Increased natural mortality at low abundance can generate an Allee effect in a marine fish

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1. Summary

Negative density-dependent regulation of population dynamics promotes population growth at low abundance and is therefore vital for recovery following depletion. Inversely, any process that reduces the compensatory density-dependence of population growth can negatively affect recovery. Here, we show that increased adult mortality at low abundance can reverse compensatory population dynamics into its opposite—a demographic Allee effect. Northwest Atlantic cod (Gadus morhua) stocks collapsed dramatically in the early 1990s and have since shown little sign of recovery. Many experienced dramatic increases in natural mortality, ostensibly attributable in some populations to increased predation by seals. Our findings show that increased natural mortality of a magnitude observed for overfished cod stocks has been more than sufficient to fundamentally alter the dynamics of density-dependent population regulation. The demographic Allee effect generated by these changes can slow down or even impede the recovery of depleted populations even in the absence of fishing.

2. Introduction

Negative density-dependent population dynamics promotes population growth at low abundance [1]. A reduction in the strength of this compensatory density-dependence can negatively affect per capita population growth, thus inhibiting recovery. Predation, for example, is known to vary with prey abundance, such that a declined prey population can experience elevated rates of natural mortality at low abundances (type-II functional response [2]). It has been postulated that such a shift in predation pressure was experienced by some severely depleted Northwest...
Atlantic cod (*Gadus morhua*) stocks that have since shown few signs of recovery [3–5], particularly in the Southern Gulf of St. Lawrence, where mortality unrelated to fishing has more than tripled during the past two decades [4,6]. This high rate of natural mortality is capable of driving a cod stock to extinction, even in the absence of fishing [7].

While much of the previous research on mortality changes in cod has focused on quantifying mortality increases and identifying their correlates [4–6] as well as projecting future population development [7], here we approach the topic by considering the potential relevance of an Allee effect. Specifically, we investigate whether an increase in adult mortality at low abundance is a mechanism that can reverse compensatory population dynamics into its opposite, a demographic Allee effect. A demographic Allee effect is characterized by a shift from a negative to a positive correlation between *per capita* population growth rate and a metric of population abundance, as the latter declines [8]. In other words, a population that experiences a demographic Allee effect has lowered growth ability at low abundance. Lack of recovery of many overfished stocks [9] has drawn attention to the question of how common Allee effects might be in marine species and to what extent they can impede the recovery [10]. One means by which Allee effects can be manifest is through interspecific interactions [11]. Whether an Allee effect can be generated by one such interaction—predation—is a fundamental question that might illuminate not only the general mechanisms underlying a population’s or species’ ability to recover from depletion but also causes that could potentially account for the lack of recovery of Northwest Atlantic cod.

3. Material and methods

To explore how population dynamics respond to increased predation at low abundance, we used an individual- and process-based modelling approach parametrized for Newfoundland’s northern stock of Atlantic cod (for detailed description of the model and its parametrization, see [12]). The model describes individual life histories through von Bertalanffy (VB) growth curves and known correlations between VB parameters, length at maturity and asymptotic body size. At each time step (year), the model simulates the demographic processes of mortality, growth, maturation and reproduction on an individual basis. Demographic stochasticity is accounted for in each of the processes. The genetic basis of the life histories is modelled through 10 diploid loci and their classical Mendelian inheritance. The model incorporates density-dependence in growth and reproduction. Juvenile production is compensatory such that it increases at low population abundances in accordance with a Beverton–Holt stock-recruitment curve for cod (electronic supplementary material, figure S1) [13].

We partitioned instantaneous rates of adult natural mortality into an overall mortality parameter (*M* = 0.12) experienced by all individuals from age 3 years onwards and a parameter representing the survival cost of reproduction (*SC* = 0.1), experienced by all mature individuals. At abundances below 20% of population carrying capacity (*K*), we increased *M* by 0, 50, 75 or 100% to mimic four scenarios of predation-mortality increase. Cod population dynamics were simulated at annual time steps. The population was first exposed to fishing (*F* = 0.25; using a logistic selectivity curve, based on body size) until it had declined below 5% of *K*, after which fishing ceased. During the subsequent recovery period, *per capita* population growth rates were recorded for each cohort, as well as the population biomass in the year the cohort was born. Simulations were replicated with versions of the model that did and did not allow for the exploited cod population to evolve in response to changes in mortality. For the former, life histories could evolve during the fishing and recovery periods, whereas life histories remained unchanged in the non-evolving simulations.

4. Results

Following the relaxation of fishing, populations began to rebuild towards their equilibrium levels. In the absence of mortality increase at abundances below 20% of *K*, realized population dynamics reflected the underlying compensatory juvenile production, as shown by high *per capita* population growth rates (*r*) at very low abundance (figure 1a,b). However, as mortality at low abundance increased, compensation weakened and population dynamics shifted to become depensatory such that an Allee effect became evident. This effect is shown by the values of *r* plotted against abundance: when looking at low abundances, a mortality increase of 50% yielded a relatively flat pattern (figure 1c,d), whereas an increase of 75% caused the values of *r* to drop as the abundance declined (figure 1e,f). These patterns were similar in both the evolving and non-evolving simulations, although the average values of *r* were often slightly lower in the evolving simulations.
Figure 1. Projected per capita population growth rates ($r$) at varying population abundances. Values of $r$ estimated for individual cohorts are plotted as grey dots against the population biomass (expressed as % of carrying capacity, $K$) in the year the cohort was born. The average growth rates calculated for abundance ranges 0–10% of $K$, ... 90–100% of $K$ are shown with black dots, and the black vertical lines encompass 95% CIs (many of these are not readily discernable because they overlap with the dot). Scenarios for the proportional increase in natural mortality at abundances below 20% of $K$ are indicated on the right of the panels and scenarios for evolution/no evolution are identified at the top of each set of panels; each panel represents the results of 20 replicated simulation runs. Simulations with 100% mortality increase did not recover (figure 2) and, therefore, a similar $r$-abundance plot could not be drawn for this scenario.

Despite lower per capita growth rates owing to increased mortality, populations experiencing a 50% mortality increase rebuilt their biomass at a pace similar to that of populations that did not experience a change in mortality (figure 2). By contrast, when mortality increased by 75%, biomass rebuilding was severely delayed; when mortality increased by 100%, population levels had attained only 10–15% of $K$ after 100 years of rebuilding (figure 2).

5. Discussion

Our results suggest that increased mortality at low abundance can generate a demographic Allee effect that slows the recovery of depleted populations. In addition to the general importance of this finding for threatened species, our work has specific relevance to the rebuilding of over-exploited populations, indicating that recovery can be significantly retarded even in the absence of exploitation. Elevated natural mortalities have been documented in four Northwest Atlantic cod stocks following their collapse in the early 1990s [5]. The mortality-increase scenarios explored here are quite conservative when compared with those estimated for the collapsed stocks. In the Southern Gulf of St. Lawrence, $M$ is estimated to
Figure 2. Temporal changes in population biomass during the fishing and recovery periods. Mortality-increase scenarios are indicated by the key and five randomly chosen replicated simulation runs are shown for each scenario. Simulations illustrated here were conducted with the non-evolutionary model version. The dashed vertical line indicates the beginning of fishing, which was continued until the population had declined to 5% of its carrying capacity, $K$.

have more than tripled during and following a period of population reduction [4]. A similar pattern of association between population size and natural mortality is evident for northern cod, which declined by an estimated 99% between 1962 and 1992 [3]. When compared with a natural mortality of less than 0.25 in the 1970s and 1980s, $M$ is estimated to have doubled in the 1990s; this increase in natural mortality persisted for more than decade, despite massive reductions in fishing mortality [14].

The findings of this study suggest that the mortality increases experienced by some Northwest Atlantic cod were sufficient to erode compensatory density-dependence in population dynamics to the extent that per capita population growth, and thus recovery rate, was severely reduced. This finding is inconsistent with the perception that Allee effects are not commonly present in marine fish and that their recovery ability should generally not be compromised by low abundance [13,15]. While this perception arose because of the challenges in detecting Allee effects (due to lack of statistical power in data and analyses), more recently it has been acknowledged that marine fish populations reduced to very low levels of abundance might indeed experience Allee effects [16]. This study provides an example of the underlying mechanisms through which a demographic Allee effect can arise and how it might impede recovery.

The exact causes for increased mortality in some depleted cod populations remain to some extent unclear and debatable, and the amount of unintentional fishing mortality through bycatches is not always well known. Nonetheless, the weight of evidence suggests that natural mortality has increased and that it can be partially attributed to predation of cod by seals [4,17–19]. In eastern Canada, concurrently with a 97% reduction in cod biomass [3], the abundance of grey seals (Halichoerus grypus) increased 50-fold [20] while that of harp seals (Pagophilus groenlandicus) has increased fourfold [21]. Despite large reductions in cod abundance, the proportion of cod in seal diets in some areas has remained substantial [17,19,22]. This general pattern suggests that relative cod mortality by seals can increase as cod abundance decreases; that is, their predator–prey dynamics exhibit elements characteristic of a type-II or type-III functional response [2]. Such ‘predator pits’, which might not be uncommon in marine ecosystems [23], can be caused by the behaviour of the prey. Southern Gulf of St. Lawrence cod, for example, undertake an autumnal migration to a comparatively restricted over-wintering region [4] where the high density of their aggregations might be sufficient to render them vulnerable to a consistent number of prey being eaten per predator.

Allee effects are clearly problematic, negatively affecting both the time [8] and uncertainty [24] of recovery. This study highlights the synergistic influence that low abundance coupled with increased mortality can have from a conservation perspective. Changes in mortality resulting from altered interspecific interactions—the mechanism proposed here—might not be uncommon. Indeed there is good evidence to suggest that increases in predator-induced mortality might be associated with low abundance in other marine fish [17]. These observations underscore the necessity of evaluating the
consequences of overfishing from an ecosystem perspective: prolonged and heavy fishing mortality combined with increasing predator abundance can lower fish population resilience and adversely affect their viability.

Data accessibility. Data supporting this article has been uploaded as part of the Supplementary Material.

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