental conditions in such plots do not vary along one of the coordinate axes. Instead, environmental variations are represented indirectly, influencing the slopes of growth trajectories, as well as the probabilities of survival. In a good growth environment, individuals will grow fast and typically hit the maturation reaction norm earlier than individuals in a poor growth environment (Fig. 1). Notice that "size" in reaction norms for age and size at maturation usually refers to body length but could just as well be based on another measure of size, such as body weight.

A point to recognize is that, strictly speaking, the reaction-norm terminology is suitable when variations in either growth or survival are mainly of environmental origin. Even though growth is very plastic in fish, heritable variation is also known to be present (see, e.g., Conover and Schultz, 1995). As discussed in detail by Dieckmann and Heino (2007), this is more of a semantic issue than a challenge to the practical utility of maturation reaction norms. For the sake of convenience, and respecting a 20-yr-old tradition, we adhere to the established use of the term maturation reaction norm.

The definition of maturation reaction norms so as to capture the effects on maturation of environmental variation in conditions for growth and survival is intri-

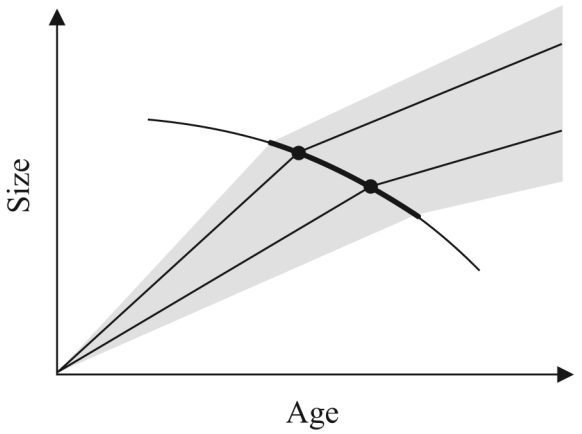

Figure 1. Illustration of a "classic" deterministic reaction norm for age and size at maturation, as introduced by Stearns and Koella (1986). The grey region indicates the distribution of growth trajectories in a population. Maturation occurs when an individual's growth trajectory hits the reaction norm. Limited variability in growth rates allows observation of only a corresponding part (thick curve) of the whole reaction norm (thin curve). Two growth trajectories are shown: the upper one represents an individual experiencing good growth conditions, which matures at an earlier age and larger size (upper circle), whereas the lower trajectory and circle correspond to an individual experiencing poor growth conditions.

cately linked to their utility in detecting evolution in the wild. First, information on environmental variation in growth and survival comes for free, as a by-product of measuring the age and size of individuals. Second, these measurements capture the ambient environmental conditions experienced by single individuals during their life before measurement. Accordingly, this information is much richer and more specific than a mere characterization of the average environmental conditions experienced by a whole population over a longer interval of observation.

At the same time, age and size are obviously only incomplete and proximate descriptors of those factors that ultimately cause maturation (Dieckmann and Heino, 2007; Marshall and Browman, 2007), so these two variables alone cannot be expected to capture all environmental variation affecting an organism's tendency to mature. For example, physiological factors such as body condition will often be relevant for maturation processes (Bernardo, 1993; Thorpe et al., 1998). The effect of these additional factors is that describing maturation tendency in terms of age and size alone will result in unexplained variation, manifest as scatter in the combinations of age and size at maturation around the mode of the corresponding maturation reaction norm. Population-level genetic variance in the reaction norms of individuals will further contribute to this scatter. Both of these probabilistic elements cannot be accounted for by the classic maturation reaction norms, which, for a given growth trajectory, imply maturation at exactly one particular combination of age and size (Heino et al., 2002b). Surmounting this shortcoming requires that the traditional deterministic concept be extended.

Rate-Based Maturation Reaction Norms.—When maturation occurs as a continuous process over time—without periodicity externally imposed, for example, by annual reproductive seasons—an organism's propensity to mature is most naturally described in terms of instantaneous maturation rates (Heino et al., 2002b). A rate-based reaction norm for age and size at maturation is therefore defined by age- and size-dependent instantaneous maturation rates. Maturation may then occur at

any combination of age and size at which immature individuals encounter positive maturation rates. As an extreme case, the classic deterministic notion described above can be recovered by rate-based reaction norms for age and size at maturation that imply vanishing maturation rates below a given curve in the age-size plane and infinite maturation rates along that curve. In reality, maturation rates will of course never reach infinity and instead will be intermediate at least for some band of age-size combinations, implying that maturation events will be scattered across the age-size plane.

Van Dooren et al. (2005) developed statistical techniques for estimating rate-based maturation reaction norms. Although these methods most naturally lend themselves to continuously observed data, they can also be used when data are collected at arbitrary, discrete time intervals. Until now, the rate-based approach has only been applied to experimental data on springtails (Van Dooren et al., 2005).

Probabilistic Maturation Reaction Norms.—A PMRN (Heino et al., 2002a,b) is defined in terms of age- and size-dependent probabilities that an immature individual matures during a given time interval (Fig. 2). A description of the entire reaction norm therefore involves specifying these probabilities for all relevant combinations of age and size. This notion is particularly useful when maturation events are observed at regular intervals, either representing natural periodicity (e.g., annual seasonality) or resulting from a choice made by the observer. The probabilistic and rate-based approaches are inherently linked: by dividing maturation probabilities for given time intervals by the length of those intervals, while making these lengths infinitely small, one obtains instantaneous maturation rates, whereas by integrating instantaneous maturation rates over the considered time intervals, one obtains the corresponding maturation probabilities. The two approaches can thus be understood as alternative representations of the same stochastic age- and size-dependent maturation process; which one is most suitable is decided by features of the natural system and by the mode of data acquisition.

The PMRN must not be confused with another maturation-related probabilistic construct often encountered in fisheries science, referred to as a maturity ogive. A maturity ogive describes the *probability of being mature*, usually as a function of age or size and occasionally of both. For such ogives, no distinction is made between in-

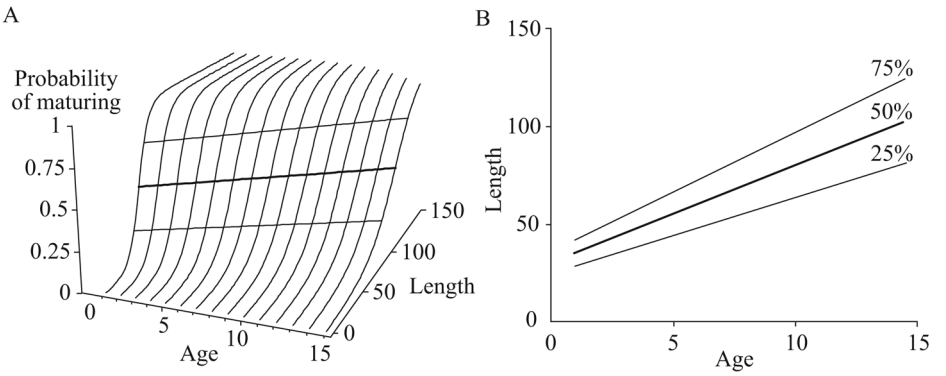

Figure 2. Illustration of a probabilistic reaction norm for age and size at maturation (PMNR) describing the probability that immature individuals will reach maturity within a certain time interval as a function of their age and size (A). When the resultant isoprobability contours are projected onto the age-size plane, a two-dimensional representation of the reaction norm is obtained (B). Three isoprobability contours are plotted in each panel.

dividuals that have matured during a given time interval and those that had matured already earlier on. Maturity ogives are affected not only by maturation but also by growth (through the probability of reaching a certain combination of age and size) and survival (through the probability of still being alive at a certain combination of age and size). In contrast, a PMRN describes the *probability of maturing* as a function of age or size. This seemingly subtle, but very fundamental, distinction is similar to the distinction between size and growth; the former refers to a state and the latter to the process that governs changes in that state. Whereas maturity ogives describe the *state of maturity*, PMRNs describe the *process of maturation*.

A PMRN can be visualized by a collection of curves following isoprobability contours. Such contours connect combinations of age and size that result in equal maturation probability. For example, the midpoint curve of a PMRN passes through all sizes for which maturation occurs with a probability of 50%. For illustration of the width of a PMRN, showing, for each age, the size interval within which the probability of maturation rises, say, from 25% to 75% (Fig. 2) is also useful. The area between such symmetrically chosen isoprobability contours is sometimes called the maturation envelope. The width of the envelope is related to the degree to which uncontrolled factors cause apparently stochastic variation in maturation tendency. Furthermore, for PMRNs estimated at the population level, genetic variability in the reaction norms of individuals manifests itself as an increased width of the population-level maturation envelope.

Note that maturation events are generally not centered along the midpoint curve. Although equating the midpoint curve with the curve passing through the mean lengths at maturation across all ages may be tempting, doing so would be incorrect (Heino et al., 2002b). In fact, the latter curve does not reveal much about the shape of the underlying probabilistic reaction norm, because it is often strongly biased toward the average growth trajectory. In many populations, most individuals will mature at ages and sizes that fall below the midpoint curve, especially when maturation envelopes are wide, midpoint curves are shallow, and maturation events are spread out over a broad range of ages. In such cases, the midpoint curve provides a suboptimal illustration of the probabilistic reaction norm as a whole, and isoprobability contours for lower probabilities should then be chosen for improved visualization.

To date, applications of maturation reaction norms have mostly considered body length as a measure of size. An alternative choice is body weight, which a priori would seem superior to body length because weight at age is a better indicator of body condition than length at age (Bernardo, 1993), but weight is also a more labile individual variable than length and may therefore fluctuate more widely over time. In particular, total body weight depends on stomach contents and on gonad development throughout the spawning cycle. Weight without viscera therefore offers a more stable measure. So far, weight-based PMRNs have been estimated for plaice (*Pleuronectes platessa* Linnaeus, 1758; Grift et al., 2007) and sole [*Solea solea* (Linnaeus, 1758); Mollet et al., 2007]. Both studies suggest that age- and weight-based maturation reaction norms display wider maturation envelopes than those produced when size is measured as body length, but both studies used total body weight. Whether other weight measures would result in better descriptions of maturation tendency remains to be seen.

Descriptions of maturation tendency may be improved by inclusion of additional explanatory variables, such as body weight (in addition to body length), condition

index, or liver index. Three-dimensional maturation reaction norms for age, condition, and length or weight have been estimated for cod (*Gadus morhua* Linnaeus, 1758, Baulier et al., 2006), plaice (Grift et al., 2007), and sole (Mollet et al., 2007). The results show, not unexpectedly, how good condition accelerates maturation, but the kind of field data available for these studies do not always make clear whether good condition is the cause or the consequence of maturation. The estimated three-dimensional PMRNs also demonstrate that the trends toward earlier maturation detected with two-dimensional reaction norms based on age and length remain for three-dimensional reaction norms, that is, when additional plastic effects are accounted for.

Detecting Fisheries-Iduced Evolution with Probabilistic Maturation Reaction Norms

Maturation reaction norms help disentangle phenotypic plasticity and genetic effects influencing maturation (Stearns, 1983; Stearns and Crandall, 1984; Stearns and Koella, 1986; Reznick 1990, 1993; Rijnsdorp, 1993a,b), because a major source of plasticity in maturation is variation in environmental conditions for growth and survival. To the extent that a maturation reaction norm is a genetic trait describing these plastic responses in maturation, contemporary differences and temporal changes in reaction norms can be interpreted as evolutionary.

The capacity of maturation reaction norms to help disentangle phenotypic plasticity and genetic change results from the way these reaction norms are constructed, by separation of the description of the maturation process itself from the description of variations in growth and survival. This property is implied by the conditioning that is inherent in the construction: maturation probability is considered conditional on having reached a certain combination of age and size (and whatever additional explanatory variables are included). Complementing the maturation process, the processes of growth and mortality instead determine the probabilities that an individual will attain such combinations of age and size—by growing to them and surviving until they are reached. These latter processes are deliberately omitted from the maturation-reaction-norm description, so that variations in average growth and mortality leave maturation reaction norms unaffected. Looked at in this way, a maturation reaction norm can be used as a filter that removes environmental variability from maturation data.

Figure 3 illustrates the logic of using maturation reaction norms for disentangling genetic and plastic changes in maturation. Variability in growth determines the part of the reaction norm that can be observed, as though a searchlight were being passed along the reaction norm: if growth improves, the reaction norm becomes observable at an earlier age, whereas it may fall out of sight at some later ages. The position of the reaction norm itself is not affected by changes in average growth rates. Instead, genetic change in the reaction norm results in its displacement (typically, fishing is expected to select for displacements in the downward direction). By comparing two maturation reaction norms over the age range in which they overlap, one can assess whether the data support a hypothesis of genetic differentiation.

Figure 3 also illustrates that information on changes in age and size at maturation is not sufficient for disentangling alternative explanations for such change. For example, declining size at maturation must not be mistaken for definitive evidence of

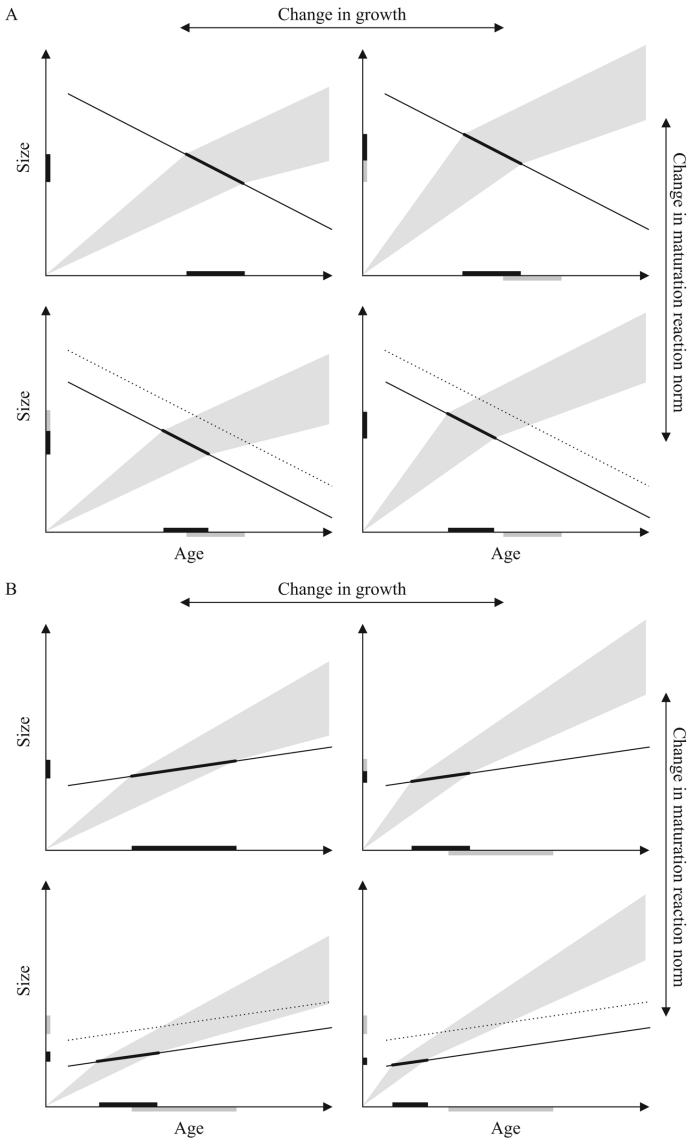

Figure 3. The logic of using maturation reaction norms (PMRN) for disentangling genetic change and growth-related phenotypic plasticity. The maturation reaction norm is assumed to be either negatively sloped (A) or slightly positively sloped (B). The upper left panels in (A) and (B) show the maturation dynamics for a baseline scenario with slow growth and with a maturation reaction norm that on average confers late maturation. The other three panels in (A) and (B) show scenarios for which the average growth rate increases (right columns) and/or the maturation reaction norm is shifted downwards (bottom rows). For clarity of illustration, linear reaction norms, piece-wise linear growth trajectories, deterministic maturation, and nonseasonal growth rates are shown. In each panel, the grey polygon indicates the range of observed growth trajectories; variability in growth lets the population "sample" a certain part (thick curve) of the maturation reaction norm (thin curve). The sampled parts determine the observed ranges of ages and sizes at maturation, as highlighted by the thick lines along the axes. The thick grey lines along the axes show these ranges for the original scenario. Where maturation reaction norms are shifted, their baseline positions are indicated by dotted lines. How variations in mortality affect the observed distribution of ages and sizes at maturation is not illustrated here. A comparison of the various panels underscores that, without knowledge of maturation reaction norms, interpreting changes in age and size at maturation remains very difficult.

evolution, nor do any changes—not even increases—in length at maturation provide definitive evidence against evolution. The reason is that both of these observations can result either from growth-related maturation plasticity or from evolution of the maturation reaction norm. A further dimension of complexity is not even visible in Figure 3, because mortality also affects mean age and size at maturation: other things being equal, increased mortality directly implies lower observed ages and sizes at maturation (fig. 4d of Heino et al., 2002b).

In light of these considerations, we can address a key question: does the observation of changes in maturation reaction norms provide sufficient evidence for underlying genetic changes in maturation tendency? The answer is, unfortunately not. Although changes in reaction norms strengthen the case for evolution, as two major sources of plasticity are accounted for, this support must not be misinterpreted as conclusive proof of evolution, for two main reasons.

First, environmental variations in growth and survival are but two important sources of maturation plasticity. Even though further variables can be included in the estimation of higher-dimensional reaction norms, one can never account for all conceivable sources of maturation plasticity. Consequently, a change in a PMRN could always be caused by changes in environmental variables that are not among those considered. Even growth-related maturation plasticity may sometimes be only imperfectly accounted for by two-dimensional reaction norms for age and size at maturation, because different growth trajectories may lead to the same combination of age and size at the time of maturation. Two-dimensional reaction norms for age and size at maturation predict the resultant maturation probabilities to be equal, independent of the shape of the juvenile growth trajectory and only affected by its endpoint, and yet individuals that have experienced good growth in the recent past may be more likely to mature than those that lately experienced poor growth, even though their current ages and sizes are the same. Such an extra dependence, making the estimation of higher-dimensional reaction norms desirable, was demonstrated by Morita and Fukuwaka (2006) for chum salmon [*Oncorhynchus keta* (Walbaum, 1792)].

Second, estimation of reaction norms is always subject to observation errors. In addition to inevitable errors due to finite samples, two other sources contribute observation error in reaction norm estimation. In particular, sampling may not have been as representative as one would wish, and furthermore, maturation reaction norms often cannot be estimated directly from raw data but only through special estimation techniques (described in the next section) that rely on some simplifying assumptions that may or may not be met accurately.

The primary effect of all these factors is to add noise to the estimation of reaction norms: the uncertainty thus introduced will only partially be reflected by usual statistical measures of uncertainty, such as confidence intervals for reaction norm midpoints. Such noise makes detection of real changes more difficult, but it does not undermine the utility of maturation reaction norms for detecting fisheries-induced evolution. The reason is that only factors that show trends that occur in parallel to those displayed by a maturation reaction norm are serious contenders for explaining the latter other than in terms of evolutionary change. Even if such factors are not identified, however, they can never be ruled out. This problem reflects a general limitation on using phenotypic data for detecting evolution. The best response to this fundamental handicap is the careful and even-handed consideration of feasible factors that could account for changes in maturation reaction norms.

Estimating Probabilistic Maturation Reaction Norms

The type and scope of available data will dictate which methods are suitable for estimating maturation reaction norms in a particular study. Depending on how much information a single individual can provide, we can distinguish among three main cases: (1) The most informative situation occurs when individuals have been followed from the immature stage to maturity, while at the same time their ages and sizes have been recorded. (2) The next-best situation arises when each individual has been observed only once, but when, in each time interval of measurements, three categories of maturation status can be distinguished: immature individuals, individuals that are maturing (or that are newly matured) during the current time interval, and individuals that matured during earlier time intervals (in other words, juveniles, first-time spawners, and repeat spawners). (3) Finally, the least informative but still useful case is that in which individuals are only classified as either immature or mature, without information that distinguishes between newly matured individuals and those that matured earlier.

Each of these cases is addressed by a different set of estimation methods. In addition, a simulation-based method that could be used when individual-level data are unavailable has been proposed by Marshall and McAdam (2007). This method has yet to be described in detail and validated, and we therefore cover it below only briefly. Figure 4 provides an overview of the key questions one must ask when determining the best estimation approach.

The main dichotomy is between measurements based on repeated observations of each individual and those based on only one. Repeated observations require an ability to gather data on the age, size, and maturity status of individual fish nondestructively. This situation is not typical of long-term sampling programs of commercially exploited fish stocks, and no maturation reaction norms have as yet been estimated from such data. Here we therefore cover this case only briefly. In contrast, substantial experience has already been gained with methods that apply when only one observation per individual is available—below, these methods are thus discussed in more detail.

Estimation Methods Based on Repeated Measurements.—The most accurate data on maturation is obtained when measurements on immature individuals can be taken repeatedly over time until they reach maturity. Age and size, as well as other variables that could affect maturation tendency, must be recorded, and the maturation event itself must be detected. A single individual will then contribute several observations on how the maturation tendency depends on age and size (and on any other variables that may have been recorded). When analyzing such data, one can (and should) account for within-individual correlations in observations, because doing so further increases the power of this type of analysis.

Such data can be dealt with by two statistical methods. The first is to use generalized linear mixed models that explain the probability of maturing as a function of the explanatory variables, with individual variability treated as a random effect. In essence, this method is an extension of the direct estimation method (see next subsection) taking within-individual correlations into account (it would also be possible—but not advisable—to use the direct estimation method and thus ignore within-individual correlations). The other option is to use maturation-rate models of the kind introduced by Van Dooren et al. (2005), which bear close resemblance to

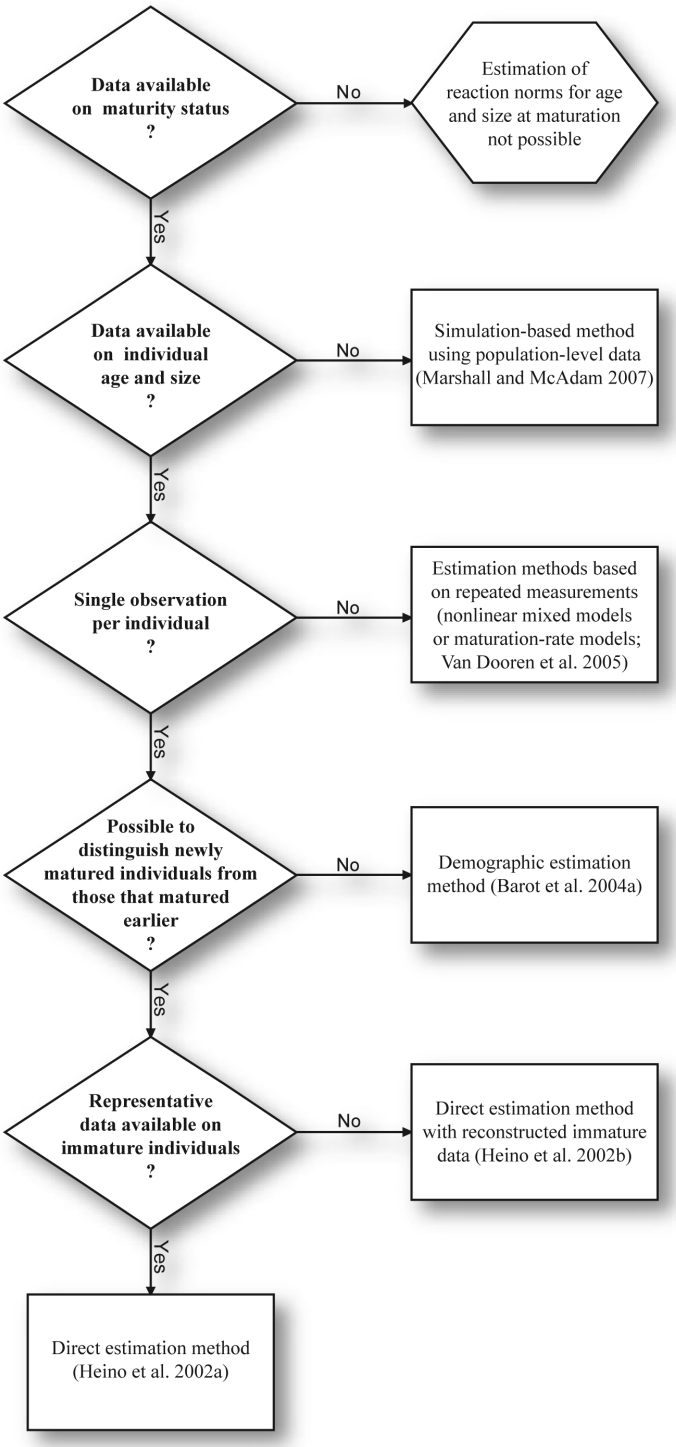

Figure 4. Flow chart for determining whether and how PMRNs can be estimated with the data available for a particular system.

survival analysis. Maturation and death are both irreversible transitions, and modeling of maturation rates can therefore be carried out in close analogy with modeling of hazard rates in survival analysis. The maturation-rate approach is somewhat more complex, but also more flexible, than using generalized linear mixed models.

The main drawback of estimation methods based on repeated measurements is that obtaining suitable data requires the highly reliable recurrent identification of individuals and the possibility of observing their maturation status without significantly hampering, or even destroying, these individuals, usually feasible only in controlled experiments and in mark-recapture studies. So far, these methods have therefore only been applied to laboratory experiments with small invertebrates amenable to automated measurements.

Direct Estimation Method.—The direct estimation method follows directly from the definition of PMRNs (hence the name): numbers of immature and newly mature individuals sharing a certain combination of age and size can be seen as resulting from independent realizations of the probabilistic maturation process. This method is easy to grasp, and no special assumptions are needed.

Within a single age class, estimation of the PMRN amounts to fitting a regression curve to such data to describe, across all relevant sizes, the size-specific proportions of maturing individuals. Logistic regression is commonly applied for this purpose and amounts to fitting a sigmoid curve, known as the logistic function, to the data (Fig. 5). The simplest useful data consist of a representative sample of individuals with known size and maturation status from a single cohort at one point in time. The simplest logistic model to be fitted to the data then is $\text{logit}(p(s)) \sim c_0 + c_1 s$, where p is the probability of maturing; $\text{logit}(p)$ denotes the logit function $\log_e(p/(1-p))$, which is the inverse of the logistic function; s is size; and c_0 and c_1 are the two regression

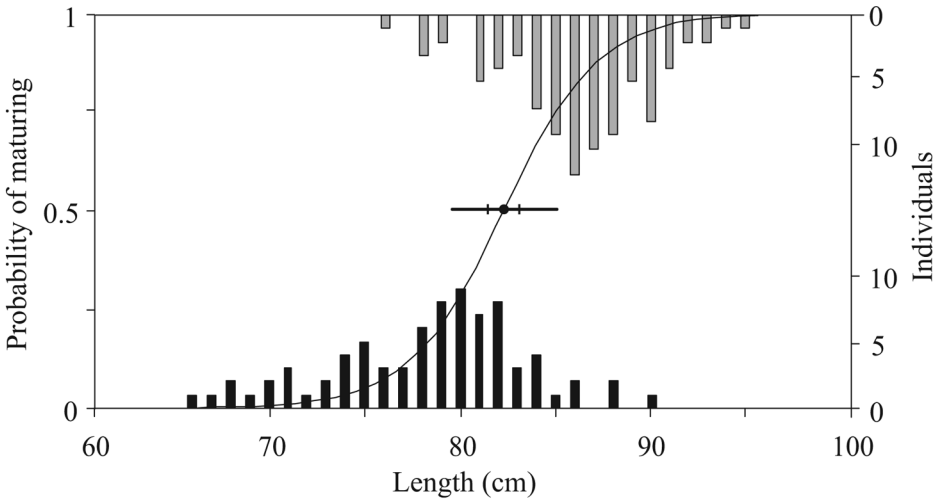

Figure 5. An example of data on maturation status (immature or maturing) and size of individuals drawn from one age group, together with the fitted logistic curve for the probability of maturing. The grey histogram at the top shows the size distribution of maturing (or newly matured) individuals in the sample; the histogram at the bottom shows the size distribution of immature individuals. The size at which the probability of maturing reaches 50% is highlighted by the filled circle at the inflection point of the logistic curve. The 95% confidence interval of this midpoint (illustrating the uncertainty in its estimation) is given by the horizontal error bar around the midpoint, whereas the size interval within which the maturation probability increases from 25% to 75% (illustrating the maturation envelope) is shown as a thick horizontal line.

parameters to be estimated. Logistic regression models can easily accommodate data with more complex structure, e.g., several age classes and cohorts or other explanatory variables (see Heino et al., 2002b, for examples).

Experience shows that the logistic function fits maturation data well, but no biological reason mandates this particular choice. Other choices include the error function and complementary log-log functions (Collett, 2003). The error function is also known as the cumulative normal function and results from the integration of a normal probability distribution. For statisticians, the default choice is the logistic function, for theoretical reasons, but the error function (together with its inverse, the probit function) facilitates linking the estimated parameters of population-level maturation reaction norms to quantitative genetics models that assume genetic variation to follow normal distributions (Heino, Dieckmann, and Ernande, unpubl. data). Nevertheless, all three functions are similar in their sigmoid shape and in practice tend to be indistinguishable. Also, more flexible sigmoid functions have been shown to make no practical difference to the statistical fit (Grift et al., 2007).

Maturation reaction norms based on field data are most naturally defined for cohorts, yet the direct method allows estimating maturation reaction norms even from a single snapshot of data. A word of caution is then warranted: if cohort-specific reaction norms contributing to such a snapshot are not similar, the reaction norm estimated from the snapshot may have a spurious shape (in the sense that this shape may not be representative of the shape of the reaction norm of any of the contributing cohorts); an analogous caveat applies to the estimation of growth trajectories.

A drawback of the direct estimation method is that its data requirements are rather stringent. First, both immature and maturing individuals must be sampled representatively, within those age classes in which maturation occurs. Doing so may be difficult if immature and mature individuals exhibit different behavior or spatial distribution. Sometimes, combining different data sources may help. For example, maturation reaction norms for Northeast Arctic cod, *G. morhua,* were estimated with data originating from two surveys, one covering mostly spawning fish and the other covering mostly juveniles (Heino et al., 2002b). In that study, a correction for different sampling effort in the two surveys was required, using information on the "true" proportions of immature and mature fish taken from maturity ogives. Dunlop et al. (2005) presented another example of how to meet the data requirements of the direct estimation method by combining data from various sources.

A further prerequisite of the direct estimation method that is not easily fulfilled is the need to distinguish between newly matured individuals and those that matured earlier. This distinction is rarely possible for iteroparous species when only routine methods of measurement are used, but for species or populations with long spawning migrations, newly matured individuals and those that matured earlier can sometimes be distinguished on the basis of "spawning checks" in otoliths or scales. Unfortunately, the presence of spawning migrations tends to go hand in hand with the limitation that representative juvenile observations are not available. Similarly, distinguishing newly matured individuals is trivial for semelparous species, but these too tend to be highly migratory, so data are collected mostly on maturing or mature individuals.

Fortunately, when representative juvenile data are missing, it can still sometimes be reconstructed, provided that certain auxiliary data are available (Heino et al., 2002a). Such reconstruction is possible because first-time spawners were juveniles in

the previous season. On the basis of information on how individuals grow, the size distribution of first-time spawners can be projected back in time to yield an estimate of their size distribution as juveniles. Together with information on "true" proportions of juveniles and adults at each age (as provided by age-specific maturity ogives) or on mortality, one can reconstruct juvenile size distributions of a certain cohort by iteratively working backwards from the age at which the last individuals of a cohort matured. Tests with artificial data demonstrated that this back-projection method can usually be expected to yield fairly robust results (Heino et al., 2002a).

A combination of the direct-estimation method with such back-projection has been applied to Northeast Arctic cod (Heino et al., 2002a,c), Norwegian spring spawning herring (*Clupea harengus* Linnaeus, 1758; Engelhard and Heino, 2004), and chum salmon (Morita et al., 2005). In cod, juvenile size distributions were back-projected on the assumption that individual growth trajectories were linear and pointing toward the origin (Jørgensen, 1992). In herring and chum salmon, individual growth trajectories were back-calculated from scale measurements, so the immature size distributions could be constructed without any specific assumptions about the shape of growth trajectories.

Once individual growth patterns have been estimated through otolith-based or scale-based back-calculations, the results may help detect an individual's age at maturation and thus whether it is newly matured or not. Dunlop et al. (2005) applied discriminant analysis to classify maturity status in smallmouth bass (*Micropterus dolomieu* Lacépède, 1802), using data on individuals with maturation histories known from recaptures to establish the discrimination scheme. Even without such extra data, back-calculated individual-level growth trajectories together with population-level maturity ogives may provide sufficient information for estimating an individual's age at maturation. This method has been applied to grayling [*Thymallus thymallus* (Linnaeus, 1758)] and trout (*Salmo trutta* Linnaeus, 1758) in Norway. Preliminarily results are presented by Haugen and Vøllestad (in press), but this approach has not been verified yet through tests with artificial data or through comparison with independent empirical data.

Demographic Estimation Method.—The least informative, but still very useful, data arise when individuals are only classified as either immature or mature, without distinction between newly matured individuals and those that matured earlier. In these cases, information on the maturation process is still not lost, but one must dig deeper for it.

The basis for what we call the demographic estimation method (Barot et al., 2004a,b) is that the processes of recruitment, growth, mortality, and maturation jointly determine a population's demographic composition in terms of maturity status, age, and size. Further, because we are interested in a probability (describing maturation tendency), only relative numbers matter, so recruitment can be ignored. With only three demographic processes left in the game, the maturation process can be inferred from demographic composition, provided sufficient information on the other two processes, growth and mortality, is available.

The appeal of the demographic estimation method is in that it relies on typical fisheries data, including measurements of individual age, size, and maturity status, but without distinguishing first-time spawners from repeat spawners. Such appeal, however, comes at a price. Data on growth and mortality are required because these too affect the composition of a population. When such data are not available, simpli-

fying assumptions must be invoked. At the very least, mean growth increments must be known. The more information can be extracted from the data, the less must be assumed. In all applications of the demographic estimation method to date, mortality has been assumed to depend on age, but independent of maturity status and size within an age group (in this case, the absolute level of mortality is inconsequential and can therefore be ignored). Similarly, growth has been assumed to depend on age, but independent of maturity status and size within an age group. Analyses with artificial data suggest that the demographic estimation method is not very sensitive to modest violations of these assumptions (Barot et al., 2004a). Even if further data on, say, mortality, are available, one must judge whether a potential gain in accuracy is not outweighed by loss in precision when adding noisy data to the analysis.

In practice, the demographic estimation method is implemented through a stepwise procedure (Barot et al., 2004a). First, one estimates how maturity depends on age and size. The estimates are obtained through logistic regression and are referred to as age- and size-specific maturity ogives. The second step is to estimate age-specific growth increments. In the third step, age- and size-specific maturation probabilities are obtained from the following equation, which uses the maturity ogive $o(a,s)$ and the growth increments $\Delta s(a)$ as inputs,

$$p(a,s) = \left[o(a,s) - o(a-1, s-\Delta s(a))\right] / \left[1 - o(a-1, s-\Delta s(a))\right]$$

After estimating the PMRN $p(a,s)$, one can easily derive from it descriptive parameters for reaction-norm midpoints and maturation envelopes. The fourth and final step is to quantify estimation uncertainties through resampling techniques, thus providing confidence intervals for all estimated parameters.

The most significant limitation of the demographic estimation method is that it requires relatively large samples to operate robustly (Barot et al., 2004a) and involves assumptions that may sometimes be difficult to validate (see above). Nevertheless, this approach has become the most frequently used estimation method for PMRNs and has been applied successfully to several stocks of cod (Barot et al., 2004b; Olsen et al., 2004, 2005) and American plaice [*Hippoglossoides platessoides* (Fabricius, 1780); Barot et al., 2005] in the northwest Atlantic, as well as to stocks of plaice (Grift et al., 2003), haddock [*Melanogrammus aeglefinus* (Linnaeus, 1758); Wright, 2005], and sole (Mollet et al., 2007) in the North Sea.

As mentioned above, maturation reaction norms are best defined for cohorts. The conditions under which a single snapshot can yield an estimate of the maturation reaction norm are more restrictive for the demographic estimation method than for the direct estimation method. Because the demographic method is based on comparing a population's compositions at two consecutive time steps, estimations based on a single snapshot involve the assumption that neither growth increments nor maturity ogives have changed.

The demographic method can easily be extended to account for explanatory variables other than age and size (Grift et al., 2007). Variables that have been considered include temperature and body condition; the latter was measured either as Fulton's morphometric condition index or as liver condition index (Baulier et al., 2006; Grift et al., 2007; Mollet et al., 2007).

Estimation Methods Based on Demographic Simulations.—Whenever data are informative enough to allow use of one of the three aforementioned main

estimation methods, one could also resort to an alternative approach based on computationally intensive methods. The idea is to assume a wide array of candidate reaction norms that span what is considered feasible, then for each candidate reaction norm to compare the predicted maturation data to the observed data, and finally to select the reaction norm that results in the best-matching predictions. One must therefore set up a demographic Monte Carlo simulation that generates artificial data with a structure similar to that of the empirical data being analyzed. For example, if data on the age distribution and length distribution of mature individuals are available, the model can easily generate that same kind of data as its output. As with the demographic estimation method described above, realistic predictions require good knowledge of growth and mortality, as inaccuracies in these input data will result in biases in the output.

Until now, this approach has only been applied to Northeast Arctic cod (Marshall and McAdam, 2007). An advantage of the method proposed by Marshall and McAdam (2007) is that it allows estimation of PMRNs when only population-level data—the age-length key and the age-specific maturity ogive—are available, thus extending the range of situations in which PMRN estimation is possible, but because this method relies on less informative data than the methods using individual-level data, results can be expected to be less accurate. Also, this method has not yet been rigorously tested. Results for Northeast Arctic cod show trends in the PMRNs similar to those obtained previously on the basis of individual-level data (Heino et al., 2002c), but the thus estimated PMRNs have a significant negative slope, which does not agree with the positive slope obtained before on the basis of individual-level data.

Work-Arounds for Data-Poor Situations.—Ideally, inferences about evolutionary changes in maturation tendency should be based on a time series of cohort-specific PMRNs, yet, many situations can occur in practice in which data availability is less than ideal. Three common cases are described below, together with possible work-arounds.

First, data may be too scarce and noisy for estimation of maturation reaction norms for each cohort. This situation is actually very common and not necessarily a major obstacle. When the direct and demographic estimation methods are applied, estimations are usually not carried out separately for individual cohorts but for the collection of all cohorts simultaneously, by inclusion of cohort as an explanatory variable in the statistical models employed. Unless the model includes all possible interactions with cohort, the shape of the maturation reaction norm is then not estimated fully flexibly from cohort to cohort. Such simplifications are not to be taken too lightly, because they can exclude the possibility, for example, that the midpoints of maturation reaction norms evolve more slowly at later ages or that their widths evolve. Another possibility for coping with sparse data is to pool cohorts, so that the subsequent analysis has a temporal resolution of, say, 5 or 10 cohorts. If the underlying data are heterogeneous, such pooling may cause artifactual features in the estimates, but as the evolution of maturation reaction norms is usually not exceedingly rapid, modest pooling is presumably quite safe.

Second, reaction norm estimation may not be possible for the full length of a time series, because data are too scarce or age information is incomplete. If the overall shape of the maturation reaction norm can be established reliably for one point in time (possibly on the basis of additional data), the interpretation of the entire time series can still be strengthened under the assumption that the overall shape of the

maturation reaction norm has not radically changed over time. As Figure 3 illustrates, the changes expected in age and size at maturation are quite different for different shapes of the underlying maturation reaction norm. Armed with this information, one can ask which processes could, and which could not, account for the available observations. To support such qualitative reasoning, one could use simple demographic Monte Carlo simulations to check, for example, what the quantitative influence of mortality variations would be.

Third, no age data may be available. The attractive properties of maturation reaction norms are to a large extent due to the use of the combination of age and size at maturation as an indicator of average juvenile growth rate. Because of the importance of growth-related phenotypic plasticity for observed changes in maturation, age and size must be considered as the minimal set of explanatory variables for the estimation of truly useful maturation reaction norms. If age is not known, one could still estimate PMRNs for length at maturation, perhaps in conjunction with some other explanatory variables, but these "ageless" reaction norms are bound to lack the appeal that reaction norms for age and size at maturation possess. Our best recommendation would be to proceed at the level of qualitative analysis, using approaches such as those discussed in the previous paragraph and taking advantage of whatever information is available on growth, maturation, and mortality.

Applications

Probabilistic maturation reaction norms have been estimated for at least 20 populations of 10 species of marine and freshwater fishes (Table 1). Most studies are based on contiguous time series, but a few compare distinct periods of time, and one study compares four populations of recent common ancestry. With few exceptions, all these studies suggest changes in maturation that cannot be accounted for by purely demographic responses in conjunction with growth-related phenotypic plasticity. Thereby, the case for evolutionary change in maturation tendency is considerably strengthened.

Other common observations have also emerged from these PMRN studies. First, PMRNs typically possess a negative slope; Northeast Arctic cod is currently the only exception. Slow-growing individuals therefore have a greater tendency to mature at small sizes than fast-growing ones. Second, where PMRNs have been estimated for both males and females, either male and female reaction norms are similar or male reaction norms describe a higher tendency to mature at smaller sizes. Third, temporal trends in PMRNs documented so far are mostly monotonic—only the cod stocks in Newfoundland–Labrador show signs of reversals after the corresponding fisheries there were closed (Olsen et al., 2004, 2005). Fourth, no simple correspondence can be discerned between the total exploitation pressure and the magnitude of PMRN evolution: some heavily exploited stocks show only insignificant responses (herring), whereas some moderately exploited stocks show significant responses (American plaice). This result suggests that factors other than total exploitation pressure are important for fisheries-induced evolution: depending on their life-history characteristics and exploitation pattern, stocks must be expected to differ in their susceptibility to fisheries-induced evolution.

Table 1. Overview of studies that estimated probabilistic maturation reaction norms (PMRNs) to help interpret maturation trends.

Species	Population or stock	Sex (C = combined)	Time span	Data coverage	PMRN trends suggesting evolutionary change?	Reference
Atlantic cod, *Gadus morhua*	Northeast Arctic	C	1932–2006	Time series	Yes	Heino et al. 2002c, in prep.
	Eastern Baltic	F, M	1991–2005	Time series	Yes	Vainikka et al., unpubl. data
	Georges Bank	F, M	1970–1998	Time series	Yes	Barot et al., 2004b
	Gulf of Maine	F, M	1970–1998	Time series	Yes	Barot et al., 2004b
	Northern (2J3KL)	F, M	(1977–) 1981–2002	Time series	Yes	Olsen et al., 2004, 2005 Baulier et al. 2006
	Southern Grand Bank (3NO)	F, M	1971–2002	Time series	Yes	Olsen et al., 2005
	St. Pierre Bank (3Ps)	F, M	1972–2002	Time series	Yes	Olsen et al., 2005
Haddock, *Melanogrammus aeglefinus*	Georges Bank	F, M	1968–2002	Time series	Yes	O'Brien et al., unpubl. data
	North Sea	F	1977–1999	Two periods	Yes	Wright, 2005
Plaice, *Pleuronectes platessa*	North Sea	F	1957–2001	Time series	Yes	Grift et al., 2003
American plaice, *Hippoglossoides platessoides*	Labrador–NE Newfoundland (2J3K)	F, M	1973–1999	Time series	Yes	Barot et al., 2005
	Grand Bank (3LNO)	F, M	1969–2000	Time series	Yes	Barot et al., 2005
	St. Pierre Bank (3Ps)	F, M	1972–1999	Time series	Yes	Barot et al., 2005
Sole, *Solea solea*	Southern North Sea	F	1958–2000	Time series	Yes	Mollet et al., 2007
Atlantic herring, *Clupea harengus*	Norwegian spring-spawning	C	1935–2000	Time series	Yes, weak	Engelhard and Heino, 2004
	North Sea	F, M	1990–2006	Time series	Yes, weak	Enberg and Heino, 2007
Small yellow croaker, *Pseudosciaena polyactis*	Yellow Sea	C	1985–2001	Two periods	Yes[a]	Heino, Yin, and Dieckmann, in prep.
Chum salmon, *Oncorhynchus keta*	Shari River, Hokkaido, Japan	F, M	1992–1997	Time series	No[b]	Morita et al., 2005
Grayling, *Thymallus thymallus*	Lake Lesjaskogsvatnet, Norway	C	1903–2000	Two periods	Yes	Haugen et al., unpubl. data
	Four lake populations of common origin in Lesja region, Norway	C	1990s	Four populations	Yes	Haugen et al., unpubl. data
Smallmouth bass, *Micropterus dolomieu*	Opeongo Lake, Ontario, Canada	M	1936–2002	Two periods	No	Dunlop et al., 2005

[a]Preliminary results
[b]Empirically established PMRN from data in 1992–1997 was used to interpret changes over a 50-yr period.

Concluding Remarks

Proving the occurrence of fisheries-induced evolution requires showing that what looks like evolutionary change is genetically based and that fishing is one of the drivers of that change. Generally, neither one of these two requirements can be fully satisfied in observational field studies. The virtue of maturation reaction norms is that they can often help address these requirements, enabling researchers to make best use of available data. In particular, maturation reaction norms greatly facilitate the identification of growth-related phenotypic plasticity (Stearns and Koella, 1986; Heino et al., 2002b). Growth-related plasticity has previously been considered the most likely, or at least the most parsimonious, explanation of worldwide trends toward earlier maturation (Trippel, 1995).

The value of maturation reaction norms for detecting fisheries-induced evolution is in large part due to their being based on an environmental indicator that arises, the world over, as a by-product of standard measurements in fisheries surveys: with information on both age and size, the average growth rate of an individual is known. Reaction norms for age and size at maturation can naturally be criticized because age and size are explanatory variables far removed from the actual physiological variables triggering maturation (Marshall and Browman, 2007). Although this concern is valid in principle, a venerable scientific tradition of using age and size for predicting life-history transitions such as maturation and metamorphosis shows that in practice age and size can actually serve as remarkably useful proxies (Wilbur and Collins, 1973; Policansky, 1983; Stearns, 1983; Stearns and Crandall, 1984; Stearns and Koella, 1986; Reznick, 1993; Rose, 2005). One must also ask how useful adamancy about using more ultimate explanatory variable for characterizing the maturation process would be, when sufficiently long time series of the quantities thus required are clearly simply not available for most stocks of interest.

The estimation methods needed for dealing with common types of available data are now all in place (Heino et al., 2002a,b; Barot et al., 2004a; Van Dooren et al., 2005) and have already been widely applied (Table 1; see also Jørgensen et al., 2007). The results suggest that changes in age structure and growth-related plasticity are usually not sufficient to account for observed maturation trends, showing unequivocally that other processes with analogous effects on maturation must be at play in all the populations covered by these studies. As the observed maturation trends are in line with the theoretical expectations resulting from the hypothesis of fisheries-induced evolution (Law and Grey, 1989; Law, 1991; Heino, 1998; Ernande et al., 2004; Gårdmark and Dieckmann, 2006) and because devising alternative explanations that would plausibly apply across species and regions is difficult, we conclude that fisheries-induced evolution currently offers the most probable and parsimonious explanation of the documented widespread trends in the maturation reaction norms of exploited stocks. To avoid misunderstandings, we stress that this conclusion does not imply that all changes in estimated PMRNs should be interpreted as being evolutionary or driven by fishing: even if fisheries-induced evolution explains the "big picture," short-term fluctuations in estimated PMRNs must be expected to remain, caused by plastic effects not accounted for, an imperfect match with simplifying estimation assumptions, variable sampling, and measurement error.

Fisheries-induced evolution is an example of rapid anthropogenic evolution. Changes documented in PMRNs have even been conjectured to be too rapid to be

evolutionary (Law, 2007). Although short-term PMRN fluctuations must be interpreted as noise, the signal in decadal PMRN trends implies evolutionary rates in darwins (Jørgensen et al., 2007) that are well in line with other examples of anthropogenic evolution (Hendry and Kinnison, 1999; Hendry et al., 2008). Evolutionary rates in haldanes, a more refined measure (Hendry and Kinnison, 1999), have so far been estimated only in one study, yielding rates that are higher than most previously published rates (Olsen et al., 2004). This increase might be explained by estimates in haldanes' being normalized relative to a trait's phenotypic standard deviation. Because phenotypic variability in PMRNs is reduced by removal of growth-related plasticity, the resulting estimates in haldanes are elevated relative to other quantitative traits for which phenotypic standard deviation is not reduced accordingly (Olsen et al., 2004).

Life-history theory suggests the occurrence of fisheries-induced evolutionary changes in traits other than those affecting maturation. For all of these traits, disentangling, to the extent possible, genetic and plastic components of change poses a key challenge. In line with decades of research in ecology and genetics, such disentanglement will require the estimation of suitably defined reaction norms of phenotypic plasticity. For traits other than those affecting maturation, doing so will be difficult, largely because obtaining relevant environmental data at the individual level is very difficult. For example, phenotypic plasticity in growth trajectories has not yet been examined from the perspective of reaction norms. An absence of clear-cut global trends in the somatic growth rates of fish (Hilborn, 2008) also suggests that evolutionary trends in growth are either weak or absent or that such trends are masked by plastic and demographic effects. Where difficulties in disentangling evolutionary and environmental components of phenotypic change cannot be resolved, contrasting interpretations of life-history patterns are bound to persist (e.g., Ricker, 1981, 1995; Bigler et al., 1996).

Reaction norms for age and size at maturation capture the main effects of growth-related and survival-related phenotypic plasticity. Reaction-norm analysis has helped overcome two decades of stagnation in understanding maturation trends in exploited fish stocks observed around the globe. Other sources of plasticity that are not accounted for in a particular study can always turn out to be responsible for observed changes in reaction norms, so a careful analysis of such factors is required before one can conclude that changes in a reaction norm are likely to be evolutionary. We suggest that the best way of dealing with such factors, data permitting, is to include them in the reaction norm estimation: although age and size have widely been used as explanatory variables, additional or alternative explanatory variables are readily incorporated (Heino et al., 2002b; Dieckmann and Heino, 2007). Additional factors considered to date include growth history (Morita and Fukuwaka, 2006), body weight (Grift et al., 2007; Mollet et al., 2007), liver condition index (Baulier et al., 2006), and temperature (Mollet et al., 2007). Such extensions not only strengthen the reaction norm analysis of fisheries-induced evolution but also further our general understanding of factors affecting maturation processes in fish.

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