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Bistability in a size-structured population model of cannibalistic fish—a continuation study

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Abstract

By numerical continuation of equilibria, we study a size-structured model for the dynamics of a cannibalistic fish population and its alternative resource. Because we model the cannibalistic interaction as dependent on the ratio of cannibal length and victim length, a cannibal experiences a size distribution of potential victims which depends on its own body size. We show how equilibria of the resulting infinite-dimensional dynamical system can be traced with an existing method for numerical continuation for physiologically structured population models. With this approach we found that cannibalism can induce bistability associated with a fold (or, saddle-node) bifurcation. The two stable states can be qualified as 'stunted' and 'piscivorous', respectively. We identify a new ecological mechanism for bistability, in which the energy gain from cannibalism plays a crucial role: Whereas in the stunted population state cannibals consume their victims, on average, while they are very small and yield little energy, in the piscivorous state cannibals consume their victims not before they have become much bigger, which results in a much higher mean yield of cannibalism. We refer to this mechanism as the 'Hansel and Gretel' effect. It is not related to any individual 'choice' or 'strategy', but depends purely on a difference in population size distribution. We argue that studying dynamics of size-structured population models with this new approach of equilibrium continuation extends the insight that can be gleaned from numerical simulations of the model dynamics.

Keywords: Size-dependent cannibalism; Physiologically structured population model; Fold bifurcation; Continuation; Mean yield; Eurasian perch; Infinite-dimensional dynamical system; Infanticide

'*Every morning the woman crept to the little stable, and cried*: "*Hansel, stretch out your finger that I may feel if you will soon be fat*".' (Grimm and Grimm, 1884)

1. Introduction

Theoretical studies have shown that the population dynamic consequences of cannibalism may be manifold. Gurtin and Levine (1982) showed that cannibalism can regulate a population that would otherwise grow exponentially. Cannibalism may have various effects on the stability of populations; on the one hand it may induce population cycles (Diekmann et al., 1986; Hastings, 1987; Magnússon, 1999) or chaos (Costantino et al., 1997), on the other hand it may dampen cycles that are caused by other interactions (van den Bosch and Gabriel, 1997; Claessen et al., 2000). Cannibalism is also known to induce multiple stable states (Botsford, 1981; Fisher, 1987; Cushing, 1991, 1992). A striking example of the latter is the so-called life boat mechanism, which enables a cannibalistic population to persist under conditions where a non-cannibalistic, but otherwise identical population would go extinct (van den Bosch et al., 1988; Henson, 1997). Finally, cannibalism can have major impacts on population size distribution and individual life history (Fisher, 1987; Claessen et al., 2000, 2002, and below).

Although there are exceptions, cannibals are generally larger than their victims (Polis, 1981). As a consequence, many theoretical studies of cannibalism use physiologically structured population models (e.g., Diekmann et al., 1986; van den Bosch et al., 1988; Cushing, 1992; Claessen et al., 2000), in which the roles of cannibals and victims are determined on the basis of stage, age or size

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(but see Kohlmeier and Ebenhöh, 1995, for an unstructured case). In order to obtain analytically tractable or numerically solvable models, very clever simplifying assumptions have been made. For example, by assuming that only the rate of recruitment is affected by egg cannibalism, Gurtin and Levine (1982) were able to reformulate their age-structured model as three ODEs (see also Diekmann et al., 1986). The conditions for the life boat mechanism have been derived for a general age-structured model (van den Bosch et al., 1988). Yet van den Bosch et al. (1988) did the numerical study of the dynamics for a special case, in which they could rewrite the model as a system of six delay differential equations.

In this article we study a model which is basically a simplification of a size-structured model, studied in Claessen et al. (2000, 2002), for the population dynamics of cannibalistic Eurasian perch. Two ingredients that we consider essential in the biology of our system have been left unaltered; size-dependent cannibalism and size-dependent competition for a dynamic, alternative resource. Due to our choice to model these ecological interactions realistically, our size-structured model cannot be reduced to a finite set of (delay) differential equations. The main reason for this is that we assume that the cannibalistic interaction depends on the ratio of cannibal length and victim length. As a consequence, a cannibal experiences a size distribution of potential victims which depends on its own body size. Since body size is a continuous variable, the cannibalistic interactions can only be described by an infinite-dimensional object (i.e., a function of body size).

So far the dynamics of this system have been studied with numerical simulations (Claessen et al., 2000, 2002), using the *Escalator Boxcar Train* (EBT) method (de Roos et al., 1992; de Roos, 1997). By studying the dependence of asymptotic dynamics on parameter values, several interesting effects of size-dependent cannibalism and competition on population dynamics have been revealed. For example, in competition-induced population cycles cannibalism may result in the coexistence of a 'dwarf' size class and a 'giant' size class (Claessen et al., 2000). Not only the effect of the cannibalistic tendency per se but also the effect of its size-dependent nature have been addressed. For example, in cannibalism-regulated, stable populations, the maximum victim size that cannibals can capture may determine whether the population is stunted or contains giant cannibals (Claessen et al., 2002). An obvious drawback of simulations is that only stable equilibria and other attractors can be found. Yet, finding unstable equilibrium curves can be helpful to understand the dependence of population dynamics on parameters, for example if an attractor may disappear through a fold or saddle-node bifurcation (e.g., van den Bosch et al., 1988; Cushing, 1992).

Our aim here is to complement our previous results with an analysis of equilibrium curves and thus to provide more insight into the population dynamics observed in simulations. Since we cannot explicitly express the equilibrium of our model in terms of its parameters, we study equilibrium curves by numerical continuation (i.e., by tracing an equilibrium while varying one or more parameters). For models of ODEs or discrete maps numerical methods for continuation of equilibria and stability analysis are readily available (e.g., Kuznetsov, 1995), but this is not the case for physiologically structured population models ('PSPMs' hereafter, Metz and Diekmann, 1986). One difficulty with PSPMs is that they are in principle infinite dimensional, as functions of individual state (e.g., size) enter the definition of the population state. Our continuation approach is based on a general method for numerical equilibrium continuation of PSPMs, recently introduced by Kirkilionis et al. (2001). Although the method is developed for finite-dimensional environments (e.g., the abundances of k food sources), we show how it can be used to approximate an infinite dimensional environment (e.g., the continuous size distribution of prey) that is needed to describe size-dependent cannibalism.

This article covers both new ecological results and a new (numerical) method of analysis and therefore it is structured as follows. In Section 2, we define our model of size-dependent cannibalism. In Section 3, we first give a non-technical outline of the continuation approach. The rest of that section (starting with Section 3.2) is more technical and gives a detailed description of the continuation approach. Section 4 describes the results of the model analysis. The discussion (Section 5) treats the ecological results as well as the merits of the continuation method.

2. The model

Our model describes the dynamics of a size-structured, cannibalistic population and its unstructured, alternative resource population. The parameter values we use (Table 1) are based on piscivorous fish, in particular Eurasian perch (*Perca fluviatilis*), and zooplankton (*Daphnia* spp.) (Claessen et al., 2000). In our model, we assume that the physiological state of an individual is completely determined by its body length x. Vital rates such as food ingestion, metabolism, reproduction and mortality are assumed to depend entirely on body length and the condition of the environment. The population size distribution is denoted by $n(x)$ and the density of the alternative resource by R. All individuals are born with the same length x_b, and are assumed to mature upon reaching the size x_f. Reproduction is assumed to be continuous (in time) which implies that

Table 1
Model symbols: state variables[a] and constant parameters. Parameterization[b] for Eurasian perch (*Perca fluviatilis*) feeding on a zooplankton resource (*Daphnia* sp., length 1 mm) and conspecifics. All parameters except r and K refer to individual level processes

Symbol	Default value	Unit	Interpretation
Variables			
x	—	mm	Individual length
$n(x)$	—	l^{-1}	Population size distribution
R	—	g l^{-1}	Resource density
Parameters			
x_b	7	mm	Length at birth
x_f	115	mm	Length at maturation
λ	9×10^{-6}	g mm^{-3}	Length-weight scaling constant
α	7.0×10^{-4}	l day^{-1} mm^{-4}	Planktivory attack rate scaling constant
x_p	160.0	mm	Maximum length of planktivory
β	0.5	l day^{-1} mm^{-2}	Cannibalistic voracity
δ	0.06	—	Lower limit of predation window
ε	0.5	—	Upper limit of predation window
ϕ	0.2	—	Optimum of predation window
c_a	0.6	—	Assimilation efficiency
c_r	0.5	—	Efficiency of offspring production
ξ	1.7×10^{6}	day g^{-1} mm^{-3}	Digestion time scaling constant
ρ	2.5×10^{-7}	g day^{-1} mm^{-3}	Metabolic rate scaling constant
κ	0.7	—	Allocation coefficient
μ_0	0.01	day^{-1}	Background mortality rate
s	1	g^{-1}	Starvation mortality coefficient
r	0.1	day^{-1}	Zooplankton population growth rate
K	3×10^{-3}	g l^{-1}	Zooplankton carrying capacity

[a] The time argument has been left out from notation of variables, because we study steady states.
[b] For references: see Claessen et al. (2000).

the size distribution $n(x)$ is continuous. Note that in our notation we ignore all time dependencies (e.g., $n(x)$ rather than $n(t,x)$) because we consider equilibria only.

The assimilation rate is assumed to follow a size-dependent, type II functional response

$$F(x)=c_a\frac{\gamma(x)}{1+H(x)\gamma(x)}, \tag{1}$$

where c_a is the assimilation efficiency, $\gamma(x)$ is the sum of the encounter rates with conspecific and alternative prey mass, and $H(x)$ is the size-dependent digestion time per gram of prey mass (Table 2). The encounter rate with alternative prey mass is assumed to be

$$\gamma_a(x)=A(x)R.$$

We assume that the attack rate on the alternative resource $A(x)$ is dome shaped, positive for the length interval $(0,x_p)$ and reaches a maximum at length $x_p/2$. At length x_p the function $A(x)$ and its slope become zero (Table 2).

The encounter rate with conspecific prey mass is obtained by integration over the size distribution of potential victims. We assume that a cannibal of length x can capture victims with lengths y in the range $\delta x<y<\varepsilon x$, and that the encounter rate with conspecific prey mass hence is

$$\gamma_c(x)=\int_{\delta x}^{\varepsilon x}\beta x^2T(x,y)w(y)n(y)\,dy. \tag{2}$$

Table 2
The model: individual level functions

Body mass	$w(x)=\lambda x^3$
Zooplankton attack rate	$A(x)=\begin{cases}\alpha x^2(x-x_p)^2 & \text{if } x\leqslant x_p\\ 0 & \text{otherwise}\end{cases}$
Cannibalism window tent function	$T(c,v)=\begin{cases}\frac{v-\delta c}{(\phi-\delta)c} & \text{if } \delta c<v\leqslant\phi c\\ \frac{\varepsilon c-v}{(\varepsilon-\phi)c} & \text{if } \phi c<v\leqslant\varepsilon c\\ 0 & \text{otherwise}\end{cases}$
Cannibalistic maximum attack rate	$v(x)=\beta x^2$
Total encounter rate	$\gamma(x)=\gamma_a(x)+\gamma_c(x)$
Zooplankton encounter	$\gamma_a(x)=A(x)R$
Cannibalistic encounter	$\gamma_c(x)=\int_{\delta x}^{\varepsilon x}\beta x^2T(x,y)w(y)n(y)\,dy$
Food intake rate	$F(x)=c_a\frac{\gamma(x)}{1+H(x)\gamma(x)}$
Digestion time	$H(x)=\xi x^{-3}$
Maintenance requirements	$M(x)=\rho x^3$
Growth rate in length	$g(x)=\begin{cases}0 & \text{if } \kappa F(x)<M(x)\\ \frac{1}{3\lambda x^2}[\kappa F(x)-M(x)] & \text{otherwise}\end{cases}$
Reproductive rate	$b(x)=\begin{cases}c_r(1-\kappa)F(x)w(x_b)^{-1} & \text{if } x>x_f\\ 0 & \text{otherwise}\end{cases}$
Total mortality	$\mu(x)=\mu_0+\mu_s(x)+\mu_c(x)$
Cannibalistic mortality	$\mu_c(x)=\int_{x/\varepsilon}^{x/\delta}\frac{\beta y^2T(y,x)}{1+H(y)\gamma(y)}n(y)\,dy$
Starvation mortality	$\mu_s(x)=\begin{cases}s[M(x)-\kappa F(x)] & \text{if } \kappa F(x)<M(x)\\ 0 & \text{otherwise}\end{cases}$

The term βx^2 is the maximum cannibalistic attack rate of a cannibal of length x, reached only for the optimal victim size $y=\phi x$ (with $\delta<\phi<\varepsilon$). The parameter β hence reflects the species-specific tendency to cannibalize, which we refer to as the cannibalistic voracity. The tent-shaped function $T(x,y)$ accounts for the effect of suboptimal victim sizes $y\neq\phi x$. It takes values between 0 and 1 if $\delta x<y<\varepsilon x$, and is zero outside this range (Table 2). We refer to the parameters δ and ε as the lower and upper limits of the cannibalism window, respectively (Claessen et al., 2000). Thus, the product $\beta x^2 T(x,y)$ equals the attack rate of a cannibal of length x on a victim of length y. The mass of an individual of length x is assumed to scale with body volume, $w(x)=\lambda x^3$.

We use a quadratic relation for the maximum cannibalistic attack rate, even though a sigmoidal function would describe the observed maximum cannibalistic attack rate better (L. Persson, pers. com.). Such a sigmoidal function would, however, be computationally more costly. Data show that small individuals (age 0–1 years, length 7–50 mm) cannibalize little while the maximum attack rate levels off at very large sizes (>250 mm). In Claessen et al. (2000) we used a function that describes the leveling-off well ($x^{0.6}$), but overestimates the cannibalistic potential of small individuals. This had no important consequences since that model assumed pulsed reproduction, such that cannibalism was practically impossible up to the age of 1 year. In the present context with continuous reproduction, the fact that the function $x^{0.6}$ overestimates cannibalism by very small individuals becomes problematic. Hence, we chose to use a quadratic relation, although it overestimates cannibalism by very large individuals. With a more realistic sigmoidal function, similar results would be obtained as long as the accelerating part of the function resembles the quadratic relation; but the maximum size in the population would be smaller.

We assume that a fraction κ of assimilated energy is allocated to growth and maintenance (Kooijman and Metz, 1984), and the remainder to reproduction. The growth rate in mass is obtained by subtracting the metabolic rate from the energy intake rate. Assuming that the metabolic rate scales with body volume like ρx^3, the growth rate in length becomes

$$g(x)=\frac{1}{3\lambda x^2}(\kappa F(x)-\rho x^3). \tag{3}$$

The length for which the metabolic rate equals the intake rate allocated to growth ($\kappa F(x)=\rho x^3$) is referred to as the maximum length, denoted by x_{max}.

We assume that all individuals allocate the fraction $1-\kappa$ of the assimilation rate to reproduction. For juveniles ($x<x_f$), this energy is assumed to be used for development of reproductive organs (Kooijman, 1993). For adults, the per capita birth rate is calculated by dividing the investment in reproduction by the energy cost of producing a single newborn:

$$b(x)=\begin{cases} c_r(1-\kappa)F(x)w(x_b)^{-1} & \text{if } x\geqslant x_f,\\ 0 & \text{otherwise,}\end{cases} \tag{4}$$

where the conversion efficiency c_r takes into account losses due to egg respiration.

The mortality rate is assumed to be the sum of a constant background mortality rate μ_0, a size-dependent mortality rate caused by cannibalism, and a starvation mortality:

$$\mu(x)=\mu_0+\mu_c(x)+\mu_s(x). \tag{5}$$

In accordance with (2), the cannibalistic mortality rate is defined as

$$\mu_c(x)=\int_{x/\varepsilon}^{x/\delta}\frac{\beta x^2 T(y,x)}{1+H(y)\gamma(y)}n(y)\,dy. \tag{6}$$

In equilibrium individuals cannot grow beyond the maximum sustainable size, so for an equilibrium analysis we do not have to consider starvation mortality. However, in population cycles individuals may go through periods of food shortage and starvation. For such cases we assume that starvation mortality rate increases linearly with the difference between the metabolic rate and the food assimilation rate

$$\mu_s(x)=\begin{cases} s[\rho x^3-\kappa F(x)] & \text{if } \kappa F(x)<\rho x^3,\\ 0 & \text{otherwise,}\end{cases}$$

where s is a proportionality constant. The individual-level model is summarized in Table 2 and the PDE formulation for the population-level model is presented in Table 3.

We assume that the alternative resource population is unstructured. In our model it follows semi-chemostat dynamics extended with a term to account for the effect of consumption by the structured population,

$$\frac{dR}{dt}=r(K-R)-\int_{x_b}^{\infty}\frac{A(x)R}{1+H(x)\gamma(x)}n(x)\,dx$$

with $A(x)$, $H(x)$ and $\gamma(x)$ as defined in Table 2.

Table 3
The model: specification of the dynamics of population state variables[a]

PDE	$\frac{\partial n}{\partial t}+\frac{\partial gn}{\partial x}=-\mu(x)n(x)$
Boundary condition	$g(x_b)n(x_b)=\int_{x_f}^{x_{max}}b(x)n(x)\,dx$
Resource dynamics	$\frac{dR}{dt}=r(K-R)-\int_{x_b}^{x_{max}}\frac{A(x)R}{1+H(x)\gamma(x)}n(x)\,dx$

The individual-level functions are listed in Table 2. Parameters are listed in Table 1.

[a] Note that the time argument has been left out from variables and functions.

3. The continuation approach

3.1. Non-technical outline

As mentioned in the Introduction, in this article we study our model (Table 3) by numerical continuation of equilibrium curves. Continuation methods for ODEs are well established but for PSPMs they are still a very recent development. We use the continuation method of Kirkilionis et al. (2001) which is based on the fundamental distinction in PSPMs between the state of an individual and the condition of its environment. The idea is that if the environment is prescribed properly, the life history of an individual (in terms of the development of its body size, survival, energy reserves, etc.) can be calculated without direct knowledge about other individuals. In this view, 'environment' mediates all interactions that affect the fate of an individual. The set of rules which dictates how the state of an individual changes in response to the environment is referred to as the individual level or *i*-level model (Metz and Diekmann, 1986). With such a distinction between environment and individual state, an equilibrium of the PSPM requires an environmental condition which gives rise to a life history consistent with this environmental condition, in the sense that the population growth rate equals zero and, moreover, the extant population generates the environmental condition by feedback.

We illustrate this with an example. Consider a size-structured population in which individuals interact with each other only through exploitative competition, i.e., through their impact on food density (e.g., de Roos et al., 1992; de Roos, 1997, or our model with $\beta = 0$). The environment of an individual is then fully characterized by the food density. Once a constant food density is given, the life history of an individual is determined as well, because it depends on the food density only. This shows that if the environment is known, individuals can be considered to be independent. Once the life history is known, we can formulate the necessary conditions for equilibrium. In this example, they are the requirements (i) that on average an individual exactly replaces itself and (ii) that the total population consumption rate equals renewal rate of the food resource. Denoting the per capita lifetime reproduction by R_0, the total consumption rate by X and the renewal rate by Y, these conditions can be specified as $R_0 - 1 = 0$ and $X - Y = 0$, respectively. These are two equations in the two unknowns P, the population birth rate and R, the resource density.

We refer to the variables P and R as *input* (I) variables, and to $R_0 - 1$ and $X - Y$ as *output* (O) variables (Diekmann et al., 1998, note that our definition of input and output is different). Quite generally the life history and the feedback yield a map

$$f : \mathbb{R}^k \to \mathbb{R}^k, \qquad I \mapsto O, \tag{7}$$

which we call the 'input–output map'. An equilibrium can be found via an input $I^* \in \mathbb{R}^k$ for which the equilibrium and feedback conditions

$$f(I^*) = 0$$

hold. In our example this is a two-dimensional map, with the food density R and the population birth rate P as input variables and $R_0 - 1$ and $X - Y$ as output variables. The population birth rate is an input variable because it determines the total population size and hence the total population consumption rate.

The condition $f(I^*) = 0$ can now be used in numerical continuation. Applying the method of Kirkilionis et al. (2001) we would trace the equilibrium food density and the total population birth rate while varying one free parameter. In this case the continuation problem is hence only two dimensional, although the population size distribution is still an infinite-dimensional object.

As pointed out above, in our model of size-dependent cannibalism the interactions depend directly on body size. This means that the definition of environment should include functions of body size, such as the size-dependent mortality rate due to cannibalism, denoted $\mu_c(x)$ where x is body size. The input variable for our model of cannibalism is therefore infinite dimensional. Kirkilionis et al. (2001) developed their continuation method for cases in which the environment is finite dimensional. In Sections 3.5 and 3.6 we show in detail how our model with an infinite-dimensional interaction environment can be studied with the method of Kirkilionis et al. (2001).

We are not able to evaluate the stability of equilibria during the continuation. For information on stability we therefore rely on EBT simulations of the model as summarized in Table 3. In a biologically meaningful way this method discretizes the continuous population distribution by subdividing the population into cohorts of individuals of similar age. The method is described in detail in de Roos et al. (1992) and de Roos (1997).

Sections 3.2–3.6 treat the new continuation methodology in detail. The biologically interested readers may skip these sections and continue with Section 4.

3.2. Life history as an input–output map

In this section we show how elements from the *i*-level model outlined in Section 2 can be used to construct a life history if the appropriate input is given. We subdivide the life history into three aspects; survival, growth and reproduction. The probability to survive to age a is denoted $S(a)$ and is the solution of the ODE

$$\frac{dS}{da} = -\mu(x(a))S(a), \quad S(0) = 1, \tag{8}$$

where the function $\mu(x)$ is the size-dependent mortality rate. The growth trajectory, denoted $x(a)$, is the solution of

$$\frac{dx}{da} = g(x(a)), \quad x(0) = x_b, \tag{9}$$

with $g(x)$ the growth rate in length. The expected, cumulative reproduction up to age a, denoted $B(a)$, is the solution of

$$\frac{dB}{da} = b(x(a))S(a), \quad B(0) = 0, \tag{10}$$

in which $b(x)$ is the size-dependent, per capita birth rate. The expected, lifetime reproductive output, denoted R_0, is then given by

$$R_0 = B(\infty). \tag{11}$$

Due to the occurrence of $x(a)$ in (8) and (10), (9) has to be solved first, then (8), and finally (10). Alternatively, the ODEs (8)–(10) can be solved simultaneously.

Together, $S(a)$, $x(a)$ and R_0 define a life history. It should be noted that the rates $\mu(x)$, $g(x)$ and $b(x)$ may depend on the environment. If the latter is known the life history is set. Below we make the dependence on the environment explicit.

3.3. In the absence of cannibalism

In equilibrium and in the absence of cannibalism (i.e., $\beta = 0$) the mortality rate (5) reduces to the constant background mortality rate, $\mu(x) = \mu_0$ (due to the assumption of equilibrium there is no starvation). The growth rate $g(x)$ and the fecundity $b(x)$ depend on the alternative resource density only ((3) and (4)). This implies that the resource density R alone is required as input to integrate (8)–(10).

In equilibrium each individual must exactly replace itself which translates into the condition $R_0 = 1$. Additionally, the total consumption rate of the size-structured population must equal the renewal rate of the alternative resource which constitutes the second equilibrium condition. To obtain the total consumption rate from the life history, we use the fact that in equilibrium the consumption rate of the entire population equals the total (lifetime) consumption of a single individual multiplied with the population birth rate. This follows from the fact that in equilibrium we can write the population age distribution as $m(a) = S(a)P$, where P is the population birth rate. We can use this to calculate integrals over the size axis in terms of integrals over the age axis,

$$\int_{x_b}^{x_{max}} z(x)n(x)\,dx = P\int_0^{\infty} z(x(a))S(a)\,da, \tag{12}$$

where $z(x)$ is some weighing function, e.g., the per capita consumption rate of alternative resource.

We denote the expected, cumulative consumption up to age a with $\theta(a, R)$. It can be calculated in parallel with (8) and (9) by integrating

$$\frac{d\theta}{da} = \frac{A(x(a))R}{1+\gamma(x(a))H(x(a))}S(a), \quad \theta(0, R) = 0 \tag{13}$$

(cf.(1)). The total population consumption rate of alternative resource is then the product of P and $\theta(\infty, R)$. Note that P is required as an input variable, since it cannot be derived from the life history.

Now we can formulate the input (I) and output variables (O) used for the input–output map (7). As input we need the resource density R and the population birth rate P,

$$I_1 = R, \tag{14}$$

$$I_2 = P. \tag{15}$$

The output variables are the equilibrium conditions,

$$O_1 = R_0(I) - 1, \tag{16}$$

$$O_2 = r(K - I_1) - I_2\theta(\infty, I_1). \tag{17}$$

Then, for the non-cannibalistic case we have a two-dimensional map, $f : \mathbb{R}^2 \to \mathbb{R}^2$ like (7). The equilibrium condition $f(I^*) = 0$ can be used to continue an equilibrium I^* with one free parameter by applying the method of Kirkilionis et al. (2001).

3.4. In the presence of cannibalism

With cannibalism ($\beta > 0$) the rates $\mu(x)$, $g(x)$ and $b(x)$ in (8)–(10) all depend on the environment in a size-dependent manner. This dependence results from the occurrence of the cannibalistic mortality rate $\mu_c(x)$ in (5) and of the encounter rate with conspecific prey $\gamma_c(x)$ in (3) and (4). It implies that, in addition to I_1 and I_2 (14) and (15), these two functions are required as input for the calculation of a life history,

$$I_\mu(x) = \mu_c(x), \tag{18}$$

$$I_\gamma(x) = \gamma_c(x). \tag{19}$$

On the basis of this extended input I we can again calculate the life history with (8)–(10). From the obtained survival function $S(a)$, the growth trajectory $x(a)$ and the population birth rate P it is possible to construct the population size distribution $n(x)$, using (12). This enables us to use (2) and (6) to compute the functions $\mu_c(x)$ and $\gamma_c(x)$ that correspond to this extended input. If we denote the result of these calculations by $\mu_c(x, I)'$ and $\gamma_c(x, I)'$, emphasizing their dependence on the input, we obtain two more output relations:

$$O_\mu(x) = I_\mu(x) - \mu_c(x, I)', \tag{20}$$

$$O_\gamma(x) = I_\gamma(x) - \gamma_c(x, I)'. \tag{21}$$

With these input and output variables we can now define an input–output map like (7), such that $f(I^*)=0$ defines an equilibrium point. However, because I and O contain functions of body size, the map is infinite dimensional.

3.5. Continuation

For continuation purposes the infinite–dimensional input–output map has to be approximated by a finite-dimensional one. In order to obtain a finite-dimensional map, we represent the functions $\mu_c(x)$ and $\gamma_c(x)$ by their values at the fixed sizes $x_1, x_2, \ldots, x_k$. The values $\mu_c(x_i)$ are used as input,

$$I_3=\mu_c(x_1),$$

$$I_4=\mu_c(x_2),$$

$$\ldots$$

$$I_{k+2}=\mu_c(x_k). \tag{22}$$

We choose x_k such that $x_{max}<x_k$ to account for the entire range of body sizes. In the calculation of the life history (8)–(10), the value of $\mu_c(x)$ is approximated by linear interpolation between the values at the two nearest x_i and x_{i+1}. Because we assume that cannibals are always larger than their victims, the function $\gamma_c(x)$ is not actually needed as input; when during the integration of (8)–(10) the growth rate of a cannibal must be calculated, the size distribution of its potential victims can be reconstructed from results obtained previously in the integration (Section 3.6).

The output variables are then defined by (16) and (17) together with

$$O_3=I_3-\mu_c(x_1,I)',$$

$$O_4=I_4-\mu_c(x_2,I)',$$

$$\ldots$$

$$O_{k+2}=I_{k+2}-\mu_c(x_k,I)'. \tag{23}$$

In Section 3.6 we give more details of the finite-dimensional approximation and show how to calculate the new estimates $\mu_c(x_i,I)'$. The input–output map (7), the input and output variables ((14)–(17) and (22)–(23)), and the life history as specified with (8)–(11) and (13) were used for numerical continuation to obtain the results in Section 4. We can apply numerical continuation techniques (Kuznetsov, 1995), for example to continue the equilibrium with β as bifurcation parameter. An initial estimate of I^* for the case $\beta=0$ was obtained from numerical integration.

3.6. Discretization

As mentioned in Section 3.5, for numerical continuation the function $\mu_c(x)$ is represented by its values at the fixed sizes $x_1, x_2, \ldots, x_k$. Here we show how in principle from given estimates $\mu_c(x_i)$ and $\gamma_c(x_i)$ new estimates can be calculated. To facilitate the distinction between the size $x(a)$ of the 'focus' individual, whose life history is calculated, and the fixed sizes used in the discretization, we denote the fixed sizes by y_i rather than x_i. To avoid excessive notation we suppress the dependence of functions on the input variable I.

During the integration of (8)–(10), we can calculate the cumulative cannibalistic mortality rate inflicted on a victim of length y_i, denoted by $M_i(a)$, as

$$\frac{dM_i(a)}{da}=\frac{\beta x(a)^2 T(x(a),y_i)}{1+H(x(a))\gamma(x(a))}S(a)P, \quad M_i(0)=0. \tag{24}$$

A new estimate of $\mu_c(y_i)$ is then given by $\mu_c(y_i)'=M_i(\infty)$. Note that x refers to cannibal length, and that the function $\gamma(x)$ occurs in the right-hand side of this differential equation. The latter means that the current, discretized estimate $\gamma_c(y_i)$ is used in the calculation of the new estimate $\mu_c(y_i)'$. Also, the current estimate $\mu_c(y_i)$ is used in this calculation, because it affects $S(a)$.

In a similar way the cumulative mass-encounter rate, $G_i(a)$, with a cannibal of fixed length y_i can be obtained by solving

$$\frac{dG_i(a)}{da}=\frac{\beta y_i^2 T(y_i,x(a))}{1+H(y_i)\gamma(y_i)}w(x(a))S(a)P, \quad G_i(0)=0. \tag{25}$$

Note that x is now victim length. The new estimate of $\gamma_c(y_i)$ is then given by $\gamma_c(y_i)'=G_i(\infty)$. Again, the current estimates $\gamma_c(y_i)$ and $\mu_c(y_i)$ have to be used in the right-hand side of this ODE.

Since the functions $\mu_c(x)$ and $\gamma_c(x)$ are represented at k fixed body lengths, we need to solve $2k$ ODEs simultaneously with (8)–(10). Although this is possible, it is computationally costly if the number of discretization points $x_1, x_2, \ldots, x_k$ is large. An alternative method is based on the idea that during the calculation of the life history we can reconstruct the entire population size distribution (see Section 3.3 and below). This allows for the derivation of a discrete approximation to the function $n(x)$ while integrating the ODEs (8)–(10). The values $\gamma_c(y_i)$ and $\mu_c(y_i)$ of the cannibalistic encounter rate and cannibalistic mortality rate at length y_i can subsequently be obtained by substituting for $n(x)$ its discrete approximation into (2) and (6), respectively. This method is computationally more efficient and was used to obtain the results in Section 4.

Theoretically, the precise choice of the discretization points does not matter although the accuracy of the approximation obviously depends on their resolution. In our calculations we chose them equidistant as $y_i=x_b+i\Delta$ with $\Delta=1$ mm (note that $x_b=7$ mm and x_{max} often lies between x_f and 350 mm). After some initial experimentation we found that $\Delta=1$ mm generally

gave a reliable result without causing too much computational effort. With too large a value of Δ the continuation process failed to find an equilibrium.

Analogous to the representation of the size distribution in the EBT method for numerical integration (de Roos et al., 1992), the continuous population size distribution is approximated by a number (q) of delta-functions: we divide the size distribution into q length classes of width Δ and represent each length class by the total number of individuals it contains, denoted N_i,

$$N_i = \int_{x_b+(i-1)\Delta}^{x_b+i\Delta} n(x)\,dx \quad \text{for } i = 1 \ldots q, \tag{26}$$

and by the average length of these individuals, denoted X_i,

$$X_i = \frac{1}{N_i}\int_{x_b+(i-1)\Delta}^{x_b+i\Delta} xn(x)\,dx \quad \text{for } i = 1 \ldots q. \tag{27}$$

The calculation of the life history provides us with *exact* values for N_i and X_i. To calculate N_i we integrate the product of survival function and population birth rate,

$$\frac{d\sigma}{da} = S(a)P, \quad \sigma(0) = 0, \tag{28}$$

simultaneously with the integration of (8)–(10). The integration proceeds in stages over the length intervals $[x_b + (i-1)\Delta, x_b + i\Delta]$ and is stopped exactly at reaching the upper bound of this interval using an ODE integration technique with event localization (Hairer et al., 1993). Let a_i refer to the age at which the length $x_b + i\Delta$ is reached, with $i = 1, \ldots, q$ and $a_0 = 0$. The number of individuals N_i is then obtained as

$$N_i = \sigma(a_i) - \sigma(a_{i-1}) \quad \text{for } i = 1, \ldots, q. \tag{29}$$

For the calculation of the average length in each size class X_i, we keep track of the integral of the product of length and survival,

$$\frac{d\pi}{da} = x(a)S(a), \quad \pi(0) = 0. \tag{30}$$

Then the average length is given by

$$X_i = \frac{(\pi(a_i) - \pi(a_{i-1}))P}{N_i} \quad \text{for } i = 1, \ldots, q. \tag{31}$$

The quantities N_i and X_i for $i = 1, \ldots, q$ constitute an exact, discrete representation of the population size distribution $n(x)$. Using this representation the value of $\mu_c(y_i)$ can be approximated as

$$\mu_c(y_i) \approx \sum_{j=1}^{q} \frac{\beta X_j^2 T(X_j, y_i) N_j}{1 + \Gamma_j},$$

where

$$\Gamma_j = A(X_j)R + \beta X_j^2 \sum_{k=1}^{q} T(X_j, X_k)\lambda X_k^3 N_k$$

(cf. de Roos et al., 1992).

Similarly, an estimate of $\gamma_c(y_i)$ can be obtained as

$$\gamma_c(y_i) \approx \beta y_i^2 \sum_{j=1}^{q} T(y_i, X_j)\lambda X_j^3 N_j$$

(cf. (2)). Note that the function $T(y,x)$ equals 0 for $y > \varepsilon x$. Therefore, when the value $\gamma_c(y_i)$ is needed in the integration of the life history ODEs, all the quantities N_i and X_i that are involved in non-zero terms in the summation above have been calculated already. Hence, the values $\gamma_c(y_i)$ can be obtained from the set of values N_i and X_i that are previously obtained in the integration and do not have to be specified as input variables.

4. Results

An obvious way to study the effect of cannibalism on population dynamics is to vary the strength of cannibalism, tuned by the parameter β (cf. Cushing, 1991; Claessen et al., 2000). However, recent model analysis has shown that the size-dependent nature of cannibalism, reflected in the cannibalism window $T(x,y)$, has important implications as well (Claessen et al., 2002). In particular it appears that population dynamics are very sensitive to the lower limit of the cannibalism (δ) but less so to the maximum (ε) and the optimum (ϕ) of the window (although ε has a major effect on life history). Since empirical work has shown that the value of δ varies between species of cannibalistic fish (Mittelbach and Persson, 1998), these results allow for between-species comparison of the effect of cannibalism on population dynamics (e.g., Claessen et al., 2002). Motivated by these findings we choose β as bifurcation parameter for $\delta = 0.06$ (the default for Eurasian perch) but repeat the continuation for various fixed values of δ.

The results of a continuation analysis with β as free parameter for a fixed $\delta = 0.06$ are shown in Fig. 1, in which the equilibrium state is characterized by the resource density R, the population birth rate P and the ultimate length x_{max}. The figure shows that initially P increases rapidly with β, which is associated with an increase in per capita fecundity as well as an increase of the number of adults. The initial decrease of the alternative resource density is associated with an increase of the number of small individuals, which consume the resource (result not shown). The resource density then increases up to a maximum which reflects that the total consumption rate decreases up to that point. After $\beta = 0.144$, both P and the number of adults decline, whereas the average per capita fecundity increases with β. The lower panel shows that the maximum size in the population always increases with β, which relates to combined effect of more alternative

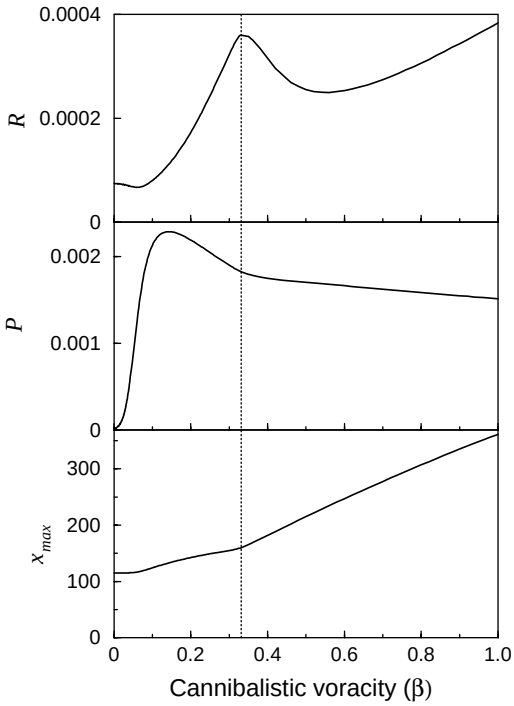

Fig. 1. One-parameter continuation of the equilibrium with β (the cannibalistic voracity) as free parameter. Continuation started in the absence of cannibalism ($\beta = 0$, $R = 7.44 \times 10^{-5}$ and $P = 1.29 \times 10^{-5}$). Parameters: $\delta = 0.06$, Table 1.

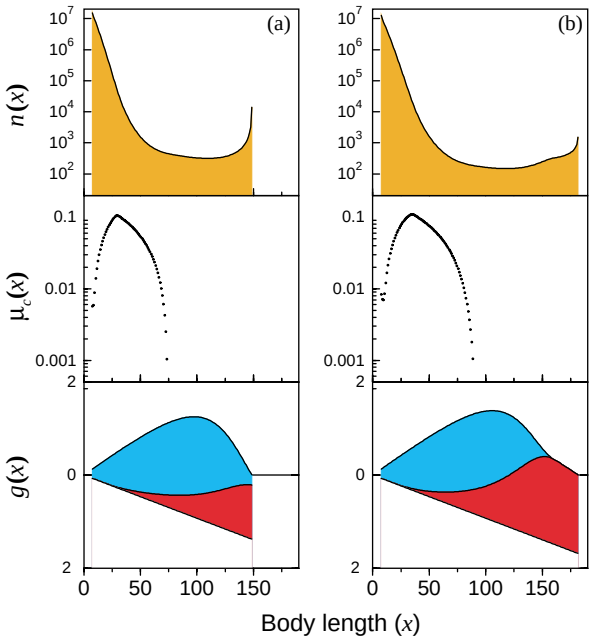

Fig. 2. Aspects of the equilibrium size-structure. (a) $\beta = 0.25$, (b) $\beta = 0.4$. Upper panels: population size-distribution $n(x)$. Middle panels: the interaction variables (22), representing the cannibalistic mortality rate $\mu_c(x)$. Lower panels: the growth rate subdivided by food type. The net growth rate (top border of colored area) is obtained by adding the intake rate (colored area) to the (negative) metabolic rate (lower border of colored area). For each x the height of the colored area corresponds to the total energy intake rate. The contributions from planktivory (blue area) and piscivory (red area) are indicated. With $\beta = 0.25$, individuals cannot grow beyond the "planktivory niche"—even the largest individuals consume the alternative resource. With $\beta = 0.4$ individuals $x > 160$ mm are in the "piscivory niche"—they consume conspecifics exclusively.

resource and the energy gain from cannibalism. The vertical, dotted line in Fig. 1 marks a critical value of β where R attains a maximum and the equilibrium curve changes slope abruptly ($\beta = 0.3316$). This point corresponds to the point where the maximum length in the population (x_{max}) equals the size for which the attack rate on the alternative resource becomes zero. This occurs at length x_p which equals 160 mm (Tables 1 and 2).

To better understand the abrupt change around the critical value of β we have a closer look at the population structure for two fixed values of β, below and above the critical value, respectively. For $\beta = 0.25$ and 0.4 the population structures are represented in Fig. 2. The figure show the population size distribution $n(x)$, but also the input function $\mu_c(x)$ (6). For both values of β the population size distribution is U-shaped. The reason is the high growth rate at intermediate sizes ($g(x)$, Fig. 2), which causes accumulation of individuals close to the ultimate size. In the lower panels the contributions from feeding on conspecifics and on the alternative resource to the individual growth rate $g(x)$ are indicated. With a β below the critical value even the largest individuals feed on the alternative resource (Fig. 2a), whereas for a β beyond it the largest individuals feed exclusively on conspecifics (Fig. 2b). The size interval for which individuals feed exclusively on conspecifics, that is (x_p, x_{max}), is referred to as the 'piscivory niche' (cf. Claessen et al., 2002). The size interval with a positive food intake rate from the alternative resource is referred to as the 'planktivory niche'. Thus, the vertical dotted line in Fig. 1 marks the opening of the piscivory niche.

4.1. The effect of the cannibalism window

In our model the body length of the smallest victims that a cannibal can take is defined as a fraction δ of its own length ((2) and Table 2). For five values of δ, Fig. 3 shows results of continuations with β as free parameter. Note that Fig. 1 was made with $\delta = 0.06$, the estimate for Eurasian perch (Claessen et al., 2000). For reference, in Fig. 3 also the constant maturation size (x_f) and the maximum size of planktivory (x_p) are indicated, together with the maximum size (x_{max}) which depends on β. We make four observations from Fig. 3:

1. If β is decreased to zero, the maximum length approaches x_f.

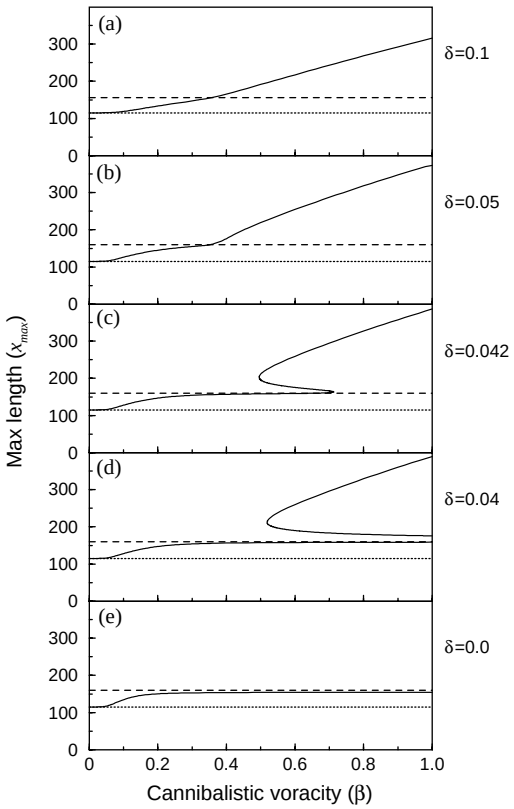

Fig. 3. Continuation of the equilibrium with β as free parameter for different values of δ (the lower limit of the cannibalism). Other parameters as in Table 1. Solid curve: maximum size in equilibrium (x_{max}). Dotted line: maturation length ($x_f = 115$ mm). Dashed line: maximum size for planktivory ($x_p = 160$ mm).

2. If β is increased from zero and δ is large, the maximum size x_{max} increases and at high β individuals reach giant sizes.
3. If β is increased from zero and δ is small, the maximum size approaches x_p, or a value just below it.
4. If β is increased from zero and δ is intermediate the intersection of x_{max} with x_p is followed by a fold bifurcation. For any β in between this fold bifurcation and a second one (at lower β) there exist three equilibria.

Below we address these four results separately.

Result 1. To understand the first result, consider a population that consists of individuals that start reproducing at age A with constant rate $\bar{b}$. Assume a constant mortality rate $\bar{\mu}$. The lifetime reproduction is then

$$R_0 = \int_A^\infty \bar{b} e^{-\bar{\mu}a} da = \frac{\bar{b}}{\bar{\mu}} e^{-\bar{\mu}A}$$

The condition $R_0 = 1$ implies that the relation $A = \frac{1}{\bar{\mu}} \ln(\frac{\bar{b}}{\bar{\mu}})$ must hold in equilibrium. From this we can see that if the mortality rate becomes very small, the equilibrium condition requires that A becomes very large and/or $\bar{b}$ becomes very small, i.e., population regulation occurs either through decreasing juvenile survival or adult fecundity.

Now consider our full model. One effect of letting β go to zero is that the total mortality rate decreases toward the background mortality rate. Of course, in this model both the maturation age and the birth rate are not parameters but they depend on the resource density R. The age at maturation decreases with R and fecundity at a given size increases with R. This correlated dependence of age at maturation and fecundity implies that if we assume $\beta = 0$ and let the background mortality rate μ_0 become very small, the equilibrium condition can be fulfilled only by decreasing the resource density. The resource density in equilibrium, however, should be at least sufficiently high to attain an ultimate size larger than the maturation size. Analogous to the age-structured case discussed above, we can hence expect that with decreasing μ_0 the resource density approaches the minimum density required to reach x_f. In turn, this implies that the maximum size approaches x_f as the mortality rate approaches zero. We have verified this by choosing the background mortality rate as free parameter and assuming $\beta = 0$. Continuation shows that if μ_0 is decreased and $\beta = 0$, the resource density indeed approaches the level at which growth becomes zero at size x_f (see also de Roos et al., 1990).

In conclusion, if we let the cannibalistic voracity β decrease to zero, the maximum length in the population approaches the maturation length because the total mortality rate becomes very small. This result therefore depends on our assumption of a low background mortality rate.

Result 2. In Fig. 3a and b the maximum size in the population is positively correlated with β over the entire range of β. Before the intersection of x_{max} and x_p this is partly because the density of the alternative resource increases with β (cf. Fig. 1). Beyond this point, however, it is entirely due to the cannibalistic energy gain which increases with β. The latter is true despite that the total biomass of the population decreases with β.

Result 3. The convergence of the maximum length x_{max} to a value just below x_p when the cannibalistic voracity (β) is increased from zero (e.g., Fig. 3e) is easily explained if we assume that cannibals do not gain energy by eating conspecifics. A negligible energy gain from cannibalism may result if cannibals consume very small victims only, which is a possibility if δ is sufficiently small. Killing conspecifics without an energy gain is often referred to as 'infanticide' (Hausfater and Hrdy, 1984). Under this assumption, increasing β merely increases the mortality rate of victims, resulting in a lower overall population density. As a consequence, the rate of consumption of alternative resource by the

population decreases with β, and hence the density of this resource increases. As the resource approaches its carrying capacity the consumers attain a maximum size which turns out to be close to the size where the attack rate on this resource becomes zero, i.e., close to x_p.

If we make the assumption of infanticide and also that the resource is at its carrying capacity, then with the parameters in Table 1 the maximum size is $x_{max} = 151.86$ mm. In our model and assuming $\delta = 0$, the maximum size converges to 155.13 mm when β is increased (Fig. 3e). The difference between these two values is due to the gain from cannibalism in the latter case. Even though the density of victims decreases to zero when β becomes very large, the per capita energy gain from cannibalism at a given size turns out to converge to a constant value. The reduced victim density appears to be exactly balanced by the increased cannibalistic attack rate, which scales with β.

With a gain from cannibalism, the asymptotic value of x_{max} when β becomes very large depends on δ. For example, with $\delta = 0.03$ this value is $x = 156.89$, and with $\delta = 0.04$ it is even larger (cf. Fig. 3d and e). A heuristic explanation of this is that if δ is small, many victims will be killed while they are still very small and hence have low energetic value. Cannibals with a higher δ 'spare' the smallest victims, but consume them when they have become more nutritious. Thus, at the same overall rate of killing, the gain from cannibalism is larger with a larger δ because the captured victims are larger.

The fact that the asymptotic value (for $\beta \to \infty$) of x_{max} increases with δ implies that there is a critical value of δ, for which the maximum size in the population equals the maximum size for planktivory (x_p). A population with a maximum size so small that it is in the planktivory niche ($x_{max} < x_p$) we characterize as 'stunted'. Above this critical δ the curve of x_{max} intersects with x_p at some value of β. For example, with $\delta = 0.042$ this intersection occurs at $\beta = 0.6555$ (Fig. 3c). Although in Fig. 3d it seems that with $\delta = 0.04$ the maximum size approaches an asymptotic value below x_p, it intersects with x_p at $\beta = 3.40$. What happens after intersection with x_p is the subject of the next paragraph.

Result 4. Whereas with $\delta = 0.1$ and 0.05 the intersection with x_p merely increases the slope of the equilibrium curve, with $\delta = 0.042$ and 0.04 the intersection is followed by a fold bifurcation (Fig. 3). In the latter two cases, the fold bifurcation occurs at $\beta = 0.7131$ and 6.139, respectively. In both cases, the maximum size at the fold bifurcation is just above the maximum size in the planktivory niche, $x_{max} \approx 163$. There is a second fold bifurcation at a lower β, and in between the two folds there are three equilibria for each parameter combination.

If the size distribution of a population extends into the piscivory niche (i.e., $x_{max} > x_p$) we say the population is in the 'piscivorous' state, as opposed to the stunted population state in which piscivory (i.e., cannibalism) is only a minor contribution to the energy budget of individuals (e.g., Fig. 2a). Fig. 3 shows that the transition from the stunted to the piscivorous population states can be associated with a fold bifurcation. An important consequence of the occurrence of fold bifurcations in the equilibrium curve is bistability. That is, for a given set of parameters (i.e., a given species in given conditions) the population can be in either one of the two states; stunted or piscivorous. In the next section we study the stunted and piscivorous states in more detail, and address the question of what determines whether the intersection of x_{max} and x_p is followed by a fold bifurcation or not.

4.2. Comparison of 'stunted' and 'piscivorous' population states

In the regions with bistability (Fig. 3c and d) numerical integration of the model (Table 3) shows that the equilibria at the upper branch, corresponding with piscivorous population states (i.e., largest x_{max}), are stable near the left fold bifurcation only (Section 4.4). Equilibria at the middle branch (i.e., intermediate x_{max}) are found to be always unstable, while equilibria at the lower branch, corresponding to stunted population states, turn out to be stable. In this section we therefore restrict ourselves to comparing stunted population states from the lower equilibrium branch with the stable, piscivorous equilibrium states near the left fold bifurcation at the upper branch.

For a specific choice of the parameters in a region with bistability ($\delta = 0.04$ and $\beta = 0.7$, cf. Fig. 3d) we compare the stunted and the (upper) piscivorous population states ($x_{max} = 158.1$ and 295.4 mm, respectively). Fig. 4 shows different aspects of the size structure of the two states. First of all, the size distribution (Fig. 4a) is U-shaped in both cases but obviously wider in the piscivorous state. The total biomass of the structured population in the piscivorous state is approximately twice the total biomass of the population in the stunted state (not shown). Fig. 4a shows that this difference is associated with (i) a larger density of small individuals in the piscivorous state, and (ii) the existence of individuals in the piscivory niche. The aspects (i) and (ii) are not independent since more individuals in the piscivory niche leads to a higher population fecundity, and thus to a higher inflow of small individuals.

The different population size distributions in the two equilibrium states give rise to different size-specific, cannibalistic mortality rates (Fig. 4b). In the piscivorous

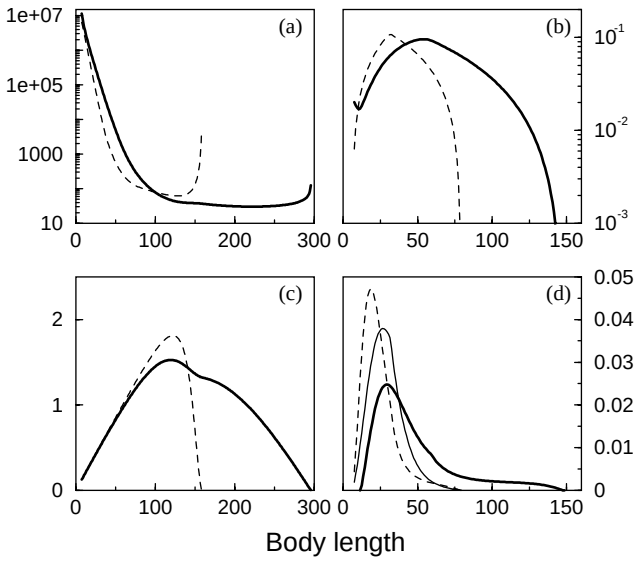

Fig. 4. Comparison of the population structure in the stunted (dashed curves) and piscivorous (solid curves) equilibrium states, for $\delta = 0.04$ and $\beta = 0.7$ and other parameters as in Table 1. (a) Population size distribution $n(x)$. (b) Cannibalistic mortality rate $\mu_c(x)$. (c) Growth rate $g(x)$. (d) Relative distribution $V(x, y)$ of ingested victim mass over victim length (horizontal axis is victim length y), as found in the diet of three types of individuals. Dashed: the largest cannibals in the stunted population (i.e., $x = 158.1$ mm). Thick solid: the largest cannibals in the piscivorous population ($x = 295.4$ mm). Thin solid: cannibals of length $x = 158.1$ mm in the piscivorous population. The distribution is scaled such that the area under the curve equals one.

state the peak occurs at a higher victim size than in the stunted state ($x = 54.5$ versus 31.5 mm, respectively). The magnitude of the peaks are comparable in both states (0.096 and 0.108 day^{-1} in piscivorous and stunted, respectively). Yet the shift to larger victim sizes gives cannibals in the piscivorous state a much larger gain from cannibalism than in the stunted state. Even if we discount the effect of the higher victim density in the piscivorous state, the expected benefit from cannibalizing a single individual is higher in this equilibrium (see below and Fig. 5). This effect is entirely due to the fact that cannibals 'spare' victims until they are more nutritious.

On the 'stunted' branch of the equilibrium curve the resource density approaches the carrying capacity as β is increased (see result 3), whereas on the 'piscivorous' branch the resource remains relatively low (Fig. 6). In a stunted population the density of alternative resource is therefore higher than in a piscivorous population. A consequence of this is that juveniles grow faster in the stunted state than in the piscivorous state (Fig. 4c), despite the fact that in the latter case the diet is supplemented with cannibalistic food.

In order to compare the size-specific harvesting by cannibals in the stunted and piscivorous population states, we define a function that describes the length distribution of the conspecifics in the stomach of a cannibal of length x, denoted by $V(x, y)$ where y refers to victim length,

$$V(x,y) = \frac{T(x,y)w(y)n(y)}{\int_{x_b}^{\infty} T(x,z)w(z)n(z)\,dz}.$$

Note that the distribution is defined as a relative frequency (i.e., area under the curve equals one), and is weighted by victim mass. Fig. 4d shows the distribution of ingested conspecific prey mass over victim length for three types of individuals. It shows that for the largest cannibals in both population states the bulk of the ingested conspecific prey mass comes from victims that are smaller than 50 mm: 96% in the stunted state, and 69% in the piscivorous state. Yet in the piscivorous state the victim distribution is wider and the peak occurs at a larger victim length than in the stunted state. Even cannibals of the same size take larger victims in the piscivorous state than in the stunted state. This reflects that on average cannibals 'spare' victims until they have reached a larger size in the piscivorous state. Of course this is not the result of a choice of the cannibals but is entirely due to the different population size distributions in the two states. Combined with the higher victim density (Fig. 4a), this explains the larger ingestion rate from cannibalism and, eventually, the larger growth rate of cannibals in the piscivorous state than in the stunted state (Fig. 4c).

4.3. Costs and benefits of cannibalism

To quantify the verbal argument of 'sparing' victims by cannibals, we compare the costs and benefits of cannibalism for equilibria at the different values of δ and β shown in Fig. 3. (Diekmann et al., 2003) derive expressions for the costs and benefits in case only adult individuals cannibalize juvenile conspecifics. We follow their derivation, adding corrections for the fact that in our model cannibalism by juvenile individuals can take place as well. Consider a newborn individual. One effect of cannibalism is that its survival probability is reduced, such that the survival probability up to a specific age is always lower in the presence of cannibalism. Let $S_c(a)$ denote the probability that the newborn individual has escaped cannibalism upon reaching age a. $S_c(a)$ can be calculated analogously to (8) by integration of the ODE:

$$\frac{dS_c}{da} = -\mu_c(x(a))S_c(a), \tag{32}$$

with initial condition $S_c(0) = 1$. Note that in this ODE we only take into account the cannibalistic mortality $\mu_c(x(a))$. The costs of cannibalism can be represented by the probability that the newborn individual at some moment in its life falls victim to a cannibal (and hence does not die of background mortality). These

costs equal

$$1 - S_c(\infty). \tag{33}$$

Considering a newborn individual the benefits can be represented by the expected per capita biomass loss due to cannibalism, given as

$$\int_0^\infty \mu_c(x(a))w(x(a))S(a)\,da.$$

The value of this integral, and those following below, can be calculated using the approach presented in Section 3. This expected biomass loss to cannibalism can be expressed in terms of the quantity of food which is required for the production of a single offspring, that is, in units of

$$c_p = \frac{c_r(1-\kappa)c_a}{\lambda x_b^3}\,\mathrm{g}.$$

In this expression, c_a converts the amount of ingested biomass into the amount of assimilated biomass, $1-\kappa$ equals the fraction of assimilated energy that an adult spends on reproduction and c_r represents the conversion efficiency with which offspring with weight λx_b^3 are produced from the energy allocated to reproduction. Since cannibalism by juvenile individuals occurs as well, not all biomass loss due to cannibalism is directly converted into new offspring. To correct for juvenile cannibalism we calculate the fraction of cannibalistic biomass loss ingested by juveniles as

$$C_j = \frac{\int_0^{a_j} F_c(x(a))S(a)\,da}{\int_0^\infty F_c(x(a))S(a)\,da}, \tag{34}$$

in which $F_c(x(a))$ represents the cannibalistic assimilation rate:

$$F_c(x) = c_a \frac{\gamma_c(x)}{1 + H(x)\gamma(x)}$$

(cf. (1) and (2)) and a_j is the age at which the maturity threshold is reached, i.e.,

$$x(a_j) = x_f.$$

Taken together, the benefits of cannibalism in terms of newly produced offspring equal

$$\Phi = c_p(1 - C_j)\int_0^\infty \mu_c(x(a))w(x(a))S(a)\,da. \tag{35}$$

We refer to the difference between the benefits and costs of cannibalism $\Phi - (1 - S_c(\infty))$ as the *net benefit*. This net benefit measures the balance between the additional reproduction and additional mortality due to cannibalism. It should be noted that this quantity only measures instantaneous effects of cannibalism; the indirect effect of additional growth is not included, but may be important since growth influences mortality, food intake and fecundity.

Fig. 5a depicts the net benefit of cannibalism for equilibria at the different values of δ and β shown in Fig. 3. First, this figure shows that the net benefit is always negative. Second, it shows that the net benefit sharply decreases from zero and reaches a minimum for values of β around 0.05. Third, for higher values of β the net benefit ultimately approaches zero, but remains significantly lower if $\delta = 0$. Fourth, if the equilibrium curve is folded, the figure clearly shows that in the piscivorous state the net benefit is close to zero but significantly higher than in the stunted state.

Obviously, for $\beta = 0$ both costs and benefits equal 0. As Fig. 5c shows, for low values of β up to 80% of all biomass loss due to cannibalism is ingested by juvenile individuals. These juvenile individuals do not produce offspring, but use the energy for the development of reproductive organs (see Section 2). In terms of new offspring the benefits of cannibalism are therefore low, while the costs in terms of the expected reduction in survival are increasing. Even though the net benefit in terms of additional reproduction and mortality is negative, cannibalism can be seen as a positive density-dependent mechanism for these values of β on the grounds that a cannibalistic population (i.e., one with $\beta > 0$) can persist at lower values of alternative resource than a non-cannibalistic population (i.e., one with $\beta = 0$; see Fig. 5b). This positive effect apparently operates more strongly for larger values of δ and is entirely due to the indirect benefit of cannibalism increasing juvenile (and adult) growth.

For larger values of β the density of alternative resource needed for persistence of a cannibalistic population is always larger than the resource density required by its non-cannibalistic counterpart (Fig. 5b). Hence, cannibalism acts as a negative density-dependent mechanism. The higher resource densities observed for higher β values imply that the net benefit of cannibalism in terms of additional reproduction and mortality is necessarily negative. This negative net benefit becomes negligible, however, with a piscivorous population state for very high values of β. For these values of β adult reproduction on the basis of alternative resource intake becomes negligible (results not shown). Hence, the model approximates a situation in which the alternative resource is used for juvenile growth and development only, while adults have an exclusively cannibalistic diet.

Overall Fig. 5a shows that the net benefit of cannibalism is larger if δ is larger, that is, if cannibals 'spare' victims until they have become more nutritious. Interestingly, this effect can also occur due to the population size distribution alone, as is shown for the cases with bistability (Fig. 5a). Here, a wider size distribution implies a larger net benefit. The occurrence of bistability and the associated two fold bifurcations suggests the presence of a positive feedback mechanism. A likely candidate for this mechanism is the fact that the

benefits of cannibalizing a large victim are higher in the sense that it leads to a higher production of new offspring as well as to increased growth of the cannibals, which in turn means that they will capture even larger victims. Since the piscivorous state depends on a high population fecundity, a high net benefit may be essential in maintaining this population state. In the piscivorous state even adult individuals are increasingly likely to become a victim of cannibalism (Fig. 5d). In comparison with the stunted population state, the piscivorous state is also characterized by that a larger fraction of the cannibalistic intake is ingested by juvenile individuals (Fig. 5c). Hence, cannibalism by both large, adult and small, juvenile individuals is higher in the piscivorous state at the expense of cannibalism by large juveniles and small adults.

4.4. Comparison with the EBT method

With the EBT method we have examined dynamics of our model for specific parameter values (e.g., Fig. 6). First of all, the congruence of the two methods confirms the validity of the continuation method laid out in Section 3. Second, the EBT method allows us to judge the local stability of the equilibria in different parts of the equilibrium curve. Close to the left fold bifurcation (e.g., $\beta_{fold} = 0.519$ in Fig. 3d), equilibria on the upper branch are stable whereas equilibria on the lower branch from the fold bifurcation (i.e., intermediate x_{max}) are not. This behavior is consistent with the exchange of stability which generically occurs at fold bifurcations (Hale and Koçak, 1991).

Fig. 6 also shows that the piscivorous equilibrium destabilizes at $\beta \approx 0.755$ and that a limit cycle exists. The amplitude of the cycle increases with β and the cycle disappears at $\beta \approx 0.875$. Beyond this point trajectories started from the piscivorous state end up near the stunted state. With other choices of parameters a similar pattern was found (results not shown).

4.5. Robustness

We tested the robustness of our results by trying many different parameter combinations. Different values of ξ, μ_0, K and x_p all gave the same qualitative pattern. An interesting result is that when x_p is smaller, the δ-range of bistability is larger (and shifted to higher δ).

5. Discussion

5.1. Population dynamical consequences of cannibalism

It is well known that cannibalism may induce alternative stable states in structured population models (Botsford, 1981; Fisher, 1987; van den Bosch et al., 1988; Cushing, 1991). Fisher (1987) has found bistability in a discrete-time model of a fish population with size-dependent cannibalism and competition. The ultimate size that individuals reach differs between the two stable states. Interestingly, in the equilibrium with large individuals the population density is low whereas in the equilibrium with small individuals the density is high, a result opposite to our results. The 'stunted' state in his model is maintained by severe intracohort competition among young-of-the-year (YOY) individuals, which retards their own growth at high densities. Cannibalism is restricted to 1-year-old individuals on YOY and increases with the body size of cannibals. In this model, bistability occurs only if the rate of cannibalism increases sufficiently abruptly with cannibal length (Fisher, 1987). This can be understood as follows. A lower YOY density leads to less competition and hence larger, but fewer, cannibals next year. If the rate of cannibalism increases sufficiently rapidly with cannibal length, this outweighs the effect of the reduced number of cannibals, and the net effect of a lower YOY density is an increased YOY mortality. Thus, a sufficiently strong dependence of cannibalistic attack rate on cannibal body size leads to a positive feedback loop between current and next year YOY density, which can induce bistability.

A major conceptual difference between the way cannibalism is modeled in Fisher's model and our model concerns the energy gain from cannibalism. Cannibalism in the model of Fisher (1987) can be considered infanticide (Hausfater and Hrdy, 1984) because it does not provide a direct energy gain. Consequently, individual growth and fecundity are not directly affected by cannibalism, only indirectly through competition.

In our model the energy gain from cannibalism creates a positive feedback loop via fecundity and growth. Basically, a high victim density may result in a large intake rate from cannibalism, leading to high fecundity, which in turn leads to a high victim density. The immediate effect of a large intake rate on instantaneous fecundity is complemented by an indirect effect via individual growth. A high intake results in a high individual growth rate, and hence large body sizes. Increased body size strengthens the feedback loop in two ways which relate to absolute and relative body size, respectively. First, with absolute body size the handling time per unit of prey mass decreases and the maximum cannibalistic attack rate increases (Table 2). Consequently, the intake rate generally increases with body size. Since fecundity is proportional to the intake rate (4), it also increases with body size. Second, the effect of relative body size is that larger individuals exploit their victims at a larger size and hence more efficiently. The net benefit of cannibalism is higher in the piscivory state than in the stunted state (Fig. 5), because in the

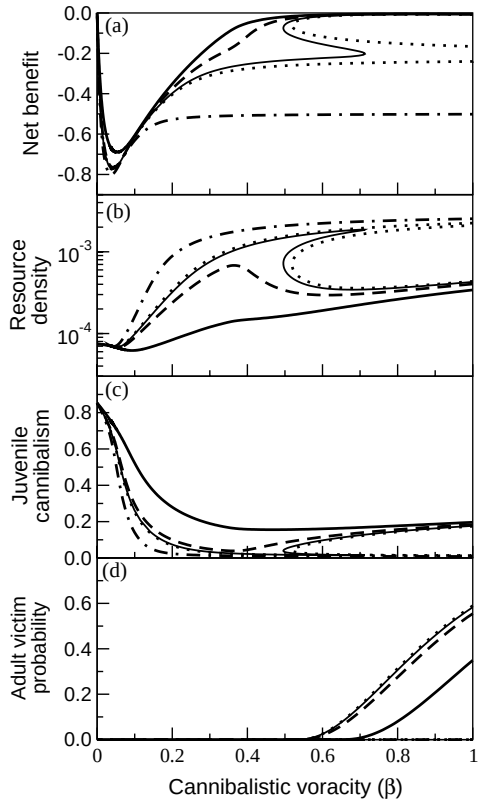

Fig. 5. (a) The net benefit of cannibalism as a function of the cannibalistic voracity β. The net benefit equals the difference between the cannibalistic benefits, i.e. the expected number of offspring per newborn individual resulting from cannibalism and the cannibalistic costs, i.e. probability that an individual during its lifetime falls victim to a cannibal (see Section 4.3). (b) Resource density. (c) Fraction of the biomass loss to cannibalism, which is ingested by juvenile individuals. (d) Probability for a just maturing individual to die of cannibalism during its adult life. Thick solid: $\delta = 0.1$; Dashed: $\delta = 0.05$; Thin solid: $\delta = 0.042$; Dotted: $\delta = 0.04$; Dot-dashed: $\delta = 0.0$.

piscivory state cannibals 'spare' victims until they have become bigger. We refer to this effect as the 'Hansel and Gretel' effect because, to our best knowledge, that tale is the first account of the idea to postpone cannibalism until the victim has become more nutritious. The Hansel and Gretel effect adds to the positive feedback loop and is hence one of the mechanisms responsible for the observed bistability.

Fisher's (1987) model and our model are examples of two different mechanisms that may give rise to bistability in cannibalistic (or infanticidal) populations. In Fisher's model the feedback between cannibalistic mortality and competition for food plays a crucial role, whereas in our model the energy gain-mediated feedback between victim density, fecundity and growth is essential. Other authors have reported bistability through cannibalism as well (Botsford, 1981; van den Bosch et al., 1988; Cushing, 1991, 1992). The model proposed by Botsford (1981) incorporates infanticide and the mechanism of bistability is similar to the one in the model of Fisher (1987). The models of van den Bosch et al. (1988) and Cushing (1991, 1992) deal with cannibalism with an energy gain. The positive feedback between population density and per capita fecundity or survival is the driving force of bistability in these models. In conclusion, we argue that the different reports of multiple equilibria in structured models of cannibalistic populations fall into two groups, one that deals with infanticide and one that incorporates the energy gain from cannibalism.

Our model belongs to the second group. There are, however two important differences with the models of van den Bosch et al. (1988) and Cushing (1991, 1992). The first and most important is that in our model the size-dependent nature of cannibalism introduces an additional feedback, by increasing the net benefit of cannibalism as the size distribution of cannibals shifts to larger sizes (the Hansel and Gretel effect), which has not been identified before. Second, in our model we did not find the life boat effect. That is, we found no subcritical bifurcation at the extinction threshold when the carrying capacity was decreased, although we tried a wide range of values of the parameters β, δ and μ_0. Of course we cannot rule out the possibility of the life boat effect for all parameter combinations, but at least for values that are relevant for perch and possibly other piscivorous fish it does not occur.

In summary, the new ecological mechanism we found is the Hansel and Gretel effect. It amounts to a feedback mechanism that depends on the population size distribution, size-dependent cannibalism and individual growth: The net benefit of cannibalism increases as the size distribution of cannibals shifts to larger sizes. Positive feedback may arise from the positive mutual effect of the gain from cannibalism on individual growth. The feedback is amplified by the positive relation between body size and fecundity. The novelty in our model that allows for this mechanism to emerge is the combination of a number of elements that had already been studied before, but not in conjunction: energy gain from cannibalism, size-dependent cannibalism and food-dependent growth. If any of these elements is lacking, the Hansel and Gretel effect cannot be found.

Based on empirical data (Mittelbach and Persson, 1998; Persson et al., 2000) we assume in our model that a cannibal cannot capture conspecifics with body lengths smaller than a fraction δ of its own length. We can compare the results with different values of δ in the light of the 'Hansel and Gretel' effect. A cannibalistic population with a higher value of δ consumes on average larger victims, resulting in a higher net benefit of cannibalism (Fig. 5). If one would start with a population in the piscivorous state (e.g., with $\delta = 0.042$, $\beta = 0.6$) and if by some process the range of potential victims would be enlarged by decreasing the lower limit

of the cannibalism window, then at some critical point (in this case, $\delta = 0.033$) the piscivorous state would disappear through a fold bifurcation, leaving the population in the stunted state. This scenario illustrates the crucial role of postponing cannibalism for the maintenance of the piscivorous population state. As an example of such a process, removal of submerged vegetation reduces the opportunity of YOY to hide from cannibals and may effectively lead to a lower δ. Alternatively, individuals with a lower δ (but otherwise the same) have larger access to food; one might expect, therefore, that natural selection favors individuals with lower δ, resulting in evolution toward the fold bifurcation.

In our discussion we frequently make use of the distinction between cannibalism and infanticide, based on the presence or absence of an energy gain to cannibalism, respectively. From a modeling perspective, ignoring the energy gain from cannibalism can be a welcome simplification, which may explain its frequent usage (e.g., Hastings, 1987; Costantino et al., 1997; van den Bosch and Gabriel, 1997; Briggs et al., 2000). From a biological point of view infanticide may be a reasonable approximation to cannibalism. However, our results indicate that 'infanticide', in the form of a small or even negligible gain from cannibalism, may be the result of population dynamics, rather than a generic aspect of the species under study. In our model, if the cannibalistic population is in the 'stunted' state the energy gain from cannibalism plays an insignificant role. However, perturbing the system may cause the population to reach the piscivorous state, a phenomenon which would have been impossible to predict under the assumption of infanticide. Thus, a model that assumes infanticide as a stand-in for cannibalism may miss some interesting results.

5.2. Continuation versus simulation

In Section 3 we have described a method for numerical continuation of a physiologically structured population model with a complex (infinite dimensional) interaction environment. In Section 4, we have shown results obtained with this method. We think our results clearly illustrate how using this method can increase the level of understanding about the dynamics of a structured population model, as compared to studying simulations alone. First, since both stable and unstable equilibria can be continued, two fold bifurcations could be localized. Moreover, continuation revealed that both fold bifurcations lie on the same equilibrium curve (although the second fold bifurcation may disappear to infinity).

Second, Fig. 6 suggests that from numerical simulations alone one might conclude that piscivorous equilibria exist only in a small interval of parameter values, and do not exist at high values of the cannibalistic voracity (β). Our continuation study shows, however, that the equilibria do exist but that they are unstable after a limit cycle arises.

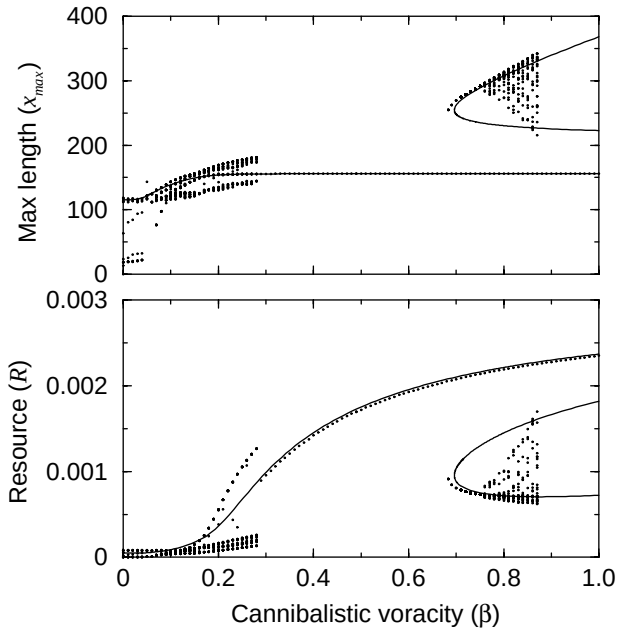

Fig. 6. Comparison of continuation results with results obtained with numerical simulations with the EBT method. $\delta = 0.03$.

Third, the fact that we study equilibria allowed us to use the conditions for equilibrium to interpret observed patterns. We applied this in the explanations of results 1 and 3 (see Section 4.1). It allowed us to compare biologically meaningful summary statistics, such as the cannibalistic net benefit, over the entire range of parameter values. The latter would have been impossible in simulations of our model since equilibria are unstable for large portions of parameter space (e.g., Fig. 6), and the interpretation of 'net benefit' in the context of population cycles is a lot more complicated than in the case of equilibria. In short, the discovery of the 'Hansel and Gretel effect' as an explanation of bistability in cannibalistic populations has to be attributed to this new method.

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