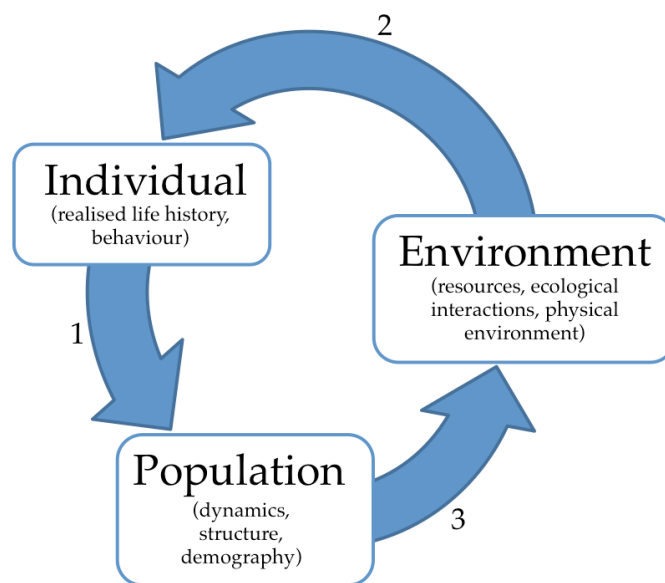


Ecology and dynamics of size-structured populations

STRU, Master 2 Ecologie-Biodiversité-Evolution (EBE)



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November 14, 2012

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1 Introduction

A struggle for existence inevitable follows from the high rate at which all beings tend to increase. (...) As more individuals are produced than can possibly survive, there must in every case be a struggle for existence, either one individual with another of the same species, or with the individuals of distinct species, or with the physical conditions of life. Darwin (1859), page 90¹

1.1 The struggle for existence

Darwin (1859)'s theory of evolution by way of natural selection is, in retrospect, a very ecological vision of evolution. As the driving force of evolutionary change, Darwin identifies the "struggle for existence", which he considers to be the natural extension of the doctrine of Malthus to the natural world. He considers this struggle for existence to be the logical result of two basic observations: the tendency of populations to grow exponentially ("the principle of geometrical increase"); and the tendency of exponentially growing populations to be kept in check by ecological interactions such as food depletion, predation or epidemics. Without heritable variation, the struggle for existence simply results in a regulated population, either at a stable equilibrium or fluctuating. Evolution kicks in as soon as heritable variation between individuals of the same population exists. Allowing for heritable variation, some lineages will be more efficient at exploiting the resources or escaping their natural enemies, and are likely to outcompete the less efficient types in the population. Darwin's term for the

¹Darwin (1859) page numbers refer to the Modern Library Paperback edition, 1998

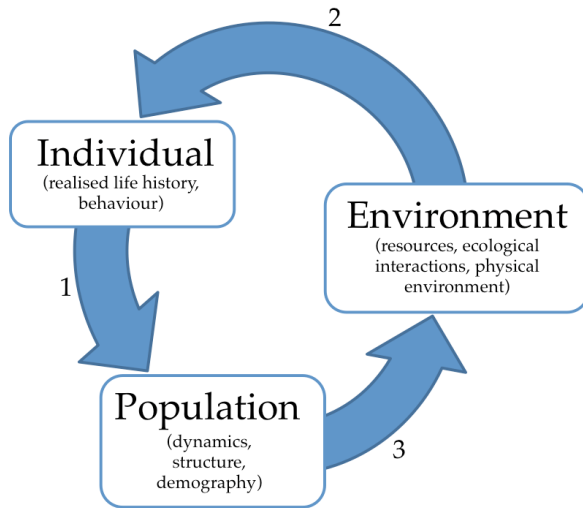


Figure 1: The environmental feedback loop. **Arrow 1:** Population dynamics are governed by the demographic properties of individuals, such as fecundity, survival, individual growth, migration rate. **Arrow 2:** These individual properties depend, in part, on the state of the environment (food density, abundance of predators, temperature, etc). The influence of the environment on individual vital rates and life history is called a reaction norm. **Arrow 3:** Collectively, individuals influence the environment, e.g., through exploitation of resources (whereas single individual has a limited impact on its environment).

latter process, "survival of fittest", is less revealing of the ecological nature of the evolutionary process than the "struggle for existence".

The idea of the *environmental feedback loop*, the central theme of this text, and described in the next paragraph, is fully analogous to the Darwin (1859)'s description of the struggle for existence.

1.2 The environmental feedback loop

The idea of the environmental feedback loop (Fig. 1) is the following. Individuals within a population (or a community) interact with each other in direct and indirect ways. Usually, in population biology, this is modelled by direct and indirect density dependence. Density-dependence can be introduced in many different (ad hoc) ways; a catalogue would be impossible and unuseful. Yet for certain (theoretical) purposes, it may be useful to have a generic way to represent and model such density dependence; which could pave the way to the development of more general concepts, theories and numerical methods. For example in the theories of adaptive dynamics and of physiologically structured populations, a general concept of density dependence is very useful (as illustrated further on in this document).

The idea proposed by Odo Diekmann and his co-authors (Diekmann et al., 1998, 2001, 2010), is to make an explicit distinction between the population dynamic behaviour of individuals (for given environmental conditions) and the dynamics of those environmental conditions. Connecting these two parts of the system results in a feedback loop since the behaviour of individuals (and their dynamics) depends on the environmental conditions (e.g., food abundance), whereas those conditions depend on the individuals that use the environment (e.g., through consumption) (Fig. 1).

The idea is to define the "environment" in such a way, that, knowing the environment, the individuals can be considered independently from each other. That is, if the environment is known, the population dynamically relevant behaviour of an individual (rates of reproduction, growth, survival, migration) can be known as well. All information about density dependence is hence captured in the definition of the environment and how the environment and the individuals mutually influence each other. This means that in addition to "individuals" and "environment", two processes need to be specified: how do the behaviour

and life history of individuals depend on the environment; and how does the environment change in response to the (collective) behaviour of individuals. The first process can be seen as a reaction norm, whereas the second process, referred to as the population impact on the environment, depends not only on the quality of the individuals (in terms of their age, size, location, trait values, etc) but also on the quantity of individuals. Whereas a single individual often has negligible impact on its environment, a population or community of individuals generally has a strong impact (possibly resulting in a struggle for existence).

Mathematically, applying this idea means that we obtain a linear model for the (short-term) population dynamics. I will illustrate this with a very simple example: logistic growth. Assume an unstructured population in which individuals reproduce with rate $\rho(E)$ and die with rate $\mu(E)$, where E refers to the condition of the environment. In continuous time, the population dynamics can then be written down as

$$\frac{dN}{dt} = r(E) N \quad (1)$$

where $r(E) = \rho(E) - \mu(E)$. In other words, for any given constant environment E , the population will grow (or decline) exponentially. This corresponds to Darwin's observation that all populations tend to grow with a (high) "geometrical ratio of increase". To give a concrete example for sake of illustration, we can assume that the environment affects reproduction only, in a linear way: $\rho(E) = \rho_0 E$ and $\mu(E) = \mu_0$. This trivial choice of vital rate functions may reflect that the reproduction rate depends linearly on food density, here captured by the environmental variable E . Then to complete the model, we need an equation for the dynamics of the environment. In particular, we need to know how E depends on the state of the population. Assuming that the food dynamics are very fast, and hence that food availability is always in steady state with the current consumer population size N , we simply specify a direct relation between population size and food availability. For example:

$$E = 1 - \frac{N}{\kappa} \quad (2)$$

The assumption that E decreases as N increases reflects the struggle for existence, that is, the geometrical growth of populations leads to a deterioration of environmental conditions (resource depletion). In this case, the reduction of E with N is a model of intraspecific competition, but similar examples can be given for other ecological interactions such as interspecific competition or predation.

Of course in this particular case, the population dynamic model is more easily presented in the traditional form of the logistic growth,

$$\frac{dN}{dt} = rN \left(1 - \frac{N}{K}\right) \quad (3)$$

with $K = \kappa(1 - \mu_0/\rho_0)$ and $r = \rho_0 - \mu_0$. However, making the environmental feedback loop explicit has the advantage of obtaining a general, linear, equation for the population dynamics. For example, when studying the model above in the context of adaptive dynamics equation, (1) can be used to find the expression for the invasion fitness, which is an extension of the function $r(E)$. This is true also for more complex models: writing the ecological model in the form of the environmental feedback loop (representing all relevant density-dependent

interactions), results in a model formulation that is naturally designed for extending it in the direction of adaptive dynamics (Diekmann, 2004).

In the context of the more complex physiologically structured populations, this representation has already paved the way for the development of general numerical techniques for the continuation and stability analysis of such models (Kirkilionis et al., 2001; de Roos et al., 2010; Diekmann et al., 2010; Diekmann and Metz, 2010). An example (not the general mathematical theory) of the latter is given below (section 2.4).

1.3 Why study structured populations?

A great deal of ecological theory is based on simple Lotka-Volterra type models, which usually assume that all individuals within a population are identical. This is obviously a very useful simplification, which has led to important results on the dynamics of populations and communities (see almost any textbook on ecology). Yet we all know that most populations are structured in different ways: spatially, sexually, by age, size, reproductive status, epidemiological status, etc. Here I focus on two kinds of population structure: spatial structure and size structure. Spatial structure is, intuitively, likely to have important implications, which are quite different for ecological and evolutionary dynamics, respectively. Ecologically, spatial structure means that individuals interact with resources and other individuals in a small, local part of the environment. In other words, the environmental feedback loop depends on the spatial location. This has been shown to affect ecological dynamics in different ways: pattern formation, dampening of population cycles, invasion waves, etc (Diekmann et al., 2000). Evolutionarily, one particular relevance of spatial structure is that it may lead to reproductive isolation of spatially separated sub-populations. For example, the classical distinction between allopatric and sympatric speciation refers to the spatial structure of populations. Spatial structure also directly influences the fitness of different types, albeit mediated through the environmental feedback loop (Ferriere and Le Galliard, 2001; Lion et al., 2011).

Body size is an important structuring variable, since many ecological properties of individuals depend strongly on their body size, according to so-called allometric scaling rules (Peters, 1983). An example of the ecological relevance of body size is provided by a typical life history of a Eurasian perch (*Perca fluviatilis*). At birth an individual has a body mass of about 2 mg and a length of 6-7 mm. After finishing its yolk sac, it starts feeding on zooplankton. When reaching several cm in length, it starts including macroinvertebrates in its diet. It matures at around 10-11 cm (around 10 g). If lucky, the individual can grow further, and soon will no longer feed on zooplankton. Instead, it continues feeding on macroinvertebrates, and will also include small fish in its diet. In fact, it can include any fish in its diet that is between about 5 and 45 % of its own body length (which means it can start becoming cannibalistic from about 10 cm in length). Perch can reach lengths up to 80 cm (in Lake Windermere for example), and weigh up to several kilograms. Clearly, the position in the food web of an individual may change significantly throughout its life time, while body mass may increase by up to a factor of 10^6 . From trophic and metabolic points of view, a newborn and an old individuals are hence quite different "organisms". This illustration should make it intuitive that size structure is likely to have implications for population dynamics.

So it is as with mountain climbing: Why do we want to model population structure? Because it is there. The existence of population structure in natural populations merits the question of what are the consequences of this structure. The consequences of size structure

and, more generally, physiological structure, on the dynamics of populations and communities has received quite some attention since the 1980s, in particular since the seminal work by Gurney and Nisbet (1985), Metz and Diekmann (1986), de Roos (1997) and Kooijman (2000). Since, it has been shown that size structure may have a wide range of dynamical consequences, the most basic of which is the emergence of generation cycles, also known as cohort cycles (Gurney and Nisbet, 1985; de Roos, 1997; Persson et al., 1998), but also alternative stable states (Persson et al., 2007), catastrophic behaviour (De Roos and Persson, 2002), ecological suicide (Van de Wolfshaar et al., 2008), population dynamics-induced size bimodality (Claessen et al., 2000), and others. This work has shown that the range of dynamical behaviour of size-structured populations is richer than that of unstructured models such as the Lotka-Volterra type models. This is no surprise, of course, as size-structured models are more complex and, in particular, they feature more complex and higher-dimensional environmental feedback loops than unstructured models. While this complexity explains the interesting, new types of population dynamics observed in these models, the complexity also poses some limitations. Studying models of multiple, interacting size-structured populations quickly becomes exceedingly difficult. Yet the qualitatively different types of dynamics predicted by size-structured models leads to new research questions as to its occurrence and importance in natural populations. Physiologically structured population models, described below, allow us to pose a new kind of questions in population ecology. Turning this around, for certain questions relevant to ecology and evolution, the use of structured populations is necessary. In section 2 I argue for this point of view with some examples of research on size-structured populations.

The case of genetic population structure (meaning heritable variation between individuals), is of course a special case, as suggested above (section 1.1). Without genetic structure the struggle for existence results in population regulation only. With such structure, it will naturally induce evolutionary dynamics in conjunction with the ecological dynamics. Genetic structure, in the sense of genotypical differences between individuals, is also important for a range of dynamical phenomena such as speciation, hybridisation, reproductive isolation, and ecological polymorphism, including ecotypes. Genetical structure may depend to a large extent on the type of inheritance (sexual reproduction, haploid/diploid, single/multi-locus traits, dominance, etc). It may also interact with spatial structure, such as for example local adaptation and maladaptation in spatial gradients.

2 Size-structured populations

2.1 Different types of population cycles

Murdoch et al. (2002) distinguish two frequently occurring types of population cycles: predator - prey cycles and generation cycles (also referred to as single-generation cycles). **Predator-prey cycles**, such as the cycles predicted by the Lotka-Volterra model, are a well-known kind of population dynamics that results from time delays that are inherent in the predator-prey interaction: a high prey density results in predator growth which means that in the future, the predator will have a high abundance. This means that high predator density occurs with a certain delay after a high prey density. Similarly, once the prey population is depleted by the predator, the predator population will decline, but this will take some time. Finally, once the predator is reduced to low densities, the prey population will grow, but again, this pop-

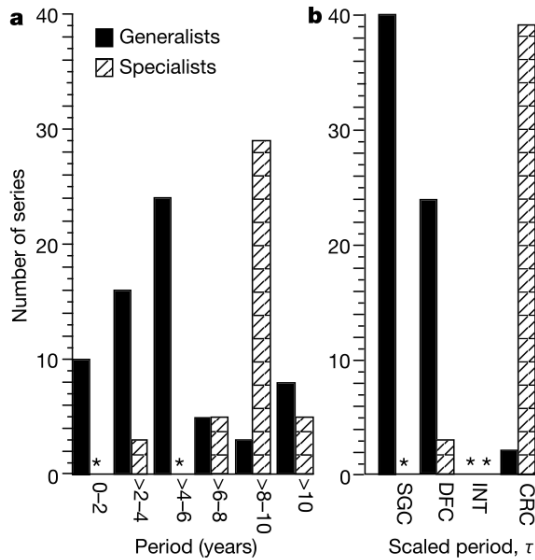


Figure 2: Cycles classified by period. Asterisk indicates zero series in the class. a, Number of cyclic populations with various periods, in years. b, Distribution of cycles among classes defined by scaled period. SGC, single-generation cycles ($\tau = 1$); DFC, delayed-feedback cycles ($2 \leq \tau \leq 4$); CRC, consumer-resource cycles (period in years $\geq 4T_C + 2T_R$). No cycles fall in the intermediate class (INT) between single-species and consumer-resource cycles. From: Murdoch et al. (2002)

ulation growth takes several generations. The mechanisms underlying predator-prey cycles requires at least several generations to complete a cycle period.

By contrast, **generation cycles** are population cycles with a periodicity of a single to two generations only. Such population cycles result from direct density dependence between differently sized individuals of the same population (or individuals in different stages such as juveniles and adults).

(Murdoch et al., 2002) argue that predator-prey cycles are likely to be present in **specialist** predators. Specialist predators and their prey form tightly coupled consumer-resource interactions. By contrast, **generalist** predators depend much less on the density-dependence of their prey, since recruitment to the prey population is unrelated to prey abundance. This weakens the link between prey and predator dynamics. Rather, they argue, generalists should be characterised by intraspecific density dependence that arises from interactions between individuals within the generalist population.

The two types of population cycles can theoretically be distinguished by the cycle periodicity (Murdoch et al., 2002). Predator-prey cycles are expected to have a periodicity of at least $4T_C + 2T_R$, where T_C and T_R are the maturation times of the consumer and resources, respectively. Intuitively, this can be understood by considering the classical Lotka-Volterra model and its predator-prey cycles. Each cycle consists a phase of exponential predator growth, depleting the prey population; followed by a phase of predator exponential decline; followed by exponential prey growth. Each bit of exponential growth or decline requires at least a few generations to complete. By contrast, generation cycles have a periodicity of one generation (exceptions include 0.5 generations or 2 generations per cycle).

(Murdoch et al., 2002) analyse a large number of time series from cyclic populations in nature and show that almost all cycling, generalist consumers examined have periods that are consistent with one-species dynamics Fig. 2. Thus generalist consumers indeed behave as if they were one-species populations, and a one-species model is a valid representation for generalist population dynamics in many-species food webs.

2.2 Physiologically structured population models

The theory of physiologically structured population models takes into account that individuals may differ from each other by their physiological state (e.g. age, size, body condition) (Metz and Diekmann, 1986; de Roos, 1997; de Roos and Persson, 2001; Diekmann et al., 2001). In this theory, physiological development (e.g. growth, maturation) is assumed to depend on the current state of the environment in terms of food availability, abundance of competitors and predators, etc. By specifying how, in turn, the environment is affected by the action of the entire population (Fig. 1) a model of population dynamics with plastic, density-dependent life history is obtained. The theory of PSP models is particularly well-suited to study the interaction between population dynamics and life history (Persson et al., 1998; Claessen et al., 2000, 2002).

We can define **life history** as the history of the physiological state of an individual. Variation in life history which is caused by variation in environmental conditions is referred to as **life history plasticity**. Many environmental factors that influence life history vary in both space and time. First, some of these factors (e.g. food density) interact with the population such that population fluctuations result in environmental fluctuations which feed back onto life history. In fluctuating populations, life histories of individuals born in different years can therefore be entirely different (e.g. size-dimorphism in fish: Claessen et al., 2000; Persson et al., 2003). Second, the environmental factors are often distributed heterogeneously in space (Hanski and Gilpin, 1997). Life histories of individuals living in different regions may differ even in the absence of genetic variability (e.g. lizards: Adolph and Porter, 1993; Sorci et al., 1996).

PSP models make an explicit distinction between **state variables** at different levels: the individual level (**i-state variables**); the population level (**p-state variables**) and the environment (the E variables as introduced above). To formulate a PSP model that takes into account both plastic life history and the population feedback loop, it is first necessary to decide:

- which i-state variables define an individual (e.g., age, size, sex, ...)
- which state variables define the E environment (e.g., resource abundance; abundance and possibly size distribution of competitor and/or predators; temperature, ...)

And then to define the environmental feedback loop (Fig. 1) by specifying:

- how individuals develop (in terms of the i-states) given the current state of the environment.
- how the state of the environment changes under the collective influence of all the individuals (the population)

The most frequently used formulation consists of a set of ordinary differential equations for the continuous dynamics of the i-state variables. A widely used example, the Kooijman-Metz model, is described below.

Table 1: Basic assumptions for the KM model. The weight-length relation, attack rate, digestion time, maintenance rate and mortality rate are basic (“empirical”) functions; they can be parameterised with empirical laboratory data. For starvation mortality: $x_C(R)$ is a critical length above which starvation mortality applies, defined in the main text.

Weight-length	$w(x) = \lambda x^3$
Attack rate	$A(x) = \alpha x^2$
Digestion time	$H(x) = \xi x^{-2}$
Maintenance	$M(x) = \rho x^3$
Holling type II	$F(x) = \frac{A(x) R}{1 + H(x) A(x) R}$
Total mortality	$\mu(x) = \mu_0 + \mu_s(x)$
Starvation	$\mu_s(x) = \begin{cases} s(x - x_C(R)) & \text{if } x > x_C(R) \\ 0 & \text{otherwise} \end{cases}$

2.3 The Kooijman-Metz model

The Kooijman-Metz model (referred to as the KM model) was first formulated by Kooijman and Metz (1984) to describe the population dynamics of a size-structured *Daphnia* population; see de Roos (1997) for a detailed model formulation and analysis. Since then it has been often applied to a range of species, including fish and springtails. The KM model comes in various degrees of detail, and the one below is quite detailed. The reason is that this version is more “biological”, that is, the functions describing individual-level properties (maintenance, attack rate, digestion rate, length-weight relation) can be easily interpreted and even measured in lab experiments. The presentation of the KM model also illustrates the role of the environmental feedback loop in PSP model formulation.

The KM-model describes the dynamics of a size-structured population and its unstructured resource (food) population. We assume that the physiological state of an individual is completely determined by its body length x : this is the **i-state variable**. 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_0 , and are assumed to mature upon reaching the size x_f . Reproduction is assumed to be continuous (in time) which implies that the size distribution $n(x)$ is continuous.

The functional response The food intake rate follows a size-dependent, type II functional response

$$F(x) = \frac{A(x) R}{1 + H(x) A(x) R} \quad (4)$$

Table 2: The Kooijman-Metz model: individual level functions. These functions are derived from the assumptions in Table 1.

Holling type II	$F(x) = \nu x^2 \frac{R}{R_H + R}$
Growth rate	$g(x) = \begin{cases} 0 & \text{if } c\kappa F(x) < M(x) \\ \gamma \left(x_\infty \frac{R}{R_H + R} - x \right) & \text{otherwise} \end{cases}$
Birth rate	$b(x) = \begin{cases} \beta x^2 \frac{R}{R_H + R} & \text{if } x > x_J \\ 0 & \text{otherwise} \end{cases}$
Total mortality	$\mu(x) = \mu_0 + \mu_s(x)$
Starvation	$\mu_s(x) = \begin{cases} s(x - x_C(R)) & \text{if } x > x_C(R) \\ 0 & \text{otherwise} \end{cases}$

where $A(x)$ is the attack rate, and $H(x)$ is the size-dependent digestion time per gram of prey mass (Table 1).

The attack rate is assumed to increase proportional to surface area: $A(x) = \alpha x^2$. The handling time is assumed to be determined by the digestion rate, which also increases proportional to surface area. The digestion time is the inverse of the digestion rate, so we have $H(x) = \xi x^{-2}$. Now we substitute these functions into equation (4):

$$F(x) = \frac{\alpha x^2 R}{1 + \xi x^{-2} \alpha x^2 R} \quad (5)$$

This can be rewritten as follows:

$$F(x) = x^2 \frac{\alpha R}{1 + \xi \alpha R} \quad (6)$$

and as follows:

$$F(x) = \nu x^2 \frac{R}{R_H + R} \quad (7)$$

where $\nu = 1/\xi$ and $R_H = \frac{1}{\alpha \xi}$. The composite parameter R_H is often called the half-saturation resource density, whereas νx^2 corresponds to the size-dependent maximum food intake rate.

The growth rate The energy intake rate is obtained by multiplying the food intake rate by a food-to-energy conversion efficiency c . We assume that a fraction κ of assimilated energy is allocated to growth and maintenance, and the remainder to reproduction (the "kappa-rule", Kooijman and Metz, 1984; Kooijman, 2000). The growth rate in mass is obtained by

subtracting the metabolic rate from the energy intake rate.

$$\frac{dw}{dt} = c\kappa F(x) - M(x) \quad (8)$$

Substituting the equations for the functional response and the maintenance rates gives:

$$\frac{dw}{dt} = c\kappa\nu x^2 \frac{R}{R_H + R} - \rho x^3 \quad (9)$$

which can be simplified as

$$\frac{dw}{dt} = x^2 \left(c\kappa\nu \frac{R}{R_H + R} - \rho x \right) \quad (10)$$

Assuming that the body weight scales with the cube of body length ($w(x) = \lambda x^3$) then the growth rate in *length* is

$$\frac{dx}{dt} = \frac{dw}{dt} \frac{dx}{dw} = \frac{dw}{dt} \frac{1}{3\lambda x^2} \quad (11)$$

which if we substitute equation (10), gives:

$$\frac{dx}{dt} = \frac{1}{3\lambda} \left(c\kappa\nu \frac{R}{R_H + R} - \rho x \right) \quad (12)$$

Note that the two x^2 terms have cancelled out. Again, we rewrite this equation:

$$\frac{dx}{dt} = \gamma \left(x_\infty \frac{R}{R_H + R} - x \right) \quad (13)$$

in which two new composite parameters appear: the growth rate parameter $\gamma = \rho/(3\lambda)$ and the ultimate size $x_\infty = c\kappa\nu/\rho$.

Equation (13) shows that, for a given resource density R , the growth rate decreases linearly with body length. Growth stops when an individual reaches a maximum size that depends on R , which equals:

$$x_M(R) = x_\infty \frac{R}{R_H + R} \quad (14)$$

The ultimate size x_∞ is the maximum body size attained under extremely high food conditions, that is, if the food intake rate is at its maximum for all sizes.

Reproduction The energy that is allocated to reproduction equals

$$c(1 - \kappa)\nu x^2 \frac{R}{R_H + R} \quad (15)$$

For juveniles, this energy is assumed to be used for the development of reproductive organs. For adults, this energy is converted into offspring, by dividing it by the cost to produce a single offspring. The cost to produce one offspring is assumed to be equal to the body weight of an offspring (x_0) multiplied by a conversion parameter c_r :

$$\text{cost} = c_r \lambda x_0^3 \quad (16)$$

For adults, the per capita birth rate is calculated by dividing the investment in reproduction by the energy cost of producing a single newborn:

$$b(x) = \frac{c(1-\kappa)\nu}{c_r\lambda x_0^3} x^2 \frac{R}{R_H + R} \quad (17)$$

We simplify the notation by defining a new composite parameter: $\beta = \frac{c(1-\kappa)\nu}{c_r\lambda x_0^3}$, resulting in the following equation for the reproduction rate:

$$b(x) = \begin{cases} \beta x^2 \frac{R}{R_H + R} & \text{if } x \geq x_J, \\ 0 & \text{otherwise,} \end{cases} \quad (18)$$

where x_J is the size at maturation, such that individuals smaller than x_J are juveniles and individuals with $x > x_J$ are adults.

Starvation In equilibrium individuals cannot grow beyond the maximum size $x_M(R)$, so for an equilibrium analysis we do not have to consider starvation and starvation-induced mortality. However, in population cycles individuals may go through periods of food shortage and starvation. We have to specify what happens to the energy budget when an individual is starving.

Remember that an individual's energy intake rate is $cF(x)$ (which depends on the food density R) and that its maintenance rate is $M(x)$. We have assumed that a fraction κ of the energy intake is allocated to maintenance and growth. Here we add the condition that this is the case only if this fraction of the energy intake is sufficient to cover maintenance, that is if $\kappa cF(x) > M(x)$. It follows from equation (13) that this is the case for individuals smaller than the maximum body for the given food conditions, i.e., if $x < x_M(R)$.

For a given food density we can compute a "starvation" body size, denoted by $x_S(R)$, which is the body size for which the energy intake rate exactly equals the maintenance rate. Individuals bigger than this size ($x > x_S(R)$) do not have enough energy to cover their maintenance rate, and they are hence **starving**. The size at which starvation begins equals

$$x_S = \frac{x_M(R)}{\kappa} \quad (19)$$

Note that this definition means that the maximum size is always smaller than the starvation size ($x_M(R) > x_S(R)$).

Individuals larger than the maximum size $x_M(R)$ follow a modified energy allocation rule: they use their energy first to cover maintenance, and what remains (if any) is used for reproduction. In this situation, the individual hence does not grow ($dx/dt = 0$). Its reproduction rate becomes

$$b(x) = cF(x) - M(x) \quad \text{if } x_M(R) < x < x_S(R) \quad (20)$$

Mortality The mortality rate is assumed to be the sum of a constant background mortality rate μ_0 , and a starvation mortality:

$$\mu(x) = \mu_0 + \mu_s(x) \quad (21)$$

Table 3: The Kooijman-Metz model: specification of the dynamics of p-state variables. The individual-level functions are listed in Table 2.

PDE	$\frac{\partial n}{\partial t} + \frac{\partial gn}{\partial x} = -\mu(x) n(x)$
Boundary condition	$g(x_0) n(x_0) = \int_{x_f}^{\infty} b(x) n(x) dx$
Resource dynamics	$\frac{dR}{dt} = r(K - R) - \int_{x_0}^{\infty} \frac{A(x)R}{1 + H(x)A(x)R} n(x) dx$

For individuals that are approaching the starvation size $x_S(R)$, we assume that they suffer an additional mortality rate, $\mu_s(x)$, that increases linearly with size as the starvation size is approached. The starvation mortality applies to individuals with a body size larger than a critical size $x_C(R)$ which is equal to

$$x_C(R) = x_S(R) - q_s [x_S(R) - x_M(R)] \quad (22)$$

The starvation mortality is given by:

$$\mu_s(x) = s(x - x_C(R)) \quad \text{if } x > x_C(R) \quad (23)$$

Individuals beyond the starvation size are assumed to die immediately.

The resource dynamics 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,

$$\frac{dR}{dt} = r(K - R) - \int_{x_0}^{\infty} \frac{A(x)R}{1 + H(x)A(x)R} n(x) dx \quad (24)$$

with $A(x)$ and $H(x)$ as defined in Table 1.

The individual-level model is summarized in Table 2 and the PDE formulation for the population-level model is presented in Table 3. The list of parameters and their values can be found in Claessen and de Roos (2003).

PSP models such as this one are often studied by numerical intergration (simulation). An efficient method for this is the Escalator Boxcar Train (EBT) (de Roos, 1997). In order to simulate the model, the population size distribution $n(x)$ needs to be discretised, and the EBT method does so in a natural way by keeping track of a (variable) number of cohorts; for each cohort, the EBT integrates ordinary differential equations for the cohort abundance and the i-state variables (body length x in this case). There are two typical population dynamic behaviours of the KM model: a stable equilibrium and generation cycles. Generation cycles are discussed in more detail below (section 2.6).

2.4 Equilibrium analysis of the KM model (advanced)

This section illustrates how the environmental feedback loop formulation can help doing an equilibrium analysis of a physiologically structured population model. The general theory for this method can be found in Diekmann et al. (1998); Kirkilionis et al. (2001); de Roos et al. (2010); Diekmann et al. (2010). Here I will just outline how using the environment E can be used to reduce an infinite-dimensional problem to a two-dimensional problem.

The environmental feedback loop How does all this (section 2.3) illustrates the principle of the environmental feedback loop? The interaction environment, denoted by E in the Introduction, is here defined as the resource density (R). On the one hand, knowing R , the life history of an individual is entirely specified: its growth trajectory is obtained by integration of the growth rate; its reproductive output is obtained by computing its per-capita, size-dependent birth rate; its survival curve can be obtained by integrating the size-dependent per capita mortality rate. This illustrates the statement that once E is known, individuals can be considered in isolation (despite the presence of density dependent interactions). On the other hand, the impact of an individual on its environment can also be computed, by integrating its feeding rate along its life history (see below for equations). Since this represents the impact on the environment by a single individual only, we need a measure of the total population size in order to complete the description of the environmental feedback loop (Fig. 1). A convenient measure is the total population birth rate P (that is, P is the product of the number of individuals and their total expected reproductive output). Then, multiplying the cumulative consumption rate with P gives the consumption rate of the whole population. In other words: the impact of the population on its environment (Fig. 1).

Life history as an input-output map In more mathematical terms, the above paragraph can be made explicit as follows. Elements from the individual-level model outlined above 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

$$\frac{dS}{da} = -\mu(x(a)) S(a), \quad S(0) = 1, \quad (25)$$

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

$$\frac{dx}{da} = g(x(a)), \quad x(0) = x_0, \quad (26)$$

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

$$\frac{dB}{da} = b(x(a)) S(a), \quad B(0) = 0, \quad (27)$$

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

$$R_0 = B(\infty) \quad (28)$$

Due to the occurrence of $x(a)$ in (25) and (27), (26) has to be solved first, then (25), and finally (27). Alternatively, the ODEs (25-27) can be solved simultaneously. Together, $S(a)$, $x(a)$ and R_0 define a life history. In other words, the recipe for translating a given environmental condition into the corresponding life history (arrow from "Environment" to "Individual" in Fig. 1) is by solving equations (25-27).

The next thing we need to do, is to find the return map, that is from a given life history back to the impact on the environment. For that we need two ingredients: the life history and the number of individuals (since the impact is determined by the whole population collectively). For a single individuals with a given life history, the expected, cumulative consumption up to age a , is denoted with $\theta(a, R)$. It can be calculated in parallel with (25-26) by integrating

$$\frac{d\theta}{da} = \frac{A(x(a))R}{1 + A(x(a))RH(x(a))} S(a), \quad \theta(0, R) = 0 \quad (29)$$

(cf. (4)). The total population consumption rate of alternative resource is then the product of P and $\theta(\infty, R)$. Note that for this computation P is required as an input variable, since it cannot be derived from the life history. In other words, the population impact on the environment given a certain life history (arrow from "Individual" passing through "Population" to "Environment" in Fig. 1) equals $P\theta(\infty, R)$.

A merit of this way to characterise the environmental feedback loop, is that it provides us with a low-dimensional definition of the population dynamical equilibrium, and a tool to compute this equilibrium using continuation techniques. It should be noted that the population-level model (Table 3) is an infinite-dimensional object (i.e., a continuous function). Characterising the population equilibrium with the function $n(x)$ therefore does not lend itself to numerical equilibrium analysis. By contrast, the environmental feedback loop is characterised by only two (unkown) variables: the food density R and the population birth rate P . Now observe that the population dynamic equilibrium of the KM model can be charcterised by two criteria, being the requirements that each individuals replaces itself ($R_0 = 1$) and that the resource is at equilibrium ($r(K - R) = P\theta(\infty, R)$). These are two equations in the two unknowns R and P .

Following Diekmann et al. (1998, 2010), we refer to the unkown variables $R := I_1$ and $P := I_2$ as the input variables I (where I is the vector of the input variables), and to the equilibrium conditions $R_0 - 1 := O_1$ and $r(K - R) - P\theta(\infty, R) := O_2$ as output variables O (the vector of ourput variables). Then the environmental feedback loop yields the map

$$f : \mathbb{R}^k \rightarrow \mathbb{R}^k; \quad I \mapsto O \quad (30)$$

which is referred to as the input-output map. (In this particular example, $k = 2$ since we have two input and output variables. See Claessen and de Roos (2003) for another example with $k \approx \infty$). An equilibrium can be found via an input $I^* \in \mathbb{R}^k$ for which the equilibrium and feedback conditions

$$f(I^*) = 0 \quad (31)$$

hold. The condition $f(I^*) = 0$ can now be used in numerical continuation. The continuation method of Kirkilionis et al. (2001) can trace the equilibrium as a function of one free parameter.

More recent development of the mathematical theory has enabled stability analysis of

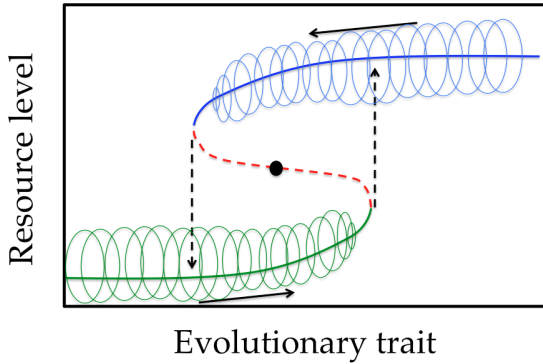


Figure 3: A schematic representation of “evolutionary cycling” caused by ecological hysteresis in a Kooijman-Metz model with a juvenile delay and an ontogenetic niche shifts (i.e., between two resources). The ecological attractors are generation cycles driven by the interaction in the first niche (blue attractor) or generation cycles driven in the second niche (green attractor). The evolutionary singular point is located on the unstable equilibrium curve connecting the green and blue attractors (black dot). The generation cycles disappear through a bifurcation close to the saddle-node bifurcations (“tipping points”). The black arrows indicate direction of evolutionary change of the trait u . The dotted arrows indicate ecological attractor shift.

the equilibrium along the traced equilibrium curve, and in particular of the detection of a Hopf bifurcation and the two-parameter continuation of the Hopf bifurcation (de Roos et al., 2010; Diekmann et al., 2010). These are the first steps towards more general numerical continuation tools for PSP models. Given the enormous contribution for the study of models based on ODEs with continuation tools such as AUTO and Content/Matcont, this is a very promising development.

Applications An application of this method is illustrated in Fig. 3 (page 16). The continuation method enables us to trace the equilibrium curve of a PSP model while varying a model parameter. Even if the equilibrium is unstable, and even if the curve folds backwards (as in Fig. 3), the equilibrium can be traced. It is clear that such an unstable equilibrium curve, in between two alternative stable states, would be impossible to find by simulation only. In Claessen and de Roos (2003) we use this method to study the influence of the size-dependent nature of a cannibalistic interaction on the equilibrium. The method allows us to detect two fold bifurcations (similar to the folded curve in Fig. 3) in the equilibrium curve. The found equilibrium curve helps interpreting the population dynamics observed in simulations (which are limited to stable equilibria and other attractors). In particular, we are able to identify the “biological” process that causes bistability in the cannibalistic model: only if the cannibals spare their smallest victims, i.e., if the victims are invulnerable to cannibalism up to a critical size, then cannibals are able to reach giant sizes. If, by contrast, cannibals are able to include even the smallest individuals in their diet, then they are bound to reach a maximum body size not much bigger than their maturation size. We called this the “Hansel and Gretel effect”.

2.5 Generation cycles

A very general result from PSP modelling is that intra-specific competition tends to cause “generation cycles”, also known as “cohort cycles” (Gurney and Nisbet, 1985; de Roos, 1997; Persson et al., 1998; Claessen et al., 2000; de Roos and Persson, 2003). This kind of population cycles is distinct from the better-known predator-prey cycles, also referred to as “consumer-resource cycles” or “delayed-feedback cycles”. The two types of population cycles can the-

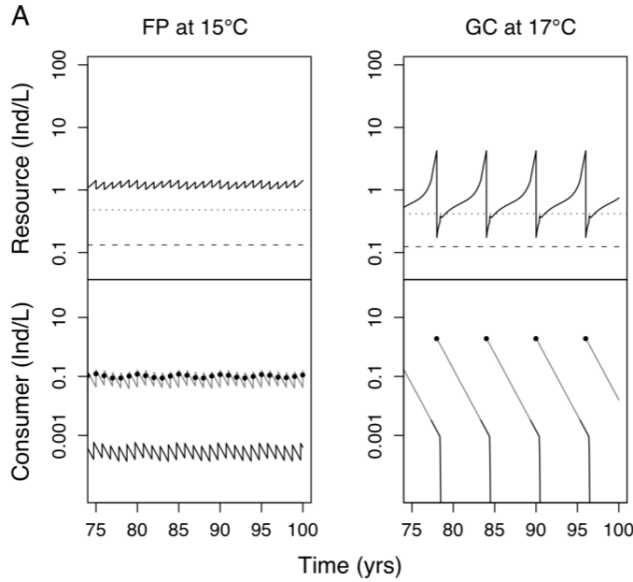


Figure 4: Population dynamics of the model of Ohlberger et al. (2011) at temperatures 15°C and 17°C (for the case without cannibalism). Top plots: predicted resource density (solid lines) and critical resource densities (defined as zero-growth resource level) for newborns (dashed lines) and mature fish (dotted lines). Bottom plots: predicted consumer density of newborns (circles), juveniles (grey lines) and adults (black lines). The left panel shows fixed point (FP) dynamics, whereas the right panel shows recruit-driven generation cycles (GC), here also referred to as single-cohort cycles. From: Ohlberger et al. (2011)

oretically be distinguished by the cycle periodicity (Murdoch et al., 2002). Predator-prey cycles are expected to have a periodicity of at least $4T_C + 2T_R$, where T_C and T_R are the maturation times of the consumer and resources, respectively. Intuitively, this can be understood by considering the classical Lotka-Volterra model and its predator-prey cycles. Each cycle consists a phase of exponential predator growth, depleting the prey population; followed by a phase of predator exponential decline; followed by exponential prey growth. Each bit of exponential growth or decline requires at least a few generations to complete. By contrast, generation cycles have a periodicity of one generation (exceptions include 0.5 generations or 2 generations per cycle).

The basic mechanism of generation cycles is an inter-generational conflict: each generation replaces its parental generation through intra-specific competition. The mechanism can be illustrated by considering an example. In Ohlberger et al. (2011) we present a model of a size-structured fish population. Under certain conditions (e.g., 17°C in Fig. 4) the populations exhibits cycles of which the periodicity corresponds to the maturation time. The figure shows that the birth of each new generation (black dots, lower panel) is followed by a sudden depletion of the zooplankton population (resource, upper panel). The adult portion of the population goes extinct rapidly, starved to death by the low resource abundance. The new generation remains juvenile for several years, during which it declines exponentially due to a constant mortality rate, and the individuals grow in size. The resource gradually increases (due to the decline of the juvenile cohort). The juveniles mature in their 5th year, and the following spring they reproduce the next generation.

The underlying mechanism of this type of cycles depends on the size scaling of the ecological properties of individuals. The basic, size-dependent functional relations in the model are similar to that of the Kooijman-Metz model (Table 1), i.e., a type II functional response, a hump-shaped attack rate on zooplankton, an increasing maintenance rate with body size, decreasing digestion time with body size. From these basic ingredients we can derive a dependent relationship, which is referred to as the "critical resource density" $R^*(x)$. As its notation suggests, this quantity is analogous to Tilman (1982)'s R^* , except that in our case it is a

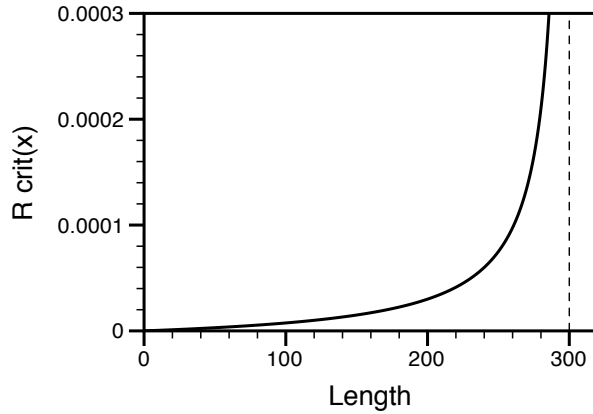


Figure 5: The critical resource density $R^*(x)$ as a function of body length x , derived from the Kooijman-Metz model. Parameters used: $x_\infty = 300$, $R_H = 1.5 \cdot 10^{-5}$. Note the asymptote at $x = 300$. It follows from the energy budget model that individuals larger than this size cannot maintain themselves at any food concentration, since energetic requirements exceed the maximum energy uptake capacity.

function of body size rather than a single measure for the whole population. As in Tilman's theory, an individual's competitive ability is measured by $R^*(x)$: the lower its value, the better the individual can deal with severely competitive situations. In the case of the fish model, $R^*(x)$ is an increasing function of body size. This results mainly from the fact that maintenance requirements increase faster with body size than the feeding rate. This result is general for PSP models that have been parametrised for particular fish species (Persson et al., 1998; de Roos and Persson, 2003; Persson and De Roos, 2006). More theoretically, we can note that the feeding rate is generally a surface-limited process and hence likely to scale with the body mass to the power $2/3$, whereas maintenance is a mass-limited process and hence likely to scale linearly with body mass (Kooijman, 2000) (even though an alternative theory postulates a $3/4$ scaling rule, Brown et al., 2004). Thus, also based on these simple theoretical considerations, $R^*(x)$ is expected to increase with body size (even under the $3/4$ scaling rule!).

Generation cycles result from the asymmetric competition between differently sized individuals, if $R^*(x)$ is sufficiently steeply increasing (or decreasing) with body size (Persson et al., 1998; de Roos and Persson, 2003). In our example, $R^*(x)$ increases with body size and hence newborns are competitively superior to adults. A sufficiently high fecundity of adults then automatically results in generation cycles.

Generation cycles occur even in more complex models that include multiple ecological interactions. Cannibalism has the potential to dampen generation cycles, because it allows adults to reduce the competition with newborns in two ways: killing newborns reduces the abundance of competitors, and eating newborns provides extra energy (Claessen et al., 2000). Yet even with strong cannibalism generation cycles may be present, even though they are modified by the cannibalistic interaction; for example, the period length may be longer, and the population may contain very big, cannibalistic individuals in addition to the cohort of juveniles that drive the periodicity (Claessen et al., 2000; Persson et al., 2004).

2.5.1 The critical resource density in the KM model

The equation for the individual growth rate, as derived in section 2.3 can be used to derive an expression for the critical resource density $R^*(x)$. From equation (13) we see that growth stops ($dx/dt = 0$) whenever the following relation between the resource density R and body

size x holds:

$$x_{\infty} \frac{R}{R_H + R} - x = 0, \quad (32)$$

which can be rewritten as the following definition of the critical resource density:

$$R^*(x) = \frac{xR_H}{x_{\infty} - x} \quad (33)$$

This relation is plotted in Fig. 5. It shows that the critical resource density is a monotonically increasing function of body size. This is a result from the assumptions that the maintenance rate (which is proportional to x^3) increases faster with body size than the food intake rate (which is proportional to x^2). In other words, when the resource decreases due to increasing consumption, the first individuals to stop growing and to start starving are the biggest individuals in the population. Hence, juvenile individuals can continue to grow in size down to lower resource concentrations than adults can. This explains that whenever a wave of maturation results in a high rate of collective reproduction, that the adult segment of the population starves to death, giving rise to a period with low reproduction (Fig. 6).

The combination of a high reproductive output, causing strong competition for food, and the size-dependent critical resource density is hence the underlying reason for the observed single-generation cycles. From this it follows that parameters that are likely to induce single-generation cycles in the KM model are those that accentuate the size-asymmetry between newborn and adult individuals; and those that tend to increase the level of competition. Thus, generation cycles can be induced by, for example, increasing the size at maturation x_J , by decreasing the mortality rate μ_0 , or by increasing the carrying capacity of the resource K .

Persson et al. (1998) showed that single-generation cycles can also result from a $R^*(x)$ that is a *decreasing* function of body size (i.e., the opposite of Fig. 5). In that case, newborns are the weakest competitors. Large juveniles and adults then depress the resource density below the R^* of newborns, thus impeding successful recruitment of newborns to the adult stage. The resident adult stage continues reproducing until it has sufficiently been reduced in abundance (due to background mortality) to relieve the resource concentration to a level above the newborns R^* . Once the newborns are able to grow, they will establish themselves as the new generation that will dominate the population until its decline. Persson et al. (1998) called such cycles "resident-juvenile cycles", as opposed to "recruit-driven cycles", their term for single-generation cycles as depicted in Fig. 4 and Fig. 6.

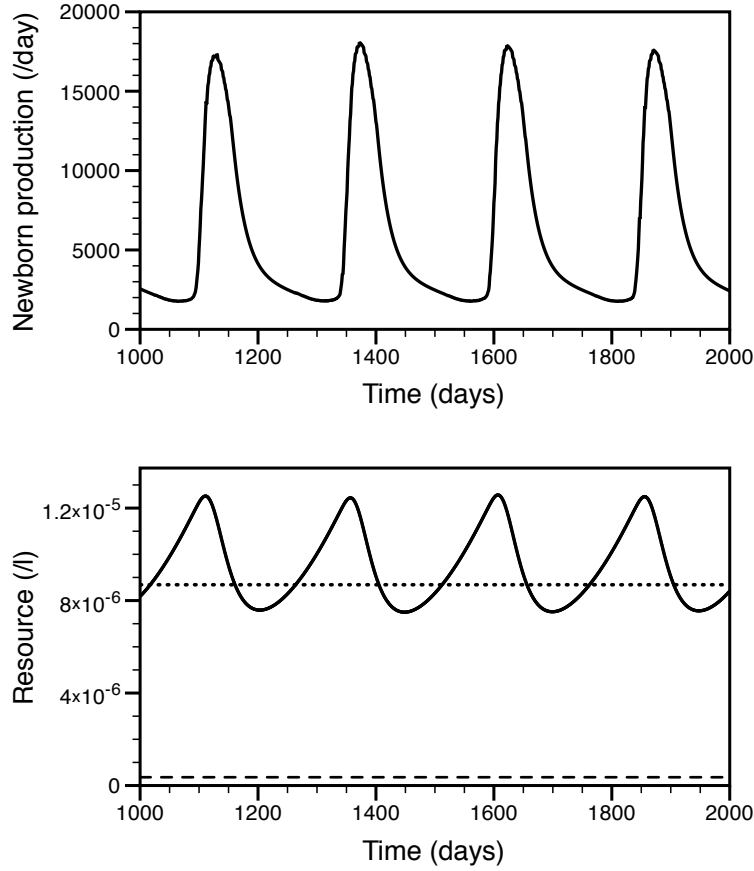


Figure 6: Time series of the KM model displaying single-generation cycles. **Top panel:** the rate of newborn production, equal to the sum of the reproduction rates of all mature individuals in the population, $\int_{x_J}^{\infty} b(x) n(x) dx$. **Bottom panel:** the resource density R . Note that following a wave of high reproduction, the consumption by the newborns depresses the resource level below the critical resource level of maturing individuals (dotted horizontal line = $R^*(x_J)$). The newborn's own R^* is much lower (dashed line = $R^*(x_0)$).

2.6 The effect of temperature on population dynamics

A first study of the effect of temperature on population cycles was published by Vasseur and McCann (2005). They used a simple, unstructured bioenergetics model to determine the influence of temperature on a consumer-resource interaction in order to predict the consequences of temperature changes on the dynamics and persistence of consumer populations. Their results indicate that warming is likely to destabilize consumer-resource interactions and that the qualitative response of the population dynamics depends on whether individual metabolic rate increases faster or slower with temperature than ingestion rate. Their model is a first step toward a bioenergetics theory of the impact of climate change on food web dynamics. The simplicity of their model allows them to analyse the model in quite some detail. The drawback is that they cannot address the size-dependent influence of temperature on organisms, and its consequences. In particular, empirical evidence suggests that small and large individuals do not respond equally to temperature. For example, in cold environments, large individuals have a higher metabolic efficiency compared to small individuals (Kozłowski et al., 2004). Van de Wolfshaar et al. (2008) present the first size-structured population model that accounts explicitly for seasonal temperature effects on vital rates. They show that the combined temperature- and size-dependence of vital rates may have fatal consequences for winter survival of both individuals and the population as a whole. However, they did not study the effect of changing temperature on population dynamics, the objective of the modelling exercise described here (Ohlberger et al., 2011).

Life history and population dynamics of a number of freshwater fish species including roach (*Rutilus rutilus*), Eurasian perch (*Perca fluviatilis*), yellow perch (*P. flavescens*) and Northern pike (*Esox lucius*), have been modelled with a model in which the i -state of a fish is defined by two variables: the amount of irreversible mass (x) and reversible mass (y) (Persson et al., 1998; Claessen et al., 2000, 2002; de Roos and Persson, 2001; Persson et al., 2004; Persson and De Roos, 2006). The model assumes that if x and y are known, all ecological functions can be derived from these two quantities: x and y hence completely define the state of an individual. For example, total body mass equals $w = x + y$, gonad mass equals $y - qx$ (where q is a constant), body length depends on x only, the search rates for different types of food are functions of x only. The state of the population is defined as the distribution of the number of individuals over the individual state (e.g., $n(x, y)$). Inspired by the biology of temperate freshwater fish, these models assume that reproduction occurs in a pulsed way during spring. At this moment, the gonad mass of adult individuals is converted into newborns. Together, the newborns form a new "cohort" of identical individuals. Thus the population consists of a variable number of cohorts, each described by a set of differential equations for the dynamics of x , y and N_i , where the latter is the abundance of cohort i . Cohorts disappear from the population when their abundance drops below a trivial threshold (e.g., a single individuals per lake). In these models, the environment is characterised by the population densities of prey (e.g., zooplankton) and, possibly, predators (e.g., cannibalistic conspecifics). Population feedback arises from the assumption that the dynamics of the environment depend on the state of the fish population: consumption by the fish depletes the zooplankton population and possibly causes mortality of small fish. Here I will not give any detailed description of these models, which can be found in the original publications cited above and in Ohlberger et al. (2011).

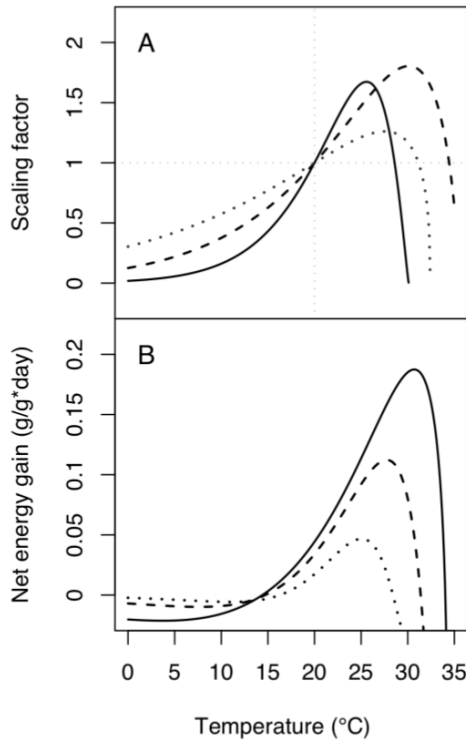


Figure 7: Temperature dependence terms (A) for perch consumption (solid), perch metabolism (dashed) and zooplankton growth rate (dotted), and the individual net energy gain (B) as a function of temperature for perch of body weight 0.1g (solid), 1g (dashed) and 10g (dotted). Thin dotted lines (A) indicate calibration to a value of 1 at 20°C (see text). The body size of perch was set to 8.2g, the optimal size for prey attack. The net energy gain for differently sized perch (B) was calculated at a zooplankton density of 2 Ind/L, which resembles rather low resource levels where exploitative competition in perch is expected to be high. From: Ohlberger et al. (2011)

2.6.1 Modelling the effect of temperature

What are the consequences of climate change on population dynamics? Or more generally, what is the relation between abiotic factors and ecological dynamics? Answering these questions may provide clues for testing theoretical predictions of our ecological theories with empirical data. And it may provide clues for how ecosystems respond to environmental change. Modelling dynamical consequences of the physiological response to temperature change requires the ability to translate individual-level physiological processes into population level dynamics. The framework of PSP models allow us to do so. The model formulation focusses on the description of individual-level properties of individuals such as the maintenance requirements, feeding rate, etc. Parameters are often numerous in such models (see the long tables in many of the above cited articles). Yet most of these parameters are fairly "easy" to measure in lab settings. The model then provides independent population-level predictions of the emergent dynamics. This contrast with more simplistic modelling frameworks, such as the Lotka-Volterra and derived models, which form the basis of theoretical ecology. Finding the temperature dependence of parameters of such simpler models may be more difficult than finding all parameters of a PSP model. For instance, the parameters r and K of the logistic growth equation (a frequent ingredient of Lotka-Volterra type models) are essentially population-level quantities, and are hence inherently impossible to measure without actually measuring the population level dynamics in the lab or field.

The goal of the modelling exercise in Ohlberger et al. (2011) is to make the basic eco-physiological functions in the above described "fish" model (Claessen et al., 2000, 2002) temperature dependent; the consumption rate (parametrised by the attack rate and the digestion time), the metabolic rate, and the zooplankton renewal rate. To do so, we multiplied each function by a temperature-dependent scaling factor (Fig. 7). We use empirically-based

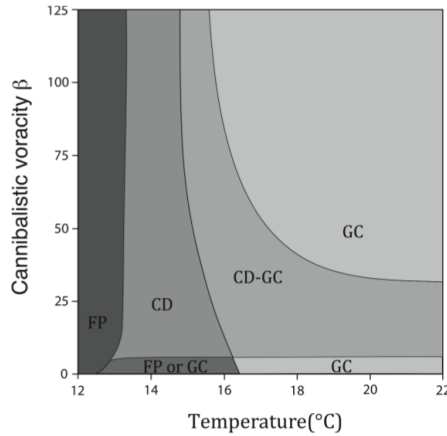


Figure 8: The regime diagram of the PSP model in Ohlberger et al. (2011). GC=generation cycles. FP=fixed point dynamics. CD=cannibal-driven dynamics. CD-GC=alternation of CD and GC dynamics (both types of dynamics are unstable). FP or GC=coexistence of two attractors (corresponding to FP and GC).

scaling relations rather than the more commonly used theoretical model for temperature dependence of chemical reactions, referred to as the Boltzmann factor or the Van 't Hoff-Arrhenius equation (Brown et al., 2004). The latter equation is one of the elements of the metabolic theory of ecology. However, looking into the literature on fish ecology, we found that the actual measurements of the temperature dependence of these relations deviates significantly from the theoretical (chemical) model. In particular, all three empirical relations (Fig. 7) display a maximum (at different optimum temperatures) rather than a monotonically increasing shape, as is the case of the Arrhenius equation.

Note that in the model of Ohlberger et al. (2011), these functions are size and temperature-dependent. Despite this complexity, we obtained fairly simple and seemingly general results: increasing temperature tends to increase the level of intra-specific competition and tends to result in the onset of generation cycles. That is, on a temperature gradient we expect generation cycles at high temperatures and stable populations ("fixed point dynamics") at low temperatures. This result is illustrated with the example of the predicted population dynamics at two different temperatures (Fig. 4). At 15°C, the population displays so-called fixed point dynamics (FP), which means that the within-year year dynamics are more or less the same from year to year. (Note that reproduction is pulsed at the beginning of the growing season). Such FP dynamics are characterised by the coexistence of a large number of cohorts (age classes). Intra-specific competition is too weak to result in the exclusion of some cohorts by other ones. By contrast, at 17°C, the model displays typical generation cycles, in this case characterised by the existence of a single cohort during most of the cycle (except during a short period following reproduction).

While this provides only two examples, a more general result can be found in Fig. 8. In fact the model we studied includes cannibalism as well as competition (in Fig. 4 cannibalism is assumed to be absent). Cannibalism was included in the analysis to assess the generality of the effect of temperature on generation cycles, since we know generation cycles occur without and with cannibalism, although in modified form (see above). The figure clearly shows that increasing temperature tends to result in generation cycles irrespective of the level of cannibalism. The amplitude of population fluctuations is predicted to increase with temperature, in both cannibalistic and non-cannibalistic populations Ohlberger et al. (2011).

Throughout the studied temperature range (12-22°C), the model assumes that the resource population growth rate increases with temperature. Yet the model predicts an overall

decrease of the mean resource density (i.e., the long-term average given a fixed temperature), caused by increased food intake by consumers, and increased consumer total reproduction rate (Ohlberger et al., 2011). The lower resource level at high temperatures reflects the increased level of intra-specific competition. A direct consequence of the different temperature dependencies of the vital rates at the individual level is that energy gain increases faster with temperature for small individuals (Fig. 7). Therefore, cool conditions favour big individuals, while warm conditions favour small ones. Increasing temperature hence reinforces the competitive advantage of small over large individuals, which enhances the mechanism that causes generation cycles (Persson et al., 1998).

Above we have seen that generation cycles are often the result of the fact that the critical resource density $R(x)$ increases with body size (which is the case for all fish for which the data exist, Persson and De Roos, 2006). The size dependence of intraspecific competition, however, changes with temperature. The net energy gain of an individual increases faster with temperature at smaller sizes (Fig. 7), thereby magnifying the competitive advantage of small over large individuals. This effect is reflected in the general observation that optimum growth temperatures (which for immature fish can be assumed to be equal to those of the net energy gain) decrease with increasing body size (Kozłowski et al., 2004). This has been reported for several fish species (Karas and Thoresson, 1992; Bjornsson and Steinarsson, 2002; Imsland et al., 2006) and other ectotherms such as amphipods (Panov and McQueen, 1998). Thus, this functional form of the temperature-size relationships may be valid for other fish species and possibly ectotherms in general.

Our conclusion on the effect of temperature on intra-specific competition and the resulting generation cycles are in line with those of Vasseur and McCann (2005): higher temperatures are predicted to destabilise population dynamics. Recall that that the model of Vasseur and McCann (2005) concerned consumer-resource (predator-prey) cycles. Interestingly, these two types of population cycles (generation cycles vs predator-prey cycles) are caused by ecologically and dynamically very different processes (size-asymmetric competition vs delayed feedback through the predator-prey interaction), and characterised by very different cycle periodicities (Murdoch et al., 2002). Whether these predictions hold for more complex food webs than the ones modelled in these studies remains to be seen. Yet these predictions can be put to the test relatively easily, by comparing population dynamics across temperature gradients, albeit natural ones or in the laboratory (Ohlberger et al., 2011).

The environmental feedback loop In the model of Ohlberger et al. (2011), the environmental feedback loop includes two different ecological interactions: competition via depletion of the zooplankton resource; and cannibalism. Competition for zooplankton is an indirect density-dependent interaction, that operates through the E -variable R . The impact of R on individual life history is via the size-dependent functional response (similar to equation (4)). The impact of the population on this E -variable is through the population-level, total feeding rate, a very similar expression to equation (24). Cannibalism is a direct-dependent interaction, in the sense that cannibalistic mortality and feeding rates depend directly on the current population abundance and size distribution. For this interaction, the E -variable hence contains the entire population size distribution. The impact of this E -variable on the individual life history is mediated by a complex function that takes into account the body sizes of any pair of interacting cannibals and victims (Claessen et al., 2000, 2002, 2004), see equation $A_c(c, v, x, y, T)$ in Table 2 of Ohlberger et al. (2011)), as well as the abundances of

victims and cannibals.

3 Some conclusions

This text has described and illustrated a theoretical framework (PSP models) that allows us to model the dynamics of size-structured populations. Size-dependent interactions may give rise to a number of dynamical behaviours that go beyond the scope of dynamics of unstructured models such as Lotka-Volterra type models. I have given the example of generation cycles: asymmetric competition between small and large individuals may give rise to population cycles with a periodicity of one generation.

A characteristic of populations with plastic life history is that the environment (and hence the environmental feedback loop) determines, to a certain extent, the realised phenotype of individuals, in terms of their growth trajectory and possibly other physiological traits such as corpulence. If the environment is constant, all individuals will have the same life history. If it is periodic (as in generation cycles), life histories will vary periodically, too. If the environment is stochastic and variable between individuals this will result in between-individual variability in life histories.

In other words, the struggle for existence determines not only population dynamics, but also realised phenotypes. The relation between phenotype and environment is called a reaction norm. It is, of course, not a new idea, but the above illustrates how PSP models account for such reaction norms, and rather, how they emerge from the intricate relation between the state of the environment, and the state of the population (i.e., the environmental feedback loop).

4 Exercises

1. Install the EBTtool on your computer. Download the software package from the home page of André de Roos, University of Amsterdam, The Netherlands.
2. Use the pre-coded Kooijman-Metz model, available from the STRU web page in the package TDebt-forstudents.zip. Read Vincent Le Boulrot's *Guide rapide d'utilisation* and *Exercice d'introduction*, as an introduction to the EBTtool. Follow the instructions.
3. Generation cycles and $R^*(x)$.
 - (a) Modify the code to obtain in your output file (.out) the values of $R^*(x)$ for newborns (size $x_B = LB$) and for maturing individuals (size $x_J = LJ$).
 - (b) Add these column into the figure of the resource dynamics (you should obtain two horizontal lines, corresponding to $R^*(x_B)$ and $R^*(x_J)$, respectively (see Fig. 6).
 - (c) Analyse the graph of the resource dynamics. Compare with the graphs of juvenile and adult dynamics. What can you conclude from these figures about the effect of competition on small, large, juvenile and adult individuals? What does this tell you about the mechanisms underlying the generation cycles? (NB the default parameter setting should give rise to single-generation cycles).
 - (d) Increase the mortality rate to obtain a stable equilibrium. How is the resource level compared to $R^*(x)$ in a stable equilibrium?
4. Single-generation cycles vs Lotka-Volterra type predator-prey cycles. Use the parameter settings for Daphnia, from de Roos (1997).
 - (a) Modify the code: change the semi-chemostat resource dynamics into logistic growth.
 - (b) Explore the parameters K (carrying capacity) and MUC (background mortality rate) and try to find the two types of population cycles. Remind yourself of the characteristics of the two types of population cycles.
 - (c) Things to look at:
 - juvs and adults in/out of phase?
 - is the resource R close to $R^*(x)$?
 - the period of the cycle (how many generation times?) cf Murdoch et al. (2002)
 - (d) Make a plot of juveniles vs adults. Compare this graph for the two types of cycles.
 - (e) Make a plot of total population vs resource density. Compare this graph for the two types of cycles.
 - (f) Run a bifurcation over the carrying capacity.

5 Mini-projects

1. **Size-dependent harvesting.** Harvesting such as fisheries usually is size-specific: often the harvesting targets larger individuals. Fishing gear such as gill nets are size-selective; all fish smaller than a certain size swim through the nets, whereas big ones get captured. Modify the code of the KM model (chemostat or logistic resource dynamics?) in order to implement a size-threshold above which individuals are subject to harvesting. That is, an individual of size $x > x_H$ has a higher (fixed) mortality rate, which correspond to the risk to become harvested. Explore the dynamics depending on the size-threshold, as well as on the harvesting intensity. Possible questions to address:
 - (a) Can you identify the so-called Maximum Sustainable Yield?
 - (b) What happens to the population cycles (either Lotka-Volterra or single-generation cycles) when you impose the harvesting mortality?
 - (c) What happens (pop dynamics and size structure) if you change life history to account for an evolutionary response to harvesting, i.e.,
 - earlier maturation at a smaller size?
 - increased energy investment into reproduction?
 - How do these changes influence the MSY?

NB to answer to this question, you should not try to model the evolutionary dynamics, but you should change the value of some parameters, in the expected direction based on a life history evolutionary response to harvesting. See the lecture by Eric Edeline.
2. **The paradox of enrichment.** Explore the KM model in the context of this classic idea from ecology. Use the exercise 4 above as a basis.
3. **Add a resource to the KM model.** Try to implement a size-dependent difference in the exploitation of the two resources (i.e., a size-dependent preference). Start by copying all equations and parameter values from the first resource and use them for the second resource. Once you got this working, you can start introducing differences between the resources (in terms of how the consumer uses them).
4. **Add a predator to the KM model.** Assume that the predator attacks all consumers with the same, size-independent attack rate.
 - (a) How does the predator influence the generation cycles of the KM model?
 - (b) Advanced: The predator could have a size-preference; it may target either the smallest or the largest individuals (or some other function of body size). What is the effect of such a size preference?
5. **Biomass overcompensation.** You can use the KM model to test the validity of the predictions made by the much simpler stage-structured biomass model (De Roos et al., 2007, 2008). Does the KM model behave according to the predictions made with this theory that has been discussed during the lectures?

6 References

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