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Fisheries-Induced Evolution in Cannibalism Promotes Collapses of Fish Populations

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Abstract

The understanding that evolutionary responses to fisheries can be rapid is supported by observational, experimental, and modeling studies. To date, however, research on fisheries-induced evolution has mainly focused on traits related to life histories. Selective changes in other traits, especially in those related to ecological interactions, are understudied. One such interaction that is especially frequent in fish populations is cannibalism. Potentially, cannibalism has important consequences for fisheries, due to the effects of cannibalism on population size-structure and dynamics. Here we study the eco-evolutionary dynamics of a cannibalistic fish population in response to increasing fishing mortality. Three observations are made: *i*) high rates of cannibalism lead to reduced fishing yield as a function of fishing mortality, *ii*) fishing mortality selects for increasing rates of cannibalism, which leads to population collapse at lower fishing mortalities and *iii*) cannibalism evolution undermines the rescue effect maturation evolution can offer in response to fishing mortality. These results are obtained by using a physiologically structured population model (PSPM), based on cannibalistic populations of Arctic char (*Salvelinus alpinus*). The PSPM describes the ecological and bioenergetic processes of char individuals as a function of their body size and the state of the environment. Selection pressures driving evolutionary change result from the feedback between population level resource use and autonomous resource renewal.

6.1 – INTRODUCTION

Fisheries impose high levels of size-selective mortality, which can lead to rapid evolutionary changes in the targeted fish stocks (Heino et al. 2015; Heino and Dieckmann 2009; Law 2000). Such fisheries-induced evolution can either work directly, promoting traits that increase the likelihood of successful reproduction before getting caught, or indirectly, by altering the environment under which certain traits have evolved and thus changing the optimal value of these traits. Research on contemporary evolutionary changes in fish stocks have mainly focused on changes in life-history traits that directly confer a selective advantage under a high mortality regime for large individuals. Such changes include decreasing size and age at maturation, slower growth rates and higher reproductive investments (Enberg et al. 2009, 2012; Heino and Dieckmann 2009; Marty et al. 2015). Theoretically these changes are understood from a life-history perspective that focuses on optimizing lifetime reproductive output, but such a perspective largely ignores the ecological feedback between the harvested population and its environment. This is unfortunate because potentially there are many traits that experience a change in selective pressure resulting from an indirect effect of fisheries through the altered ecological feedback loop.

Indirect evolutionary effects of fisheries-induced mortality can be expected to affect traits that are related to ecological interactions. Harvesting fish stocks inevitably reduces standing stock biomass and therefore alleviates competition between remaining individuals (Amundsen et al. 1993). Decreased competition can lead to increased growth and/or reproductive rates, which changes the population size distribution. In turn, the ecological feedback loop of the population with its environment will lead to changes in food availabilities (Amundsen et al. 2007; De Roos and Persson 2013). Resource profitability will shift and this potentially leads to an adaptive response in the traits related to feeding. Currently, research on fisheries-induced evolution has not considered selective changes in such indirectly selected ecological traits, as opposed to life-history traits.

An important ecological feedback commonly observed in fish species is cannibalism, which is regularly defined as the killing and eating of conspecifics (Elgar and Crespi 1992; Fox 1975; Polis 1981; Smith and Reay 1991). Cannibalism is mostly a size-dependent interaction where large individuals kill and eat small conspecifics. Because fish species often grow considerably in size during their life, they are especially prone to the effects of cannibalism. While many fish species are cannibalistic to some extent there is substantial variation in the importance of cannibalism for regulating population and community dynamics (Andersson et al. 2007; Claessen et al. 2000; Persson et al. 2000, 2003). Some species only suffer from egg cannibalism for which the energetic gains of the cannibals are limited, while in other stocks a major part of the diet in terms of biomass is derived from cannibalism (Persson et al. 2004).

Cannibalism in cod (*Gadus morhua*) can remove up to 40% of a cohort and contributes significantly to the diet of adult individuals (Andersen et al. 2016; Neuenfeldt et al. 2000). Moreover, even interpopulational differences exist, such that individuals from some populations are more prone to cannibalize than individuals of other populations of the same species (Griffiths 1994; Klemetsen et al. 2003). The mechanism behind this variation is currently not well understood, but the variation itself suggests that the cannibalistic behavior should be regarded as adaptive and possibly has a genetic basis (Amundsen et al. 1999; Wagner et al. 1999).

The population dynamical effects of cannibalism include population regulation, population (de)stabilization, bistability and changes in the size distribution of the population (reviewed in Claessen et al. 2004). Claessen et al. (2004) furthermore described four defining ecological aspects of cannibalism, namely victim mortality, energy extraction for the cannibal, size or age dependency and competition. However, these aspects of cannibalism are not in a straightforward way related to the population dynamical effects (Claessen et al. 2004). Therefore, to draw general conclusions about potential fisheries-induced evolution in cannibalism and thus arrive at sensible management strategies for cannibalistic fish populations the ecological and evolutionary effects of cannibalism should be studied in detail. This requires an ecological perspective that includes an accurate description of individual-level energetics and their dependence on body size and food availability, since such an approach provides the cornerstone for understanding the link between individual-, population-, and community-level dynamics (Persson et al. 2014).

Besides the need for an accurate ecological description of cannibalistic fish populations the different selection pressures acting on cannibalism should be considered. From an ecological point of view the evolution of cannibalism is predicted to depend on the profitability of cannibalistic prey in relation to the profitability of alternative prey types (Getto et al. 2005; Polis 1981). This profitability is determined both by prey availability and the (time) costs of handling and/or digesting prey. Getto et al. (2005) showed that the evolutionary onset of cannibalism in such an ecological context can be derived from an optimal foraging criterion, which states that the profitability of cannibalistic food should exceed the average intake of non-cannibalistic individuals. Hence, if handling times for the various food resources differ, the evolution of cannibalism can be inhibited if more profitable alternative prey items are available. However, the profitability of cannibalistic prey is often higher compared to alternative prey, since cannibalistic prey is readily available and biomass composition of prey and predator are very similar, if not identical, which results in high digestion efficiencies (Polis 1981). From an ecological point of view selection on cannibalism is therefore expected to be positive, as long as cannibalistic prey is readily available. It is this availability

of conspecific, as well as alternative prey that might be affected by fisheries-induced mortality and hence indirectly affect the evolution of cannibalism.

Here we use a modeling approach to study the effect of fisheries-induced mortality (increased mortality that mainly targets large individuals) on a cannibalistic fish population. Specifically, we ask the question how fisheries-induced mortality affects selection on the cannibalistic propensity of individuals and how this affects the fisheries yield curve. For this purpose a detailed ecological model of the life history of Arctic char (*Salvelinus alpinus* L.) is developed. This species is known to be highly cannibalistic, especially in the northern part of its range and is commercially targeted mainly by sports fishermen (Amundsen et al. 1999; Griffiths 1994; Klemetsen et al. 2003; Svenning and Borgstrøm 2005). The adopted model framework is that of physiologically structured population models (PSPMs: De Roos 1997; De Roos et al. 1992; Metz and Diekmann 1986), since this framework allows to incorporate both an accurate description of individual-level bioenergetics and the ecological feedbacks between char individuals and their environment. Size-dependency of the cannibalistic interaction is modeled according to observed size relationships between Arctic char cannibals and their victims. Cannibalizing conspecifics is assumed to increase mortality rates due to the acquisition of pathogens and parasites. We find that increased fisheries-induced mortality selects for increasing rates of cannibalism. This leads to reduced fishing yields and a population collapse at lower fishing mortality compared to non-cannibalistic populations or population with non-evolving cannibalistic rates. Furthermore, cannibalism evolution undermines the rescue effect that maturation evolution can offer in response to fishing mortality.

6.2 – Model and method

Model description

A physiologically structured population model (PSPM: De Roos 1997; De Roos et al. 1992; Metz and Diekmann 1986) describes ecological processes such as feeding, growth and reproduction on the level of the individual organism. These individual-level dynamics can depend on both the (physiological) state of the individual (*i*-state) and the state of the environment (*E*-state). The population-level dynamics are simply the collected action of all consumer individuals in the population, in addition to the dynamics of the environment. Hence, all biological assumptions pertain to the behavior and/or physiology of individuals, in addition to the specification of the dynamics of the environment in which these individuals live. For this particular PSPM we largely follow the approach and model specification as presented by Byström et al. (2004), Byström and Andersson (2005) and Claessen et al. (2000).

As a representative cannibalistic species Arctic char (*Salvelinus alpinus*) is chosen. This species is commercially harvested in some parts of its circumpolar distribution and has a variable lifestyle and ecology in which cannibalism is expressed in several instances (Froese and Pauly 2016; Klemetsen et al. 2003). Cannibalism is especially important for structuring high Arctic and alpine populations of Arctic char, in contrast to the more temperate ones (Griffiths 1994). The combination of variation in the extent of cannibalism and commercial fisheries makes Arctic char an ideal species for studying fisheries-induced evolution of cannibalism. In the PSPM, char individuals are distinguished by their body mass in grams, which is the variable that features as *i*-state. The environment consists of a zooplankton resource in the water column, which is available mainly for small individuals, and a benthic resource of macro-invertebrates from which all individuals can feed (Byström et al. 2004; Jansen et al. 2003). In addition to resource feeding, large char individuals are cannibalistic and feed on smaller char individuals. The total amount of food encountered by a char individual of mass w when searching for prey is given by:

$$\gamma_{\text{tot}}(w) = \gamma_{\text{z}}(w) + \gamma_{\text{m}}(w) + \gamma_{\text{c}}(w) \tag{6.1}$$

where $\gamma_{\text{z}}(w)$ is the zooplankton encounter rate, $\gamma_{\text{m}}(w)$ is the macrobenthic inverte-brate encounter rate and $\gamma_{\text{c}}(w)$ is the cannibalistic encounter rate. For simplicity we assume that char individuals can simultaneously search for all three food sources. The encounter rates for the two non-cannibalistic resources are the products of the attack rates for those resources and the resource densities:

$$\gamma_{\text{z}}(w) = a_{\text{z}}(w)\,R_{\text{z}} \tag{6.2a}$$

$$\gamma_{\text{m}}(w) = a_{\text{m}}(w)\,R_{\text{m}} \tag{6.2b}$$

where the R_{z} is zooplankton density in the water column in g m^{-3} and R_{m} is benthic macro-invertebrate density in g m^{-2}. Feeding on zooplankton is a volume related processes, while benthic feeding is a surface related process. The resource attack rates are size-dependent functions and given by:

$$a_{\text{z}}(w) = A\left(\frac{w}{w_0}\exp\left(1 - \frac{w}{w_0}\right)\right)^{\alpha} \tag{6.3a}$$

$$a_{\text{m}}(w) = \chi_1 w^{\chi_2} \tag{6.3b}$$

The attack rate for the zooplankton resource is a hump-shaped function of body mass and is derived from foraging experiments with differently-sized char individuals feeding on a zooplankton prey (Byström et al. 2004; Jansen et al. 2003). In this function the parameter A represents the maximum attack rate in m^3 day^{-1} which is

attained at a body size of w_0 grams. The steepness of the hump-shaped function with increasing body size is controlled by the parameter α. Equation (6.3a) has been shown to be an appropriate way of modeling zooplanktivory in several other fish species such as cod, roach and perch (Claessen et al. 2000; Persson et al. 1998) and has become a well-established function in studies of fish population dynamics (De Roos and Persson 2013). Byström and Andersson (2005) show that the attack rate for benthic macro-invertebrate feeding is best described by a power law function of char body mass (equation (6.3b)), in which χ_1 is the attack rate constant in $\mathrm{m}^2 \, (\mathrm{g}^{\chi_2} \, \mathrm{day})^{-1}$ and χ_2 the attack rate scaling exponent.

The cannibalistic encounter rate $\gamma_c(w)$ describes the rate at which a single char individual of body mass w encounters suitably sized prey conspecifics when searching for prey. This rate is the product of the allometric attack rate function $v(w) = \beta w^\theta$, which describes the scaling of the cannibalistic attack rate with predator body mass w, and a cannibalism interaction kernel, which models the dependency of the cannibalistic encounter rate on prey availability in biomass and predator catch success. The prey availability is the total prey biomass weighted with the vulnerability of prey to cannibalism. The predator catch success for cannibalistic fish species has been shown to be maximal at predator and prey sizes that are positively correlated (see Claessen et al. 2000, and references therein). To allow for this effect the cannibalistic interaction kernel is a weighted sum of three separate interaction kernels that each describe the probability that a cannibal of length l^c will catch a prey (victim) of length l^v upon encounter. Each of these three interaction kernels is the product of a prey vulnerability function, which is assumed to be a Gaussian function of prey body size in length l^v with mean μ^v and standard deviation σ^v, $N_v\left(l^v | \mu_i^v, \sigma_i^v\right)$, and a predator success rate function, which is assumed to be a Gaussian function of predator body size in length l^c with mean μ^c and standard deviation σ^c, $N_c\left(l^c | \mu_i^c, \sigma_i^c\right)$. The three interaction kernels differ in their values for μ^v, μ^c, σ^v and σ^c, while the contribution of each kernel is weighted with probability p_i. To transform body size in length l to body size in weight w the length-weight relationship $l(w) = \lambda_1 w^{\lambda_2}$ is used. The cannibalistic encounter rate for a char individual with body mass w is thus given by:

$$\gamma_c(w) = v(w) \sum_{i=1}^{3} p_i N_c\left(l(w) | \mu_i^c, \sigma_i^c\right) \int_0^\infty N_v\left(l(y) | \mu_i^v, \sigma_i^v\right) y \, n(y) \, \mathrm{d}y \qquad (6.4)$$

Here y is the body mass of prey individuals and $n(y)$ is the population size distribution.

Besides searching for prey the char individuals are assumed to spend time digesting or handling prey items and this process ultimately sets the upper level of food intake when prey availability is high. Handling times for Arctic char are reported in Byström et al. (2004) and follow a power law relationship with body mass $h(w) = \xi_1 w^{-\xi_2}$.

Total ingested food $I(w)$ is then given by Hollings disc equation:

$$I(w) = \frac{\gamma_{\text{tot}}(w)}{1 + h(w)\gamma_{\text{tot}}(w)} \qquad (6.5)$$

The ingested food is assimilated with efficiency σ_a, which gives the assimilated energy $E_a(w) = \sigma_a I(w)$. Maintenance costs $E_m(w)$ are modeled with a power law function of body size:

$$E_m(w) = m_1 w^{m_2} \qquad (6.6)$$

We follow a net-production dynamic energy budget model, which means that maintenance costs are paid from assimilated energy before this can be used for growth and/or reproduction (Lika and Nisbet 2000; Enberg 2012). Growth and/or reproduction are thus dependent on surplus energy production, which is the difference between the rate of energy assimilation and the maintenance rate: $E_g(w) = E_a(w) - E_m(w)$. Juveniles with $w < w_f$ invest all surplus energy into growth, while adults ($w \geq w_f$) invest a constant fraction κ of the surplus energy into growth and $(1 - \kappa)$ into reproduction. Growth $g(w)$ is then given by:

$$g(w) \begin{cases} \max\left(\kappa E_g(w), 0\right) & \text{if } w \geq w_f \\ \max\left(E_g(w), 0\right) & \text{otherwise} \end{cases} \qquad (6.7)$$

and the reproductive rate $b(w)$, or the number of offspring produced per unit time is given by:

$$b(w) = \begin{cases} s_e \sigma_r (1 - \kappa) E_g(w) w_b^{-1} & \text{if } w \geq w_f \\ 0 & \text{otherwise} \end{cases} \qquad (6.8)$$

where w_b is the size at birth, σ_r is the reproductive efficiency and s_e is the egg survival.

Total mortality rate is composed of size-dependent background mortality rate $\mu_0 + \mu_1 \exp(-w/w_m)$, mortality due to cannibalism $\mu_e(w)$, costs of cannibalism $\mu_c(w)$ and harvesting mortality $\mu_F(w)$. Mortality due to cannibalism for an individual of mass w depends on the chance of falling victim to a cannibalistic individual of size y. This is a function of the predator catch success and the vulnerability of the prey, in addition to the number of predators and the value of their functional response:

$$\mu_e(w) = \sum_{i=1}^{3} p_i N_v \left(l(w)|\mu_i^v, \sigma_i^v\right) \int_0^\infty \frac{\upsilon(y) N_c \left(l(y)|\mu_i^c, \sigma_i^c\right)}{1 + h(y)\gamma_{\text{tot}}(y)} n(y) \, dy \qquad (6.9)$$

In addition there are mortality costs related to cannibalism because we assume that eating cannibalistic prey increases mortality rates. These increased mortality risks are a phenomenological representation of the chance of acquiring pathogens or parasites that reduce survival. Attracting pathogens and parasites has been shown to be a

negative side-effect of cannibalism that can potentially hinder or stabilize the evolution of cannibalism (Polis 1981). Cannibalistic survival costs are assumed proportional to the ingested biomass of cannibalistic prey with proportionality constant s_c:

$$\mu_c(w) = \frac{s_c \gamma_c(w)}{1 + h(w)\gamma_{tot}(w)} \tag{6.10}$$

Harvesting mortality mainly targets individuals that exceed the threshold body mass w_h, but smaller individuals can still be targeted as accidental by-catch. This is captured by the size-dependent harvesting function:

$$\mu_F(w) = \frac{\mu_h}{1 + e^{-w+w_h}} \tag{6.11}$$

Total mortality rate is then given by:

$$\mu(w) = \mu_0 + \mu_1 \exp\left(-\frac{w}{w_m}\right) + \mu_e(w) + \mu_c(w) + \mu_F(w) \tag{6.12}$$

Resource dynamics result from an autonomous resource renewal process and the foraging of all the individuals in the population. Following Claessen et al. (2000) and Byström et al. (2004), the resource growth is modeled as a semi-chemostat process, such that resource growth rate linearly decreases from a maximum resource growth rate at low resource densities. Resource foraging is given by the integral of the population size distribution weighted by the resource ingestion rates:

$$\frac{dR_z}{dt} = r_z(K_z - R_z) - \int_0^\infty \frac{\gamma_z(w)}{1 + h(w)\gamma_{tot}(w)} n(w)\,dw \tag{6.13}$$

$$\frac{dR_m}{dt} = r_m(K_m - R_m) - \int_0^\infty \frac{\gamma_m(w)}{1 + h(w)\gamma_{tot}(w)} n(w)\,dw \tag{6.14}$$

Population dynamics of the char consumers are summarized in the following partial differential equation:

$$\frac{\partial n(w)}{\partial t} + \frac{\partial g(w)n(w)}{\partial w} = -\mu(w)n(w) \tag{6.15}$$

This partial differential equation describes that growth $g(w)$ leads to displacement of mass along the body size axis and mortality $\mu(w)$ decreases the population size distribution. Equation 6.15 is accompanied by a boundary condition describing the inflow of newborn individuals at $w = w_b$:

$$g(w_b)n(w_b) = \int_0^\infty b(w)n(w)\,dw \tag{6.16}$$

Model parameterization

Model parameters are summarized in table 6.1. Parameters for the resource feeding of char (A, α, w_0, χ_1 and χ_2) are taken from Byström and Andersson (2005) and P. Byström (*pers. comm.*) who parameterized the attack rates of differently-sized char individuals on both zooplankton and macrobenthic invertebrate by means of foraging experiments. For the transformation of attack rates from the experimentally measured rates per second to those used in the model per day, it was assumed that char individuals forage for 12 h per day (Byström and Andersson 2005). Handling time parameters (ξ_1 and ξ_2) were derived by Byström et al. (2004) from data in Jobling et al. (1993) on growth rates of Arctic char under *ad libitum* food conditions. The latter author also provided the parameter values for the maintenance rate constant (m_1) and exponent (m_2). The cannibalism attack rate constant β is varied during analysis and the allometric scaling exponent θ is set to 0.64 following Claessen et al. (2000). Assimilation efficiency σ_a includes the conversion of food to assimilated biomass and also costs for specific dynamic action. The estimate of $\sigma_a = 0.61$ from Persson et al. (1998) for the zooplanktivore roach (*Rutilus rutilus*) is adopted here for char. The value for efficiency of converting body mass into newborn mass, $\sigma_r = 0.5$, is also adopted from Persson et al. (1998). Size-independent background mortality rate μ_0 is derived from Vøllestad and L'Abée-Lund (1994) who provided estimates of natural mortality rates of different char populations. Their mean value of 0.0017 day^{-1} is adopted here as default value for μ_0. In addition, because small fish are often thought to be more at risk of mortality than larger individuals, char are assumed to suffer from a mortality rate that decreases with size. No estimates for size-dependent mortality could be found from the literature so values of $\mu_1 = 0.02$ and $w_m = 10$ were adopted as default. Egg survival probability s_e is set be 0.1 and the cost of cannibalism is set to $s_c = 0.002$. This latter parameter scales the costs of cannibalistic feeding in terms of additional mortality. For low values of s_c positive directional selection on the cannibalistic rate will lead to ever higher values of cannibalism. On the other hand, too high values of s_c will lead to negative directional selection towards non-cannibalistic individuals. Preliminary model analysis showed that $s_c = 0.002$ leads to stabilizing selection on the cannibalistic rate at zero fisheries-induced mortality, which is the appropriate default setting for studying the effect of fisheries-induced mortality on the evolution of the cannibalistic rate. The fraction of energy allocated to post-maturation growth, κ, is set default to 0.6. Parameters related to resource growth are all taken from Byström et al. (2004) and the length-weight relationship parameters were kindly supplied by P. Byström (*pers. comm.*).

Parameters used in the cannibalistic interaction kernel are derived from stomach content data described in Amundsen (1994); Finstad et al. (2001); Hammar (1998); Malmquist et al. (1992) and kindly provided by P. A. Amundsen and P. Byström (*pers.*
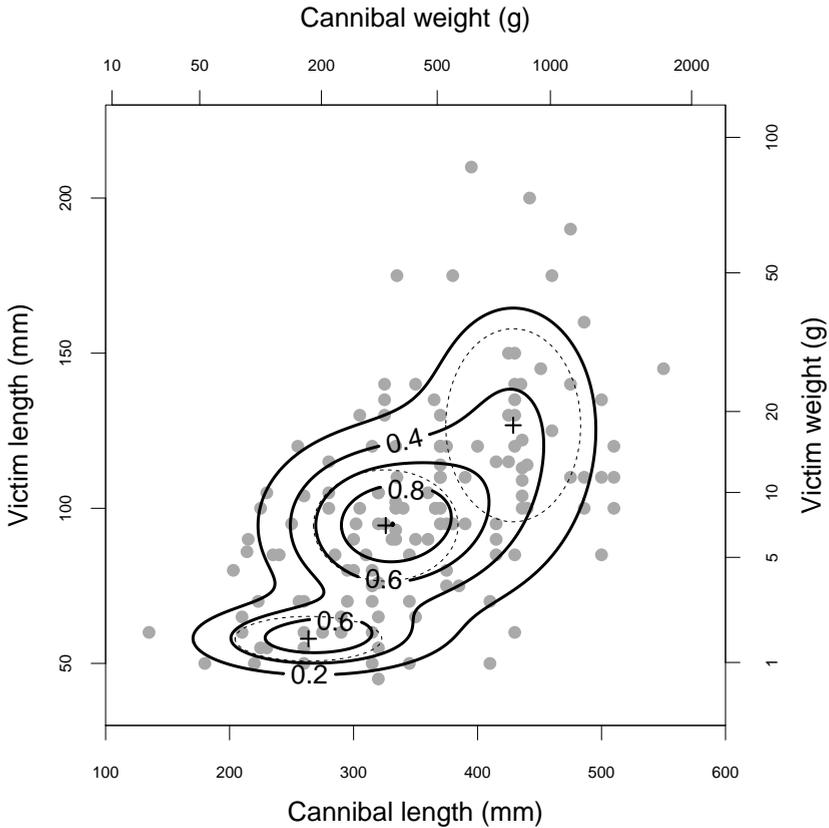
FIGURE 6.1 – Observed relationship between cannibal length (mm) and victim length (mm) for Arctic char (*Salvelinus alpinus*). Data from Malmquist et al. (1992), Hammar (1998), Finstad et al. (2001) and Amundsen (1994) and kindly provided by P. A. Amundsen and P. Byström. The '+'-symbols and dashed contour lines indicate the means and standard deviations of the three bivariate Gaussian functions. The solid contour lines are the linear combinations of these three Gaussian functions weighted with probabilities 0.15, 0.51 and 0.34 for bottom left, middle and upper right Gaussian function, respectively (table 6.1).

comm.). These data represent pairs of cannibal and victim length observations and give an indication of the size-dependency of the cannibalistic interaction. A total of 135 observations were obtained. A Gaussian bivariate mixture model is used to obtain density estimates of this cannibalistic interaction kernel (Fraley and Raftery 2002). The mixture model allowed for differences in variance between the different Gaussians while using a diagonal covariance structure. We used the Bayesian information criterion (BIC) to select the appropriate number of clusters. This resulted in three bivariate Gaussian distributions which each give the incidence probability of a canni-

TABLE 6.1 – Model Parameters

Parameter	Value	Unit	Interpretation	Reference
w_b	0.057	g	Size at birth	P. Byström *unpublished results*
w_f	Varied	g	Size at maturation	this study
w_0	7.15	g	Size at maximum zooplankton attack rate	Byström and Andersson 2005
w_m	10	g	Size scaling size-dependent mortality	this study
w_h	10 or 100	g	Size scaling fisheries-induced mortality	this study
α	0.65	–	Zooplankton attack rate scaling	Byström and Andersson 2005
A	14	$\mathrm{m^3\ day^{-1}}$	Maximum zooplankton attack rate	Byström and Andersson 2005
χ_1	0.6	$\mathrm{m^2\,(g^{\chi_2}\ day)^{-1}}$	Macrobenthos attack rate constant	Byström and Andersson 2005
χ_2	0.3	–	Macrobenthos attack rate scaling	Byström and Andersson 2005
ξ_1	5.3	$\mathrm{day\ g^{-(1+\xi_2)}}$	Handling/digestion time constant	Byström et al. 2004; Jobling et al. 1993
ξ_2	-0.66	–	Handling/digestion time exponent	Byström et al. 2004; Jobling et al. 1993
m_1	0.06	$\mathrm{g^{(1-m_2)}\ day^{-1}}$	Maintenance scaling constant	Jobling et al. 1993; P. Byström *unpublished results*
m_2	0.63	–	Maintenance scaling exponent	Jobling et al. 1993; P. Byström *unpublished results*
β	Varied	$\mathrm{m^3\ day^{-1}\ g^{-\theta}}$	Cannibalism maximum attack rate	this study
θ	0.64	–	Cannibalistic size-scaling	Claessen et al. 2000
σ_a	0.61	–	Assimilation efficiency	Persson et al. 1998

Continues on next page

Table 6.1 – *Continued from previous page*

Parameter	Value	Unit	Interpretation	Reference
σ_r	0.5	–	Gonad to egg conversion efficiency	Persson et al. 1998
μ_0	0.0017	day^{-1}	Size-independent background mortality	Vøllestad and L'Abée-Lund 1994
μ_1	0.02	day^{-1}	Size-dependent background mortality	this study
μ_h	Varied	day^{-1}	Harvesting mortality scalar	this study
s_e	0.1	–	Egg survival probability	this study
s_c	0.002	g^{-1}	Cost of cannibalism	this study
κ	0.6	–	Allocation constant	this study
λ_1	50.3	mm g$^{-\lambda_2}$	Length-weight scalar	P. Byström *pers. comm.*
λ_2	0.32	–	Length-weight exponent	P. Byström *pers. comm.*
r_z	0.05	day^{-1}	Zooplankton growth rate	Byström et al. 2004
r_m	0.05	day^{-1}	Macrobenthos growth rate	Byström et al. 2004
K_z	0.1	g m^{-3}	Maximum zooplankton density	Byström et al. 2004
K_m	3.0	g m^{-2}	Maximum macrobenthos density	Byström et al. 2004
μ_i^v	58, 95, 127	mm	Means of prey vulnerability	this study
μ_i^c	264, 328, 431	mm	Means of predator catch success	this study
σ_i^v	7.30, 18.08, 31.21	mm	Standard deviation of prey vulnerability	this study
σ_i^c	59.22, 59.06, 53.83	mm	Standard deviation of predator catch success	this study
p_i	0.15, 0.51, 0.34	–	Proportion of Gaussian i	this study

balistic event given the vulnerability to cannibalism as a function of victim length and the catch success as function of cannibal length. The mean and standard deviation of Gaussian i for victim vulnerability are indicated with μ_i^v and σ_i^v respectively, and similarly for cannibal catch success with μ_i^c and σ_i^c, respectively. The probability of a certain predator-prey length point belonging to Gaussian i is indicated with p_i. The predator-prey pairs length data together with the contour lines of the cannibalism interaction kernel are shown in figure 6.1.

Evolutionary dynamics

Evolutionary dynamics are studied by using the framework of adaptive dynamics (Dieckmann and Law 1996; Geritz et al. 1998; Metz et al. 1995). This framework assesses the invasion success of mutant phenotypes in populations exhibiting ecological dynamics that are determined by the resident phenotype. Mutations are assumed to be small and the ecological dynamics are assumed to occur on a faster timescale than the evolutionary process. Adaptive dynamics hence assumes that the ecological dynamics have reached their stable, long-term attractor (e.g., equilibrium or limit cycle) before the next mutation occurs. Evolutionary endpoints or evolutionary singular strategies (Geritz et al. 1998) can be detected by following the selection gradient in trait space until the selection gradient on all evolutionary traits vanishes. Adaptive dynamics therefore can be used to identify evolutionary endpoints and how these evolutionary endpoints depend on other model parameters. However, heritability is not taken into account and adaptive dynamics therefore does not give realistic predictions about how fast these evolutionary endpoints are reached.

Model analysis

The parameters that are allowed to evolve in this model are β, the scalar of cannibalistic attack rate and w_f, the size at maturation. Evolutionary dynamics of these two parameters are studied for varying levels of the fishing mortality constant μ_h and for two values of the minimum size threshold for fishing w_h. The effect of increasing fishing mortality on ecological dynamics is explored for different levels of cannibalism and minimum size threshold for fishing. Subsequently we detect and continue the evolutionary equilibrium of the cannibalistic rate β as a function of the fishing mortality constant μ_h, for different values of the size at maturation (w_f) and the minimum size threshold for fishing (w_h). Finally, we compare the response to increasing fishing mortality between two scenarios; one of joined maturation and cannibalism evolution (both β and w_f evolve) and the other with only maturation evolution and a fixed cannibalistic rate.

Model analysis is carried out with PSPManalysis, which is a freely available software package specifically designed for demographic, equilibrium and evolutionary

analysis of physiologically structured population models (De Roos 2016). The package contains numerical routines for equilibrium computation and numerical curve continuation as described in (De Roos et al. 2010; Diekmann et al. 2003; Kirkilionis et al. 2001). In addition, during curve continuation it detects and classifies evolutionarily singular strategies according to the theory as presented by Geritz et al. (1998) and allows for following selection gradients in parameter space by solving the canonical equation of adaptive dynamics (Dieckmann and Law 1996; Durinx et al. 2008).

The PSPManalysis package is an equilibrium continuation tool that assumes the ecological dynamics to be an equilibrium point. However, more complex ecological dynamics such as periodic fluctuation (limit cycles) and chaotic dynamics have frequently been observed in population models that include cannibalism (Claessen et al. 2004). We therefore assess the stability and nature of the ecological attractor by using the Escalator Boxcar Train method (De Roos 1988; De Roos et al. 1992). With this method we confirmed that all the equilibria as calculated by the PSPManalysis are indeed stable point equilibria.

6.3 – Results

High rates of cannibalism lead to reduced fishing yield as a function of fishing mortality

The consequences of an increased cannibalistic rate on the ecological dynamics are twofold: total population (biomass) density decreases and the maximum size of the individuals within the population increases (figure 6.2). The decrease in population density is caused by increased mortality from cannibalism that reduces age-specific survival rates. The reduction of population density reduces the impact on the zooplankton and macrobenthos resources and hence relaxes resource competition. Therefore, the individuals that do not fall victim to cannibalism profit both from cannibalizing smaller individuals and from increased resource densities. This allows the surviving individuals to grow faster and to larger sizes than individuals in non-cannibalistic populations.

Figure 6.2 (bottom panels) show that increasing the rate of cannibalism leads to lower fisheries yield as a function of fishing mortality. However, this result is dependent on the minimum size threshold for fishing (w_h). For $w_h = 10$ (figure 6.2; left panels), the highest yield is achieved when the cannibalistic rate is nearly zero ($\beta = 0.0001$) and the yield decreases with increasing β, irrespective of the fisheries-induced mortality (μ_h). For $w_h = 100$ (figure 6.2; right panels) the yield is always highest for $\beta = 0.2$, intermediate for $\beta = 1$ and $\beta = 0.0001$ and lowest for $\beta = 5$. Population extinction only occurs in case the minimum size threshold is below the size at maturation ($w_f < w_h$; figure 6.2 left panels). For a high minimum size threshold the increased fisheries-induced mortality is applied only to large individuals that have
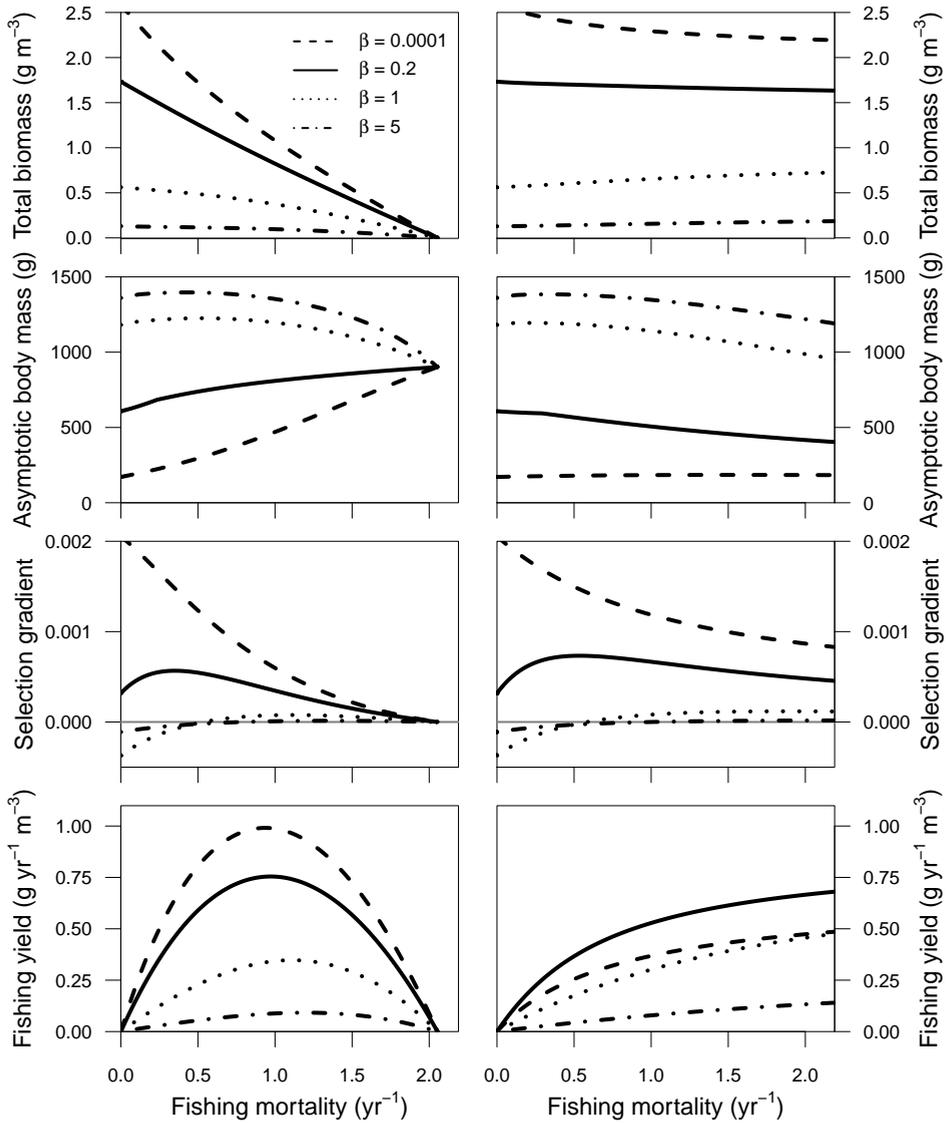
FIGURE 6.2 – Population equilibrium response to increasing fishing mortality (μ_h; on x-axis) mainly targeting individuals above 10 g (w_h = 10 g; left panels) or mainly targeting individuals above 100 g (w_h = 100 g; right panels). Different lines represent different levels of cannibalism (see plot legend in top left panel). Top row panels show total population biomass in g m^{-3}, second row panels show the asymptotic body mass in g, third row panels show selection gradient on the cannibalistic rate constant (β) and bottom row panels show fishing yield in g yr^{-1}m^{-3}. All other parameters are at their default value (table 6.1)

a negligible contribution to the population birth rate. Population biomass densities therefore stay approximately constant or only slightly decrease when cannibalistic rates are low. For a higher cannibalistic rate ($\beta = 1$; figure 6.2; right panel), population biomass density even slightly increases due to the increased survival that results from the increased fishing of the cannibalistic individuals.

Fishing mortality selects for increasing rates of cannibalism, which leads to population collapse at lower fishing mortalities

Figure 6.2 (third row panels) shows the selection gradient on the cannibalistic rate constant β. For low values of the fishing mortality constant (μ_h) the selection gradient is negative for high cannibalistic rates and positive for low cannibalistic rates. Hence, in this region the selection on the cannibalistic rate is stabilizing. This changes into positive directional selection for high cannibalistic rates at higher values of μ_h. The selection gradient on β is still positive, although small, for $\beta = 5$. Top panels of figure 6.3 show the evolutionary equilibrium value of β as a function of the fishing mortality constant μ_h. This is the value of β for which the selection gradient on β equals zero. Increasing the fisheries-induced mortality constant μ_h leads to an increase in the evolutionary equilibrium value of β, up to an asymptote at around $\mu_\mathrm{h} = 0.85$ for $w_\mathrm{h} = 10$ (figure 6.3; left panel) and around $\mu_\mathrm{h} = 1.25$ for $w_\mathrm{h} = 100$ (figure 6.3; right panel). Beyond these points the selection on the cannibalistic rate is no longer stabilizing, but strictly positive.

The increasing evolutionary equilibrium value of β with increasing fisheries-induced mortality rates causes the population to be driven down both by the direct mortality of fishing and by the increasing rates of cannibalism. Comparing the scenario with cannibalism evolution (figure 6.3) to the scenario with different, but fixed cannibalistic rates (figure 6.2) shows that, for $w_\mathrm{h} = 10$, the point of population extinction is at a much lower value for the fishing mortality constant μ_h when the cannibalistic rate evolves. More strongly, for $w_\mathrm{h} = 100$, cannibalism evolution leads to population extinction at a fishing mortality constant of $\mu_\mathrm{h} \approx 1.2$ (figure 6.3; right panels), while population extinction does not occur for any reasonably levels of fishing mortality in case cannibalistic rates are fixed (figure 6.2 right panels).

Cannibalism evolution undermines the rescue effect maturation evolution can offer in response to fishing mortality.

Figure 6.4 explores the population response to increased fishing mortality by comparing three scenarios: *i*) fixed values of size at maturation and cannibalism (left panels figure 6.4), *ii*) only evolution in the size at maturation with fixed values of the cannibalistic rate (middle panels figure 6.4), *iii*) evolution in both the size at maturation and the cannibalistic rate (right panels figure 6.4). The fixed parameters are in all cases
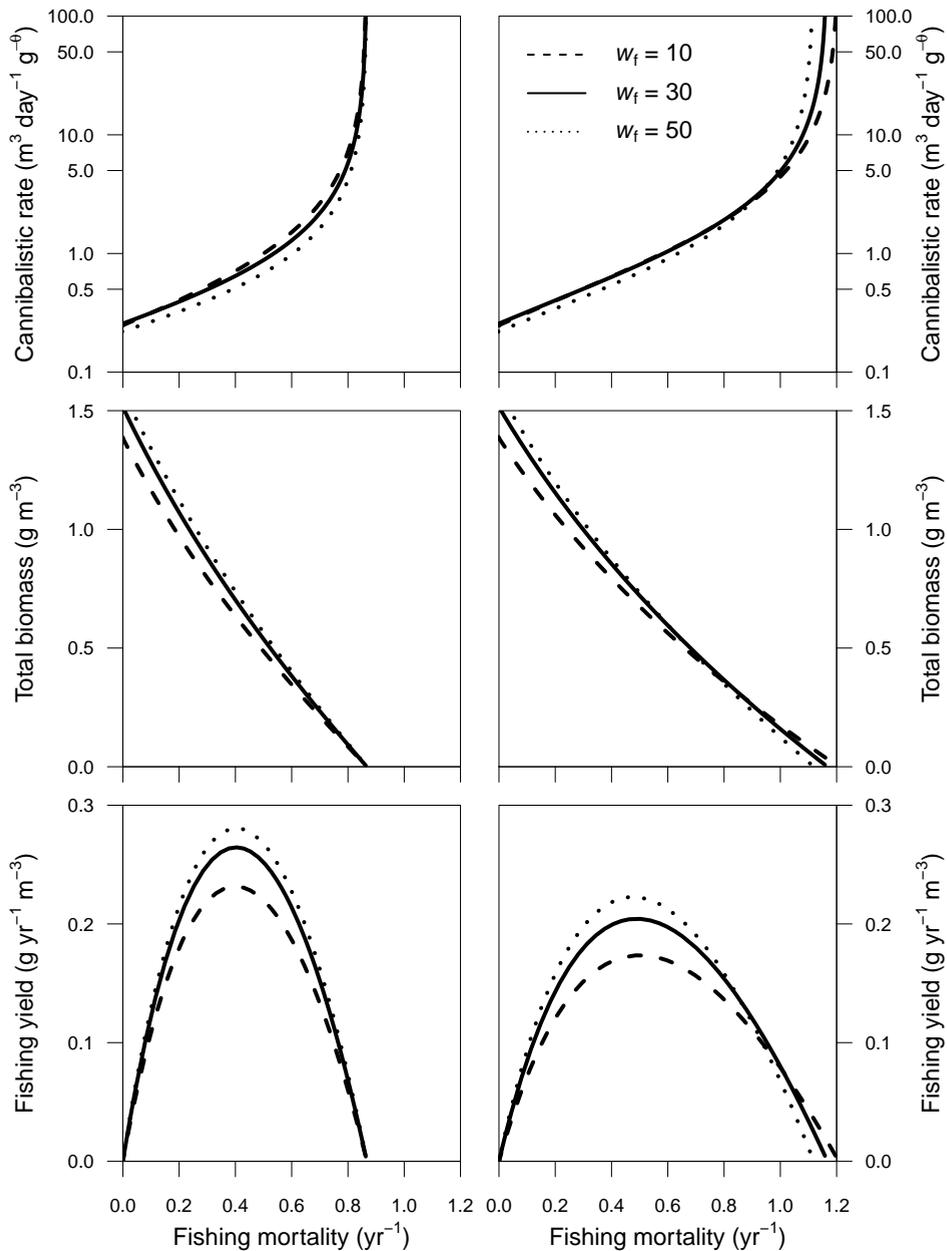
Figure 6.3 – Increasing fishing mortality (μ_h; on *x*-axis) leads to an increase in the evolutionary equilibrium of the cannibalistic rate constant (β), shown in top panels. Middle panels show total population biomass density and bottom panels show the fishing yield. In the left panels fishing mainly targets individuals above 10 g ($w_h = 10$ g) and in the right panels fished individuals are mainly above 100 g ($w_h = 100$ g). The different lines indicate different values for the size at maturation (w_f), shown in the legend in the top right panel.

fixed at their evolutionary equilibrium value for zero fishing mortality. Comparing the solid lines in the left and middle panels reveals that maturation evolution can offer a rescue effect and move the fishing mortality threshold at which population extinction occurs to higher values. This is achieved through an evolutionary response of the size at maturation to values below the minimum threshold size for fishing. This evolutionary rescue effect only occurs for $w_h = 10$, since at $w_h = 100$ there is no population extinction. Maturation evolution, however, does slightly decrease the yield curve for $w_h = 100$. Comparing the middle and right panels reveals that the rescue effect of maturation evolution is nullified when the cannibalistic rate also evolves. Similar to figure 6.3, the evolutionary equilibrium value of β increases rapidly with increasing fishing mortality and the population is driven down by both an increase in the rate of cannibalism and by the direct effect of the increased fishing mortality. Maturation evolution is unsuccessful in preventing this process.

6.4 – Discussion

By means of a physiologically structured population model (PSPM: De Roos 1997; De Roos et al. 1992; Metz and Diekmann 1986) we explore how cannibalism modifies the impact of fishing mortality on populations of Arctic char. For constant resource productivity, increasing cannibalism leads to declining population densities, while the surviving individuals experience increased growth rates and reach large asymptotic sizes. These ecological effects of cannibalism have been described before (Claessen and De Roos 2003; Claessen et al. 2000; Van Kooten et al. 2007). We show that high rates of cannibalism lead to decreasing fisheries yields, irrespective of the imposed fishing mortality. Furthermore, allowing for the evolution of cannibalism drastically alters the ecological response to fishing. When fisheries-induced mortality increases, the selection on cannibalistic rate changes from stabilizing selection to positive directional selection. The evolutionary response to high fishing mortality leads to increasing rates of cannibalism and this causes an additional decrease in population densities, next to the direct effect of fishing. Consequently, allowing for evolution of cannibalism substantially decreases the fishing mortality threshold at which the population goes extinct. Maturation evolution towards lower sizes at maturation is shown to increase population persistence at high fishing mortality, but only when cannibalistic rates do not evolve. Cannibalism evolution nullifies this rescue effect of maturation evolution.

Increasing fishing mortality changes the stabilizing selection on the cannibalistic rate that occurs at no or low exploitation levels into positive directional selection. This change in selection drives the rapid increase in cannibalistic rate and the concomitant decrease in population biomass. The stabilizing selection on cannibalism at low fishing mortality is a consequence of the trade-off between the gain of cannibalism
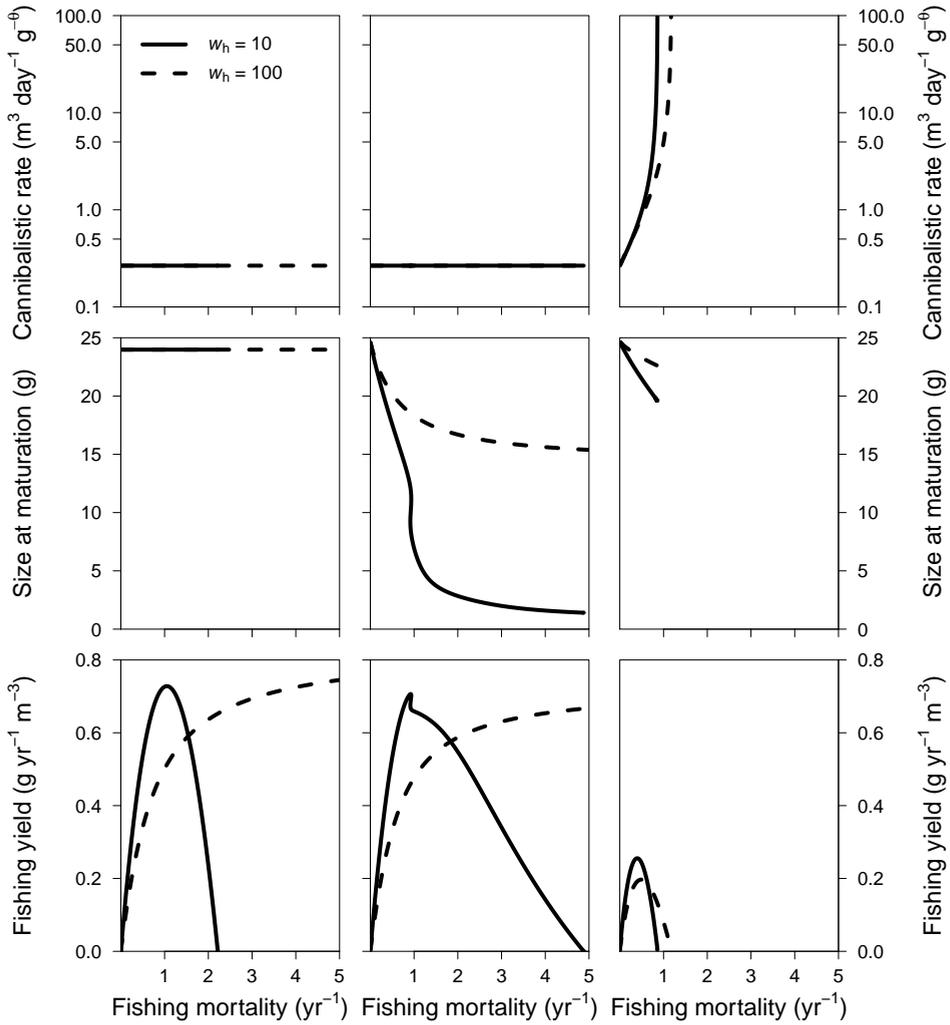
FIGURE 6.4 – Cannibalism evolution nullifies the evolutionary rescue effect of evolution in the maturation size threshold. Left panels show the response to increasing fishing mortality (μ_h; on the x-axis) for a fixed size at maturation and a fixed cannibalistic rate. Both parameters are fixed at their evolutionary equilibrium value for $\mu_h = 0.0$. Middle panels show the response in case of evolution only in the size at maturation with β fixed at its evolutionary equilibrium value at $\mu_h = 0.0$. Right panels show the response of joined evolution in the size at maturation and the cannibalism rate constant β. All other parameters are at their default value (table 6.1). For $w_h = 10$ there is an evolutionary rescue effect of the evolution in maturation size that delays the population extinction point to higher μ_h-values (compare solid lines in left and middle panels). This effect is nullified in case cannibalism evolution also occurs (solid lines in middle and right panels). For $w_h = 100$ (dashed lines), there is no population extinction, so there cannot be an evolutionary rescue effect. Cannibalism evolution also in this case leads to population extinction at low fishing mortality (dashed line in right panels)

in terms of extra ingested resources and the increased mortality that results from the cannibalistic feeding. Increasing fishing-induced mortality on large individuals disrupts the balancing selection on the cannibalistic rate and favors ever higher rates of cannibalism. Because survival is an exponentially decreasing function of the mortality rate, it decreases much faster with an increase in mortality rates when mortality rates are low, compared to a similar increase in mortality rates when mortality rates are high. The additional mortality costs of increasing cannibalism in terms of a reduction in the expected remaining lifespan of the cannibal, are therefore larger at low fishing mortality rates, than when fishing mortality rates are high. Therefore, cannibalizing conspecifics mainly increases resource intake rates and only marginally reduces survival at high fisheries-induced mortality.

Studies on fisheries-induced evolution aim to link high mortality rates imposed by fisheries to evolutionary changes in traits that affect the probability of individuals of getting caught by fisheries. Fisheries mainly target large and mature individuals and this imposes direct selection pressure for faster life histories, which means earlier maturation at a smaller size, increased reproductive investment and decreased post-maturation growth (Heino et al. 2015). Currently, changes in the maturation schedule form the most prominent evidence for the occurrence of fisheries-induced evolution. In addition, changes in behavior are certainly known to affect the susceptibility of individuals to fishing, but it is proven difficult to link this to fisheries-induced evolution in wild populations (Heino et al. 2015). Selection on behavioral traits can either be direct by selecting individuals that display a certain type of behavior, or indirect by changing the conditions under which the particular behavior has evolved (Heino et al. 2015). The tendency to cannibalize is an example of a trait on which fishing mortality exerts indirect selection, as fishing mortality devaluates individual survival and thereby disrupts the balancing selection on cannibalistic rate. Currently, there are few examples of indirect selection on behavioral traits due to fishing mortality, as most behavioral traits are either directly selected for (e.g. selection on bold or active individuals: Biro and Post 2008; Diaz Pauli et al. 2015; Klefoth et al. 2012) or behavior correlates with other traits on which selection acts (e.g. selection on growth alters swimming performance and vulnerability to predation in Atlantic Silverside *Menidia menidia*: Chiba et al. 2007; Lankford Jr. et al. 2001). Probably there are many traits on which fishing can exert indirect selection, but the tendency to cannibalize can be considered especially important since high rates of cannibalism severely impact stock productivity and fisheries production.

In this study, the stabilizing selection on the cannibalistic voracity is derived from a trade-off between energetic gains which increase growth and/or reproduction and additional mortality costs. These mortality costs of cannibalism are central to the results presented here, as without them positive selection for higher rates of canni-

balism occurs under all circumstances. A frequently proposed cost of cannibalism is the increased chance of acquiring parasites or pathogens from cannibalistic prey due to host specificity and resistance to host immune systems (Pfennig et al. 1998, 1991). Other costs are the risk of getting injured during cannibalistic feeding (Polis 1981), but this cost is not necessarily restricted to cannibalism as it can also occur during heterospecific predation. Kinship relationships are also hypothesized to decrease cannibalistic tendencies in some species as discussed by Pfennig (1997). In light of Hamilton's inclusive fitness concept, not eating a conspecific can be considered as a cost of missed energy and nutrients, while it provides a benefit to the conspecific. Cannibalism should therefore evolve hand in hand with kinship recognition, or other mechanisms that offer safeguards against eating your own kin (Pfennig 1997).

In this study, the evolution of cannibalism is limited by mortality costs that are derived from parasite and/or pathogens build-up from the cannibalistic feeding (Pfennig et al. 1998, 1991). These costs have been quantified experimentally in the tiger salamander (*Ambystoma tigrinum*), where individuals suffered from reduced survival to metamorphosis and growth rates when feeding on diseased conspecifics in contrast to either diseased heterospecifics or healthy individuals (Pfennig et al. 1998). Arctic char is known to be a potential final host for a number of parasites that have either zooplankton or the macrobenthic invertebrate *Gammarus lacustris* as intermediate hosts and the presence of these food-transmitted parasites strongly reflects past diet specialization (Knudsen et al. 1996). Char individuals in Lake Takvatn have been shown to be heavily infested with the parasitic tapeworms *Diphyllobothrium dendriticum* and *D. ditremum*, which have copepods as intermediate hosts and fish as secondary hosts and with the nematode *Cystidicola farionis* which has the amphipod *Gammarus lacustris* as its only intermediate host. The second larval phase of *D. dendriticum* and *D. ditremum* in fish can re-establish in piscivorous char, which suggests that cannibals can build up high density of these parasites (Knudsen et al. 1996). Another study by Knudsen et al. (2002) found indirect evidence for parasite-induced mortality of old char individuals (> 10 years) resulting from infections with *C. farionis*, which can reach up to several thousands of worms in a single fish. The study by Knudsen et al. (2002) was inconclusive on whether these high parasite loads were derived from feeding on *Gammarus lacustris* or whether piscivorous feeding was the main transmission route. In contrast, Amundsen et al. (2003) conclude that *Cystidicola* spp. are long-lived parasites that are relatively harmless and only have a small impact on host survival at very high densities. The relation between parasite prevalence and diet in Arctic char was also studied by (Hammar 2000) who concluded that cannibalism in a population on Svalbard resulted in the accumulation of *D. ditremum* and that this increased age-dependent mortality rates. Char individuals that exclusively fed on the large amphipod *Gammaracanthus lacustris* showed a lower parasite burden and faster growth. Amundsen (2016) also

ascribes high infestations of *Diphyllobothrium* spp. parasites in large char individuals to high rates of cannibalism and piscivory. In summary, there is sufficient empirical evidence that cannibalistic behavior may lead to an accumulation of large numbers of parasites, which can increase mortality. However, the current research is inconclusive on whether parasite-induced mortality plays a role in stabilizing selection on cannibalistic behavior.

Whether or not cannibalistic behavior has a clear genetic component and is hence heritable has been the topic of much discussion. In some animal species cannibalism is considered a completely phenotypically plastic response that is triggered by specific environmental conditions such as food shortage. For example, in the tiger salamander (*Ambystoma tigrinum*) some individuals develop a distinct cannibalistic phenotype but only in response to high densities of conspecifics (Pfennig 1997). Variation in cannibalistic rates between populations can also point to genetic differences in the expression of cannibalism. Baur (1994) showed interpopulational variation in rates of egg cannibalism in the land snail *Arianta arbustorum*, but did not exclude environmental differences as a possible explanation. Stevens (1989) and Giray et al. (2001) showed that cannibalism is a heritable trait and that artificial selection for cannibalism in laboratory strains of the flour beetle *Tribolium confusum* can increase the expression of cannibalism. The environmental and genetic component of cannibalism in a natural population of the ladybird beetle *Harmonia axyridis* was studied by Wagner et al. (1999), who showed significant genetic variation in the expression of cannibalism. In addition, cannibalism was selected for only in low food environments where it reduced larval development time, which potentially reduces risks of predation. Also for Arctic char there are clear interpopulational differences in the cannibalistic tendency, which are found to increase with latitude (Amundsen 1994; Amundsen et al. 1999; Griffiths 1994), but there is no agreement on whether these have a clear genetic background (Amundsen et al. 1999; Svenning and Borgstrøm 2005). Char individuals from Arctic lakes can be highly cannibalistic but this can also be explained by the very low productivity and absence of other fish species (Amundsen et al. 1999). However, several more recent studies describe separate cannibalistic Arctic char morphs that have distinct feeding habits and morphologies, suggesting that sympatric speciation events could be responsible for the evolution of cannibalistic behavior (Amundsen 2016; Berg et al. 2010; Borgstrøm et al. 2015; Finstad et al. 2006; Florø-Larsen et al. 2014; Hammar 2000; Knudsen et al. 2016). These cannibalistic morphs have morphologies that are related to piscivory such as large jaws and robust skulls (Knudsen et al. 2016) and mature at a larger size compared to co-occurring dwarf morphs that feed only on invertebrates (Amundsen 2016; Finstad et al. 2006; Florø-Larsen et al. 2014). These adaptations to a cannibalistic lifestyle certainly suggest that genetic factors play a role (Florø-Larsen et al. 2014).

The modeling framework used here deviates from models used in several other studies on fisheries-induced evolution (Dunlop et al. 2009, 2007; Enberg et al. 2009). The modeling studies that predict evolutionary changes in life-history characteristics such as age and size at maturation or growth and reproductive investments as a consequence of fishing-induced mortality have mainly used individual-based eco-genetic models as developed by Dunlop et al. (2009, 2007). In the eco-genetic approach the life-history processes such as growth and reproduction depend only on the current state of the individual and are hence independent of food availability. This seems inappropriate for fish species, since growth and reproduction are known to be highly variable and dependent on food availability (Sebens 1987). This strong dependence of individual performance on food availability especially holds for Arctic char of which the typical environment is oligotrophic to ultraoligotrophic (Amundsen et al. 2007). Moreover, Arctic char is often the only fish species present in high Arctic and alpine freshwater systems (Klemetsen et al. 2003). In such a setting the density-dependence that arises from the ecological feedback loop between food-availability and growth, survival and reproduction is likely to be important (Amundsen 2016; Amundsen et al. 2007). Therefore, we used the framework of physiologically structured population models, which allows for a detailed description of the ecological environment and the dependence of individual life-history rates on this environment.

What is gained in ecological realism is however lost at the evolutionary side. The framework of adaptive dynamics assumes that evolution is mutation limited (Metz 2012) and does not incorporate standing genetic variation and measures of heritability as used in the quantitative genetics approach of the eco-genetic models. Adaptive dynamics allows for the identification of evolutionary endpoints and thus provides a qualitative understanding of the direction of selection and the evolutionary attractors of the ecological system. Transforming these into realistic rates of evolution requires estimates about heritability and existing genetic variation. These are notoriously difficult to measure and it therefore remains to be addressed if the rapid increase in the cannibalistic rate with increasing fishing mortality is realistic for natural populations.

Nonetheless this study shows the importance of considering the consequences of indirect selection pressures resulting from fishing mortality. It specifically demonstrates that fishing cannibalistic species can lead to unexpected rapid decline of population productivity and fisheries production when cannibalistic rates are under stabilizing selection. Furthermore, these results strengthen earlier warnings that the low productive Arctic and alpine systems where such cannibalistic char populations occur are especially vulnerable to overexploitation, due to the high catchability of these large individuals (Berg et al. 2010; Florø-Larsen et al. 2014). Management of exploited Arctic char populations should therefore not only carefully monitor changes on the population level, but also address changes in individual behavior and life-history.

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