

# The cod that got away

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Commercial fishing can reduce the age and size at which fish mature. But it has been unclear whether this reflects changes in genes or in physical responses to the environment. A look at Atlantic cod provides an answer.

Can fisheries be thought of as uncontrolled experiments in evolution? In other words, do they produce genetic change? Scientifically at least, the suggestion shouldn't cause too much bother. Evolution by natural selection involves pressures that kill off individuals with 'unfavourable' inherited traits, while those with more 'favourable' features survive and reproduce. Fisheries would seem to provide such a pressure: most fisheries target the largest and oldest individuals, so fish that are genetically predisposed to mature at larger sizes and older ages are more likely to be caught before they can reproduce. Such selective harvesting should theoretically favour early- and small-maturing genetic types — a consequence reminiscent of other circumstances<sup>1,2</sup> in which humans have unintentionally selected against that which they desire most.

Nonetheless, many fisheries scientists and managers seem reluctant to acknowledge the potential for fishing to elicit genetic change<sup>3</sup>, despite supportive experimental<sup>4</sup> and field<sup>5,6</sup> data. On page 932 of this issue<sup>7</sup>, however, Olsen *et al.* provide compelling evidence of genetic change in one of the most notoriously overfished stocks, northern Atlantic cod (*Gadus morhua*; Fig. 1), arguing that such change preceded the collapse of the stock.

The growing number of over-exploited marine fishes worldwide has created numerous opportunities to examine whether the collapse of fish populations is associated with genetic responses to fishing. Among these populations, few have declined more than the Atlantic cod that range from southern Labrador to the northern half of Newfoundland's Grand Bank. By the early 1990s, the numbers of northern cod had declined by 99.9% relative to their abundance in the early 1960s<sup>8</sup> — a rate of decline almost unmatched among living terrestrial and aquatic species. Concomitant with this decrease in population size were significant reductions in age and size at maturity.

Having been documented repeatedly in exploited populations, it is incontestable that fishing can lead to significant changes in life-history traits, such as age and size at maturity. The question is whether these changes are phenotypic (reflecting non-genetic variation in physical or behavioural characteristics) or genetic. Consider age at maturity, for instance. As the density of a population declines, relaxed competition for food and



Figure 1 Atlantic cod: test case for studying human-induced genetic change.

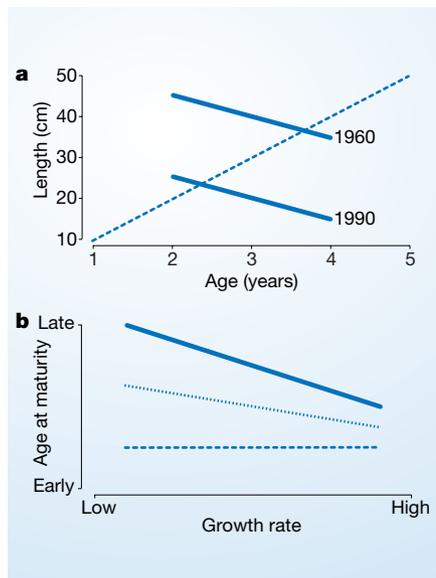
space should lead to individuals growing at an increased rate. Fish generally respond to increased growth by maturing earlier in life. Thus, fishing could lead to earlier maturity solely as a consequence of variable phenotypic responses to growth. Alternatively, however, by selecting against individuals whose genes predispose them to breed at older ages and larger sizes, fishing might genetically alter exploited populations.

To disentangle phenotypic life-history responses to fishing from genetic responses, Olsen *et al.*<sup>7</sup> use a new method<sup>9</sup> that allows them to detect significant changes in age and size at maturity independently of the effects that growth and survival can have on patterns of maturation. The method involves estimating 'probabilistic reaction norms' (Fig. 2a, overleaf), which describe the likelihood that immature individuals of a given age and size will mature during a specific time interval, assuming that that size and age are theoretically enough to allow maturation. It is this assumption — that the individuals are already old enough and big enough to mature — that renders probabilistic reaction norms independent of the effects of growth and survival on maturation. (Although these trajectories are not 'reaction norms' in the strictest sense of the word, as they would then describe how individual genotypes respond to environmental change<sup>10</sup>, Olsen and colleagues' terminology is not unprecedented<sup>11</sup>, and can be justified if

there is a large environmental component to variability in individual growth.)

Olsen *et al.* find that, before the northern cod population collapsed, there was a decline in reaction-norm midpoints (the ages and sizes at which the proportion of mature fish is 50%). In other words, the norms shifted towards younger ages and smaller sizes. The authors assert that this decline is consistent with the hypothesis<sup>12</sup> that heavy fishing pressure selected against genotypes that predispose cod to maturing later and larger. They also observe that these life-history changes are not associated with increased growth rate, which could lead to earlier maturity. Together, these findings suggest that genetic change provides the most parsimonious explanation for why age and size at maturity declined in northern cod.

Of potentially greater interest is the implication that fishing produced a genetic change in the way in which northern cod genotypes respond to environmentally induced variability in growth rate. The question of whether fishing can mould the shapes of reaction norms (an indicator of this type of change) was first raised a decade or so ago<sup>13</sup>. The fitness benefits of delayed maturity — more eggs for females, more mates for males — decline as the risk of death from exploitation increases. So one might predict a 'flattening' of reaction norms with regard to age at maturity, such that individuals would be favoured if they reproduce as early in life



**Figure 2 Fishing out life histories.** a, As Olsen *et al.*<sup>7</sup> describe, the probability of an individual maturing at a specific age or size can be determined from where the negatively sloped probabilistic reaction norms (solid lines), which represent the age and size at which 50% of a population reaches maturity, intersect the positively sloped growth function (dashed line), which relates length to age. In this hypothetical example, an individual growing at the average rate in 1960 would intercept the 50% maturation probability contour — the reaction-norm midpoint — at about 3.6 years and 38 cm. Following intensive fishing, an individual growing at the same rate in 1990 matures at 2.3 years and 22 cm. b, How traditional reaction norms might look in an unfished population (solid line), and as fishing increases to low (dotted line) and high (dashed line) levels. (Growth rate is often used as a proxy for environmental change in the study of reaction norms for organisms that continue to grow after maturity.)

as possible, and expend higher reproductive effort at that age<sup>14</sup>, irrespective of growth rate (Fig. 2b). With sufficiently high mortality, the potential for phenotypes to change might decrease, leading to relatively invariant phenotypic life-history responses to environmental variability — a prediction borne out recently by work on European grayling fish<sup>15</sup>.

In any case, the potential for fishing to generate evolutionary change within harvested populations can no longer be seriously discounted. This may well be the most enduring contribution of Olsen and colleagues' research<sup>7</sup>. If evolutionary change in response to harvesting proves to be the rule rather than the exception for exploited species, we must begin to address questions concerning the magnitude of evolutionary change, the reversibility of such change, and its consequences for sustainable harvesting, population recovery and species persistence. As with

unintentional selection by humans against, for instance, large animals and antibiotic-susceptible pathogens, the long-term repercussions of fishing are almost certainly more complicated than previously believed. ■

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