The cod that got away

Jeffrey A. Hutchings

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[1,2] 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[3], despite supportive experimental[4] and field[5,6] data. On page 932 of this issue[7], 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[8] — 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 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.

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

To disentangle phenotypic life-history responses to fishing from genetic responses, Olsen *et al.*[7] use a new method[9] 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[10], Olsen and colleagues' terminology is not unprecedented[11], 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[12] 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[13]. 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

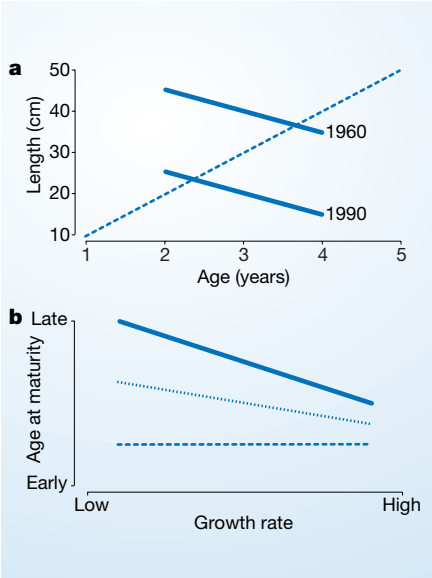

Figure 2 **Fishing out life histories.** a, **As Olsen *et al.*[7] describe, the probability of an individual maturing at a specific age or size can be determined from where the negatively sloped probabilistic reaction norms (solid lines), which represent the age and size at which 50% of a population 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[14], 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[15].

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[7]. 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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1. Coltman, D. W. *et al. Nature* **426,** 655–658 (2003).
2. Stockwell, C. A., Hendry, A. P. & Kinnison, M. T. *Trends Ecol. Evol.* **18,** 94–101 (2003).
3 Stokes, K. & Law, R. *Mar. Ecol. Prog. Ser.* **208,** 307–309 (2000).
4. Conover, D. O. & Munch, S. B. *Science* **297,** 94–96 (2002).
5. Handford, P., Bell, G. & Reimchen, T. *J. Fish. Res. Bd Can.* **34,** 954–961 (1977).
6. Sinclair, A. F., Swain, D. P. & Hanson, J. M. *Can. J. Fish. Aquat. Sci.* **59,** 361–371 (2002).
7. Olsen, E. M. *et al. Nature* **428,** 932–935 (2004).
8. COSEWIC (Committee on the Status of Endangered Wildlife in Canada). *COSEWIC Assessment and Update Status Report on the Atlantic Cod,* Gadus morhua (COSEWIC, Ottawa, 2003). Available at www.sararegistry.gc.ca
9. Heino, M., Dieckmann, U. & Godø, O. R. *Evolution* **56,** 669–678 (2002).
10. Schlichting, C. D. & Pigliucci, M. *Phenotypic Evolution: A Reaction Norm Perspective* (Sinauer, Sunderland, 1998).
11. Stearns, S. C. & Koella, J. C. *Evolution* **40,** 893–913 (1986).
12. Hutchings, J. A. *Can. J. Fish. Aquat. Sci.* **56,** 1612–1623 (1999).
13. Stokes, T. K., McGlade, J. M. & Law, R. (eds) *The Exploitation of Evolving Resources* (Springer, Berlin, 1993).
14. Hutchings, J. A. in *Handbook of Fish Biology and Fisheries* Vol. 1. *Fish Biology* (eds Hart, P. J. B. & Reynolds, J. D.) 149–174 (Blackwell, Oxford, 2002).
15. Haugen, T. O. & Vøllestad, L. A. *J. Evol. Biol.* **13,** 897–905 (2000).

Astrophysics

Jump-start for a neutron star

Duncan Lorimer

Radio emission from one of the neutron stars in the 'double-pulsar' system is strangely enhanced in two sections of its orbit — stimulated, perhaps, by radiation from its companion.

One of the most exciting discoveries in astronomy in recent times was that of the binary system[1] J0737−3039, and its confirmation as a 'double-pulsar' system earlier this year[2]. Pulsars are rapidly spinning neutron stars that form during the supernova explosions of massive stars; although their masses tend to be slightly larger than that of our Sun, their radii are only about 15 km. For the first time, both neutron stars in this binary have been identified as radio pulsars — one that spins about its rotation axis every 22.7 milliseconds (which I shall refer to as 'A') and another ('B') that spins with a period of 2.77 seconds.

This duo promise to surpass even the original Nobel-prizewinning pulsar in a binary system[3] as a testing ground for relativity, but they are also a fantastic laboratory for studying pulsar emission. The intense magnetic fields of pulsars accelerate charged particles around them, causing the emission of beams of radiation that sweep the sky like the rotating beams of a lighthouse. Already there are intriguing observations[2] of the emission from the double-pulsar system — in particular that pulsar B seems to emit most strongly in two separate parts of its orbit. On page 919 of this issue, Jenet and Ransom[4] offer an explanation for this strange effect, in a model that will have important implications for our understanding of this binary system.

The rotation periods of pulsars increase over time, reflecting the loss of rotational kinetic energy of the spinning neutron star as it emits a 'wind' of electromagnetic radiation along its emission beams. The difference in spin properties of the neutron stars in the double-pulsar binary means that their winds carry away energy at significantly different rates: the rate of loss of energy from A is some 3,000 times greater than that from B. This, and the compactness of the pulsars' orbit, implies that the energy carried in the respective winds from A and B is actually balanced inside the emission region of B (ref. 2). As a result, the energetics of A can be expected to dominate the system.

The panels of Fig. 1 show the geometry of the double-pulsar system, as seen from above the orbital plane. The two stars hurtle around their common centre of mass every 2.4 hours, at 0.1% of the speed of light. The two regions of strongest radio emission from pulsar B are indicated in orange in Fig. 1d. Because observers on the Earth are looking at the system nearly edge on, essentially in the same plane as the orbit, it is not surprising that the emission from B is strongest when it is closest to the Earth and A is furthest away. But it is not immediately obvious why there is a clear break in the emission between the two parts of the orbit.

Jenet and Ransom[4] postulate that the emission from B is somehow stimulated — jump-started into action — when the lighthouse beam of A sweeps through B's emission region. The authors make the reasonable assumption that A's beam is a wide, hollow cone[1] whose size and opening angle can be determined directly. It is then a relatively straightforward geometrical exercise to show that pulsar B intercepts A's beam at precisely the points of the orbit where increased emission is observed[2]. From current observations, the various angles in the system are constrained such that they fit two slightly different solutions of Jenet and Ransom's model, both of which produce the effect shown in Fig. 1.