

Fisheries-induced evolution of neutral and selected genetic markers

Lise Marty

Fisheries Unit, French Research Institute for the Sustainable Exploitation of the Sea (Ifremer), Nantes, France

Goal

To develop a multi-trait individual-based eco-genetic model for studying the roles of neutral and adaptive fisheries-induced evolution in harvested fish populations.

Background and motivation

Various studies – based on field observations, experiments, and theoretical models – have highlighted that, on top of its demographic impact on harvested populations, fishing might induce individual life-history traits to evolve (e.g., Jørgensen et al. 2007; Hutchings and Fraser 2008; Kuparinen and Merilä 2007). Because of the very high levels of fishing mortality and its size-selectivity, individuals with slower growth rate, earlier maturation at smaller size, and higher reproductive investment are predicted by life-history theory to be selectively favoured. In line with such predictions, experiments have shown that unexpectedly rapid harvest-induced evolution of life-history traits is possible (e.g., Conover and Munch 2002; Reznick et al. 1996; Reznick et al. 2005). Furthermore, changes in life-history traits, particularly in the age and size at maturation, have been reported in a number of harvested fish stocks. These changes in maturation can have two origins. First, fisheries-induced evolution due to selective forces imposed by fishing may have occurred. Alternatively, such changes may result from phenotypic plasticity alone, if, for example, a fisheries-induced reduction in stock biomass relaxes intraspecific competition, and thereby leads to faster individual growth and hence to earlier maturation.

In an effort to disentangle phenotypically plastic changes in maturation in response to fishing from genetic changes, probabilistic maturation reaction norms (PMRNs) have been introduced to characterize maturation independently of growth and survival (e.g., Barot et al. 2004; Heino et al. 2002; Olsen et al. 2004; Grift et al. 2007). Size-selective fishing mortality can cause PMRNs to shift toward smaller ages and sizes at maturation. Such a shift may be evolutionary, because PMRNs account for phenotypic plasticity resulting from changes in average juvenile growth rates, which are causing a major part of environmental variation in maturation (Ernande et al. 2004). However, even when estimating PMRNs, evidence for fisheries-induced maturation evolution is only available at the phenotypic level, whereas one would ideally want to know whether genes underlying maturation tendency have evolved at the molecular level. Unfortunately, no genes specifically coding for maturation schedules have as yet been identified. Their identification is the goal of ongoing work that, in the longer run, might provide the necessary molecular tools for assessing genetic evolution underlying phenotypic maturation changes.

In parallel to these phenotypic studies of adaptive evolution, others have investigated the influence of fishing pressures on the neutral genetic diversity of specific stocks, using DNA extracted from archived collections of otoliths or scales. These

studies, based on neutral genetic markers (mostly microsatellite loci and/or mitochondrial DNA), have shown a loss of neutral genetic diversity in several harvested populations, including orange roughy (Smith et al. 1991), red drum (Turner et al. 2002), New Zealand snapper (Hauser et al. 2002), and North Sea cod (Hutchinson et al. 2003). While neutral genetic markers are insensitive to selection, studying their variation has many applications, including genetic mapping of a population's geographic structure, stock identification, and phylogeny estimation. Neutral genetic markers are influenced by genetic drift, which is the change in the genetic composition of a population by purely random processes unrelated to selection pressures; these processes are especially important when for populations of small size. Genetic drift also influences genes coding for life-history traits influenced by fisheries-induced evolution. To disentangle these two causes, changes in the genetic composition of adaptive genetic markers have to be assessed against the baseline of changes observed in neutral genetic markers.

In summary, fishing mortality may increase genetic drift and generate selection pressures, both affecting the composition of coding genes in exploited fish stocks, whereas only the former affects the composition of neutral genetic markers. In turn, changes in the neutral genetic markers leave traits unchanged, whereas changes in coding genes modify phenotypes.

Research questions

The aim of this project is to study the effects of commercial fishing on the evolution of PMRNs, somatic growth rates, and reproductive investments, by developing an individual-based eco-genetic model with an explicit description of genetic transmission during sexual reproduction. In this model, individuals will carry neutral genetic markers and genes coding for the considered life-history traits. This will allow studying the interplay between neutral and adaptive evolution in the context of fishing.

Specifically, the following research questions will be addressed:

- What are the effects of different kinds of fishing pressures on neutral genetic diversity? Changes in neutral diversity are caused by increases in genetic drift due to decreases in population size. The extent to which different fishing practices, characterized by their selectivity and intensity, may have different consequences in terms of neutral genetic diversity is currently unknown.
- What are the consequences of changes in neutral genetic diversity for the ecological properties of a stock? Traditional approaches in population genetics interpret changes in neutral genetic diversity as being related to stock characteristics such as population size, reproductive potential and success, spawning stock biomass, or level of recruitment. In particular, decreases in neutral genetic diversity are interpreted as indicators of detrimental changes in these characteristics. However, so far no formal link between neutral genetic diversity and the ecological properties of stocks has been established in a realistic model.
- What are the relative strengths of fisheries-induced genetic drift and adaptive fisheries-induced evolution on the evolution of the composition of coding genes in exploited fish stocks? Changes in this composition may result from either mechanism, which complicates the analysis of empirical genetic data. Additionally, po-

tential decreases in genetic diversity are often presented as potentially hampering the adaptive genetic responses of life-history traits. Ecologically and genetically detailed and sufficiently realistic models are needed to predict the strengths of the two mechanisms of genetic change, as well as to develop and test new statistical methods to deal with empirical data.

- What are the links between changes in the composition of neutral genetic markers and coding genes? Because of the ease and cost-effectiveness of carrying out molecular analyses on neutral markers, it would be highly desirable to develop operational tools for inferring some relevant changes in the composition of coding genes in exploited stocks from observed changes in neutral markers. If this were feasible, neutral molecular markers could be used to establish early-warning signals for detecting significant and relevant fisheries-induced evolutionary changes in the composition of coding genes of exploited stocks.

Methods and work plan

To study the effect of size-selective harvesting on the genetic composition of a stock, we will devise a generic individual-based eco-genetic model with allelic inheritance. Below, the model is described in two parts, focusing in turn on the ecological setting and genetic architecture.

Ecological setting

The ecology of fish stocks will be described based on the following ingredients.

To model somatic growth, we will use a generic biphasic growth model based on energy allocation principles developed by Quince et al. (2008), generalizing a closely related model previously proposed by Lester et al. (2004). Unlike Lester et al.'s model, Quince et al.'s model allows the rate of mass acquisition to scale with any power of somatic mass (instead of being fixed at a power of $2/3$ as in Lester et al.'s model) and the gonado-somatic index (GSI) to vary with an individual's age. These extensions are important, since the scaling exponent of mass acquisition in fish often exceeds $2/3$, and since fish often show an increase in GSI with age. Mass acquisition will be density-dependent and will decrease nonlinearly with population biomass. This means that both somatic growth and fecundity will be affected by density dependence.

The transition from the juvenile to the adult part of the life cycle will be determined by a PMRN. For the sake of simplicity, we will assume a linear PMRN with constant width, described by its intercept, slope, and width.

Fecundity will equal gonad weight divided by the weight of an egg, with gonad weight being derived from the growth model. The number of new individuals recruiting to the population at age 1 will be determined from a Beverton-Holt stock-recruitment function.

Natural mortality will consist of two components: a size-independent mortality due to, for example, diseases and parasites; and a size-dependent mortality nonlinearly decreasing with body size (according to a power function or exponential function), with the decrease primarily being due to the diminished vulnerability of larger fish to predators.

In addition to natural mortality, individuals will be subject to size-dependent fishing mortality. The size-selectivity of fishing will take different shapes, which will allow considering the different selection pressures resulting from different gears: a sigmoid curve will be used to model trawl selectivity, and a normal curve will be used for gillnet selectivity.

We will consider the evolution of three life-history processes: somatic growth, maturation, and reproductive investment, described by a total of five adaptive traits – three traits related to growth and reproductive investment, and two traits related to maturation. The traits describing growth and reproductive investment will be the maximum annual increase in size as determined by energy acquisition, the proportion of acquired energy that is devoted to somatic growth in the first adult year, and the relative annual increase in reproductive investment during adulthood. The parameters related to maturation will be the PMRN intercept and the PMRN slope.

Genetic architecture

We will focus on diploid organisms. The inheritance of adaptive traits will be described either through quantitative characters or through the transmission of alleles according to Mendelian laws. The inheritance of neutral markers will always be described by allelic transmission. Under quantitative inheritance, offspring trait values will vary around mid-parental trait values according to a normal segregation-recombination-mutation kernel. Under allelic inheritance, haploid gametes will be formed during meiosis by independently drawing one of the two homologous alleles at each locus, representing the effect of genetic recombination. Reproduction will occur randomly between pairs of mature individuals, and the fusion of two random gametes will create new offspring.

Under allelic inheritance, each of the five adaptive traits will be controlled by several loci, each of them being polymorphic (i.e., exhibiting more than one allele). The effect of these loci will be additive (neglecting dominance and epistasis), but we will consider two types of distribution of genetic effects across loci: either a uniform distribution of small effects of equal magnitude, thus following the classical assumption of quantitative genetics, or an exponentially decreasing distribution of effects, thus matching empirical results for quantitative trait loci (QTL), where a few major loci have strong effects while many other minor loci have weaker effects. The number of loci and of alleles per locus will be set as model parameters, and we will determine how many loci are needed to achieve stable results. To obtain values of the adaptive genetic traits, allelic values will be summed over all corresponding loci.

Neutral loci will be treated analogously, without affecting the expression of adaptive traits. The number of neutral loci, of alleles per locus, and of their mutation rates will be set in accordance with empirical results.

Work plan

As a first step, we will calibrate the allelic inheritance of adaptive genetic traits by using a uniform distribution of small phenotypic effects across loci, to obtain results similar to those for the quantitative inheritance of adaptive genetic traits.

As a second step, we will study neutral and adaptive evolution under varying fishing selectivity and mortality. We will (i) assess changes in neutral diversity and investigate whether we can find consistent patterns between neutral diversity and the eco-

logical properties of fish stocks, (ii) evaluate the relative contribution of neutral and adaptive fisheries-induced evolutionary changes of life-history traits, and (iii) investigate whether we can identify correlations between changes in a stock's composition of neutral genetic markers and coding genes.

Time permitting, as a third step, we will carry out an analogous investigation using a genetic architecture with exponentially decreasing effects of loci and compare results with the previous one.

Relevance and link to EEP's research plan

This project aims to assess the influence of size-selective fishing on the genetic composition and the ecological properties of commercially exploited stock. It is therefore directly linked to EEP's focus on *Evolutionary Fisheries Management*. In addition, the development of an allelic model with rich ecological detail is a novel contribution to existing applications of eco-genetic models using quantitative inheritance.

Expected output and publications

The results of this work will be included in my PhD thesis and are intended for publication as a co-authored research article in an international scientific journal.

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