



LETTERS

edited by Jennifer Sills

Conserving Top Predators in Ecosystems



THE NEWS FOCUS STORY “WOLVES AT THE DOOR OF A MORE dangerous world” (V. Morell, 15 February, p. 890) discusses whether the proposed delisting target for the Northern Rockies wolf population will ensure its long-term demographic and genetic viability. We would like to add that viability should not be the sole objective of a species-conservation plan. Another objective—often overlooked—should be to restore and maintain the ecological functionality of the species in its ecosystem.

Recent results from long-term research [including some following the wolf reintroduction in Yellowstone (1)] have shown that top predators can play some unexpected but nevertheless crucial roles in ecosystems. For example, by checking the densities of abundant generalist mesopredators, they can indirectly support species at lower trophic levels (2), and

by preventing irruptions of ungulate populations, they can help restore vegetation (3). Top predators can also buffer some effects of climate change (4), drive senescence of prey (5), and frame river channel dynamics (6).

Conservation plans for predators should take this broader view of ecological roles into account instead of focusing solely on a species' viability by numbers.

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The Role of Fisheries-Induced Evolution

IN THEIR POLICY FORUM (“MANAGING EVOLVING fish stocks,” 23 November 2007, p. 1247), C. Jørgensen *et al.* propose evolutionary impact assessments (EvoIAs) as a general tool for managing evolving resources. The basis for their proposal is that fisheries-induced evolution (FIE) is the most important driver of changes in life-history characteristics of heavily exploited marine fishes. Although Jørgensen *et al.* give the impression that this is well established, the evidence supporting FIE unfortunately remains circumstantial and is

often open to alternative interpretations (1).

To make the case for EvoIAs, Jørgensen *et al.* present a selective set of studies—those concluding that FIE was a likely cause of the observed changes, after considering some environmental effects (see their table S2). In doing this, they excluded results that do not support their case [e.g., (2, 3)]. Furthermore, because FIE is often a matter of interpretation [e.g., (3, 4)] and the authors of the Policy Forum are strong advocates of FIE, the majority of the studies on life-history traits included in table S2 were their own. Their analysis does not represent a consensus opinion developed from critical scrutiny of the studies currently available.

Some component of phenotypic change is undoubtedly genetic and caused by fishing. The challenge remains to determine how important this is relative to other environmental and trophic drivers. A truly precautionary approach to fisheries management must allow for FIE in the longer term. However, EvoIA should be one of several tools used to address the many pressing problems facing fisheries managers.

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IN A RECENT POLICY FORUM, “MANAGING evolving fish stocks” (23 November 2007, p. 1247), C. Jørgensen *et al.* propose that evolutionary impact assessment should be adopted as a tool to manage evolving fish stocks. This is a well-motivated idea in principle, but their reasoning relies entirely on the assumptions that fisheries-induced evolution (FIE) occurs commonly and that it is an undisputable fact. Neither of these assumptions is true. None of the studies of exploited fish populations in their article have provided genetic evidence for the observed phenotypic changes. Because evolution is by definition a change in the genetic constitution of a population, an evolutionary change cannot be postulated without demonstrating a genetic basis for the observed phenotypic shift. In fact, phenotypic changes in mean trait values due to simple environmental inductions are common (1), as are cases where populations are not evolving despite strong directional selection acting on heritable traits (2). Furthermore, several studies have shown that observed phenotypic shifts in exploited fish popula-

tions are fully consistent with simple environmentally induced changes (3, 4).

We are inclined to believe that some of the case studies listed in the Jørgensen *et al.* Policy Forum might indeed turn out to be cases of FIE if genetic data were to become available. However, until that proof is provided, the claims about FIE are nothing but “adaptive storytelling” (5). As pointed out by S. J. Gould and R. C. Lewontin three decades ago (5), unwillingness to consider alternatives to adaptive stories, reliance on plausibility as a criterion for accepting speculative tales, and failure to consider adequately competing themes are

Letters to the Editor

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characteristics of an “adaptationist program” that seems to have become revitalized in the context of fisheries-induced “evolution.”

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Response

WE THANK BROWMAN *ET AL.* AND KUPARINEN and Merilä for their responses to our Policy Forum on fisheries-induced evolution (FIE) (“Managing evolving fish stocks,” 23 November 2007, p. 1247).

We disagree with Browman *et al.*’s interpretation of our article and with their view of the state of research in this field. FIE warrants attention because it is one of the drivers of change in exploited fish populations. We do not claim that “FIE is the most important driver” of changes in fish life histories, and

our argument in no way depends on this being the case. Ecology, evolution, and economics are linked through feedbacks and jointly determine the future of fisheries on time scales relevant for management. FIE is one of several threats to the long-term viability of fish stocks, but the potentially slow reversibility of FIE necessitates extra precaution. The evolutionary impact assessment (EvoIA) framework we proposed recognizes the need to address complementary perspectives simultaneously and is one of several tools required to achieve sustainable fisheries.

We and others (1) think that after environmental factors are accounted for, FIE is the most probable and parsimonious explanation of the remaining phenotypic changes documented for many stocks, species, fisheries, and regions. Our table S2 illustrates the wide taxonomic and geographic occurrence of FIE. Of the studies included, 19 (out of 34) had no involvement from our large group of co-authors. We explicitly listed positive findings, as they are sufficiently numerous that ignoring FIE can no longer be justified.

While we agree with Kuparinen and Merilä that direct genetic evidence for FIE in the wild

is highly desirable and practically nonexistent, we must take issue with their claim that “an evolutionary change cannot be postulated without demonstrating a genetic basis for the observed phenotypic shift.” This claim questions the fundamental assumption that scientists can make inferences about genotypes by studying phenotypes. It is worth remembering that Darwin formulated his theory of evolution with a similar assumption—that traits are heritable—nearly a century before DNA was found to carry hereditary information. Without such assumptions, evolutionary ecology could not operate. Moreover, Kuparinen and Merilä now seem to contradict their recent conclusion that “[t]heory, phenotypic observations and modelling studies all suggest that fisheries are capable of inducing evolutionary changes in life histories in harvested populations” (2).

Like Kuparinen and Merilä, we look forward to the day when direct genetic evidence can decisively determine the extent of FIE. At a practical level, however, traits affected by FIE are likely polygenic and involve unexplored genotype-to-phenotype relations. Even where changing allele frequencies are found, it might take a long time before such changes

are robustly linked to phenotypic effects.

FIE is not a universal explanation for phenotypic changes in harvested fish populations. The importance of FIE relative to other processes that induce phenotypic change will need to be evaluated case by case. One must expect, as Kuparinen and Merilä suggest, that sometimes “observed phenotypic shifts in exploited fish populations are fully consistent with simple environmentally induced changes.” For this reason, researchers of FIE have made considerable efforts to account for environmental effects and phenotypic plasticity before ascribing residual trends to FIE [e.g., (3)]. Of the two studies Kuparinen and Merilä highlighted, one kept open the possibility of FIE (4), while the other even concluded that FIE played a role (5).

Kuparinen and Merilä also refer to a famous argument from the 1970s (6) that did not stand up to scrutiny (7) and had few implications for mainstream evolutionary biology. Furthermore, Kuparinen and Merilä overlook the many, mutually complementary sources of evidence for FIE: expectations from life-history theory and quantitative evolutionary models of exploited fish; statistical analyses

of scientific-survey and fisheries time series that consider phenotypic plasticity; comparative studies of populations experiencing different fishing pressures; demonstrations of FIE in laboratory experiments; and successful engineering of life-history traits in breeding programs. Together, this is much more than “adaptive storytelling.”

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Tips for NIH

THERE ARE TWO USEFUL THINGS THE NIH could do to disseminate science information. First, NIH could ensure that every NIH-funded study had to produce some public report. This would provide an outlet for results that had not been published through conventional channels within a reasonable time. Second, NIH could make

publicly available the raw data of all funded studies, within a reasonable time after the end of funding, and with all appropriate documentation and protection of confidentiality. Instead, the NIH chooses to require collection and redundant dissemination of already-published articles and to provoke copyright battles between scientists and journals, such as those discussed in J. Kaiser's News of the Week story, "Uncle Sam's biomedical archive wants your papers" (18 January, p. 266). Future historians of science may wonder what we were thinking.

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CORRECTIONS AND CLARIFICATIONS

Random Samples: "Genes and humor" (21 March, p. 1595). The item confused the data and conclusions of two studies. The 2008 Twin Research and Human Genetics study of U.K. twins involved close to 2000 twin pairs, not 456 as reported. The 2008 Personality and Individual Differences study covered 456 U.S. twin pairs. In addition, the U.K. study showed substantial heritability for negative as well as positive humor styles, while genetic effects for negative humor in the U.S. sample were not significant.

Table of Contents: (14 March, p. 1449). In the description of the Report "Amyloid fibrils of the HET-s(218–289) prion form a β solenoid with a triangular hydrophobic core" by C. Wasmers *et al.*, "yeast prion" should have been "fungal prion."

News of the Week: "\$300 million in private money for new investigators" by J. Kaiser (14 March, p. 1469). The statement that the new HHMI awards for early career scientists are "twice the size of an NIH R01 grant" could be misinterpreted. The research portion of the HHMI award rises over 6 years from \$150,000 per year to \$300,000 per year, which is roughly equal to the average NIH R01 grant. The remainder is for salary, benefits, and funds paid to the host institution to cover occupancy costs for the scientist's space.

News of the Week: "Physicist wins open Illinois seat" by E. Kintisch (14 March, p. 1470). The article incorrectly described the position of the previous holder of the seat, Representative Dennis Hastert (R-IL). It was Speaker of the House.

News Focus: "Dueling visions for a hungry world" by E. Stokstad (14 March, p. 1474). Emile Frison's institution is named Bioersivity International, not Biodiversity International.

Editors' Choice: "Picking O over N" (29 February, p. 1163). The doi for the referenced paper should have been 10.1021/ja711349r. The complete citation for the paper is T. Ohshima, T. Iwasaki, Y. Maegawa, A. Yoshiyama, K. Mashima, *J. Am. Chem. Soc.* **130**, 2944 (2008).

Random Samples: "Mastodon on the block" (1 February, p. 551). The article stated that all male mastodons had four tusks. In fact, in some adult male mastodons there is no evidence of lower tusks.