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Stage-specific biomass overcompensation by juveniles in response to increased adult mortality in a wild fish population

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Abstract. Recently developed theoretical models of stage-structured consumer–resource systems have shown that stage-specific biomass overcompensation can arise in response to increased mortality rates. We parameterized a stage-structured population model to simulate the effects of increased adult mortality caused by a pathogen outbreak in the perch (*Perca fluviatilis*) population of Windermere (UK) in 1976. The model predicts biomass overcompensation by juveniles in response to increased adult mortality due to a shift in food-dependent growth and reproduction rates. Considering cannibalism between life stages in the model reinforces this compensatory response due to the release from predation on juveniles at high mortality rates. These model predictions are matched by our analysis of a 60-year time series of scientific monitoring of Windermere perch, which shows that the pathogen outbreak induced a strong decrease in adult biomass and a corresponding increase in juvenile biomass. Age-specific adult fecundity and size at age were higher after than before the disease outbreak, suggesting that the pathogen-induced mortality released adult perch from competition, thereby increasing somatic and reproductive growth. Higher juvenile survival after the pathogen outbreak due to a release from cannibalism likely contributed to the observed biomass overcompensation. Our findings have general implications for predicting population- and community-level responses to increased size-selective mortality caused by exploitation or disease outbreaks.

Key words: biomass model; compensatory response; infectious disease; life history; maturation; mortality; pathogen; perch (Perca fluviatilis); *reproduction*; *stage structure.*

Introduction

Increasing mortality is expected to decrease population density because individuals are removed from the population. However, besides the negative direct effect on population numbers, mortality also releases the surviving individuals from competition for resources. If growth, maturation, and/or reproduction are food-dependent processes, this indirect density-dependent effect may lead to higher growth rates, faster maturation, and/or increased adult fecundity. Such indirect effects of mortality may result in increased stage-specific biomass production thereby compensating for the removal of individuals from the population (Werner and Gilliam 1984, De Roos et al. 2007). Experimental studies on laboratory populations have shown that stage-specific biomass remains unchanged or increases if not all individuals are subjected to increased mortality rates, for instance in water fleas (Slobodkin and Richman 1956), blowflies (Nicholson 1957), soil mites (Cameron and Benton 2004), and fish (Schröder et al. 2009). Differences in mortality rates between size classes or life stages of natural populations are commonly observed in nature and may arise from size-specific predation (Brooks and Dodson 1965, Werner and Gilliam 1984), harvesting by humans (Law 2000, Fenberg and Roy 2008), or parasite infections (Ohlberger et al. 2011*b*).

Recently developed theoretical models of stage-structured consumer–resource systems have shown that compensation (unchanged biomass) or overcompensation (increased biomass) in stage-specific biomass can arise in response to increased mortality rates (De Roos et al. 2007, 2008*b*). This phenomenon occurs in populations in which one or more of the life stages (e.g., juveniles and adults) are limited by intraspecific competition at high population densities. Biomass compensation or overcompensation emerges in the life

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stage in which individual performance is not strongly limited by competition, independent of which stage experiences increased levels of mortality. When adults experience strong competition and dominate the population biomass regulation of reproduction occurs, whereas when juveniles predominate and compete intensely for resources regulation of maturation occurs (De Roos et al. 2007, 2008*b*, Huss and Nilsson 2011). If mortality increases in reproduction-regulated systems, the net effect of the release from competition is an accumulation of juvenile biomass, because the increase in individual fecundity is more important than the increase in juvenile growth and maturation. Stage-specific biomass overcompensation can have important consequences for interspecific interactions at the community level as it may affect, for instance, the performance of predators that specialize on a specific life stage (Persson et al. 2007, De Roos et al. 2008*a*). We may thus hypothesize that in natural systems density-dependent biomass overcompensation is an important process underlying population-level responses to increased mortality caused by selective harvesting, predator increases or disease outbreaks. However, besides theoretical and experimental work on this topic, evidence of stage-specific overcompensation in natural populations is rare. Moreover, in cannibalistic populations, such as perch (*Perca fluviatilis*; Craig 1978, Le Cren 1992), the compensatory response to increased adult mortality might be modified through changes in cannibalistic interactions between the life stages.

In this paper, we investigate whether a massive pathogen outbreak in a natural fish population induced a compensatory response in stage-specific biomass production through indirect density-dependent effects. The perch population in Windermere (UK) experienced a considerable increase in mortality due to the outbreak of a perch-specific pathogen in 1976. Although the pathogenic organism itself has never been unambiguously identified, secondary infections have been described in detail (Pickering and Willoughby 1977). The pathogen induced a massive (98%) mortality in 1976 and the following years, with much higher prevalence among large, mature compared to small, immature individuals (Bucke et al. 1979) and with an estimated 10-fold increase in mortality after 1976 (Langangen et al. 2011). The population age structure remained severely truncated up to the early 2000s, indicating that the pathogen affected the perch population for many years (Edeline et al. 2008, Ohlberger et al. 2011*b*). However, population numbers did not collapse after the disease outbreak (Langangen et al. 2011), suggesting the incidence of a compensatory response at the population level.

We first present a stage-structured biomass model parameterized to our study system to predict the effect of the estimated increase in adult mortality rates on perch population biomass and age structure. The model predicts biomass overcompensation by juveniles in response to high adult mortality due to a shift in food-dependent growth and reproduction rates. Incorporating cannibalism into the model reinforces this compensatory response by further releasing adults from resource competition and by releasing juveniles from intraspecific predation. Second, using a 60-year time series of scientific monitoring of Windermere perch, we demonstrate that the high adult mortality after the pathogen outbreak was indeed associated with juvenile biomass overcompensation, with the consequence of a nearly unchanged average total population biomass. Third, a comparison of individual gonad weights showed that age-specific adult fecundity and growth increased after the disease outbreak, thereby indicating a competitive release. We argue that changes in intraspecific competition and cannibalism mediated the observed compensatory response by the juvenile stage in this population.

Material and Methods

Model description

We used a consumer–resource biomass model with a stage-structured consumer population as presented by De Roos et al. (2007, 2008*b*). This model is a simplified representation of a physiologically structured population model that accounts for a complete size-structure of the consumer population based on size-dependent individual vital rates, and thus directly translates individual-level assumptions to the population level. Juvenile (J) and adult (A) consumers feed on an unstructured resource (R) according to a type II functional response at maximum ingestion rate ($I_{\max}$). The resource population follows semi-chemostat dynamics, in the absence of consumers, with turnover rate (δ) and maximum biomass ($R_{\max}$). The change in resource biomass is described by its intrinsic growth rate and the loss through consumption by juveniles and adults:

$$\frac{dR}{dt} = \delta(R_{\max} - R) - \frac{R}{R+1}(I_{\max}J + qI_{\max}A)$$

where (q) scales the ingestion rate of adults to account for differences in foraging abilities of the two life stages, that is, whether adults are competitively superior to juveniles in terms of resource use ($q > 1$) or vice versa ($q < 1$). The net biomass production (ν) of juveniles (j) and adults (a) depend on the assimilation efficiency (σ), food-dependent ingestion rate and maintenance rate (T). It is assumed that basic metabolic demands are met before energy is allocated to somatic or reproductive growth and that all surplus energy is allocated to somatic growth in juveniles, while all surplus energy is allocated to reproduction in adults. All consumers experience a mortality rate (μ). The change in juvenile biomass is described as

$$\frac{dJ}{dt} = \nu_a^+(R)A + \nu_j(R)J - \gamma[\nu_j^+(R)]J - \mu_j J$$

where ν_j is the net biomass production of juveniles:

$$\nu_j(R) = \sigma I_{max} \frac{R}{R+1} - T$$

with

$$\nu_j^+(R) = \nu_j(R) \quad \text{if } R > \frac{1}{\sigma I_{max}/T - 1};\ 0 \text{ otherwise.}$$

Maturation rate (γ) depends on the newborn–adult size ratio (z), the net biomass production (which depends on resource abundance), and juvenile mortality rate:

$$\gamma(\nu_j^+(R)) = (\nu_j^+(R) - \mu_j)/(1 - z^{1-\mu_j/\nu_j^+(R)}).$$

The change in adult biomass is described as

$$\frac{dA}{dt} = \gamma[\nu_j^+(R)]J + \nu_a(R)A - \nu_a^+(R)A - \mu_a A$$

where ν_a is the net biomass production of adults according to

$$\nu_a(R) = \sigma q I_{max} \frac{R}{R+1} - T$$

with

$$\nu_a^+(R) = \nu_a(R) \quad \text{if } R > \frac{1}{\sigma q I_{max}/T - 1};\ 0 \text{ otherwise.}$$

We incorporate inter-stage cannibalism into the model by assuming that adults feed unselectively on the alternative resource and on juveniles. In contrast to the non-cannibalistic model, the change in resource biomass is then described as

$$\frac{dR}{dt} = \delta(R_{max} - R) - \frac{R}{R+1}(I_{max}J) - \frac{R}{R+J+1}(qI_{max}A)$$

the change in adult net biomass production is described as

$$\nu_a(R,J) = \sigma q I_{max} \frac{R+J}{R+J+1} - T$$

with

$$\nu_a^+(R,J) = \nu_a(R,J) \quad \text{if } R+J > \frac{1}{\sigma q I_{max}/T - 1};$$

0 otherwise

and juvenile mortality is described as

$$\mu_j = \mu + \frac{qI_{max}A}{R+J+1}.$$

All other equations and all parameters are the same as presented for the non-cannibalistic version of the model.

Model parameterization

We parameterized the stage-structured biomass model to perch using observational data and published literature values. Maximum mass-specific ingestion rate (0.032) was calculated based on the mean body mass (90 g) of adult perch from our data. As in De Roos et al. (2008*a*), values for mass-specific maintenance and background mortality rates follow standard quarter-power scaling laws of adult body size with proportionality constants 0.01 and 0.001, respectively. Maximum ingestion rate was assumed to be 10 times larger than maintenance rate and 100 times larger than background mortality rate. The newborn–adult size ratio was set to 0.1 based on the size when Windermere perch start feeding on their main larval prey *Daphnia* (~10 mm; Guma'a 1978) and the mean size at 50% maturation probability of male perch in Windermere (~100 mm; Ohlberger et al. 2011*b*). Based on data on asymmetric competition between perch life stages (Persson and De Roos 2006, Ohlberger et al. 2011*a*) and in accordance with Nilsson et al. (2010), we set the default value for q to 0.8. Assimilation efficiency was set to 0.5 for adult perch in approximation to Karås and Thoresson (1992). Resource turnover rate and maximum density were set to 0.1 and 2.0 (De Roos et al. 2008*a*). The last two parameters scale the total biomass, but not the juvenile to adult biomass ratio.

To compare model results and observational data, we simulate a low and a high mortality case for a cannibalistic and a non-cannibalistic population. Based on the estimated increase in adult mortality in Windermere perch after the pathogen outbreak, we increase the background mortality rate in adults by a factor 10 (Langangen et al. 2011). In order to account for uncertainty in the estimated parameter values, we run the model 1000 times by re-sampling all parameters simultaneously and randomly within a uniform ±20% range of the default value. We used the equilibrium value of each run as model output.

Data collection

Windermere, a glacial valley lake in the English Lake District, United Kingdom, is divided into a north and south basin by shallows and islands (Le Cren 2001). The perch populations in the two basins are effectively independent according to capture–mark–recapture and genetic data (Kipling and Le Cren 1984, Bodaly et al. 1989). Perch is the most abundant fish species and is preyed upon by pike (*Esox lucius*), the top predator in the lake. The scientific monitoring of perch (and pike) was initiated in the mid-1940s and continues to date with very little change in gear type and fishing methods. Perch trapping takes place for 6 weeks during spring on the spawning grounds with standard traps that are unselective for perch of 90–300 mm total length (Le Cren et al. 1977). Individual fish (1942–2003, N = 105 763) are measured for total length and sexed by internal examination, and for most of the individuals the left opercular bone is removed for age determination (Le Cren 1947). For a few fish of some of the year classes wet weight and gonad weight were also recorded. Further details on the lake, its fisheries and the scientific

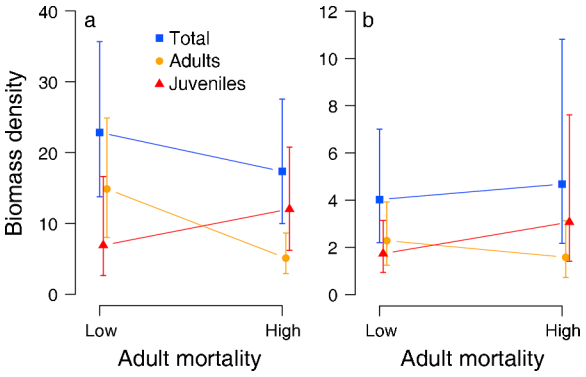

FIG. 1. Model results for (a) non-cannibalistic and (b) cannibalistic populations showing the juvenile (triangles), adult (circles), and total population (squares) biomasses at low and high mortality. High mortality refers to a 10 times higher adult mortality compared to juveniles, while all other parameters were the same in both cases. Symbols represent mean values and lower and upper error bars represent 5% and 95% quantiles from 1000 runs of simultaneously and randomly resampled parameters within a ±20% range of the default value.

monitoring of perch and pike can be found in Le Cren (2001).

Data analyses

We estimated perch biomasses based on population numbers that were taken from recent population estimations for the years 1943–2002 (Langangen et al. 2011). These estimates were obtained by fitting age-structured population models to the 60-year time series of catch-at-age data, thus including long time series before and after the outbreak of the perch-specific pathogen in 1976. The age-structured model takes into account the full cohort and the non-linearity between catch and effort for estimating the population numbers (Langangen et al. 2011). Age-specific biomasses in each year were calculated by multiplying the population estimates by the average sex, age, and basin-specific body masses from the catch data. Since only very few 1-year-old fish were caught in the traps, their numbers were estimated assuming the same mortality for age 1 fish before and after the disease outbreak (this age group was much less affected by the pathogen; Bucke et al. 1979). This is a conservative approach to estimating age 1 fish biomass, because assuming higher mortality after 1976 increases the estimated number and thus biomass for this age-class after the disease outbreak. Biomasses for the 1-year-old fish were estimated by fitting von Bertalanffy growth curves to the observed mass data on older age classes using a length–weight relationship obtained from years in which individual length and weight were recorded (44 out of 60, including years before and after the disease outbreak). Stage-specific biomasses were calculated as juvenile and adult biomasses for each year from the 1–2 and the 3–6 year-old fish, respectively. Windermere perch typically mature at age 2 or age 3, with males maturing on average slightly earlier and at a smaller size than females (Craig 1977, Ohlberger et al. 2011*b*). Due to the abrupt (10-fold) increase in mortality and the corresponding change in the demography of the perch population in 1976 (Edeline et al. 2008, Langangen et al. 2011, Ohlberger et al. 2011*b*), we pooled the biomass data into two periods, a low mortality phase before (34 years) and a high mortality phase after the disease outbreak (25 years) and calculated mean biomasses for the two phases.

We used analysis of covariance (ANCOVA) to compare regressions of gonad weight to age relationships for female perch before and after the disease outbreak in both basins. Gonad weight data were log-transformed, because they were not normally distributed (Shapiro-Wilk normality test: $W = 0.8829$, $P < 0.001$). Total population fecundity could not be estimated reliably because the catch data are biased towards mature fish (trapping on spawning grounds), which introduces high levels of uncertainty when estimating age-class specific proportions of mature vs. immature fish, and due to a shift in age at maturity before vs. after the disease outbreak.

RESULTS

The stage-structured consumer–resource model predicts biomass overcompensation by the juvenile stage in response to increased adult mortality for non-cannibalistic and cannibalistic populations (Fig. 1a, b). When background mortality in adults is low, adult biomass is higher than juvenile biomass, whereas juvenile biomass is higher when adult mortality is increased by a factor of 10. In the non-cannibalistic population, juvenile biomass production increases in response to high adult mortality due to higher total reproduction and higher juvenile growth rates (Fig. 1a). Total reproduction increases without but slightly decreases with cannibalism, while "per biomass" reproduction increases in both cases. In the cannibalistic population, however, the release from predation on juveniles reinforces the compensatory response. As a consequence, total population biomass even increases at high compared to low adult mortality (Fig. 1b). Biomass overcompensation by juveniles, that is, a decrease in adult biomass and a corresponding increase in juvenile biomass, was found in all 1000 model runs in both cases. The described pattern is therefore remarkably robust to parameter perturbations (±20%), showing that these model results apply to a broad parameter space.

Our data analysis shows that the biomass of perch in both Windermere basins decreased considerably in the age classes 3–6, but increased in the age classes 1–2 after the invasion of the pathogen (Fig. 2a, b). Although biomasses of 3–6 year-old fish decreased by 88% and 77%, total population biomasses decreased by only 14% and 10% in the north and south basins, respectively. Hence, biomasses of 1–2 year-old fish increased nearly as much as the biomasses of adult fish decreased,

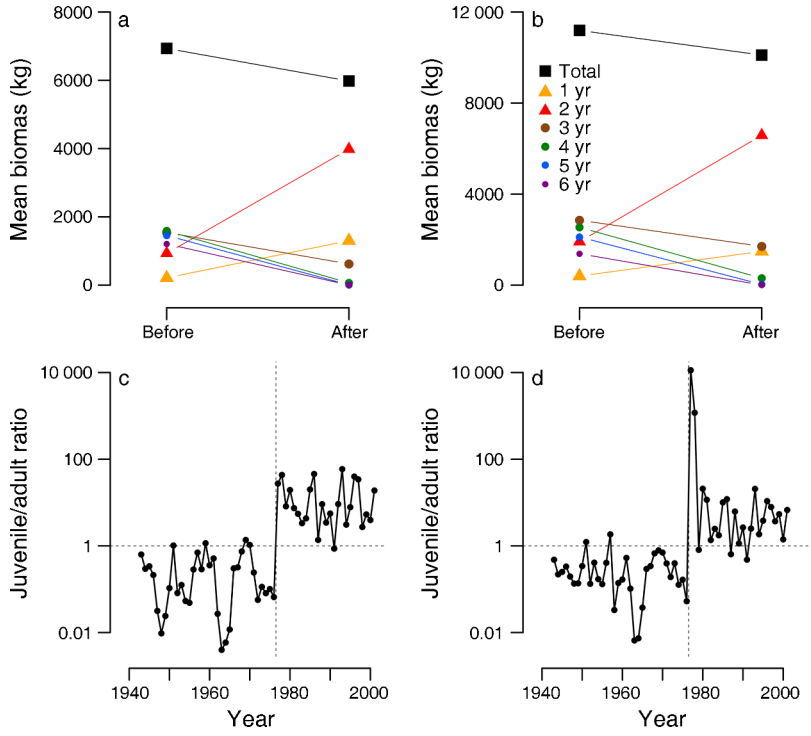

FIG. 2. Age-specific biomasses of perch before and after the disease outbreak (top) and time series of juvenile/adult biomass ratios (bottom) in the north (left) and south (right) basins of Windermere. Top panels show the average biomasses in the (a) north and (b) south basins for the total population (black squares) and the age classes 1 (orange triangles), 2 (red triangles), 3 (brown circles), 4 (green circles), 5 (blue circles), and 6 yr (purple circles). Bottom panels show the time series of juvenile/adult biomass ratios in the (c) north and (d) south basins, indicating a shift in the biomass distribution from being dominated by adults (<1) to being dominated by juveniles (>1) after the disease outbreak in 1976 (vertical dashed line).

demonstrating an overcompensatory response by the juvenile stage to the increased adult mortality. While the older age classes (3–6) were more abundant before, the younger age classes (1–2) were more abundant after the disease outbreak, and this pattern was relatively constant within each period despite large fluctuations in total population numbers (Fig. 2c, d). The juvenile-to-adult biomass ratio shifted from smaller than one to larger than one around 1976. The biomasses of 1–2 year-old fish (in percentage of total population biomass) increased from ~20% before to ~80% after the disease outbreak in both basins. Our analysis also showed biomass overcompensation by juveniles when 2-year-old male perch were classified as adults. The comparison of gonad weight data before and after the pathogen outbreak showed that age-specific fecundity increased significantly after 1976 (Fig. 3a, b). In both basins, the gonad weight to age relationships differed significantly in slope and intercept between the two periods (ANCOVA; north, $F_{1,251} = 81.2$, $P < 0.001$; south, $F_{1,413} = 31.8$, $P < 0.001$). In contrast, the gonad mass to body mass relationships did not differ significantly (north, $F_{1,251} = 0.44$, $P = 0.506$; south, $F_{1,413} = 2.71$, $P = 0.101$), indicating enhanced adult growth after the disease outbreak.

DISCUSSION

Our data analysis demonstrates strong stage-specific overcompensation in juvenile perch in response to the drastically increased adult mortality induced by the disease outbreak, with a nearly unchanged average total population biomass. The stage-structured biomass model that we parameterized to our study system predicts biomass overcompensation in response to increased adult mortality due to food-dependent shifts in growth and reproduction and, in the presence of cannibalism, due to changes in cannibalistic interactions between the life stages. The increase in age-specific adult fecundity and size at age (with an unaltered mass–fecundity relationship) after the pathogen outbreak indicates that higher growth and reproduction rates contributed to the observed biomass overcompensation in Windermere perch. Enhanced adult growth rates may result from a competitive release in reproduction-regulated systems with indeterminate growth. Published data on zooplankton abundance in Windermere (George and Hewitt 1998) suggest a slight increase in resource levels from the mid-1970s onward. However, these data are only from the north basin, do not cover the entire time series, and their interpretation is complicated by the fact that phosphorus levels in the lake have also

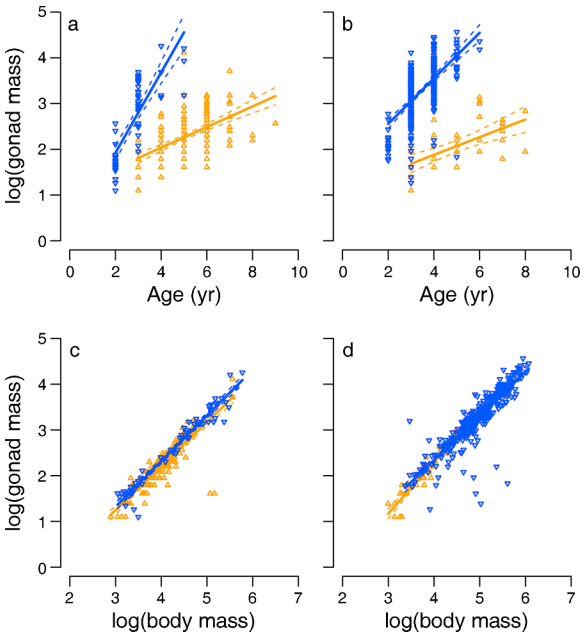

FIG. 3. Age–fecundity relationship (top) and mass–fecundity relationship (bottom) of Windermere perch. (a, b) The top panels show the gonad mass to age relationships in the (a) north and (b) south basins for before (orange triangles) and after (blue triangles) the pathogen outbreak. Dashed lines represent the 95% confidence intervals of the linear regressions. The gonad mass to age regressions differed significantly in slope and intercept in both basins (ANCOVA). (c, d) In contrast, the corresponding gonad mass to body mass regressions did not differ significantly in slope or intercept. Note that gonad mass and body mass data were measured in grams and were log-transformed.

increased at the same time (Parker and Maberly 2000). Besides a release from competition for common resources, reduced inter-stage cannibalism may also have contributed to the observed overcompensation in juvenile biomass. It is known that perch undergo ontogenetic niche shifts from feeding on zooplankton to feeding on macroinvertebrates and finally to piscivory and cannibalism (Craig 1978, Le Cren 1992). Cannibalism may enhance the compensatory response in juvenile biomass production by releasing adults from competition thereby increasing energy availability for growth and reproduction, and/or by releasing juveniles from predation thereby increasing juvenile survival. Although small perch constitute a small proportion of total diet of large perch in Windermere (McCormack 1970), we suggest that a release from inter-stage cannibalism after the disease outbreak contributed to the observed biomass overcompensation by juveniles, because adult biomass decreased considerably and because perch cannibalize less when other resources are abundant (McCormack 1970).

Several factors and processes that may be of importance in generating the observed response in Windermere perch were not included into the model. First, we do not consider evolutionary change in life-history traits, such as changes in size at maturity. Indeed, length at maturity in perch has previously been shown to have slowly increased before but rapidly decreased after the pathogen outbreak, which may have contributed to a shift in reproduction rate (Ohlberger et al. 2011*a*). Although the smaller size at maturation increased the proportion of mature 2-year-old fish, this effect can be assumed to be negligible for the biomass analyses, because the increase was only a few percent (Ohlberger et al. 2011*a*). Second, Windermere perch were commercially fished at the beginning of our time series (see Appendix), continuing until 1948 in the north and until 1964 in the south basin (Le Cren et al. 1977). However, fishing effort was relatively low (7–28% of the fish were removed in any given year) compared to those years prior to our time series and tests have shown that our results do not change qualitatively when using only data collected after commercial fishing had stopped. Third, an introduced roach (*Rutilus rutilus*) population has expanded in Windermere since the early 1990s (Winfield et al. 2008), which might conceivably have contributed to a change in perch age-structure through intensified interspecific competition. Roach were not caught by gill net surveys in 1979 and 1980 (Craig and Fletcher 1981), and they subsequently comprised only about 4% of the fish community in 1995 and about 20% in 2000 (based on fish numbers from extensive gill net surveys; I. J. Winfield, *unpublished data*). Changes in the perch biomass distribution, however, occurred around the disease outbreak and perch population numbers did not decline in response to the roach expansion.

We used a simple stage-structured biomass model that predicts the observed biomass overcompensation in the juvenile stage as a result of a shift in food-dependent reproduction and growth rates and/or due to changes in cannibalism and thus juvenile survival. Here, we assume that all surplus energy in adults is diverted to reproduction. We have evaluated this assumption by introducing a parameter that scales the energy allocation to growth and reproduction and by studying model behavior as a function of this parameter. Biomass overcompensation by juveniles becomes weaker as more energy is allocated to growth in adults, but a compensatory effect occurs for all nonzero values of the allocation parameter. Hence, relaxing this assumption does not change the model results qualitatively. In our model we also assume asymmetric competition between life stages. This assumption is supported by physiological observations and modeling results suggesting that smaller-sized perch tend to be more efficient than larger ones in exploiting a common resource (Persson and De Roos 2006, Ohlberger et al. 2011*a*). De Roos et al. (2007) showed that populations are likely to be reproduction limited when juveniles are superior to adults with respect to resource competition (or have lower mortality) and that in such systems increases in mortality induce a compensatory effect in the juvenile

stage. The extent of the compensatory response depends on the degree to which mortality rate increases, because juvenile biomass changes with adult mortality according to a hump-shaped relationship (De Roos et al. 2007, Schröder et al. 2009). In other words, if adult mortality increases above a certain level, juvenile biomass also decreases and the population will eventually go extinct. In perch, the pathogen-induced increase in mortality rate was such that the compensatory response by the juveniles nearly matched the loss of biomass in the adult stage. As a consequence, average total population biomass remained nearly unchanged.

Compensatory responses in biomass distributions between ontogenetic life stages can have far-reaching consequences for population structure, community dynamics, and food web stability. On the population level, increased size-selective mortality changes the size structure of the population and may promote stage-specific biomass overcompensation if growth and reproduction are food-dependent processes (De Roos et al. 2007, 2008*b*). On the community level, changes in size-structure affect resource, competitor and predator populations through interspecific interactions. For instance, predators may either benefit or suffer from changes in prey size distribution, depending on which size-class or life stage they target. A predator may thus promote its own population growth by feeding on the size-class that shows the overcompensatory response in biomass (emergent Allee effect; De Roos and Persson 2002), or it may facilitate the existence of another predator by feeding on a different size class (emergent facilitation; De Roos et al. 2008*a*, Huss and Nilsson 2011). Accordingly, Persson et al. (2007) showed experimentally that culling old stunted fish of a prey population, Arctic charr (*Salvelinus alpinus*), caused an increase in the availability of small-sized fish and allowed the previously almost extinct predator, brown trout (*Salmo trutta*), to recover. In Windermere, the invasion of the perch-specific pathogen may have had a similar positive effect on the predator pike. Pike feed on most of the perch size-classes, and juvenile pike potentially compete with large perch for macroinvertebrates and small perch (Frost 1954, Edeline et al. 2008). Abundance of adult pike increased considerably around 5–10 years after the pathogen outbreak in perch (see Appendix; Langangen et al. 2011), and it was shown that the disease changed the effect of perch on pike recruitment from negative to positive (Edeline et al. 2008, Langangen et al. 2011), thereby increasing pike population growth rate. Taken together, this suggests that the disease facilitated the high pike abundance by decreasing competition and increasing the biomass production of its prey. In contrast to theoretical models assuming adverse effects of pathogens and predators on a shared prey population (Hatcher et al. 2006), our results indicate that pathogens and predators might also enhance each other's performance. As a consequence of these changes in community structure, predation by pike on perch may have increased considerably and thus contributed to the high levels of mortality among adult perch that have been observed since the outbreak of the pathogen.

The presented findings, together with recent theoretical developments and experimental work, have implications for both disease control and fisheries management. Our study suggests that natural populations may respond to increased mortality rates caused by infectious diseases or harvesting by stage-specific biomass overcompensation. This has management implications for at least two reasons: biomass overcompensation may result in counterintuitive measures necessary to restore fish stocks (Persson et al. 2007), and it may render marine protected areas less efficient than expected based on models without overcompensation (Claessen et al. 2009). The population-level response depends on the increase in mortality rates, the competitive interactions within and between life stages, and the environmental conditions that determine resource availability. More studies addressing such applied issues, particularly regarding the interplay between exploitation and environmental conditions, are clearly needed.

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APPENDIX

A figure showing time series of population numbers and total fishing effort for pike and the two perch populations in the north and south basins of Windermere (*Ecological Archives* E092-187-A1).