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# Title: **Antagonistic selection from predators and pathogens alters food-web structure**

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**Abstract**

Selection can alter predator-prey interactions. However, whether and how complex food-webs respond to selection remains largely unknown. We show in the field that antagonistic selection from predators and pathogens on prey body-size can be a primary driver of food-web functioning. In Windermere (UK), pike (*Esox lucius*, the predator) selected against small perch (*Perca fluviatilis*, the prey), while a perch-specific pathogen selected against large perch. The strongest selective force drove perch trait change and ultimately determined the structure of trophic interactions. Before 1976, the strength of pike-induced selection overrode the strength of pathogen-induced selection and drove change to larger, faster growing perch. Predation-driven increase in the proportion of large, infection-vulnerable perch presumably favored the pathogen since a peak in the predation pressure in 1976 coincided with pathogen expansion and a massive perch kill. After 1976, the strength of pathogen-induced selection overrode the strength of predator-induced selection and drove a rapid change to smaller, more slowly growing perch. These changes made perch easier prey for pike and weaker competitors against juvenile pike, ultimately increasing juvenile pike survival and total pike numbers. Therefore, although predators and pathogens exploited the same prey in Windermere, they did not operate competitively but synergistically by driving rapid prey trait change in opposite directions. Our study empirically demonstrates that a consideration of the relative strengths and directions of multiple selective pressures is needed to fully understand community functioning in nature.

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## Introduction

Interacting populations often show reciprocal phenotypic changes reflecting co-adaptations. In turn, co-adaptations alter the strength and even the nature of interactions (1-3). Therefore, community structure and functioning is driven by an interplay between demography and phenotypic change (4-6). Recently, there has been considerable interest in how prey adaptive responses to predators can drive community dynamics (5-10). At the same time, it has been shown that parasites and parasite-mediated trait changes can play a crucial role in food-web structuring (11, 12). However, despite the fact that organisms are often confronted with both predators and parasites (13), there have been few attempts to understand how adaptive response to joint predation and parasitism affects food-web functioning in nature. Here, we use 50-years long time series from a whole-lake system (Windermere, UK) to show that simultaneous selection from both predators and pathogens structured the food-web in a way that could not be predicted by considering each selective pressure separately.

Windermere is a glacial valley lake of the English Lake District, divided by shallows into north and south basins of different size and productivity (14, 15). The fish community of Windermere is size-structured, with only a few numerically dominant species interacting in a mixture of competition, predation, and cannibalism termed intraguild predation (IGP) (16, 17). Perch (*Perca fluviatilis*) are the most abundant fish and are preyed upon by pike (*Esox lucius*), the top predator of the system. Small perch below 16 cm body length (~ age ≤ 2 years) feed entirely on zooplankton and macroinvertebrates, while large perch (above 16 cm body length) feed on macroinvertebrates and on their own fry (18, 19). Small pike below 20 cm body length (~ age ≤ 1.5 years) have the same diet as large perch (i.e. macroinvertebrates

and small perch), while large pike feed exclusively on fish, mostly perch of 6-9 cm body length (20). Consequently, small perch are prey for pike in Windermere, but large perch are potentially strong competitors with pike (especially with small pike).

A long-term monitoring program for Windermere perch and pike was initiated in the early 1940s. Since 1944, pike have been gillnetted during winter (14, 15, 21, 22). Perch have been caught with traps set on their spawning grounds from the end of April to mid-June (23). On each lift of a trap, the whole catch or occasionally a random fraction of the catch has been sexed, measured for total body length, and opercular bones have been removed for age determination following a validated method (24). Bone density differs between summer and winter, producing narrow bands ("checks") that are deposited on the opercular bones during the slow winter growth period. These checks then serve as an annual mark and, thus, allow the aging of individual fish (24). Pike were aged following the same method (25). The abundances of both perch and pike have been estimated annually for the 1944-1995 period, separately for each basin as well as for both small (i.e. age = 2) and large (i.e. age > 2) individuals (26) (Fig. 1A and 1B). Together with these biological data, surface water temperatures were recorded on a near daily basis and were here averaged for each year. Finally, maximum phosphorus concentration between September/October in year *y* and February in year *y*+1 was measured each year since 1945 in the north basin and since 1946 in the south basin, and was here used as a proxy for Windermere primary productivity in year *y*+1.

In 1976, a perch-specific pathogen severely impacted the perch population (Figs. 1A, 1C and 1D). Although the primary pathogenic agent remains unidentified, the disease is characterized by epidermal lesions associated with a wide variety of fungal and bacterial infections (27). The pathogen preferentially infects large, maturing (90-100% prevalence) perch over small, immature individuals (50-70% prevalence) and induced a 98 % mortality of

spawners during the 1976 reproductive period (27). By 1977, captured perch showed no external sign of disease (27), but the numbers of large perch have remained low since 1976 (Fig. 1A). Both the age structure (Fig. 1C) and mean body length (Fig. 1D) of the Windermere perch population remains severely truncated, suggesting that the pathogen is still present. Windermere perch were shifted by the pathogen from an iteroparous to an effectively semelparous population (Fig. 1C), setting the stage for increased investment into one single reproductive bout (28). Increased reproductive investment in perch is likely to have reduced somatic growth rate owing to the trade-off between body growth and reproduction (29). Additionally, in immature perch, disease prevalence is much higher on fast-growers than on slow-growers (27), indicating a trade-off between disease resistance and somatic growth (28).

Based on these observations, we predicted that pike (predator)-induced selection and pathogen-induced selection acted in opposite directions on perch body-size and somatic growth rate. Before pathogen invasion, perch somatic growth rate should have reflected the effect of increased predation due to an increase in the pike/perch ratio (Fig. 2A). After pathogen invasion, perch growth should have reflected the combined action of the two antagonistic selective forces (21). We have tested this prediction by estimating nonlinear changes in perch somatic growth rate (21). In our statistical analysis, we accounted for the effects of environmental variables known to plastically affect perch growth [i.e. primary productivity, water temperature, and perch density (23), see Material and methods] and, by using a smooth term on the *Year class* effect, we removed any *a priori* expectation concerning the shape of the temporal trend. We performed separate analyses for each basin of Windermere because the two perch populations are considered distinct (30, 31), thus providing a natural replicate for hypothesis testing. Since life-history responses to pathogens may be sex-specific (28), we also performed separate analyses for each sex. In both basins of

the lake, our results support the prediction that pike and pathogens induced selection in opposite directions on perch body-size.

## Results and discussion

Before 1976, perch somatic growth rate generally increased in both basins and in both male and female perch (Fig. 2C), in parallel with an overall increase in predation pressure (Fig. 2A). Short-term variations in predation pressure (Fig. 2A) were in remarkably close match with similar changes in perch growth in both basins (especially in males, Fig. 2C), supporting the prediction that pike selected for increased somatic growth in Windermere perch. A correlation analysis revealed that predation pressure had a statistically significant ($p < 0.05$) positive effect on perch somatic growth at lags ranging from 0 to 9 years, with the highest correlation at a 5-years lag. This lag corresponds roughly to 1.25 to 5 perch generations since male perch in Windermere may mature at age-1 but mean age of mature fish in the catch was approximately age-4. Interestingly, female perch responded less closely than male perch to variation in the predation pressure (Fig. 2C), presumably because females reached a size refuge faster than males (*Sex* effect in Table 1). Indeed, fast immature growth generally lasts longer in female than in male fish because females mature at an older age (29). After outbreak of the pathogen in 1976, Windermere perch somatic growth decreased rapidly in both basins and for both sexes (Fig. 2C) despite the fact that predation pressure remained high (Fig. 2A). This result supports the prediction that the pathogen selected for slow somatic growth in perch, and further suggests that the strength of pathogen-induced selection overrode the strength of pike-induced selection (21, 22). Finally, perch somatic growth rate in 1995 decreased to 1940s values in the north but not in the south basin (Fig. 2C), in accordance with

99 raw data observation of perch numbers (Fig. 1A), age (Fig. 1C) and size (Fig. 1D) showing
100 that the infection was more severe in the north than in the south basin.
101 Antagonistic selection from multiple consumers on their joint prey may result in
102 counterintuitive demographic effects. Indeed, while linear density-dependence predicts a
103 negative impact of multiple consumers on each other (i.e., exploitative competition),
104 antagonistic selection on a joint resource can make consumers mutually beneficial foragers (5,
105 6). In Windermere, observations are consistent with the predictions that the effects of
106 antagonistic selection overrode the effects of exploitative competition and made pike and the
107 pathogen mutually beneficial foragers. Indeed, signs of an externally similar disease on perch
108 were reported as early as 1963 (27) but the spread of the pathogen and massive perch kill in
109 1976 coincided with a peak in predation pressure in both basins (Fig. 2A). Additionally,
110 predation pressure was higher in the north than in the south basin both before and after the
111 spread of the pathogen (Fig. 2A), and the infection was more severe in the north than in the
112 south basin (see above). Therefore, by increasing the proportion of large, fast-growing perch
113 which were more sensitive to infection, pike may have facilitated the spread of the pathogen.
114 Then, by selecting against slow somatic growth in perch, pike may have prevented perch from
115 maximizing energy allocation to disease resistance (28) and may have favored the
116 maintenance of high levels of pathogen prevalence.
117 In turn, by preventing perch from reaching a size refuge the pathogen may have made
118 perch become easier prey for and weaker competitors with pike (16). This process has been
119 recently demonstrated by the artificial removal of large prey (Arctic charr *Salvelinus alpinus*)
120 from a Norwegian lake, which ultimately increased top predator numbers (Brown trout *Salmo*
121 *trutta*) (32). In Windermere, examination of trends in pike numbers suggests a similar process
122 driven by the pathogen. Indeed, at odds with a linear density-dependent effect, pike numbers
123 increased markedly after invasion of the perch pathogen in Windermere (Fig. 1B). We

predicted that juvenile pike should have most strongly benefited from invasion of the perch pathogen because (i) juvenile pike were shown from diet data to be more directly in potential competition with large perch (18-20) and (ii) juvenile pike eat at a higher rate than large pike and are thus more susceptible to competition for food (20). To test this prediction, we used pike stock-recruitment models which explored the relationship between parental stock size in year *y* and the number of age-2 pike in year *y*+2 (see Materials and methods). These models allowed us to estimate the effects of pathogen-induced trait changes in perch on the pike-perch interaction, while controlling for the effects of temperature, perch numbers and pike numbers (Table 1). As emphasized above, perch populations in the north and south basins of Windermere should be considered distinct and only about 20% of pike disperse between the two basins (14, 15). We therefore analyzed pike recruitment separately for the north and south basins. Our results clearly show that pathogen-induced trait changes in perch increased juvenile pike survival by changing perch from being mainly a competitor to being mainly a prey for pike.

Pike recruitment rate (i.e., number of recruits per spawner) increased significantly in both basins after invasion of the pathogen (*Pathogen* effects in Table 1, intercepts in Fig. 2B). This increase was not the result of a higher number of eggs produced by female pike because female pike reproductive investment decreased from 1963 to 1995 (21). Increased pike recruitment rate was also not due to a relaxation of density dependence (competition and cannibalism) in the pike population because the strength of density dependence did not change significantly (*SSB*Pathogen* interactions in Table 1, slopes almost unchanged in Fig. 2B). Therefore, increased pike recruitment rate most likely reflected increased survival of small pike due to pathogen-induced trait changes in perch. Modeling the effect of perch on pike recruitment rate supported this hypothesis. Pathogen invasion changed the effect of perch from negative to positive (*Perch*Pathogen* interactions in Table 1, slopes changing from

149 negative to positive in Fig. 2D), indicating that the pike-perch link was changed from a
150 mixture of predation and competition dominated by perch towards a simpler predator-prey
151 relationship dominated by pike. Interestingly, perch traits were more severely shifted by the
152 pathogen in the north than in the south basin (see above), driving a locally higher increase in
153 pike recruitment and steeper change in the effect of perch on pike survival (Fig, 2D, Table 1).
154 These results suggest that antagonistic selection from predators and pathogens on
155 Windermere perch body-size generated a mechanism similar to the so-called "synergy" [i.e.
156 synergistic foraging rates (5, 6, 10)] which has been modeled to arise among multiple
157 predators when there is a trade-off in the prey for behavioral avoidance of the predators (5, 6).
158 To our knowledge, our results provide the first empirical example of this synergistic effect
159 acting through prey life-history change.
160
161 **Conclusions**
162
163 It has been shown that behavioral disturbance of predation capacity and sensitivity to
164 predation in an invertebrate host (*Gammarus* spp.) by parasites can reverse species dominance
165 in an IGP hierarchy (17). Parasites have also been shown to indirectly increase algal growth
166 by reducing grazing capacity in a gastropod host (12). Our findings considerably expand the
167 scope for parasite-induced effects on ecosystems by showing synergistic effects between
168 parasites and predators acting through antagonistic selection on the prey. Antagonistic
169 selection on prey body-size (as depicted in Windermere) is a potentially strong ecosystem
170 modifier because body-size determines a host of species traits that affect the structure and
171 dynamics of food webs (4, 33). Hence, in light of the abundance of parasites across systems
172 (11), size-selective predators and pathogens are likely to play an important role in the
173 structuring and resilience of ecosystems. So far, synergistic foraging between multiple

consumers (acting through a behavioral trade-off in the prey) has been consistently modeled to favor species coexistence and food-web stability (5, 6).

Antagonistic selection leading to synergistic foraging rates has practical implications since it can magnify the effects of species introductions and human activities. For instance, invasive species will more strongly deplete native resource populations if they select in an opposite direction compared to native predators or pathogens of the resource. Antagonistic selection, by favoring species coexistence (5, 6), may also favor long-term persistence of invasive predators and parasites. In particular, our results underline that predators can influence coevolution between parasite virulence and host resistance by impeding evolution of resistance in the host (13, 34, 35). Finally, harvesting by humans often targets large individuals and induces body-size reduction in exploited populations (21). Hence, harvesting could select in an opposite direction to competitors and predators and magnify their effects. Management strategies ignoring potential effects of antagonistic selection on trophic interactions might lead to inappropriate management of ecological resources.

## Materials and methods

**Perch growth modeling.** Perch traps used for sampling were unselective for individuals ranging from 9 to 30 cm body length and thus captured both fast and slow growers for ages ranging from 2 to 6 years (23). However, age 5 and 6 perch became rare after the invasion of the pathogen in Windermere. Therefore, in order to confidently rule out possible effects of sampling bias we restricted our growth analysis to perch caught from age-2 to age-4. We modeled temporal changes in Windermere perch somatic growth rate using a generalized additive model (mgcv library of R (36)) of the form:

$$BL = \beta_0 + f_1(A) + \beta_1 Bas + \beta_2 S + \beta_3 T + \beta_4 Ph + \beta_5 P + \beta_6 Ph \times T + \beta_7 P \times T + f_2(Yc) + \varepsilon \qquad \textbf{Eq(1)}$$

where *BL* stands for body length of individual *i* and year class *Yc* (n=67,457), *A* is the individual's age at capture, *Bas* is the basin in which the individual was captured, *S* is the individual's sex, *T*, *Ph* and *P* are mean temperature, mean phosphorus concentration and mean perch density (small + large), respectively, experienced by the individual (i.e. from year *Yc* to year *Yc*+*A*), $\beta$s are slopes of the linear effects, $\beta_0$ is an intercept, $\varepsilon$ is an error term, and $f_1$ and $f_2$ are nonparametric smoothing functions (natural cubic splines fitted by generalized cross validation (36)). In the model, interactions between temperature and the other biological covariates accounted for the thermal dependence of primary productivity and competitive interactions. Plots in Fig. 2C were produced with basin- and sex-specific models as described in **Eq(1)** but in which *Bas* and *S* were dropped (north basin: n=17,321 males and n=3,279 females; south basin: n=40,904 males and n=5,953 females). 95% confidence limits around the *Yc* effect in Fig. 2C were computed using a modified wild bootstrap approach (37). Briefly, the bootstrap distribution for the effect estimate was obtained by randomly inverting the signs of the errors from the model, adding these to the fitted values, and refitting the model (repeated 500 times). To account for intra year-class correlation, all errors from a given year-class in a given bootstrap sample were either inverted or not with probability 0.5. Estimates of the main effects of *T*, *Ph* and *P* in Table 1 were obtained from a model in which the interaction terms were omitted from **Eq(1)**. We calculated predation pressure from pike on perch as the natural log of the ratio of the numbers of all (age $\geq$ 2) pike on the number of small (age-2) perch because pike target mainly small perch in Windermere (20). Finally, we tested for the link between predation pressure and perch somatic growth using correlations between the fitted *Yc* effect (from 4 basin- and sex-specific models as in Fig. 2C) and predation pressure from pike on perch in year class *Yc*-*t* where *t* varied from 0 to 16 years.

**Pike recruitment modeling**. We modeled pathogen- associated change in pike recruitment using linear stock-recruitment models (38) of the form:

$$\ln(R/SSB) = \beta_0 + \beta_1 T + \beta_2 P + \beta_3 SSB + \beta_4 SSB \times Pa + \varepsilon \qquad \textbf{Eq(2)}$$

where $R$ stands for the number of pike recruits (i.e. age-2 pike) in year $y$ and basin $Bas$ (n=50 for each basin), $SSB$ is pike spawning stock biomass (i.e. number of spawners) in year $y$-2 and basin $Bas$, $T$ and $P$ are mean water temperature and mean perch density (small + large) experienced by the recruits from year $y$-2 to year $y$, $Pa$ is the pathogen (i.e. presence or absence), $\beta$s are slopes of the effects, $\beta_0$ is an intercept, and $\varepsilon$ is an error term. We modeled changes in the pike-perch interaction using a model similar to **Eq(2)** except that $P$ and $SSB$ were inverted in **Eq(2)**. In our models, the response (natural log of the $R/SSB$ ratio) measured recruitment rate, i.e. the number of recruits per spawner (38). The $SSB$ effect in the right hand side of **Eq(2)** captured cannibalism and competition (density-dependence) in the pike population (38), and the $SSB*Pa$ interaction tested for an effect of the perch pathogen on density dependence in the pike population. The $P$ effect captured predation and competition between perch and juvenile pike, while the $P*Pa$ interaction tested for an effect of pathogen-induced trait changes in perch on the pike-perch trophic interactions. Estimation of the main effects of $T$, $P$, $SSB$, and $Pa$ in Table 1 were obtained from a model in which the interaction term was omitted from **Eq(2)**. Predicted values in Figs. 2B and 2D were computed from 2 different models as in **Eq(2)** but in which only the focal terms ($SSB$ and $Pa$ in Fig. 2B; $P$ and $Pa$ in Fig. 2D) were kept.

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**Author contributions**. E.E. and T.B.A. conceived the study. E.E. performed the statistical analyses. E.E. and T.B.A. wrote the paper in dialogue with all coauthors, all of whom participated in the interpretation of the statistical results. I.J.W., J.M.F. and J.B.J. contributed to the collection of data and made them all available to the present study.

**Author information**. The authors declare that they have no conflicts of interests. Correspondence and requests for materials should be addressed to N.C.S. (n.c.stenseth@bio.uio.no).

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**Figure legends**

**Figure 1**. Background information for pike (*E. lucius*), perch (*P. fluviatilis*) and for expansion of a perch-specific pathogen in Windermere (UK). Vertical solid lines indicate the first massive perch kill from the pathogen in 1976. (**A**-**B**) Time series for population size of perch (**A**) and pike (**B**) in the north and south basins of the lake, separated into small (age-2 years) and large (age ≥ 3 years) individuals. Horizontal lines show mean abundances before and after pathogen invasion, separately for small (dashed and dotted lines) and large (solid lines) individuals. (**C**-**D**) Time series for perch mean age (**C**) and mean body length (**D**) with 95% confidence intervals, separated by sex and basin.

**Figure 2.** Effects of predator (pike, *E. lucius*)- and pathogen-induced selection on perch (*P. fluviatilis*) trait-change and resultant impacts on pike-perch-pathogen interactions in Windermere (UK). Vertical solid lines indicate the first massive perch kill from the pathogen in 1976. Note that a peak in the predation pressure coincided with the perch kill. (**A**) Time series for the predation pressure from pike on perch in each basin of Windermere. (**B**) Effect of the perch pathogen on the link between number of pike spawners (SSB) and pike recruitment rate (i.e. natural log of number of age-2 recruits per spawner) in each basin of the lake (see also Table 1). Points represent observed data and lines represent predicted values with 95% confidence intervals. (**C**) Nonlinear temporal trends for perch somatic growth rate (in partial residuals units) with 95% bootstrap confidence intervals, accounting for the effects of environmental variation in growth conditions. Gray points represent the partial residuals for the smooth term (i.e. residuals that would be obtained by dropping the focal term from the model while leaving all other estimates fixed). Trends are provided separately for each sex and basin of the lake. (**D**) Effect of the perch pathogen on the link between perch density and pike recruitment rate (see also Table 1). Points represent observed data and lines represent predicted values with 95% confidence intervals.

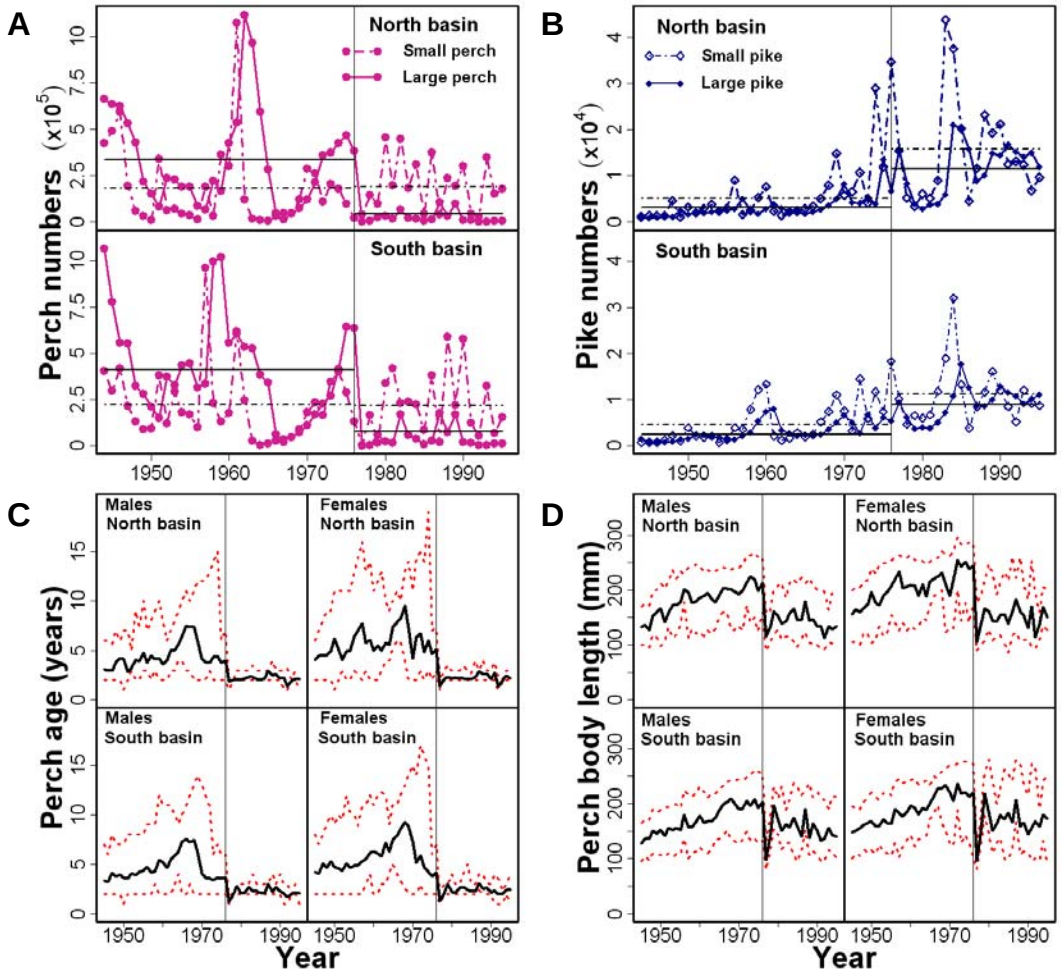
A
North basin
Small perch
Large perch
South basin
Perch numbers (x10^5)
B
North basin
Small pike
Large pike
South basin
Pike numbers (x10^4)
C
Males North basin
Females North basin
Males South basin
Females South basin
Perch age (years)
Year
D
Males North basin
Females North basin
Males South basin
Females South basin
Perch body length (mm)
Year

**Figure 1.**

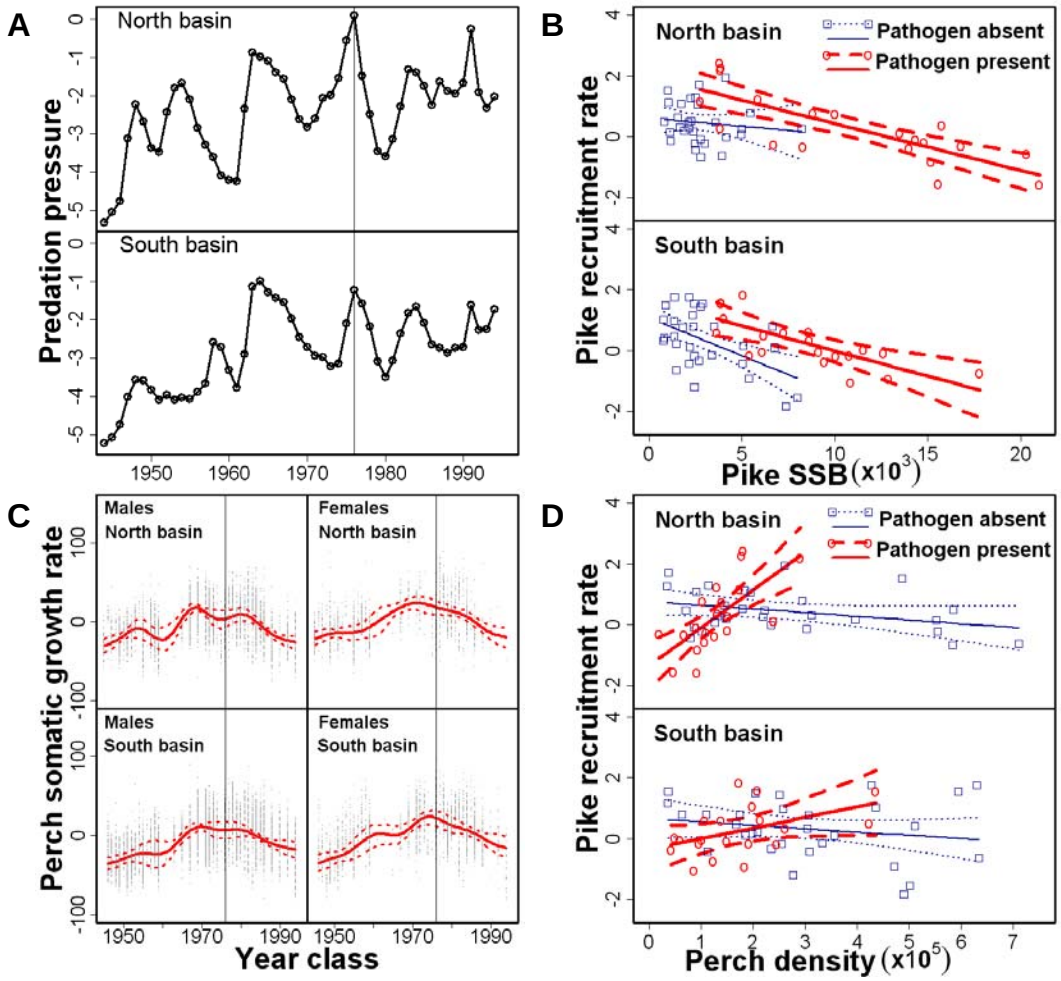
A
North basin
South basin
Predation pressure
1950
1960
1970
1980
1990
B
North basin
Pathogen absent
Pathogen present
South basin
Pike recruitment rate
Pike SSB (x10^3)
C
Males
North basin
Females
North basin
Males
South basin
Females
South basin
Perch somatic growth rate
Year class
D
North basin
Pathogen absent
Pathogen present
South basin
Pike recruitment rate
Perch density (x10^5)

**Figure 2.**

## Table legend

## Table 1. Model parameter estimates and their statistical significance (df: degrees of freedom, edf: estimated degrees of freedom of smooth term).

| Response | Effects | Estimate* | df (linear effect) or edf (smooth term) | *F* value | *P* value¶ |
|---|---|---|---|---|---|
| Perch body length (n = 67,457) | f(Age) | none | 1.992; 67,445.17 | 53593 | <0.0001 |
| | Basin (south relative to north) | 2.987 | 1; 67,445.17 | 249.97 | <0.0001 |
| | Sex (females relative to males) | 5.297 | 1; 67,445.17 | 566.25 | <0.0001 |
| | Temperature | 1.210 e+1 | 1; 67,441 | 2366.4 | <0.0001 |
| | Phosphorus | -8.071 | 1; 67,441 | 919.3 | <0.0001 |
| | Perch density | -3.688 e-5 | 1; 67,441 | 4277.9 | <0.0001 |
| | Phosphorus * Temperature | 7.016 | 1; 67,445.17 | 999.74 | <0.0001 |
| | Perch density * Temperature | -6.223e-06 | 1; 67,445.17 | 134.01 | <0.0001 |
| | f(Year class) | none | 4.749; 67,445.17 | 3877 | <0.0001 |
| Ln(Pike recruits/SSB), North basin (n = 50) | SSB (spawning stock biomass) | -1.517e-04 | 1,45 | 20.4 | <0.0001 |
| | Temperature | 2.860e-01 | 1,45 | 4.8 | 0.0344 |
| | Perch density | -7.819e-07 | 1,45 | 1.2 | 0.1259 |
| | Pathogen (presence/absence) | 8.704e-01 | 1,45 | 8.7 | 0.0051 |
| | SSB * Pathogen | -8.831e-05 | 1,44 | 1.1 | 0.3016 |
| | Perch density * Pathogen | 7.934e-06 | 1,44 | 7.7 | 0.0080 |
| Ln(Pike recruits/SSB), South basin (n=50) | SSB (spawning stock biomass) | -1.994e-04 | 1,45 | 16.6 | <0.0002 |
| | Temperature | 4.532e-01 | 1,45 | 6.1 | 0.0172 |
| | Perch density | -9.134e-07 | 1,45 | 4.2 | 0.0458 |
| | Pathogen (presence/absence) | 7.247e-01 | 1,45 | 5.6 | 0.0220 |
| | SSB * Pathogen | 7.568e-05 | 1,44 | 0.9 | 0.3577 |
| | Perch density * Pathogen | 1.273e-06 | 1,44 | 0.5 | 0.4985 |

* Parameter estimates for main effects are from models without interaction terms.
¶ Sequentially tested in case of stock-recruitment models.