Conclusion that fishing-induced evolution is negligible follows from model assumptions

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Phenotypic changes in exploited fish stocks have been reported worldwide (1), but it remains an open question how much is attributable to phenotypic plasticity (likely reversible) versus genetic change (likely slow to reverse), and whether the driver is fishing (manageable) or environmental (mostly difficult to control). Recently, Eikeset et al. (2) attempted to disentangle density dependence from fishing-induced evolution using a simulation model and concluded that trait evolution was of minor importance. This conclusion is inconsistent with highly relevant data sources: First, observed heritability of life history traits is in the range $h^2 = 0.15$–0.68 for Atlantic cod and $h^2 = 0.16$–0.29 for proportion mature at age 2 (3, 4). Heritability of age at maturation has not been quantified in Atlantic cod, but the median was $h^2 = 0.21$ across salmonids (5). We quantified heritability of age at maturation with parent–offspring regression using a similar model (6) and parameters from Eikeset et al. and got $h^2 = 0.02$. We encourage Eikeset et al. to present emergent heritability from their model (their model parameter referred to as "heritability" only affects the initial generation, whereafter emergent heritability is influenced by a range of ecological and genetic assumptions). Second, statistical probabilistic maturation reaction norm (PMRN) analysis, which accounts for density-dependent growth, suggests that PMRN midpoints have declined by 5–10 cm for all abundant ages in this stock over the period 1930–2000 (7), whereas Eikeset et al.'s best-fitting simulation model predicts a decline of only 0.5 cm (their figure S3*A*). This discrepancy requires explanation.

Time series observations are often restricted to phenotypic traits, in which the total variance V_P can be decomposed into environmental variance V_E, additive genetic variance V_A, and other V_ε, that is, $V_P = V_A + V_E + V_\varepsilon$. Because response to selection is proportional to heritability, defined as $h^2 = V_A/V_P$, conclusions about the role of evolution are sensitive to how observed V_P is partitioned between V_E and V_A. Eikeset et al. make two assumptions that inflate the role of V_E relative to V_A, thus biasing their conclusion in favor of slow evolution. First, they find density-dependent effects on growth for the latter part of the time series but apply this relationship to the whole period including early years when biomass was much higher, extrapolating the effect and thus inflating the role of V_E. Second, when modeling maturation, they included most population-level variance in the parameter for PMRN width (part of V_E), rather than as between-individual variance in PMRN intercept or slope, which would have contributed to V_A (mentioned but not resolved in their section S8). Additionally, despite detecting statistically significant contributions from North Atlantic Oscillation, temperature, and capelin biomass on growth, these factors were ignored in their simulation model so that density dependence was the only explicitly modeled mechanism affecting V_E. Statistically, density-dependent growth may then absorb environmental effects due to other drivers, and its role is thus overestimated.

Until the approach and model are better justified, it might be premature for Eikeset et al. to conclude that, with their preferred growth model, "the amount of evolution required for explaining observed maturation trends is small."

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