Ontogenesis

Eco-evolutionary perspective on life history complexity

Hin, V.

VINCENT HIN

ONTOGENESIS

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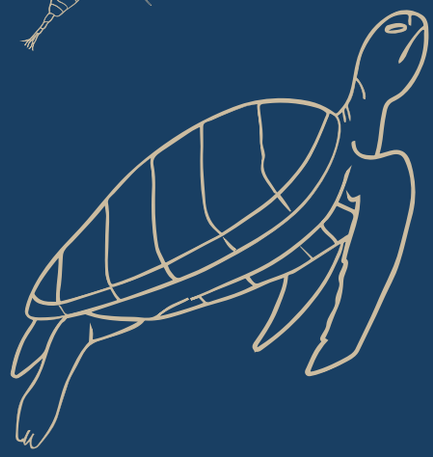



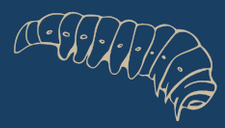

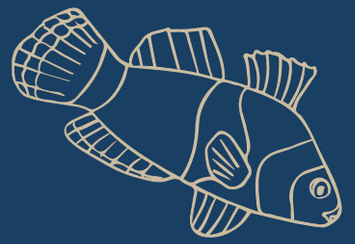

VINCENT HIN

ONTOGENESIS:

An

Eco-Evolutionary Perspective

on

Life History Complexity

by

Vincent Hin

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Chapter 1

General Introduction

Vincent Hin

Outline

The main paradigms in population and community ecology are based on mathematical models that assume equal ecological interactions for all individuals in the population. These models are also referred to as unstructured models, and have provided important insights about the functioning of natural systems. However, accounting for variation in ecological interactions between individuals (*i.e.* intraspecific variation in ecological interactions), can help to even better understand the dynamics of populations and communities. Ontogenetic development is an important source of intraspecific variation in ecological interactions. Ontogenetic development refers to the changes in size, shape, physiology or maturity status of an individual that occur during its lifetime. Recently, ecological theory has been developed that accounts for the intraspecific variation in ecological interactions that arises from ontogenetic development (De Roos et al. 2013). This body of theory uses structured population models, which in contrast to unstructured models, can account for differences between individuals. Not only are the dynamics from structured models richer, they also overthrow many of the paradigms from unstructured ecological theory.

The crucial feature that leads to the dissemblance in dynamics between structured and unstructured theory is the *ontogenetic asymmetry* in energetics and mortality of an individual. Ontogenetic asymmetry implies that the mass-specific rates of resource ingestion, biomass production and mortality, change with individual body size (De Roos and Persson 2013). Thus, in case of ontogenetic asymmetry, ontogenetic development leads to intraspecific variation in ecological interactions. Structured ecological theory readily connects ontogenetic asymmetry in individual organisms to the dynamics of populations and communities (reviewed in De Roos and Persson 2013).

The next step is to understand the eco-evolutionary implications of ontogenetic asymmetry. This thesis takes this step by studying the evolutionary causes and consequences of ontogenetic asymmetry. After this introductory chapter, chapters 2 and 3 study whether and how selection can explain ontogenetic asymmetry in a basic ecological setting. In chapters 4 and 5, the ecological and evolutionary consequences of ontogenetic asymmetry for the long-term persistence of species in more complex ecological communities are studied. Finally, chapter 6 describes an example of how evolution can lead to more complexity in ecological communities.

The remainder of this chapter shortly describes the approach and some insights from the ecological theory that is based on unstructured models. Thereafter, reasons to develop theory that includes ontogenetic development will be discussed, and examples of the consequences of ontogenetic asymmetry for the dynamics of populations and communities will be given. Finally, a more specific description of the research questions of the following chapters will be presented.

Unstructured ecological theory

A main objective in ecology is to understand mechanisms and processes that govern the dynamics of natural populations and communities for a broad range of species and systems. A theoretical approach towards this goal is to use dynamic models that describe interacting populations, such as consumer-resource models (Murdoch et al. 2003), competition models (MacArthur 1970; Tilman 1980, 1982), models of three species trophic modules (Holt and Polis 1997; McCann et al. 1998; Polis and Holt 1992; Polis et al. 1989) and models describing multi-species food webs (De Ruiter et al. 1995; May 1972; McCann 2011). Many of the paradigms about how populations and communities function and persist have been developed through the strategic use of these models.

As an example of such paradigms, modeling studies have helped to realize that the fate of species co-depends on their dynamic, biotic environment. Furthermore, species impact their own environment and in such a way affect their own success. Therefore, fitness is not a static predefined trait, but rather it depends on the functioning of species in their dynamic environment. Another example is the work devoted to studying the conditions and mechanisms that lead to stable coexistence of species communities. Theory predicts that in constant environments, the number of competing species cannot be higher than the number of resources (Gause 1934; Hardin 1960). In natural communities there are often many species coexisting on a limited number of resources, and theoretical research has been valuable in discovering the potential mechanisms that enable many species to coexist on a limited number of resources (Chesson 2000; Huisman and Weissing 1999).

One way to link consumer-resource interactions to larger food webs is to study the dynamics of trophic modules (Bascompte and Melián 2005; Kondoh 2008). These are components within the greater food web, containing three or four species in different configurations. Examples of trophic modules are the diamond food web (Wollrab et al. 2013), where species compete for a resource and share a common predator, apparent competition (Holt 1977), in which case two species only interact through a shared predator, and intraguild predation (IGP), which describes a predator and a prey that also compete for a resource (Polis and Holt 1992; Polis et al. 1989). IGP appeared to be common in food webs (Bascompte and Melián 2005; Polis 1991; Polis and Myers 1985). Numerous fish, amphibian, reptile and insect species are engaged in IGP (Persson 1988), and this interaction can also be found in zooplankton communities (Toscano et al. 2016). However, early theoretical work showed limited potential for coexistence of intraguild predators with intraguild prey. This finding stimulated more research on the mechanisms that enhance coexistence in intraguild predation systems (*e.g.* Diehl and Feißel 2000; Diehl and Feissel 2001; Mylius et al. 2001).

Many of the above models are population-level models (De Roos and Persson 2005), as their formulation begins by considering population-level processes. A population-level viewpoint considers ecosystems as trophic pyramids, in which the nodes are populations composed of identical individuals (De Roos and Persson 2005). Implicitly, this approach assumes that existing intraspecific variation between individuals within a population is negligible, in terms of its impact on population-level processes, and hence can be ignored. Therefore, all individuals are considered to have identical ecological interactions. This concerns both the *type* of interaction (*e.g.* feeding, predation or competition) as well as the *strength* of the interaction (*e.g.* rates of resource feeding, or competitive ability). For intraguild predation systems, this implies that all intraguild predators simultaneously hunt for intraguild prey and feed on the shared resource. In addition, it also implies that all intraguild predators have identical rates of resource feeding and identical predation rates. Because of the lack of internal structure in such a population, population-level models are also called unstructured models. An important consequence of the assumption that all individuals are identical, is that these models delimit population dynamics to mere changes in population numbers or total population biomass. The life histories of individual organisms are therefore only represented by the processes of reproduction and mortality, which respectively lead to an increase and a decrease in population density.

Ontogenetic development: an important life history process

However, beyond the two key processes of reproduction and mortality, an important life-history process is ontogenetic development. Ontogenetic development can be regarded as the collection of changes in the state of an individual that occur during its life, in terms of changes in size, shape, physiology, maturity status, or behavior. It is well recognized that ontogenetic development plays a major role in the life of all species, as there are no species that reproduce immediately after birth (De Roos and Persson 2013). In any species, individuals must first grow in size and develop maturity before they can commence reproduction. One of the most striking and critical features of ontogenetic development is ontogenetic body size growth. In many species, the differences in size between newborns and adults can span several orders of magnitude (De Roos and Persson 2013). Also, the preponderance of species with complex life cycles or metamorphosis is illustrative for the importance of ontogenetic development in nature.

Ontogenetic development unavoidably creates intraspecific variation in ecological interactions (DeAngelis and Mooij 2005), as the nature of many ecological processes changes as a result of ontogenetic development. For example, juveniles and adults

can compete for resources that are important for their growth and reproduction, and juveniles might be better or weaker competitors. Alternatively, in many species juveniles and adults consume different resources and occupy different habitats, either as a result of ontogenetic niche shifts or metamorphosis (Werner 1988; Werner and Gilliam 1984). Juveniles and adults may also experience differences in predation, because of different predators or different predation rates. To return to the example of intraguild predation, in many cases the competition and predation between intraguild predators and prey are separated between different life stages of the intraguild predator. In these so-called life history intraguild predation systems (LHIGP: Hin et al. 2011; Pimm and Rice 1987; Van de Wolfshaar et al. 2006), juvenile intraguild predators compete with intraguild prey, while adult intraguild predators feed on intraguild prey.

Intraspecific variation in ecological interactions that originates from ontogenetic development can have significant consequences for ecological and evolutionary processes. Population-level models cannot account for this variation, as these models do not consider differences between individuals. Studying the population and community consequences of ontogenetic development therefore requires a different modeling approach. A framework is needed that describes the important life history processes of individuals (ontogenetic development, reproduction and mortality), and subsequently translates these individual-level dynamics to the population and community level (De Roos and Persson 2001; Diekmann and Metz 2010). Such an approach is used by structured population models. In the next section I will introduce this modeling approach and subsequently describe how ontogenetic development can change the dynamics of populations and communities.

Structured ecological theory

Structured population models start by describing the ecology and key life history processes of individuals. The population and community-level dynamics then emerge from the individual-level processes (De Roos and Persson 2001; Diekmann and Metz 2010; Metz and Diekmann 1986). Structured population models keep track of the individuals separately or track the distribution that describes the (relative) abundance of different types of individuals, *i.e.* the population structure or population composition. Changes in the population composition make the dynamics of structured population models more complex compared to unstructured models. A structured modeling approach that incorporates ecological interactions with descriptions of life histories is provided by the framework of physiologically structured population models (PSPMs; De Roos 1997; De Roos et al. 1992; Metz and Diekmann 1986). The core ideas of this framework are outlined in box 1.1.

Ontogenetic (a)symmetry

In order to understand the consequences of ontogenetic development for the dynamics of populations and communities, one needs to specify the conditions under which ontogenetic development does not lead to changes in ecological interactions. For this we use the condition of *ontogenetic symmetry*, which applies if and only if the mass-specific ingestion rate, the mass-specific biomass production rate and the mortality rate of an individual do not change with individual body size (De Roos et al. 2013). Any deviation from the conditions of ontogenetic symmetry leads to *ontogenetic asymmetry*. In case of ontogenetic asymmetry, either the mass-specific resource ingestion rate, the mass-specific biomass production rate or the mortality rate changes with individual body size (De Roos et al. 2013). Ontogenetic symmetry describes the conditions under which ontogenetic development does not lead to deviations in the strength of ecological interactions. Conversely, ontogenetic asymmetry introduces changes in strength of ecological interactions during ontogeny through changes in the resource intake, biomass productivity or mortality.

There are many sources of ontogenetic asymmetry in natural populations. For example, the biomass production rate of an individual depends on both the energy assimilated from food and the amount of energy required for maintenance metabolism. When these two processes scale differently with body size, this leads to ontogenetic asymmetry through changes in the mass-specific biomass production. Also, ontogenetic asymmetry will readily result from ontogenetic diet shifts or metamorphosis, through a shift in mass-specific ingestion or mass-specific biomass production rates. Size-dependent mortality is also an important source of ontogenetic asymmetry. These different examples suggest that ontogenetic asymmetry applies to individuals in most, if not all species.

In population-dynamical equilibrium, ontogenetic asymmetry leads to a difference in productivity between different life stages. For the whole population, the rate of biomass production through growth or reproduction must necessarily equal the rate of biomass loss through mortality, in order to remain at population equilibrium. When in one life stage biomass production exceeds biomass loss, this net gain of biomass must be compensated for in the other life stage (De Roos et al. 2007). Therefore, in the latter life stage a net biomass loss must occur.

Ontogenetic asymmetry in a consumer species with a distinct juvenile and adult life stage, for example, translates into *juvenile-adult asymmetry*, when the population is at equilibrium (De Roos et al. 2013). Juvenile-adult asymmetry implies a difference in net biomass productivity between juvenile and adult life stages. When the juvenile life stage is a net source of biomass, the adult life stage is a net sink of biomass. Consequently, population-level maturation rate in biomass exceeds population-level

Box 1.1: Physiologically structured population models (PSPMs)
Ecological models that describe interacting populations and also incorporate an explicit description of individual life history (that can depend on these ecological interactions) are referred to as physiologically structured population models (PSPMs; De Roos 1997; De Roos et al. 1992; De Roos and Persson 2001; Metz and Diekmann 1986). A PSPM uses structuring variables (individual-states or *i*-states), such as age, size or energy reserves, to distinguish different individuals. Individuals can only differ with respect to their *i*-state value and individuals that have same *i*-state values are considered fully identical by the PSPM. The population of all individuals is tracked by the *p*-state (from population-state), which is a measure that describes the distribution of all individuals within the *i*-state space (the collection of all possible *i*-states). The specification of a PSPM lies in describing the *i*-state dynamics, which reflect life history processes. The rate of change of an *i*-state can depend on the current *i*-state value (*e.g.* body size, amount of energy reserve), on the state of the environment (the *E*-state, *e.g.* resource density) or on the *p*-state or some scaled version of the *p*-state (*e.g.* predator pressure from cannibalism). All assumptions of a PSPM pertain to the specification of the *i*-state dynamics or the dynamics of the environment. No further population-level assumptions are required. Ecological interactions within a PSPM framework occur between two structured populations or between a structured population and the environment (*e.g.*, a size-structured consumer population feeding of an unstructured resource). In many PSPMs, a Dynamic Energy Budget (DEB) model is used to describe the *i*-state dynamics. A DEB model describes the rates of uptake, allocation and use of energy and nutrients for energy-demanding processes within an organism, such as growth, reproduction and maintenance metabolism (Kooijman 2010). Using a DEB model confines the individual life history to what is physically possible, as organisms have to adhere to the principle of mass and energy conservation. For example, organisms need to take up energy from the environment to grow and reproduce and cannot use the same amount of energy twice. The resource input of the DEB model can be an environmental variable in the PSPM. The joint feeding of all individuals depletes resource densities, which limits growth and reproduction through the dynamics of the DEB model. In such a way density-dependence is introduced through a dynamic feedback between consumer feeding and resource renewal.

reproduction rate in biomass. The biomass turn-over through the life cycle is in this case regulated mostly by reproduction (De Roos et al. 2007). Alternatively, when the adult life stage is a net source of biomass and the juvenile stage is a net sink of biomass, the population-level reproduction rate in biomass exceeds the population-level maturation rate in biomass. In this case, the biomass turn-over is regulated mostly by maturation.

Ontogenetic development can thus lead to changes in the strength of ecological interactions through ontogenetic asymmetry. Ontogenetic asymmetry in turn leads to an asymmetry in the productivity of different life stages when the population is in equilibrium. Next, I will describe how ontogenetic asymmetry affects dynamics of populations and communities.

Population and community consequences of ontogenetic asymmetry

Structured population models have revealed considerable effects of ontogenetic asymmetry on the dynamics of populations and communities (reviewed in De Roos et al. 2013). Ontogenetic asymmetry leads to phenomena through changes in population composition. The most important consequence for community dynamics is *biomass overcompensation*, which is the phenomenon that (stage-specific) biomass density increases with increasing mortality (De Roos et al. 2007).

Biomass overcompensation occurs because mortality relaxes competition for resources and in this manner indirectly increases resource density. In case of ontogenetic asymmetry this will lead to a disproportionate increase in the production rate of the life stage that limits the population-level biomass turn-over most. As a consequence, the equilibrium biomass density in the other life stage increases. In a maturation-regulated population, mortality leads to an increase in maturation rate and this increases adult biomass density. In a reproduction-regulated population, mortality increases the reproduction rate and this increases juvenile biomass density.

Biomass overcompensation is a robust phenomenon and has been demonstrated to occur in several empirical systems (Cameron and Benton 2004; Ohlberger et al. 2011; Reichstein et al. 2015; Schröder et al. 2014). It occurs when different life stages share a resource, or feed on separate resources that differ in productivity. It occurs independent of whether mortality is increased in the sink or the source life stage, or in both simultaneously (De Roos and Persson 2013). Also, biomass overcompensation happens irrespective of life-history details and whether reproduction occurs continuously throughout the year or with a seasonal pattern (Soudijn 2016).

Biomass overcompensation differs from the Hydra effect, which describes a (time-averaged) positive mortality response in the cyclic dynamics of unstructured consumer-resource models (Abrams 2009). The essential ingredients for the occurrence of the Hydra effect are a positive correlation between resource productivity and resource density (as in logistic resource growth) and a saturating consumer functional response (Schröder et al. 2014). An increase in consumer mortality will change the amplitude and period of the consumer-resource cycle, which can lead to an increase in time-averaged consumer density. Biomass overcompensation occurs because of energetic bottlenecks in life history of the consumer and is independent of the type of resource growth, or the consumer functional response.

Biomass overcompensation creates a positive feedback between the mortality rate and the biomass density of a life stage. This allows stage-specific predators to increase their own prey availability, which results in an emergent Allee effect for the predator (De Roos and Persson 2013; De Roos et al. 2003*b*). Through a similar mechanism, two

stage-specific predators are able to mutually facilitate each other's persistence (De Roos et al. 2008*a*).

Besides biomass overcompensation, ontogenetic asymmetry can induce cohort cycles in the population dynamics (De Roos and Persson 2013). These arise through changes in competitive ability during ontogeny. Competitively superior individuals require lower resource densities to cover their maintenance needs than competitively inferior individuals. Size-dependent changes in competitive ability arise when mass-specific ingestion rates change in a different way with size than mass-specific biomass production rates. When juveniles are superior competitors, they outcompete their parents by suppressing resource density. This will lead to juvenile-driven cohort cycles (De Roos and Persson 2003). Alternatively, adult-driven cycles occur when adults are superior competitors. In this case, destabilization occurs because of an increase in the juvenile period, caused by limited food availability, combined with a sudden increase in fecundity and adult biomass when the dominant cohort matures (De Roos and Persson 2003, 2013).

The omnipresence of ontogenetic asymmetry and its strong effects on the structure and dynamics of populations and communities, raises the question of how ontogenetic asymmetry evolves. Under which conditions does selection favor ontogenetic asymmetry, and how do ecological factors affect selection on the strength and direction of ontogenetic asymmetry? Furthermore, how does selection on ontogenetic asymmetry affect the long-term potential for persistence of species and food webs? These are the main questions that will be addressed in this thesis and answering these questions requires a framework that integrates ecological interactions with the evolution of life histories. The next section will motivate such a framework and introduce the more specific research questions of the following chapters.

Studying the eco-evolutionary dynamics of ontogenetic asymmetry

There are at least two reasons why ecological interactions must be accounted for when studying the evolutionary origins of life histories. The first is that life histories are plastic and vary with ecological conditions, such as resource availability (Claessen et al. 2000; Van Kooten et al. 2007) and predation (Pfennig et al. 2010). The second reason is that optimal life histories and ecological dynamics influence each other through an eco-evolutionary feedback. As life history decisions affect ecological interactions, in turn, the ecological interactions determine which life history strategy returns the highest fitness and is selected for. This leads to a constant feedback between evolutionary change and ecological response (Ferrière and Legendre 2013; Palkovacs and Post 2008; Post and Palkovacs 2009).

Studying the eco-evolutionary dynamics of ontogenetic asymmetry thus requires an approach that incorporates both ecological interactions and eco-evolutionary feedbacks. The theory of adaptive dynamics provides such an approach. The concepts and rationale of adaptive dynamics are discussed in box 1.2. This thesis will combine the structured models as described in box 1.1, with the adaptive dynamics approach to study the evolutionary origin of ontogenetic asymmetry and its consequences for the persistence of populations. The specific research questions will be motivated next.

Chapter 2 and 3 study the evolution of ontogenetic asymmetry in a basic ecological setting. This involves a size-structured consumer population feeding on an unstructured resource. The life history of the consumer is described by a simple dynamic energy budget (DEB) model (Kooijman 2010; Lika and Nisbet 2000). In this DEB model, the rate of maximum resource ingestion and the rate of maintenance metabolism follow power functions of body mass. The exponents in these power functions determine the body-mass scaling of energy intake (through resource ingestion) and energy expenditure (through maintenance metabolism). Biomass production of the consumer equals the difference of energy intake and energy expenditure. The scaling of biomass production with body mass therefore depends on the scaling exponents of both maximum ingestion and maintenance metabolism.

Chapter 2 shows that there is strong selection towards an equal body-mass scaling of energy supply and energy expenditure, but only under limited conditions do these scaling exponents evolve exactly to ontogenetic symmetry (linear scaling of both energy supply and energy expenditure with body size). The type of ontogenetic asymmetry that evolves leads to higher mass-specific resource ingestion and mass-specific biomass production for either juveniles or adults. As a consequence, one life stage becomes a net source of biomass, while the other life stage becomes a net sink of biomass. Which life stage becomes a net source depends on the size-dependency of mortality and the extent of pre- and post-maturation growth. Furthermore, the evolved type of ontogenetic asymmetry (equal scaling exponents of maximum ingestion and maintenance metabolism) ensures that all individuals require the same resource density to cover their maintenance costs. This neutralizes intraspecific competition and stabilizes population dynamics.

Chapter 3 extends the approach of chapter 2 and studies whether ontogenetic asymmetry can arise from multiple processes that determine the body-mass scaling of energy supply. In addition to the scaling of maximum ingestion rate with body mass, in chapter 3 the scaling of energy supply is also determined by the attack rate scaling. Chapter 3 shows that ontogenetic asymmetry does not evolve from considering separate scaling processes for energy supply. This result is also consistent between two different trade-offs that are considered.

Box 1.2: Adaptive dynamics

Adaptive dynamics (Geritz et al. 1998; Metz 2012; Metz et al. 1995) is an approach for studying adaptive phenotypic evolution in ecological models that describe interacting populations. Adaptive dynamics distinguishes between resident and mutant phenotypes. Initially, the population consists only of the resident phenotype (*i.e.* is monomorphic) and resides at its population dynamical attractor (*e.g.* stable equilibrium point, limit cycle). Evolutionary change occurs when mutants with a slightly different phenotype invade and take over the population-dynamical attractor of the resident. Whether the mutant invades and replaces the resident not only depends on the phenotype of the mutant, but also on the environment in which the mutant invades. In turn, this environment depends on the resident trait, because it is the resident that determines the population dynamical equilibrium in which mutants invade. The fitness of the mutant is hence a function of the trait values of both the mutant and the resident. The tools of adaptive dynamics provide methods to quickly assess the long-term outcome of many subsequent rounds of mutant-resident interactions. The dynamics between mutants and residents make adaptive dynamics a framework in which evolutionary change explicitly depends on ecological dynamics.

Chapter 4 and 5 consider an intraguild predation (IGP) system to study the consequences of ontogenetic asymmetry in a more complex community than the basic ecological setting of chapters 2 and 3. Size-dependent interactions can readily induce ontogenetic asymmetry in IGP systems. For example, an ontogenetic diet shift in the intraguild predator (in case of life-history IGP (LHIGP); Hin et al. 2011; Rudolf 2007; Van de Wolfshaar et al. 2006) leads to a difference in the mass-specific ingestion and the mass-specific biomass production rate between juvenile and adult intraguild predators. Also, cannibalism is common in LHIGP systems and increases both adult mass-specific production and juvenile mortality.

Chapter 4 shows that the ontogenetic asymmetry that arises from cannibalism disrupts the stable persistence of intraguild predators, irrespective of the type of change in diet that the intraguild predator exhibits during its life. Cannibalism leads to a shift from a reproduction- to a maturation-regulated population. In a maturation-regulated population, juvenile intraguild predators suffer from competition with intraguild prey. With increasing levels of cannibalism, the competition in the juvenile stage becomes too severe for stable predator persistence.

Chapter 5 builds on chapter 4, by studying how ontogenetic asymmetry in resource ingestion and biomass production evolves in response to cannibalism in an IGP system. It is assumed that intraguild predators can evolve to increase juvenile biomass production and resource ingestion by increasing juvenile specialization on the shared resource. Such an increase could potentially offset the negative effects of competition in the juvenile life stage under high levels of cannibalism. Alternatively, evolution of adult specialization will increase intra- and interspecific predation rates. This leads

to an increase in ingestion and biomass production for adults. However, a genetic constraint between the life stages is assumed to prevent concurrent specialization of both life stages on their respective food source. Hence, the direction and strength of ontogenetic asymmetry in the intraguild predator can evolve through selection on an ontogenetic trade-off in resource specialization.

Chapter 5 shows that in absence of cannibalism, selection on this ontogenetic trade-off leads to an increase in specialization of one life stage, at the expense of feeding performance in the other life stage. Ultimately, this increasing specialization of one life stage shifts the community dynamics to a state in which predators can no longer persist. Consequently, selection on the ontogenetic trade-off in absence of cannibalism leads to evolutionary suicide of the intraguild predator. Cannibalism prevents evolutionary suicide by stabilizing selection on the ontogenetic trade-off in resource specialization.

Chapter 4 and 5 thus show that cannibalism has a central role in LHIGP systems. Cannibalism can inhibit persistence of intraguild predators on ecological timescales (chapter 4), but also stabilizes evolutionary dynamics and prevents evolutionary suicide (chapter 5). It is therefore important to understand the conditions that inhibit or promote the evolution of cannibalism. Chapter 6 addresses this topic in the more applied and practical context of fisheries-induced evolution. In chapter 6 a model for the population dynamics of cannibalistic Arctic char (*Salvelinus alpinus)* is formulated. With this model the eco-evolutionary interaction between cannibalism and fisheries-induced mortality on large char individuals is investigated. It is shown that fisheries-induced mortality promotes the evolution of cannibalism, by changing the stabilizing selection on cannibalism resulting from costs associated with cannibalistic feeding, into positive directional selection. This leads to a double effect of mortality on the population. The fisheries-induced mortality decreases population biomass directly, but also selects for even higher rates of cannibalism, which further reduces population density. The interaction between ecological and evolutionary effects of harvesting severely increases vulnerability of cannibalistic Arctic char populations to high levels of mortality.

Finally, chapter 7 summarizes the main findings of the preceding chapters and discusses how these results contribute to understanding the conditions under which ontogenetic asymmetry evolves in basic and more complex ecological communities.

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Chapter 2

Evolution of Size-Dependent Intraspecific Competition Yields Paradoxical Predictions on the Scaling of Metabolism with Body Size

Vincent Hin

André M. de Roos

Manuscript in preparation

Abstract

Growth in body size is accompanied by changes in foraging capacity and metabolic costs, which lead to changes in competitive ability over ontogeny. The resulting size-dependent competitive asymmetry determines population and community dynamics, but it is not understood whether natural selection favors such asymmetry in intraspecific competition. We address this question using a size-structured consumer-resource model to study the evolution of the scaling of competitive ability with body size. Strength and direction of competitive asymmetry depend on the scaling exponents of maximum ingestion and maintenance metabolism with body size. We use adaptive dynamics to study evolution of these exponents and their dependence on mortality and life-history parameters. The two exponents converge to the same value, such that all individuals are competitively equal. Furthermore, the scaling exponents respond adaptively to changes in mortality such that growth and/or reproduction increases in the life stage that is affected most by mortality. Also, decreasing size at birth leads to increased investment in juvenile growth, while increasing maximum size leads to increased investment in post-maturation growth and reproduction. The latter result provides an explanation for the variation in intraspecific scaling exponents of metabolic rate with body size as observed in nature. Data will be presented that support these predictions. However, selection towards equal scaling exponents contradicts other empirical findings and explanations for this discrepancy will be discussed.

2.1 – Introduction

Intraspecific competition is often asymmetric such that some members of the population have a large negative effect on others, but suffer relatively little from competition themselves. Furthermore, this asymmetry most often depends on individual body size. For example, small larvae of the damselfly *Ischnuru elegans* suffer from reduced growth and longer developmental times due to interference of large larvae, but not from other small larvae (Gribbin and Thompson 1990). In case of competition for resources, or exploitative competition, the competitive ability of an individual is determined by resource foraging capacity and metabolic costs, which affect the ability to grow, reproduce and withstand resource scarcity (Hjelm and Persson 2001; Persson et al. 1998; Werner 1994). Foraging capacity has various behavioral and physiological components, such as attack rate, handling time, assimilation efficiency and resource supply rates. These processes generally scale with body size in different ways (Hansen et al. 1997; Persson et al. 1998; Peters 1983). The specifics of these scaling relationships determine whether large or small individuals are competitively superior. In fish, for example, the metabolic costs generally increase faster with body size than foraging capacity (Persson and De Roos 2006). Therefore, larger individuals need higher food quantities just to cover maintenance requirements and are therefore competitively inferior resource competitors compared to small individuals (Aljetlawi and Leonardsson 2002; Hjelm and Persson 2001; Kooijman 2010; Persson et al. 1998; Werner 1988). On the other hand, large individuals are likely to have more reserves and in combination with lower mass-specific maintenance requirements may hence withstand starvation better than small individuals (Byström et al. 2006).

Understanding intraspecific competitive asymmetry and its consequences for population and community dynamics from an individual perspective requires a measure that relates individual-level competitive performance to body size. Two such measures have been proposed previously: *i*) the mass-specific biomass production rate (MBP) (De Roos et al. 2013; De Roos and Persson 2013) and *ii*) the maintenance resource density (MRD; Persson et al. 1998), referred to by some authors as the critical resource density (Byström and Andersson 2005; Lefébure et al. 2014; Persson and De Roos 2006). The MBP describes the ability of an individual to produce new biomass through growth and reproduction with a certain amount of resources, expressed per unit body mass of the individual (De Roos et al. 2013). An individual with a high MBP can be considered competitively superior to one with a low MBP, since at the same resource level it can reproduce and grow with a higher mass-specific rate. The MRD is defined as the resource level at which an individual can just cover its maintenance metabolism, *i.e.* when the MBP becomes zero (Gliwicz 1990; Persson et al. 1998). The MRD can be interpreted as an individual-level R^*-value (sensu Tilman 1980), such that

competitively superior individuals have a lower MRD. Both the MBP and the MRD are derived from the difference between assimilation and metabolic maintenance costs. The scalings of the MBP and the MRD with body size can be used to determine whether small, intermediate sized or large individuals are competitively superior (Persson et al. 1998). However, the two measures do not necessarily have the same outcome. In the case of identical scalings of assimilation and maintenance rates, the MRD is a constant function of body mass, while the MBP can still increase or decrease with size when both these scalings are allometric (different from one).

Changes in the MBP and MRD with body size can have large consequences for population dynamics, species coexistence and community structure (reviewed in De Roos and Persson 2013; De Roos et al. 2003*a*; Persson and De Roos 2013). A change in the MBP with ontogeny implies that either large or small individuals produce more new biomass per unit of existing biomass and this causes a bottleneck in the flow of biomass across the life cycle. Such an energetic bottleneck considerably influences the population size distribution (De Roos et al. 2007), as biomass accumulates in the size-range with the lowest productivity. Increasing (size- or stage-specific) mortality can alleviate such a bottleneck and lead to an overcompensatory increase in biomass of the non-limited stage (De Roos et al. 2007). This phenomenon of biomass overcompensation is shown to occur in both experimental (Cameron and Benton 2004; Schröder et al. 2009*b*) and natural systems (Ohlberger et al. 2011) and can lead to community wide effects such as emergent Allee effects (De Roos et al. 2003*b*), emergent facilitation between two size-selective predators (De Roos et al. 2008*b*) and alternative stable states (De Roos and Persson 2002; Guill 2009; Schröder et al. 2014; Van Kooten et al. 2005).

The scaling of the MRD with body size has been used to explain the occurrence of population dynamic cycles (Persson et al. 1998). When the MRD changes over ontogeny some individuals have a negative energy balance and suffer from starvation, while others have a positive energy balance and can invest in growth and/or reproduction. This destabilizes population dynamics (De Roos and Persson 2003; Persson and De Roos 2013; Persson et al. 1998). An increasing MRD with body size leads to juvenile-driven cycles in which a single dominant cohort outcompetes all other individuals in the population. A decrease in the MRD with body size causes adult-driven cycles, which arise from a retardation of juvenile growth and hence an elongation of the juvenile period due to limited food availability. When the dominant cohort matures, this leads to a sudden increase in fecundity and adult biomass (De Roos and Persson 2003).

Besides its impact on population and community dynamics, asymmetric competition can considerably influence individual life history, with potential evolutionary consequences. For example, a modeling study on Trinidian guppies by Bassar et al.

(2016) revealed that the degree of asymmetry in competition changed both the mean and the variance of the generation time and life expectancy at birth and also the variance of the lifetime reproductive success. By changing the nature of the density-dependence, asymmetric competition is suggested to influence both the direction and speed of evolutionary life history changes (Bassar et al. 2016; Mylius and Diekmann 1995). However, explicit predictions about the eco-evolutionary dynamics of competitive asymmetry were not discussed by Bassar et al. (2016). Evolutionary consequences of intraspecific competition have mainly been studied in the light of ecological character displacement, where increased competition leads to diversification in diet and morphology between individuals (Bolnick 2004; Svanbäck and Bolnick 2007). It remains unclear how the degree and direction of asymmetry in competition affects eco-evolutionary dynamics and whether natural selection would lead to symmetric or asymmetric intraspecific competition.

Both the MBP and the MRD are derived from the energy budget of an individual and are controlled by the rates of assimilation and maintenance metabolism, which generally change allometrically with body size (Glazier 2005; Kleiber 1932; Kooijman 2010; Peters 1983; West et al. 2001). Allometric scaling relationships are described by power functions that contain a proportionality constant and a scaling exponent (Glazier 2005; Kleiber 1932; Peters 1983). Two competing frameworks model ontogenetic growth and provide a value for the allometric scaling exponents of assimilation and maintenance: the ontogenetic growth model of West, Brown and Enquist (OGM-model Hou et al. 2011, 2008; West et al. 2001; Zuo et al. 2012) and Dynamics Energy Budget (DEB) theory (Kooijman 2010; Maino et al. 2014; Sousa et al. 2008, 2010; Van der Meer 2006). In both models ontogenetic growth results from the difference between resource or energy supply and maintenance costs of existing cells (Kearney and White 2012; Van der Meer 2006; West et al. 2001). In the OGM-model energy supply is proportional to the resting metabolic rate, which is assumed to scale with three-quarters power of body mass (Hou et al. 2008; Savage et al. 2004; West et al. 2001). This three-quarters scaling follows from an independent model of a distribution network that delivers resources to terminal units (capillaries). Minimization of the energetic costs in such a network leads to a fractal-like distribution network in which the number of terminal units scales with three-quarters power to body mass (West 1997; West et al. 1999). DEB theory (Kooijman 2010) describes an individual in terms of structural body volume and reserve density. Resource supply is assumed proportional to structural surface area and hence scales with a two-thirds power of structural volume for isomorphically growing organisms, while maintenance costs increase isometrically with volume. This results in Von Bertalanffy growth curves (Kooijman 1986, 2010). As Ricklefs (2003) points out, the two growth models are specific versions of

a broader class of ontogenetic growth models and discriminating between the two on the basis on growth data is often impossible (Banavar et al. 2002).

Recently, data is accumulating that indicates substantial variation in the value of the scaling exponent of metabolic rate and this variation has been related to taxonomic diversity, lifestyle in aquatic organisms (pelagic vs. non-pelagic), temperature, life stage, activity level, physiological state, predation and body shape (Burton et al. 2011; Caruso et al. 2010; Glazier 2005, 2006, 2009; Glazier et al. 2011, 2015; Hirst et al. 2014; Killen et al. 2010). Glazier (2005) argues that the diverse scaling relationships observed in nature result from diverse adaptations in combination with ecological physico-chemical constraints. This suggests that scaling exponents can change adaptively, for example through changes in body shape during ontogeny (Hirst et al. 2014; Killen et al. 2010; Ohlberger et al. 2011). Such changes would alter the degree of competitive asymmetry within the population and have substantial consequences for population and community dynamics, as well as individual life history. However, explicit predictions about the evolutionary dynamics of competitive asymmetry are missing. Therefore, the main purpose of this study is to understand the selection pressures that act on the scaling of competitive ability with body size.

We formulate an individual-level, dynamic energy budget model that describes ingestion, maintenance, growth and reproduction as a function of body mass and resource density. Both assimilation and maintenance rates follow power functions of body mass. The individual-level model is translated to a population-level model by considering the density distribution of individuals along the body mass axis, as in the framework of physiologically structured population models (De Roos 1997; De Roos and Persson 2001; Metz and Diekmann 1986). We explore how the scaling exponents of assimilation and maintenance affect population dynamics and subsequently study the evolutionary dynamics that result from selection pressures on these scaling exponents. Fitness and the direction and strength of selection arise through the feedback between an individual and its environment (Metz et al. 1992). Therefore, we use the framework of adaptive dynamics to study evolutionary change and identify evolutionary endpoints of the scaling exponents of maximum ingestion and maintenance rates (Durinx et al. 2008; Geritz et al. 1998; Metz 2012; Metz et al. 1992).

2.2 – Model description

Ecological dynamics

A dynamic energy budget (DEB) model specifies the rates of resource ingestion, maintenance, growth, reproduction and mortality of an individual, as a function of its body size and resource density. All equations of this model are shown in table 2.1. Both growth and reproduction are modeled as food-dependent processes. The competition for food is incorporated explicitly by considering a dynamic resource on which all consumer individuals feed. A net-production energy budget model is used to model the allocation of resources to the processes of maintenance, growth and reproduction (Gurney and Nisbet 1998; Lika and Nisbet 2000). In a net-production model, maintenance always takes precedence over growth or reproduction. Hence, reproduction and growth are impossible when food conditions are insufficient to cover maintenance requirements. Both maximum ingestion rate and maintenance rate are power functions of body mass. For an individual with mass s the maximum ingestion is given by $M(\frac{s}{s_r})^Q$ and maintenance rate by $T(\frac{s}{s_r})^P$. Here, M and T are, respectively, the maximum ingestion rate and maintenance rate of an individual with body mass s_r. The exponents Q and P describe how these rates scale with body mass s. An increase in Q and P implies an increase in, respectively, the maintenance rate and maximum ingestion rate for individuals with $s > s_r$ and a decrease for individuals with $s < s_r$. Individuals are born with size s_b, mature at size s_j and can reach a maximum size of s_m, when food density is sufficient. In case $s_b < s_r < s_m$ there is a trade-off between individuals with $s < s_r$ and conspecifics with $s > s_r$, for both maximum ingestion rate and maintenance rate. By default a juvenile-adult trade-off is assumed by setting $s_r = s_j$, but deviations from this assumption are explored. Besides the size-dependent maximum ingestion rate, the rate of food ingestion, $I(R, s)$, follows a Holling type-II functional response of resource biomass R with a size-independent half-saturation constant H (see table 2.1). Ingested food is assimilated with efficiency σ and maintenance costs are subtracted from assimilated biomass (Lika and Nisbet 2000). We assume that the conversion efficiency σ includes all the overhead costs involved in producing new biomass through somatic growth or reproduction. The production rate of biomass, denoted by $\Omega(R, s)$, is hence equal to the assimilated biomass minus the maintenance costs (table 2.1).

Depending on the scaling exponents of maintenance and maximum ingestion, individuals can grow to very large body sizes. To prevent this, we model adult allocation towards growth by an sigmoid function $\kappa(s)$ that decreases from one at maturation size s_j towards zero at the maximum individual body size s_m (table 2.1). Hence, asymptotic size is limited to s_m when food availability is sufficient, but this size might not be reached when food is limited. The production not spent on growth

Table 2.1 – Model Equations

Equation	Description
$I(R, s) = M \left(\frac{s}{s_r}\right)^Q \frac{R}{R+H}$	Resource ingestion
$\Omega(R, s) = \sigma I(R, s) - T \left(\frac{s}{s_r}\right)^P$	Biomass production
$\frac{ds(R, a)}{da} = g(R, s) = \kappa(s)\Omega^+(R, s)$ with $s(R, 0) = s_b$	Growth rate
$b(R, s) = \frac{(1-\kappa(s))\,\Omega^+(R, s)}{s_b}$	Fecundity rate
$\kappa(s) = \begin{cases} 1 & \text{for } s < s_j \\ 1 - 3L(s)^2 + 2L(s)^3 & \text{for } s_j \le s < s_m \\ 0 & \text{for } s = s_m \end{cases}$ with $L(s) = \frac{s-s_j}{s_m-s_j}$	Allocation function
$\mu(R, s) = \begin{cases} \mu_c + \mu_j - \frac{\Omega^-(R,s)}{s} & \text{for } s < s_j \\ \mu_c + \mu_a - \frac{\Omega^-(R,s)}{s} & \text{for } s \ge s_j \end{cases}$	Mortality rate
$G(R) = \delta\,(R_{max} - R)$	Resource growth rate

We use $\Omega^+(R, s)$ to denote $\max(\Omega(R, s), 0)$ and $\Omega^-(R, s)$ means $\min(\Omega(R, s), 0)$

is allocated to reproduction in adults, whereas juveniles ($s_b \le s < s_j$) are assumed to spend all production of biomass on growth.

Growth in mass with age a occurs only when the biomass production rate, $\Omega(R, s)$, is positive and is given by the differential equation $\frac{ds(R,a)}{da}$ in table 2.1, with the size at birth as initial condition. Similarly, reproduction only occurs for positive values of $\Omega(R, s)$ and individual fecundity (the rate of offspring production per adult) is given by the function $b(R, s)$ (see table 2.1). Mortality is represented by the function $\mu(R, s)$ which is composed of background mortality μ_c for all individuals and additional size-dependent mortality for juveniles, μ_j, and adults, μ_a (table 2.1). Furthermore, mortality increases when food conditions are insufficient to cover maintenance requirements. This starvation mortality is equal to the magnitude of the mass-specific biomass production when negative. Starvation is handled as an increase in mortality instead of a reduction in body mass. Resource growth follows semi-chemostat dynam-

ics given by the function $G(R)$, with turn-over rate δ and maximum resource density R_{max} (table 2.1).

Model equilibria were computed using the software package PSPManalysis, which is especially designed for demographic, equilibrium and evolutionary analysis of physiologically structured population models (De Roos 2016). The basic method of PSPManalysis involves the numerical integration of a set of coupled ordinary differential equations that describe the change over age a of four life-history characteristics: growth, survival, cumulative resource ingestion and cumulative reproduction. This system of ODEs is integrated repeatedly, while iteratively adjusting the value of the equilibrium resource density $\tilde{R}$ until a solution has been found that satisfies the equilibrium condition. This equilibrium condition is given by the equation $R_0 = 1$, where R_0 represents the lifetime reproductive success of a single individual, given by:

$$R_0 = \int_0^\infty b(\tilde{R}, s(\tilde{R}, a))F(\tilde{R}, a)da \tag{2.1}$$

Here, $b(\tilde{R}, s)$ is the fecundity at equilibrium and the expression $F(\tilde{R}, a)$ is the survival function at equilibrium, which follows from:

$$F(R, a) = e^{-\int_0^a \mu(R, s(R,\alpha))d\alpha} \tag{2.2}$$

Subsequently, the population-level birth rate in equilibrium, $\tilde{B}$, is calculated from the condition that in equilibrium the resource growth rate should equal total population-level foraging:

$$\delta(R_{max} - \tilde{R}) = \tilde{B}\int_0^\infty I(\tilde{R}, s(\tilde{R}, a))F(\tilde{R}, a)da \tag{2.3}$$

The integrals in equations 2.1, 2.2 and 2.3 are calculated by the PSPManalysis package through numerical integration of the ODEs for cumulative reproduction, survival and cumulative resource ingestion, respectively. With the equilibrium values for the resource density, $\tilde{R}$, and population-level birth rate, $\tilde{B}$, the consumer size distribution follows from (De Roos 1997):

$$\tilde{m}(s) = \frac{\tilde{B}}{g(\tilde{R}, s)}\exp\left(-\int_{s_b}^{s}\frac{\mu(\tilde{R}, \xi)}{g(\tilde{R}, \xi)}d\xi\right) \tag{2.4}$$

Using 2.4 juvenile (J) and adult (A) biomass are given by the integral over the size distribution, weighted with body mass s:

$$J = \int_{s_b}^{s_j} s\tilde{m}(s)ds \tag{2.5a}$$

$$A = \int_{s_j}^{s_m} s\tilde{m}(s)ds \tag{2.5b}$$

The PSPManalysis package calculates model equilibria and stage-specific biomass densities (eqs. 2.5a and 2.5b) as a function of any model parameter by means of numerical curve continuation as described in Kirkilionis et al. (2001), Diekmann et al. (2003) and De Roos et al. (2010). The equilibrium analysis was complemented by numerical studies of transient and non-equilibrium dynamics of the model using the Escalator Boxcar Train (EBT) method (De Roos 1988; De Roos et al. 1992). The EBT method calculates population dynamics as a function of time by dividing the size distribution into cohorts of similarly-sized individuals and for every timestep calculating the growth, mortality and reproduction for each cohort, as a function of the body mass of individuals within that cohort and resource density.

Evolutionary dynamics

To study evolutionary dynamics we use the framework of adaptive dynamics, which assumes timescale seperation between ecological and evolutionary dynamics and that evolution is mutation limited (Durinx et al. 2008; Geritz et al. 1998; Metz 2012; Metz et al. 1995). Evolution then boils down to a series of trait substitutions that can result in an evolutionary singular strategy (ESS). At such an ESS, the selection gradient becomes zero and the population either resides at a (local) fitness maximum (evolutionarily stable) or when at a local fitness minimum, undergoes evolutionary branching due to disruptive selection (Doebeli and Dieckmann 2000; Geritz et al. 1998). Since our model has a one-dimensional environment, the occurence of evolutionary branching is impossible and all the ESSs that we encounter are convergence and evolutionarily stable (continuously stable strategies; Eshel 1983). For the selection gradient we use the derivative of lifetime reproductive success with respect to a particular trait p (*i.e.* model parameter) that is subject to evolution. In our model, an ESS obeys the conditions $\frac{\partial R_0(\tilde{R},p)}{\partial p} = 0$. When calculating the model equilbria as a function of trait values, the PSPManalysis software package automatically detects and classifies evolutionary singular strategies according to the subdivision presented in Geritz et al. (1998). Furthermore, the PSPManalysis package is used to calculate evolutionary isoclines. Such a line shows the value of an ESS of one trait (the evolutionary parameter), as a function of a second trait (Dieckmann 2002). We calculating both the evolutionary isocline of Q as a function of P, and vice versa, the evolutionary isocline of P as a function of Q. The intersecting of these two isoclines is the CSS of both Q and P.

Model parameters

The parameters and their default values are summarized in table 2.2 and a more detailed description of the parameter derivation is given in De Roos and Persson (2013). Body size of the consumer is expressed in grams (g) and biomass densities

Table 2.2 – Model Parameters

Symbol	Unit	Value	Description
R_{max}	$mg\ L^{-1}$	30	Maximum resource density
δ	day^{-1}	0.01	Resource renewal rate
Q	–	1	Maximum ingestion exponent
P	–	1	Maintenance exponent
M	$g\ day^{-1}$	0.1	Maximum ingestion constant
T	$g\ day^{-1}$	0.01	Maintenance constant
μ_c	day^{-1}	0.0015	Background mortality
μ_j	day^{-1}	0.0	Additional juvenile mortality
μ_a	day^{-1}	0.0	Additional adult mortality
σ	–	0.5	Assimilation efficiency
H	$mg\ L^{-1}$	3	Half-saturation density
s_b	g	0.1	Size at birth
s_j	g	1	Size at maturation
s_r	g	1	Scaling reference size
s_m	g	10	Maximum size

are expressed in milligrams per liter ($mg\ L^{-1}$). Across differently-sized species, the mass-specific maximum ingestion, mass-specific maintenance and mortality rates are inversely proportional to the quarter power of adult body size (Kleiber 1932; Peters 1983; Savage et al. 2004). Taking time in days and adult body weight in grams, representative proportionality constants of these scaling relationships for invertebrate species are 0.1, 0.01 and 0.0015 for maximum ingestion, maintenance and mortality, respectively (De Roos and Persson 2013). For the parameterization we take the characteristic adult body mass to equal the size at maturation, for which we adopt a value of 1 gram. Hence $M = 0.1$, $T = 0.01$ and $\mu_c = 0.0015$. A value of 0.01 is adopted for the resource renewal rate δ, so that resource turn-over equals the mass-specifc maintenance rate of an individual with size s_j. Only a change in the ratios between these four rates changes model predictions, as changing their absolute values all with the same factor only scales the unit of time. Values for the volume related parameters are $R_{max} = 30\ mg\ L^{-1}$ and $H = 3\ mg\ L^{-1}$ (table 2.2). The value for H is derived from zooplankton grazing rates as presented by Hansen et al. (1997) and R_{max} is assumed one order of magnitude larger than H (De Roos and Persson 2013). As long as the ratio of these volume-related parameters remains constant a change in these parameters

is equivalent to a scaling of the volume in which the consumer-resource interaction takes place. This does not qualitatively change model predictions. Default parameters for size at birth and maximum size are 0.1 and 10, but these values are changed during model analysis. The scaling exponents Q and P are varied throughout the analysis, but their values are limited to the range 0.4–1.3, which is the observed range of intraspecific scaling exponents found by Clarke and Johnston (1999) for post-larval teleost fish.

2.3 – Results

Measures of competitive asymmetry

One measure for competitive ability is the mass-specific biomass production (MBP) rate. For the same resource densities individuals with a higher MBP can be considered competitively superior. According to this measure, size-independent competition only occurs when the MBP is a constant function of body mass s:

$$\frac{d}{ds}\left(\frac{\Omega(R,s)}{s}\right) = 0. \tag{2.6}$$

From the expression for $\Omega(R, s)$ in table 2.1, it can be inferred that this only holds when $Q = P = 1$.

Another measure of competitive ability is the maintenance resource density (MRD), which is the resource level at which the MBP equals zero and follows from solving $\Omega(R, s) = 0$ for R:

$$MRD = \frac{T\left(\frac{s}{s_r}\right)^P H}{\sigma M\left(\frac{s}{s_r}\right)^Q - T\left(\frac{s}{s_r}\right)^P} \tag{2.7}$$

Individuals with a lower MRD are competitively superior to those with a higher MRD. It can be easily seen from equation 2.7 that the MRD is a constant function of body mass s when $Q = P$, irrespective of the value of Q and P. Hence, under the measure of the MRD competition is symmetric when $Q = P$, while under the measure of the MBP competition is symmetric when $Q = P = 1$.

Equilibrium and evolutionary analysis of Q and P

Exploring the model equilibria as a function of Q and P shows a similar pattern. In case $P > Q$, the maintenance rate increases faster with body mass s than the maximum ingestion rate (low Q values at left-hand side in figure 2.1A-D, high P values at right-hand side in figure 2.1E-H). Consequently, the MBP decreases with size while the MRD increases with body size. In such a population, the smallest individuals are most competitive and competitive ability decreases with size. A stable equilibrium results

in which the asymptotic body size is well below the maximum possible asymptotic size (figure 2.1D,H), due to the low competitive ability of adults. Adults grow until they spend all their assimilated biomass on maintenance and cannot grow any further. This explains the coincidence of the equilibrium resource density with the MRD of the largest individuals in the population for $Q < P$ (figure 2.1A,E). Because of the low competitive ability of adults, total productivity in the adult stage is insufficient to compensate for adult biomass loss due to mortality. This results in a net biomass loss in the adult stage, which is compensated for by a net biomass gain in the juvenile stage. This is shown by the fact that the total rate of biomass recruitment to the adult stage through maturation, is larger than the total rate of biomass recruitment to the juvenile stage through reproduction (figure 2.1C,G). Juveniles therefore grow rapidly, but growth and reproduction of adult individuals is slow since they spend a considerable amount of their assimilated biomass on maintenance. As a result total population biomass mainly consists of adult individuals (figure 2.1B,F).

When the maximum ingestion rate increases faster with body size than the maintenance rate, the MBP increases with body size, while the MRD decreases with body size. This means that large individuals are competitively superior ($Q > P$; large Q values at right-hand side in figure 2.1A-D, small P values at left-hand side in figure 2.1E-H). This results in adult-driven cycles in which adults that are close to the maximum size (figure 2.1D,H) hinder growth of the newborn cohorts. Growth of newborns only occurs when background mortality has sufficiently diminished adult density to allow the resource density to increase above the MRD of newborn individuals (figure 2.1A,E). Because individuals increase in competitive ability during growth, the growing juveniles decrease resource density again and inhibit growth of later cohorts. If abundant enough, these later cohorts will either catch up with the earlier produced individuals when resource densities increase again, or die due to background and starvation mortality. Adult-driven cycles exists for $P = 1$ and $Q > 1$, as well as for $Q = 1$ and $P < 1$. The amplitude and period of the cycles increase with increasing difference between Q and P.

An continuously stable strategy (CSS) exists at the value of Q and P at which the equilibrium resource density reaches a minimum. The CSSs for Q and P are indicated with dashed vertical lines in figure 2.1 and both are within the stable region of parameter space. From this point onward we refer to the CSS-values of Q and P as $\bar{Q}$ and $\bar{P}$, respectively. We confirmed convergence stability of $\bar{Q}$ and $\bar{P}$ within the parameter range of population cycles by explicitly assessing the growth rate of mutant phenotypes in the cyclic attractor of the resident phenotype. For all values of Q and P, the mutant with a trait value closer to the CSS successfully invades and replaces the resident population. The evolutionary isoclines of $\bar{Q}$ as a function of P as well as $\bar{P}$ as a function of Q are shown in the $Q - P$-parameter space in

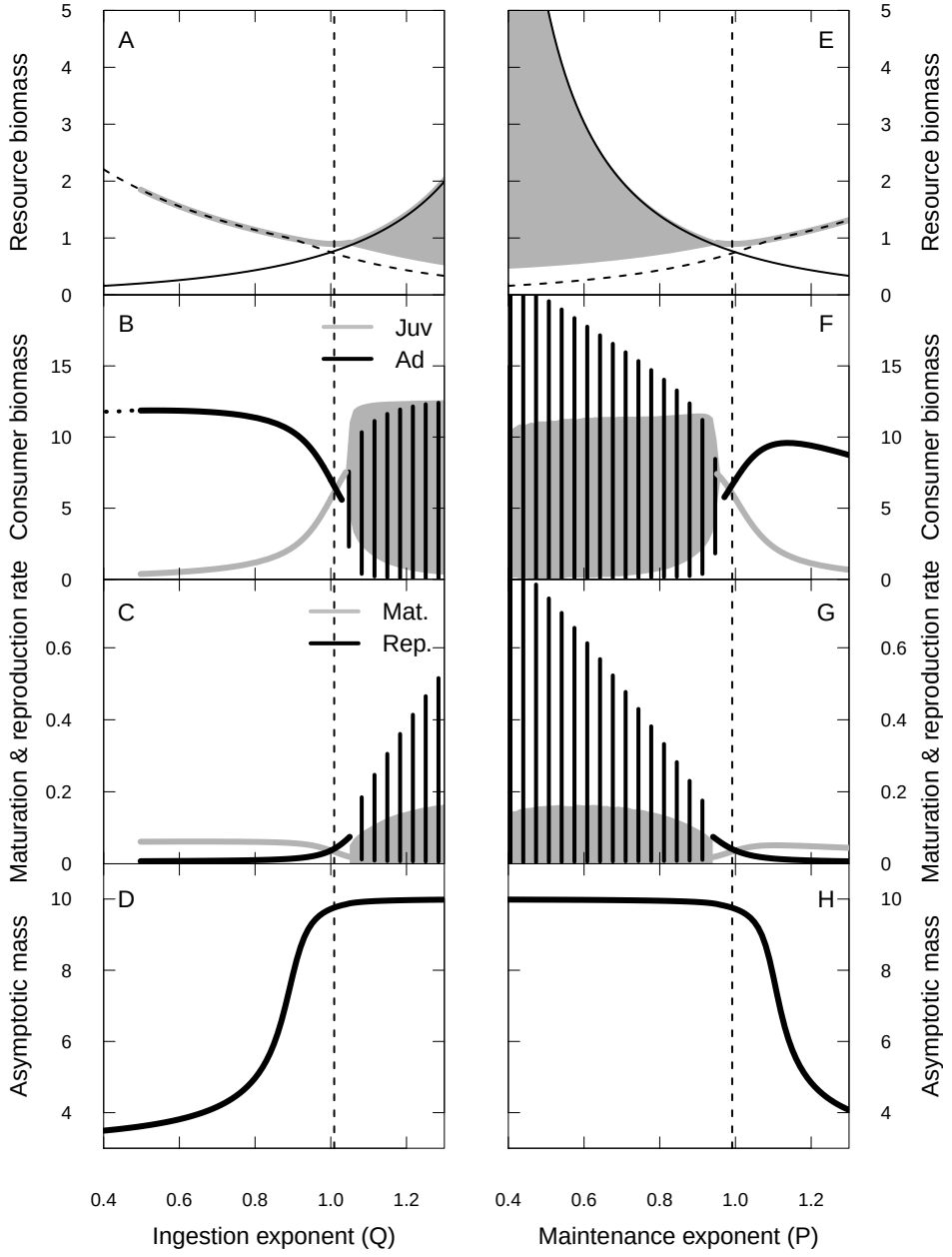

FIGURE 2.1 – Model dynamics as a function of the maximum ingestion scaling exponent Q (left with $P = 1$) and the maintenance rate scaling exponent P (right with $Q = 1$). Thick lines indicate stable model equilibria, while the solid-filled and dashed areas show the range and extent of population cycles. A,E: resource biomass (gray thick lines and shading) and the maintenance resource density (MRD) for the smallest (solid black lines) and largest (dashed black lines) individuals in the population. B,F: adults and juvenile biomass. C,G: total population reproduction rate and maturation rates in biomass. D,H: body mass of largest individuals in the population (asymptotic body size). The vertical thick dashed lines show the position of the CSS of Q in panels A:D, and the CSS of P in E:H. All other parameters as in table 2.2.

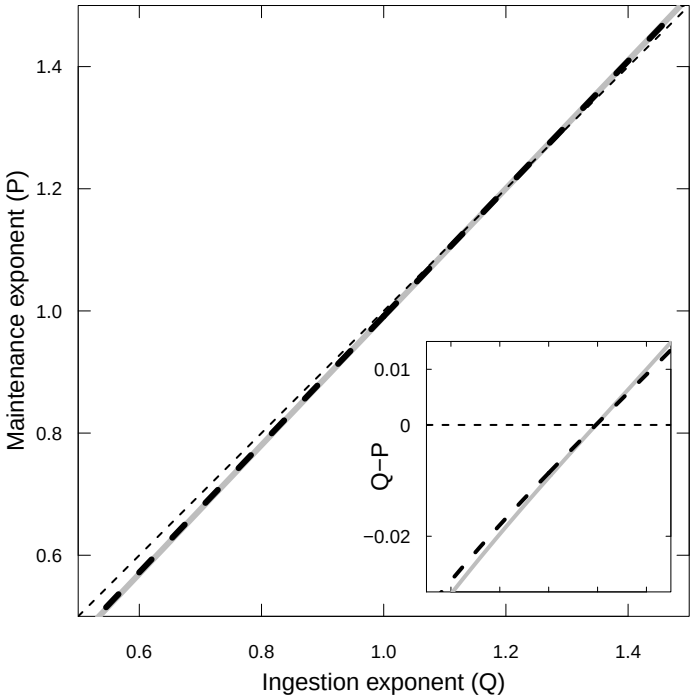

Figure 2.2 – Evolutionary isoclines, showing the value of the CSS of Q as a function of P (gray line), and the CSS of P as a function of Q (black dashed line) in the $Q-P$-plane. The thin dashed line represents the line where $Q = P$. The inset shows the difference between each of the two evolutionary isoclines and the line $Q = P$, as a function of Q (x-axis range identical to main figure). Isoclines cross exactly when this difference is zero (indicated by thin dashed line). All other parameters as in table 2.2.

figure 2.2. The isoclines appear to be on top of each other, but closer inspection reveals that they cross at $\bar{Q} = \bar{P}$ (inset figure 2.2). The small difference between the isoclines means that the CSS-value of one scaling exponent is approximately equal to the value of the other, non-evolving, scaling exponent. Therefore, evolutionary change in only one scaling exponent leads to approximately the same value of the other scaling exponent. Moreover, the evolutionary isoclines cross at zero difference, which implies that the CSS of both Q and P has the property that $\bar{Q} = \bar{P}$. For the default parameters (table 2.2), the common CSS-value is $\bar{Q} = \bar{P} \approx 1.19$. For this CSS-point the MRD does not change with body size, while the MBP increases with size, since $\bar{Q} = \bar{P} > 1.0$. Per unit biomass, larger individuals produce more new biomass than smaller individuals and the population-level biomass reproduction rate exceeds the population-level biomass maturation rate. In appendix 2.A we show that the convergence of the two exponents to the same value is independent of the assumption $s_r = s_j$, while the scaling of the MBP with body mass in the CSS-point is

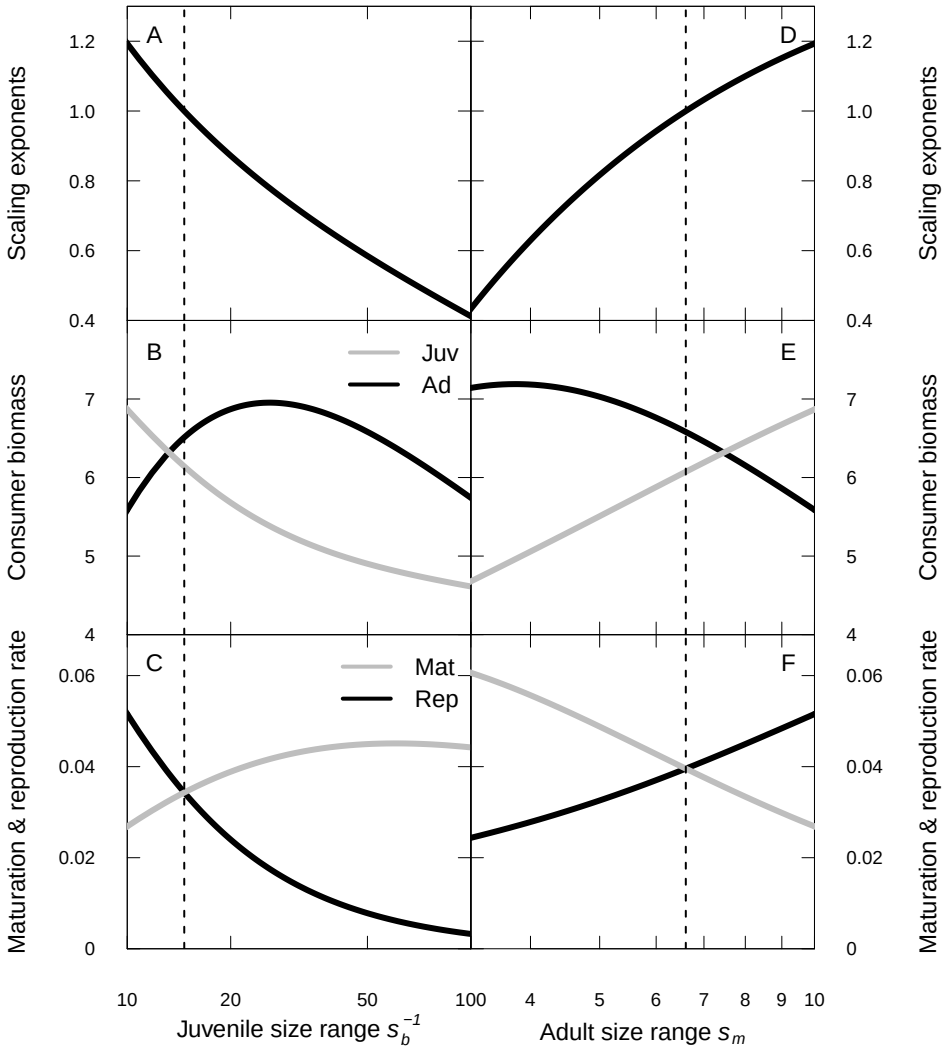

Figure 2.3 – CSS-values of scaling exponents of maximum ingestion rate Q and maintenance rate P (A,D: black line), as a function of the juvenile size range, parameterized by s_b^{-1} (A-C) and the adult size range, parameterized by s_m (D-F). A,D: common scaling exponent of maximum ingestion and maintenance rate. B,E: adult and juvenile consumer biomass. C,F: population-level reproduction and maturation rates in biomass. The vertical dashed lines indicated the value of s_b^{-1} (A-C) and s_m (D-F) where $\bar{Q} = \bar{P} = 1$.

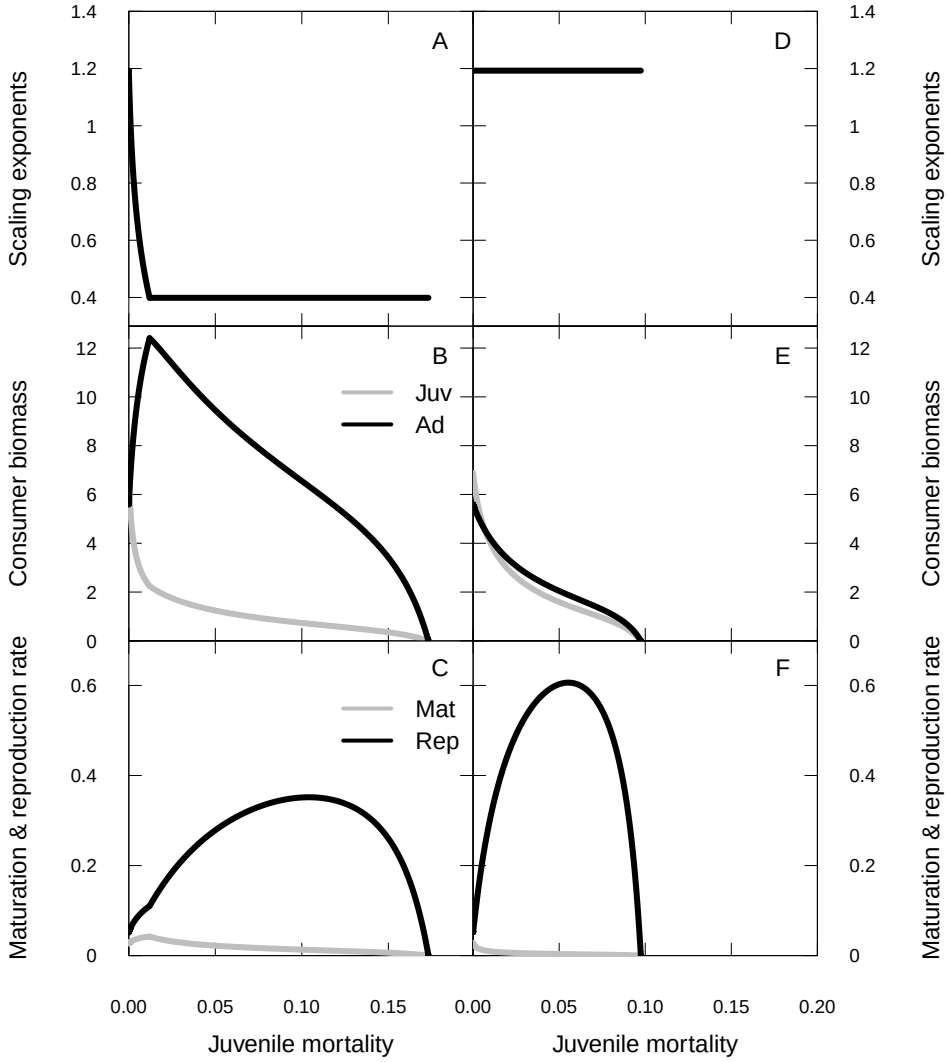

Figure 2.4 – Equilibria as a function of increasing mortality for juveniles (μ_j), for evolving (A-C) and non-evolving scaling exponents (D-F). A,D: common scaling exponent of maximum ingestion and maintenance rate. B,E: adult and juvenile consumer biomass. C,F: population-level reproduction and maturation rates in biomass. Default parameters as in table 2.2 where $\bar{Q} = \bar{P} = 1.193$ at $\mu_j = 0.0$.

dependent on the reference size s_r. Increasing the value of s_r leads to a decrease of $\bar{Q} = \bar{P}$.

The evolutionary convergence of the scaling exponents to a common CSS-value is robust against changes in the size at birth and the maximum body size. However, the value of the evolutionary endpoint is influenced by these size parameters. In general, increasing the size range of a life stage changes $\bar{Q}$ and $\bar{P}$ such that the MBP of this life stage increases. Figure 2.3 shows the effect of changing size at birth and maximum size on $\bar{Q}$ and $\bar{P}$. A decrease in s_b and hence an increase in the juvenile size-range decreases the common CSS-scaling exponent, which increases the MBP for small individuals. This leads to an increase in maturation rate and a decrease in reproduction rate (figure 2.3C). Biomass accumulates in the adult life stage and the juvenile/adult biomass ratio decreases. Alternatively, a larger adult size range increases the common value of $\bar{Q}$ and $\bar{P}$ (figure 2.3D). As a consequence, the reproduction rate increases, the maturation rate decreases and the consumer population becomes dominated by juveniles (figure 2.3E,F). Their is a combination of s_b and s_m values for which $\bar{Q} = \bar{P} = 1$. This size ratio is indicated with vertical dashed lines in figure 2.3. Only for this combination of juvenile and adult size ranges, does the model predict the MBP to be independent of body size.

The evolutionary response to increasing juvenile and adult mortality is shown in figure 2.4 and appendix figure 2.C1. This evolutionary response is compared with the response to increasing mortality when the scaling exponents do not evolve (figure 2.4D-E). In case of increased juvenile mortality and non-evolving scaling exponents, $Q = P > 1$ and this causes a larger MBP for adults compared to juveniles. This can be inferred from the population level reproduction rate being larger than the population level maturation rate (figure 2.4F). Increasing juvenile mortality initially increases reproduction rate due to increased resource availability (not shown), while maturation rate immediately decreases for non-evolving scaling exponents. In case of evolving scaling exponents, $\bar{Q}$ and $\bar{P}$ are again equal. However, additional juvenile mortality does change the common CSS-value. Increasing juvenile mortality (μ_j; figure 2.4) decreases $\bar{Q} = \bar{P}$ such that the increased loss rate of juvenile biomass is compensated for by an increased juvenile MBP. This compensatory response does not change the result that $\bar{Q} = \bar{P}$. Therefore, juveniles also suffer from increased maintenance costs due to a decrease in P. The decrease in $\bar{Q} = \bar{P}$ leads to an increase in adult biomass and even total consumer population biomass, until the exponents hit the lower constraint of 0.4. This initial increase comes about through an increase in maturation rates (figure 2.4C), while for a constant Q and P the maturation rate never increases and there is no increase in stage-specific biomass with increasing mortality (figure 2.4E,F). Further increases in mortality lead to a decrease of consumer biomass until the consumer goes extinct at juvenile mortality values that are substantially higher compared to those in

the absence of an evolutionary response. The response to increased adult mortality (μ_a) is comparable but leads to higher rather than lower values of $\bar{Q}$ and $\bar{P}$ and an increase in juvenile biomass through an increase in reproduction rate (see appendix figure 2.C1). This increase also only occurs for evolving scaling exponents. Similar to increased juvenile mortality, the consumer goes extinct at higher adult mortality rates when the scaling exponents evolve compared to non-evolving exponents (figure 2.C1).

2.4 – Discussion

Recent studies show considerable variation in the intraspecific scaling of maintenance metabolism with body size and relate this variation to ecological and environmental factors such as, among others, temperature, lifestyle, predation, body shape and activity level (Caruso et al. 2010; Glazier 2005, 2010; Glazier et al. 2011, 2015; Hirst et al. 2014; Killen et al. 2010; Ohlberger et al. 2012*b*, 2007). Metabolic rates affect competitive ability. Therefore, changes in competitive ability during ontogeny can arise through changes in the scaling of metabolic rate with body mass. The population and community effects of such size-dependent changes in competition are well documented. Here we report the first results on the evolutionary dynamics of the scaling of competitive ability with body size. In case of a trade-off in the energetics between small and large bodied individuals (newborn or juveniles versus adults), the scaling exponents of maximum ingestion and maintenance evolve to minimize the competitive asymmetry within the population. This is achieved when maintenance and ingestion scale in the same way with body size. Only in this case all differently-sized individuals are equal with respect to the resource density they require to cover their maintenance costs. We show that this result is robust against changes in the juvenile and adult size ranges (figure 2.3), size-dependent mortality (figure 2.4 and appendix figure 2.C1) and the reference size on which the scaling exponents are parameterized (appendix figure 2.A1).

The evolutionary prediction of equal scaling exponents of maintenance and maximum ingestion is at odds with the existing theories about ontogenetic growth. These theories assume an isometric increase of maintenance costs ($P = 1$) and a sublinear allometry of maximum ingestion rates ($Q = 3/4$ or $Q = 2/3$ Hou et al. 2008; Kooijman 2010; Moses et al. 2008; Van der Meer 2006; West et al. 2001, 2004). Such a combination of scaling exponents leads to a decrease in competitive ability over ontogeny, as measured by the maintenance resource density. We show that this results in negative selection on the maintenance exponent and positive selection on the maximum ingestion exponent. Also, our results show that when either Q or P is fixed, the other exponent still evolves to a value close to the evolutionarily constrained scaling exponent. Therefore, in this model there can only be a difference between both exponents

when they are both constrained and non-evolvable. Selective change in one or both exponents will eventually bring them together.

It is difficult to assess experimentally whether the scaling of maintenance rate is different from that of ingestion. One reason is that the exact maintenance costs are often hard to quantify. Measures like resting or basal metabolic rate are used frequently, but even when organisms are not feeding they can still invest in growth or reproduction. In which case the measured metabolic rate still includes overhead costs for these investments (Kooijman 1986; McCauley et al. 1990). Even if measurements of metabolic rate of non-feedings individuals would provide a reliable estimate of their maintenance costs, the resulting scaling exponents often show considerable variation (Glazier 2005). Likewise, the scaling of ingestion rate with body size or volume also varies considerably (Maino and Kearney 2015). DEB theory assumes that ingestion is a surface related process and scales with a two-thirds power of (structural) volume (Kearney and White 2012). This appears to be true for non-volant mammals, in which the small intestinal surface area scales with body mass$^{2/3}$. However, the pooled mass exponent for both non-volant mammals and birds is 0.73 and for birds alone it is even higher (Kearney and White 2012). For insects the surface-area scaling of ingestion describes the central tendency when averaged across several species, but for 8 out of the 38 species (21%) tested by Maino and Kearney (2015), the scaling exponent was significantly different from two-thirds. Due to the variation in scalings of both maintenance and ingestion rates a comparison between the two can only be informative when measurements are performed under identical conditions and for the same species or even the same population. This considerably limits the amount of information available to compare scalings of ingestion with those for maintenance rate.

A well studied case is that of *Daphnia* sp., in which the dependence of ingestion and maintenance metabolism was reviewed by McCauley et al. (1990) and Gurney et al. (1990). Maintenance rates of *Daphnia* sp. increased superlinear with body mass (> 1) due to contributions to carapace formation (McCauley et al. 1990). Ingestion rates were best described by separate functions for juveniles and adults (McCauley et al. 1990), but the overal scaling exponent was approximately 0.73. Models of ontogenetic growth in which maintenance costs increase faster with body mass than ingestion rates lead to decreasing growth rates with increasing size. Such a pattern often performs well in describing observed growth trajectories (Peters 1983; Ricklefs 2003). Equal scaling exponents would lead to an exponential growth pattern, which are less often observed (Kooijman 2010). In conclusion, the data from Daphnia and most growth patterns show that the maintenance rate scaling exceeds the ingestion rate scaling. This contradicts the evolutionary prediction of equal scaling exponents.

Another way to assess the scaling of maintenance and ingestion rates is to study the scaling of the MRD with body size directly from experiments that measure individual growth at different food levels and from this derive the food density where growth is zero (Gliwicz 1990; Kreutzer and Lampert 1999). Several studies indicate that the MRD is an increasing function of size, leading to a competitive advantages of small individuals (Aljetlawi and Leonardsson 2002; Byström and Andersson 2005; Byström et al. 2006; Hjelm and Persson 2001; Jansen et al. 2003; Lefébure et al. 2014). Aljetlawi and Leonardsson (2002) show that the MRD increases with body size in the amphipod *Monoporeia affinis* and that this increase is attributable to an decrease in handling time (which in Holling's functional response is the inverse of maximum ingestion) with mass$^{0.43}$, while resting metabolic rates increases isometrically with size. Consequently, juveniles are competitively superior to adults, which provides an explanation for the often observed fluctuating population dynamics of this species (Aljetlawi and Leonardsson 2002). The often observed increase of the MRD with body size is at odds with equal scaling exponents of maintenance and ingestion.

As shown in figure 2.4E and appendix figure 2.C1E, equal scaling exponents prevent the occurrence of biomass overcompensation. This is the increase in stage-specific biomass with increasing mortality (De Roos et al. 2007). Schröder et al. (2014) conclude that biomass overcompensation is likely to be commmon in natural systems and especially leads to increases in juvenile biomass. Overcompensation in juvenile biomass is indicative of an energetic bottleneck in the adult life stage. A higher scaling of maintenance metabolism compared to the scaling of ingestion could lead to such an energetic bottleneck for adults. A community consequence of biomass overcompensation is the emergent Allee effect, in which a size-specific predator changes the size distribution of its prey to ensure its own persistence (De Roos et al. 2003*b*; Van Kooten et al. 2005). Emergent Allee effects have been found in freshwater fish stocks (Persson et al. 2007) and cannot occur without an energetic bottleneck in the prey population. The observed stable population dynamics for equal scaling exponents provides another indication that the true exponents of maintenance and ingestion differ. As shown in figure 2.1, equal scaling exponents lead to stable population dynamics. This contradicts laboratory and empirical observations of fluctuating population dynamics for *Daphnia* sp. (McCauley et al. 2008, 1999) and the suggestion that cyclic population dynamics are widespread in nature (Murdoch et al. 2002).

In summary, the detailed studies on the physiology and population dynamics of *Daphnia* sp, the observed increase in the MRD with body size in several fish and crustacean species and the observed population and community consequences of unequal scaling exponents all support the tenet that maintenance rates increase faster with body mass than ingestion rates. This is at odds with the evolutionary predictions derived in this paper. One simple explanation for this discrepancy could be that the

scaling exponents are constrained and hence cannot evolve. There is at least some evidence against this explanation. First of all, the range of observed intraspecific scaling exponents does suggest there is variation for selection to act on (Glazier 2005; Moses et al. 2008). This variation has been related to lifestyle, activity, growth form, temperature and predation (Glazier 2005, 2006; Killen et al. 2010; Kiørboe and Hirst 2014; Ohlberger et al. 2012*b*, 2007). For example, White et al. (2011) experimentally showed that metabolic rate scales with mass$^{0.5}$ for colonies of a bryozoan species, while other colonial marine invertebrates have scaling exponents of 0.75, 1 or 1.125 (Hughes and Huges 1986; Nakaya et al. 2003). These different scaling exponents are explained by the differences in growth forms of the colonies (White et al. 2011). Two-dimensional growth at the colony edge leads to exponents around 0.5, while growth forms that deviate from circular lead to higher exponents (White et al. 2011). Also on the individual level, body shape changes can influence the intraspecific scaling of metabolic rates. As shown by Hirst et al. (2014) growth in 3 dimensions (isomorphic growth) is related to low scaling exponents, while one dimensional growth (elongation) relates to exponents around one. These findings are in favor of theories that assume metabolic scaling to be determined by transport of materials across surfaces and indicate that changes in growth form can influence the scaling of resource supply rates. Selection would hence be able to change the scaling of maximum ingestion with body size by altering the dimension of ontogenetic growth.

Secondly, the possibility that the existing variation in the scaling of metabolic rate with body size is an evolutionary adaptive and phenotypically plastic response, is discussed by Ohlberger et al. (2012*b*). These authors show that in one pair of related coregonids (*Coregonus albula* and *C. fontanae*), which live in cold environments, the scaling exponent of metabolic rate with body mass decreases linearly with temperature, while in a pair of related cyprinids (*Abramis brama* and *Rutilus rutilus*), which live in warmer waters, the body-mass scaling of metabolic rate is independent of temperature. This indicates that the response of the body-mass scaling of metabolism to temperature differs between species or taxa and can be dependent on their environment or evolutionary history (Ohlberger et al. 2012*b*). The hypothesis that evolutionary adaptions to different environments can lead to differences in scaling exponents is further illustrated by Glazier et al. (2011). These authors show that the scaling of resting metabolic rate in the amphipod *Gammarus minus* depends on the presence of fish predators. Individuals from three populations that naturally co-occur with the predatory fish *Cottus cognatus* have lower scaling exponents than individuals from two populations in which these predators are absent. The lower scaling exponents for the individuals from the predatory lakes resulted in a higher metabolic rates for small individuals and a lower metabolic rate for large individuals. Furthermore, Glazier et al. (2011) show by using field enclosures that juvenile growth

is increased and adult growth is reduced in individuals from the fish-exposed populations. This resulted in faster growth towards lower asymptotic sizes. The results of Glazier et al. (2011) correspond to our evolutionary predictions in case of increasing juvenile mortality. This lowers the CSS of the common scaling exponent of maintenance and maximum ingestion, in order to increase the mass-specific biomass production (growth) of juveniles. The increase in mass-specific production of juveniles occurs at an expense of the mass-specific production of adults. This decreases adult growth, resulting in a decrease in asymptotic body size.

More complex ecological scenarios or life histories can possibly lead to different predictions about the evolution of the scaling exponents of metabolism. The current analysis shows that the evolutionary convergence of the body-mass scaling exponents of maintenance metabolic rate and maximum ingestion rate towards the same value is robust against changes in many of the model parameters. However, it remains to be studied to which extent this result depends on the current net-production allocation scheme or the consideration of a single, shared resource. Also, increased ecological complexity by incorporating additional ecological interactions are likely to affect the predictions. For example, complexity such as ontogenetic diet shifts, prey/predator size ratios and interference competition have been shown to reduce the size-dependency of the MRD, and in this way counteract the negative competitive effect of small individuals on the large conspecifics. For example, Aljetlawi and Leonardsson (2002) show that interference competition of large *M. affinis* increases the MRD of the smaller size-classes. Also ontogenetic diet shifts can reduce the negative effects of competition for large individuals. As observed in many fish species, the attack rate of zooplankton is a humped shaped function of body size, as foraging capacities become hindered by large size differences between predators and prey (Byström and Andersson 2005; Hjelm and Persson 2001). This often coincides with the size threshold at which individuals switch to a second resource, such as benthic macroinvertebrates or other fish species. Indeed, Byström and Andersson (2005) show that the increase of the MRD for Arctic char *Salvelinus alpinus* was substantially reduced when using macroinvertebrates as prey, instead of zooplankton. Also, Jansen et al. (2003) demonstrate that the decrease in attack rate of Arctic char on zooplankton occurs at a larger size when large zooplankton prey are used and that this substantially reduces the increase in MRD with body size. Future research should point out how the evolution of size-scaling exponents depend on additional ecological complexity, such as ontogenetic diet shifts or interference competition.

Furthermore, we show that the common value of the maximum ingestion and maintenance scaling exponents depends on the mortality rates and the size-ranges of the juvenile and adult life stages (figure 2.3, 2.4 and 2.C1). Either an increase in the juvenile size-range (for species with a lower size at birth) or a high juvenile mortality

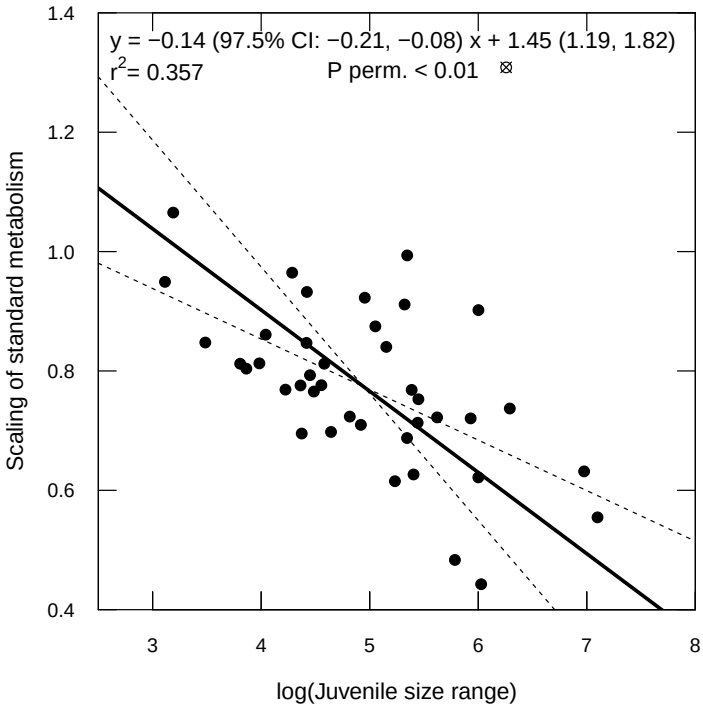

Figure 2.5 – The scaling of standard or routine metabolism with body size of 41 teleost fish species, versus the ratio of length at maturation and egg diameter, as a measure for the juvenile size-range. Data on scaling exponents are from Killen et al. (2010). See appendix 2.B for a detailed description of data collection and analysis. The lines show a RMA regression with 97.5% confidence intervals. The point indicated by the open circle with the cross is the eel *Anguilla anguilla*, which was not part of the regression equation (see appendix 2.B).

rate favor selection towards increased juvenile growth rates. Similarly, an increase in the adult size-range (for species with a higher maximum size) or a high adult mortality rate favor selection towards increased post-maturation growth and reproduction rates. On the basis of this work we therefore predict low scaling exponents for species that have a large size at maturation compared to size at birth or suffer from high juvenile mortality. Alternatively, species with a limited juvenile size-range or low juvenile mortality rates are expected to have higher scaling exponents for maintenance and ingestion, even so for species with high adult mortality or substantial opportunity for post-maturation growth. The quantitative predictions for the scaling exponents of maintenance and ingestion very much depend on these size and mortality parameters, but the qualitative trends remain unchanged.

We tested the prediction about the scaling of metabolism in relation to the juvenile size range by using data on scaling exponents of standard or routine metabolic rate for teleost fish as published by Killen et al. (2010) and Clarke and Johnston (1999) and combining these with estimates of size at maturation and size at birth. We obtained estimates for these size parameters for 41 of the 89 species in the original dataset of Killen et al. (2010). Instead of weight estimates we used length at maturation (l_{mat}) and egg diameter (l_{egg}), since these are more readily available for fish. In figure 2.5 the temperature-corrected scaling exponent of standard metabolism is plotted against the logarithm of l_{mat}/l_{egg}. The regression line in figure 2.5 results from a Ranged Major Axis model (Legendre 2014). In agreement with our prediction that the common scaling exponent of ingestion and maintenance rate decreases with an increase in the juvenile size range, figure 2.5 indeed shows a significant negative relationship between the scaling exponent of standard metabolism and the juvenile size-range, despite considerable variation in metabolic scaling exponents, egg diameters and maturation sizes (Bagenal 1971; Clarke and Johnston 1999; Kamler 2005; Killen et al. 2010). The data and references are available in appendix table 2.B1 and details on data collection and statistics are described in appendix 2.B.

In this study we ignore the proximate causes that lead to the allometric scaling of maintenance metabolism and ingestion and instead focus on the ultimate, evolutionary causes that are shaped by how individuals interact with each other through their interaction with a shared environment. Such interactions ultimately determine fitness and drive evolutionary change (Metz et al. 1992). Although this model considers a simplified individual energy budget and includes only the most simple environmental feedback, it provides a powerful and robust null model against which to evaluate evolutionary considerations regarding the intraspecific scaling of ingestion and maintenance with body size. The model predictions paradoxically contrast with a substantial amount of empirical findings, both observational and experimental. Further study should focus on the consequences of more complex ecological scenarios, as well as more complex life histories, for evolutionary dynamics of asymmetric, intraspecific competition.

Acknowledgments

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Appendix 2.A Supplementary figure

We assume a juvenile-adult trade-off in both maximum ingestion rate and maintenance rate. This trade-off is implemented by parameterizing the power functions that describe these rates, at the size at maturation. When another reference size than the size at maturation is used to fix these power functions, both exponents still evolve towards a common value. This CSS-value increases with decreasing reference size, see figure 2.A1

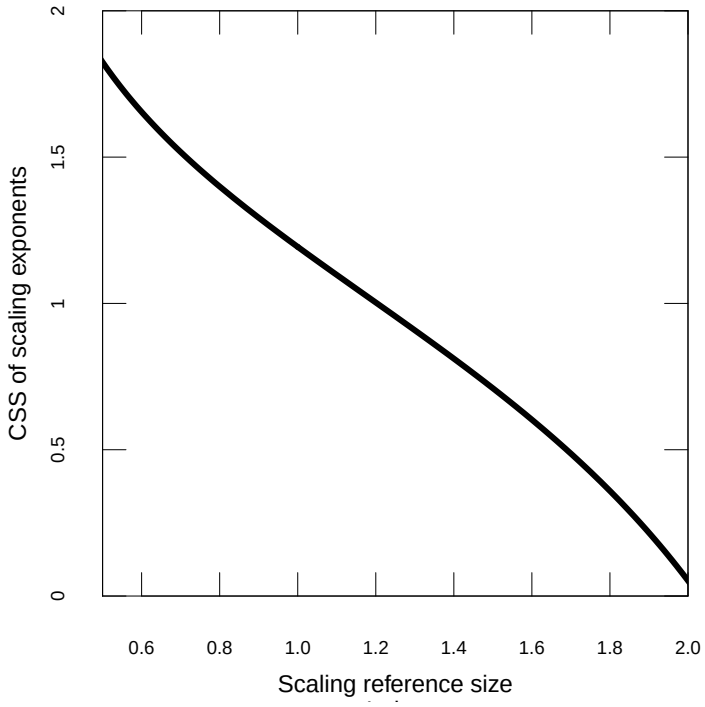

Figure 2.A1 – Value of the evolutionary equilibrium (CSS) of the common scaling exponent of maximum ingestion and maintenance rate, as a function of the scaling reference size s_r. All other parameters as in table 2.2.

Appendix 2.B Data collection and analysis

Data collection

Data on the relationship between metabolic rate and body mass were taken from the supporting information of Killen et al. (2010). This dataset provides estimates on metabolic scaling exponents (b) and metabolic activity level (L), defined as "the mass-specific metabolic rate estimated at the body mass corresponding to the midpoint of the allometric relationship" (Killen et al. 2010) for 89 species of Teleost fish. The data is an extension of the data provided by Clarke and Johnston (1999) and selection criteria handled by both Killen et al. (2010) and Clarke and Johnston (1999) were aimed at selecting rates of standard or routine metabolism. Only data on resting, post-larval, fasted animals measured in absence of additional stressors and after a 24h acclimation period were accepted (Killen et al. 2010). Additionally, the temperature (T) at which metabolism was measured should be within the natural temperature range of the species and the metabolism should be measured over a suitable body-mass range (see Clarke and Johnston 1999 and Killen et al. 2010 for a more detailed description of acceptance criteria).

To test the hypothesis that the scaling of metabolic rate changes with the ratio between size at birth and size at maturation we collected data on egg diameter (l_{egg}) and length at maturation (l_{mat}) for the species in the dataset of Killen et al. (2010). Length instead of mass estimates were used since the former are more readily available for fish. Furthermore, any pattern in the length ratios will remain intact after a non-linear transformation to mass ratios. Data collection was carried out at a species-specific level and the estimates for l_{mat} and l_{egg} are generally not from the same source, nor is any of the two directly derived from populations or individuals that were used for the metabolic rates measurements. There is considerable variation in both size at maturation and egg size between individuals of the same population, between different populations of the same species and with size and age of the parents (Bagenal 1971; Bøhn et al. 2004; Bonislawska et al. 2001; Kamler 2005; Wallace and Aasjord 1984). This variation could not be controlled for and is taken for granted since estimates performed on the same individuals for our variables of interest (l_{egg}, l_{mat} and b) were not available.

Various sources report different estimates of l_{egg} and l_{mat} for the same species and these data were averaged to arrive at a species-specific prediction. Main sources for maturation size estimates were Winemiller and Rose (1992), King and McFarlane (2003) and Fishbase (Froese and Pauly 2016). Fishbase estimates were collected using R (R Core Team 2015) with the package 'rfishbase' (Boettiger et al. 2016, 2012). Single estimates for l_{mat} were preferred over range estimates and the latter were only used when both minimum and maximum values were reported. Estimates of l_{egg} were

mainly derived from Winemiller and Rose (1992), King and McFarlane (2003) and Russel (1976). Additional searches for egg size were performed for species if size at maturation estimates were provided in the sources mentioned above. This resulted in a total of 41 species with estimates of l_{egg}, l_{mat} and b. All data and data source are reported in table 2.B1.

Data analysis

Killen et al. (2010) show an effect of the temperature (T) at which the metabolic measurements where performed on both the scaling of metabolism with body size (b) and on the metabolic level (L). Furthermore, the authors show an effect of $\log(L)$ on b. To correct for these dependencies in our analysis we calculate the effect of T on the $\log(L)$ for the original 89 species described in Killen et al. (2010). This analysis is identical to the one described in the figure S1 of the supporting information of Killen et al. (2010) and gives an ordinary least squares (OLS) regression of $\log(L) = 0.0653T + 3.17$ (slope $P < 0.0001$ and $R^2 = 0.510$). We used the residuals of the regression of $\log(L)$ against T relative to the prediction for $\log(L)$ at $T = 15°C$ to calculate temperature-corrected estimates of $\log(L)$. These temperature-corrected values are referred to as $\log(L_{Tcorr})$. We related $\log(L_{Tcorr})$ to the scaling exponents of metabolic rate of the original dataset of Killen et al. (2010). Contrary to the values of L the temperature-corrected values do not show a clear relationship with b (slope $= -0.0300$, $P = 0.176$, $R^2 = 0.0212$), indicating that the original effect of $\log(L)$ on b as reported by Killen et al. (2010) was mediated by temperature and not through an effect of $\log(L)$ directly. Due to the lack of a clear correspondence between the temperature-corrected values of $\log(L)$ on b we refrained from correcting estimates of b to the metabolic level L. Instead, we only controlled for a direct effect of T on b. This was done by recalculating the OLS regression between b and T as reported in figure S1b of Killen et al. (2010) and using the residuals of this regression equation ($b = -0.005872T + 0.875878$, slope $P < 0.0001$, $R^2 = 0.198$) relative to the predicted scaling exponent at $15°C$. These temperature-corrected values were calculated with the full dataset as reported by Killen et al. (2010) and used to test the hypothesis that the scaling of maintenance metabolism is negatively related with the ratio between size at maturation and size at birth for species for which these latter estimates were available (see table 2.B1).

We used ranged major axis (RMA) regression to calculate the correlation between $\log(l_{mat}/l_{egg})$ and the temperature-corrected scaling exponent b_{Tcorr} (main text figure 2.5). Since RMA regression is sensitive to the presence of outliers (Legendre 2014). Cook's distance was calculated from an OLS regression of b_{Tcorr} on $\log(l_{mat}/l_{egg})$. The estimates for the eel (*Anguilla anguilla*) had a Cook's distance of 0.697, which was 6.2 times the value of the second highest Cook's distance. Therefore, we de-

cided to exclude the eel from further analysis, since it has a disproportionally large effect on the estimates of the OLS regression. The RMA regression line relating the temperature-corrected scaling exponent of metabolism to the juvenile size-range is: $b_{Tcorr} = -0.14 \log(l_{mat}/l_{egg}) + 1.45$, with $n = 40$, $R^2 = 0.357$ and a one-tailed permutation test of the slope of $P < 0.01$ (main text figure 2.5). This negative correlation supports the evolutionary prediction that an increased juvenile size-range through either a smaller size at birth (measured as egg diameter) or an larger size at maturation should result in lower scaling exponents of metabolism with body size.

TABLE 2.B1 – Dataset used for figure 2.5 (main text)

Species	l_{egg} (mm)	Source	l_{mat} (mm)	Source	l_{mat}/l_{egg}	b	T(°C)	L (mg kg^{-1} h^{-1})	log(L_{Tcorr})	b_{Tcorr}
Alosa sapidissima	3.17		433		137	0.695	17.5	111.22	4.55	0.71
Ameiurus nebulosus	3.00	1,2	218	1	73	0.994	10	34.02	3.85	0.96
Anguilla anguilla	1.15	6	600	2	522	1.291	18	19.83	2.79	1.31
Bathylagus antarcticus	2.10	9	100	2	48	0.889	0.5	20.35	3.96	0.8
Brevoortia tyrannus	1.62	1,5,7	253	1,2	156	0.816	25	328.58	5.14	0.87
Catostomus commersonii	3.13	1,2	260	1	83	0.903	20	62.86	3.81	0.93
Chaenocephalus aceratus	4.13	2	429	2	104	0.78	1	26.98	4.21	0.7
Chelon labrosus	1.30	10	273	2	210	0.976	18	171.64	4.95	0.99
Coryphaena hippurus	1.43	2,7	593	2	414	0.384	25	192.52	4.61	0.44
Cyprinus carpio	1.72	1,2,6	297	1,2	173	0.781	25.1	86.29	3.8	0.84
Dorosoma cepedianum	0.87	1,2,5	282	1,2	325	0.454	20	105.38	4.33	0.48
Electrona antarctica	1.30	9	74	2	57	0.946	0.5	56.55	4.98	0.86
Eleginus gracilis	1.35	11	300	2	222	0.7	2.5	47.6	4.68	0.63
Euthynnus affinis	0.48	12	575	2	1211	0.496	25	337.86	5.17	0.55
Gadus macrocephalus	1.02	1,3	550	1,2,3	540	0.787	6.5	49.84	4.46	0.74
Gadus morhua	1.49	1,4,5,7,8	561	1,2	376	0.791	3	52.77	4.75	0.72
Gasterosteus aculeatus	1.67	1,2,6	38	1,2	23	1.008	5	168.78	5.78	0.95
Gymnocanthus tricuspis	2.00	13	90	2	45	0.909	-1.5	25.68	4.32	0.81
Hippoglossoides platessoides	2.41	4,8	298	2	124	0.794	3	16.3	3.58	0.72
Katsuwonus pelamis	1.03	1,7	414	1,2	404	0.563	25	269.1	4.94	0.62
Lepomis gibbosus	1.03	1,2	70	1	68	0.71	25	38.07	2.99	0.77
Lepomis macrochirus	1.30	1,2	102	1	78	0.717	25	34.11	2.88	0.78

Continues on next page

TABLE 2.B1 – *Continued from previous page*

Species	l_{egg} (mm)	Source	l_{mat} (mm)	Source	l_{mat}/l_{egg}	b	T(°C)	L (mg kg^{-1} h^{-1})	log(L_{Tcorr})	b_{Tcorr}
Limanda limanda	0.95	5,7,8	220	2	232	0.782	10	28.37	3.67	0.75
Mallotus villosus	1.00	1	142	2	142	0.864	25	114.2	4.08	0.92
Microstomus kitt	1.29	7,8	270	2	209	0.717	10	39.04	3.99	0.69
Mugil cephalus	0.83	7,8	333	2	404	0.855	23	177.57	4.66	0.9
Myoxocephalus scorpius	2.15	5,8	210	2	98	0.909	-1.5	43.6	4.85	0.81
Oncorhynchus mykiss	4.63	1,2,6	411	1	89	0.789	11	82.77	4.68	0.77
Oncorhynchus nerka	5.38	1,3	446	1,2,3	83	0.847	15	54.2	3.99	0.85
Oreochromis niloticus	3.60	5	193	2	54	0.754	25	45.8	3.17	0.81
Platichthys flesus	0.96	5,8	209	2	218	0.827	5	19.48	3.62	0.77
Pleuronectes platessa	3.03	4,5,8	288	2	95	0.782	14	63.16	4.21	0.78
Salmo trutta	5.01	2,6	163	2	33	0.877	10	72.4	4.61	0.85
Salvelinus alpinus alpinus	4.80	1,2,6	381	1,2	79	0.748	6	32.64	4.07	0.7
Salvelinus fontinalis	4.90	1,2	119	1	24	1.036	20	148.62	4.67	1.07
Sander vitreus	1.93	1,2	394	1	205	0.882	20	69.59	3.92	0.91
Sebastolobus alascanus	1.30	1,3	243	2,3	187	0.674	5	8.46	2.79	0.62
Theragra chalcogramma	1.40	3	387	2,3	276	0.778	5.5	67.53	4.83	0.72
Thunnus albacares	0.97	7,14	1033	2	1070	0.573	25	203.33	4.66	0.63
Trachurus trachurus	0.93	8	214	2	231	0.725	13	53.65	4.11	0.71
Zoarces americanus	3.50	15	299	2	86	0.834	8	70.46	4.71	0.79

[1] Killen et al. (2010) [2] Bagenal (1971) [3] King and McFarlane (2003) [4] ICES (2015) [5] Neuheimer et al. (2016) [6] Bonislawska et al. (2001) [7] Pauly and Pullin (1988) [8] Russel (1976) [9] Kock (1992) [10] Crosetti and Cordisco (2004) [11] Dunn and Matarese (1987) [12] Nissar et al. (2015) [13] `species-identification.org` [14] Margulies et al. (2007) [15] Steimle et al. (1999)

Appendix 2.C Supplementary figure

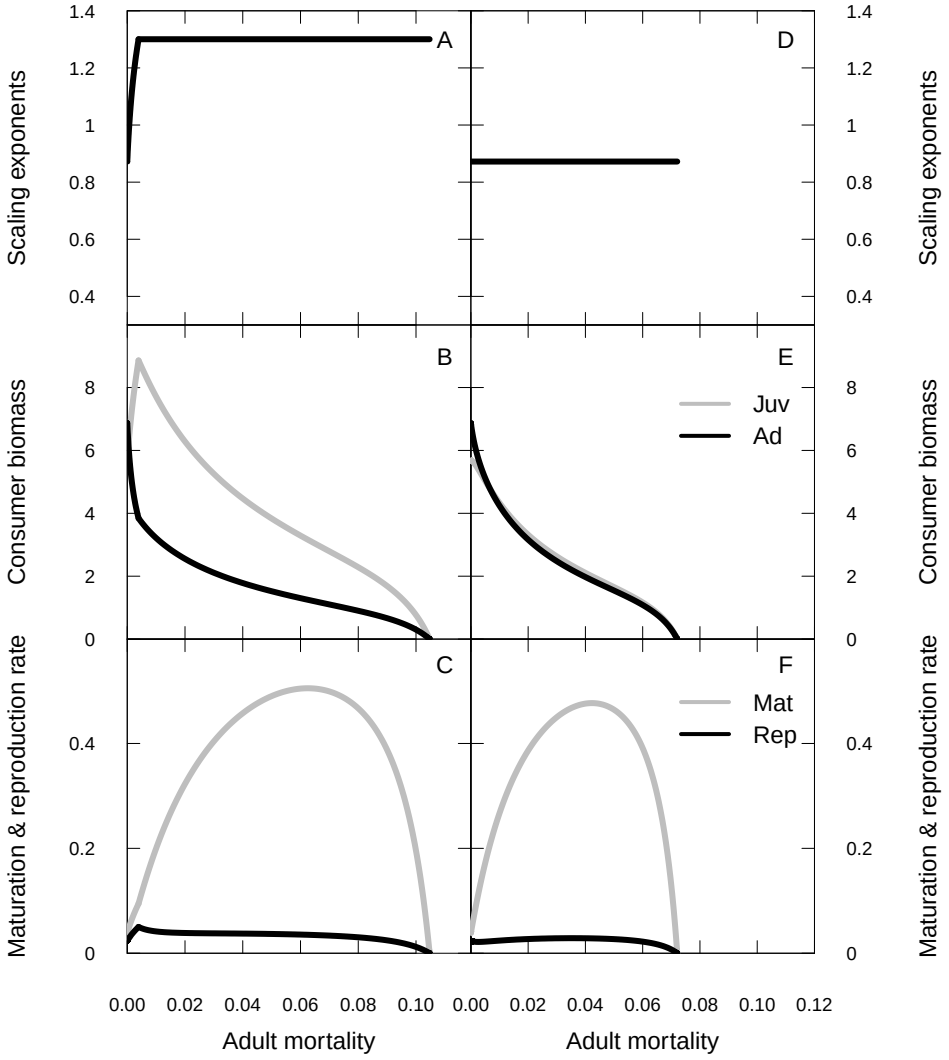

Figure 2.C1 – Equilibria as a function of increasing mortality for adults (μ_a), for evolving (A-C) and non-evolving scaling exponents (D-F). A,D: common scaling exponent of maximum ingestion and maintenance rate. B,E: adult and juvenile consumer biomass. C,F: population-level maturation and reproduction rates in biomass. Default parameters as in table 2.2, in addition to $s_b = 0.05$, for which $\bar{Q} = \bar{P} = 0.872$ at $\mu_a = 0.0$.

Chapter 3

Evolution of Metabolic Scaling

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Manuscript in preparation

Abstract

Theories about the metabolic organization of organisms use constraints on energy supply or reserve mobilization to predict how metabolic rate scales with body mass during ontogeny. Observed variation in the ontogenetic scaling of metabolism with body mass can be explained through changes in the organism's shape during ontogeny. Although observed variation in the scaling of metabolic rate with body mass is significant and depends on ecological and physiological characteristics, the adaptive consequences of such variation has received little attention. Here, we address this by using a size-structured consumer-resource model with a dynamic energy budget describing consumer energetics. We study adaptive dynamics of the body size scaling of two processes that contribute significantly to metabolic rate: energy supply (assimilation) and maintenance metabolism. Assimilation is described by a type-II functional response, which depends on two processes that each have a separate scaling relation with body size: the maximum ingestion rate and the attack rate. We either use a juvenile-adult trade-off, in which an increase of a metabolic process in one consumer life stage results in a decrease in the other consumer life stage, or an energetic trade-off, in which increasing assimilation is associated with increasing metabolic costs. We show that independent of the considered trade-off, the scaling exponents related to energy supply converge towards the same value over evolutionary time. Furthermore, under a juvenile-adult trade-off the scaling of assimilation and maintenance metabolism converge such that the food threshold for starvation becomes independent of body size. Under an energetic trade-off, however, the maintenance scaling is unaffected by the scaling of assimilation. We discuss how these results relate to the variation in the scaling of metabolic rate with body size.

3.1 – Introduction

Understanding the metabolic organization of organisms provides a powerful way to unravel the interplay between hierarchical levels of biological complexity (Brown et al. 2004; Marquet et al. 2004; Sousa et al. 2008, 2010). Many processes at the population, community and ecosystem level depend on how individual organisms take up, convert and use energy and nutrients from the environment, *i.e.* on their metabolism (Enquist et al. 2003; Kooijman 2010; Martin et al. 2013; Nisbet et al. 2008; Van der Meer 2006). Models on the metabolism of individuals that are useful for ecology take the approach of ignoring complex metabolic networks containing thousands of interacting metabolites and instead apply the principles of mass and energy conservation while considering a limited number of biomass pools (Jusup et al. 2016; Kooijman 2001, 2010). The two main approaches are the Dynamic Energy Budget theory (DEB; Kooijman 2010) and the Metabolic Theory of Ecology (MTE; Brown et al. 2004). In recent years the MTE has been successful in linking different levels of biological organization through the metabolism of individuals (Brown et al. 2004; Enquist et al. 2003; Sibly et al. 2012), but the idea of explaining higher-level patterns from individual-level energetics already dates back to the beginning of DEB theory (Kooijman and Metz 1984; Metz and Diekmann 1986).

The main determinant of an organism's metabolism is its body size and considerable research effort has been made to quantify the precise relationship of metabolic rate with body size as organisms grow (Glazier 2005, 2006). Such an ontogenetic scaling relation is often quantified with the power function $B = aM^b$, where B is metabolic rate, M is body mass across ontogeny, b is the scaling exponent and a is a proportionality constant that can depend on temperature (Gillooly et al. 2001; Zuo et al. 2012). Both DEB and MTE propose an explanation for the value of b and although the quantitative predictions of the two theories overlap, they do so for different reasons (Kearney and White 2012; Kooijman and Metz 1984; Maino et al. 2014; Van der Meer 2006).

The simplest DEB variant (the standard DEB model; Kooijman 2010) describes an isomorphically growing organism in terms of two individual state variables that represent different biomass pools: reserve and structure. Growth in structure is proportional to the difference between energy assimilation, which scales with the surface area of structure, and maintenance requirements, which is proportional to structural volume. Under constant food this leads to a Von Bertalanffy growth pattern in body length (Kooijman and Metz 1984). According to DEB theory, whole-organism metabolic rate equals the rate of all chemical transformations in an organism, which is usually quantified as heat production (Kooijman 2014; Sousa et al. 2008). Different processes contribute to this, such as overheads for feeding and digestion (specific-

dynamic action or heat-increment of feeding), overheads for growth, maintenance costs and (for endotherms) energy spent on thermogenesis (Kooijman 2014). The whole-organism metabolic rate is therefore a combination of processes that scale with body surface (assimilation and thermogenesis in endotherms) and those that scale with body volume (maintenance rate) (Kooijman 1986; Van der Meer 2006). Hence, standard DEB predicts a value of b in between two-thirds and one for growing organisms.

In the MTE, metabolic rate is defined as the rate at which organisms take up, transform and expend energy and materials (Brown et al. 2004) and is assumed to scale with an exponent of 0.75 to body mass (Savage et al. 2004; West et al. 2001). This three-quarters scaling relationship is derived from an optimization principle on the delivery of energy and materials from a central source through a space-filling, fractal-like distribution network (e.g. the cardiovascular system) towards the service regions where the metabolic work is done (Banavar et al. 1999, 2010; West 1997). The limitation of the distribution network is independent of species identity and $b = 0.75$ is therefore predicted to hold between fully grown individuals of different species (interspecific scaling, also referred to as Kleiber's law) and across ontogeny within species (ontogenetic scaling, but see Makarieva et al. 2009). During ontogenetic growth, the metabolic rate is assumed to control the rate of energy supply to cells and as such, constrains the amount of metabolic work that an organism can do (Brown et al. 2004; West et al. 2001). The energy supply is used for both maintenance of existing biomass and for the overhead costs of synthesizing new biomass in growing organisms (West et al. 2001). As in DEB theory, maintenance costs per unit biomass are assumed to be independent of body mass and maximum body size is reached when all metabolic power is spent on maintenance (Hou et al. 2011, 2008; West et al. 2001). While the metabolic rate fuels the maintenance costs and growth overheads, the generated biomass that is assembled into growing organisms and that is therefore not dissipated as heat, is derived from assimilated food (Hou et al. 2011, 2008; Makarieva et al. 2004). The rate of food assimilation does not follow a power law scaling relation, because it is assumed to match the metabolic rate plus the energy stored in newly assimilated biomass (Hou et al. 2011, 2008). The ontogenetic growth model of MTE therefore describes a demand-driven metabolic organization and is indeed mostly used to model ontogenetic growth of birds and mammals (Hou et al. 2011; West et al. 2004).

Substantial and consistent variation in b has been observed in many groups of animals, and this variation has been linked to several ecological, physiological and environmental factors (Bokma 2004; Caruso et al. 2010; Glazier 2005, 2009; Glazier et al. 2011, 2015; Killen et al. 2010, 2016; Ohlberger et al. 2012*b*). For example, scaling exponents of standard metabolic rate are significantly larger in pelagic invertebrate

species than in non-pelagic (Glazier 2005). Also, Ohlberger et al. (2012*b*) showed that evolutionary adaptive responses to temperature can affect the scaling of metabolic rate with body mass in fishes. While in the MTE the scaling is assumed fixed at $b = 0.75$, DEB allows for more variation in scaling exponents due to varying contributions of the different processes that lead to heat dissipation. It therefore seems that the observed variation in b can be better accommodated in DEB theory, than in the MTE. However, changes in b can be explained by MTE through changes in the shape of the organism during ontogenetic growth. Changes in shape alter the spatial dimensionality of the ontogenetic growth, which affects the predicted scaling of metabolic rate with body size (Kearney and White 2012). As West (1997); West et al. (1999) derive, $b = {}^{D}/_{(D+1)}$, where D denotes this spatial dimensionality. Hence, for isomorphically growing animals that exhibit equally rapid growth in 3 dimensions the value of $b = 0.75$ holds, while growing in 2 or 1 dimension(s) decreases the value of b. Interestingly, scaling considerations based on surface-area relationships, such as DEB, predict the opposite pattern; a lower dimensionality of ontogenetic growth due to increased elongation or shape flattening will increase the value of b (Kearney and White 2012). Hirst et al. (2014) show that the relationship between the dimensionality of ontogenetic growth and the scaling exponent for metabolic rate, b, matches with predictions based on surface-area theory and refutes the prediction of resource-transport network models (see also Glazier et al. 2015). So DEB and MTE can both harbor variation in the ontogenetic scaling of metabolism with body size, but DEB seems to do a better job in explaining the existing patterns of variation (Glazier et al. 2015; Hirst et al. 2014, 2017).

Although there is substantial variation in b and current theories of metabolic organization predict variation in b due to changes in the dimensionality of ontogenetic growth, there is currently no understanding about the adaptive significance of this variation (but see Ohlberger et al. 2012*b* and Glazier et al. 2015 for empirical examples). Variation in the scaling of metabolic rate over ontogeny ultimately stems from variation in the scaling of the processes that contribute to metabolic rate. These are processes related to energy supply (assimilation which fuels growth) and energy expenditure (maintenance metabolism). Okie (2013) shows that such variation can occur through adaptations that change the surface-area to volume scaling ratio, such as fractal-like surface convolutions, shape shifting through elongation, flattening and hollowing or internalization of surfaces. When applied to digestive surface areas, such adaptations can alter the scaling of assimilation rate with body mass (Okie 2013). Understanding the selection pressures on the scaling of assimilation and maintenance metabolism will contribute to understanding observed variation in b.

Here, we use a dynamic energy budget model to study the adaptive consequences of changes in the ontogenetic body size scaling of two processes that contribute signif-

icantly to metabolic rate: energy supply (assimilation) and maintenance metabolism. Assimilation depends on food density through a type-II functional response, which depends on two processes that each have a separate scaling relation with body size: the maximum ingestion (*i.e.* digestion) rate and the attack rate. Together with the scaling of maintenance metabolism there are in total three different scaling exponents: maximum ingestion scaling, attack rate scaling and maintenance rate scaling. We study selection on each of these exponents in dependence on the other two exponents. To constrain the possible evolutionary outcomes, we use either one of the two different trade-offs: a juvenile-adult trade-off or an energetic trade-off. Under a juvenile-adult trade-off an increase in a scaling exponent leads to an increase of the relevant process for adults, but to a decrease for juveniles. With the energetic trade-off, changing a scaling exponent leads to a response of the relevant process in the same direction for all differently sized organisms. There are, however, energetic costs associated with the maximum ingestion rate and attack rate. These costs represent metabolic costs of maintaining a large digestive machinery (for a higher maximum ingestion) and activity costs (for a higher attack rate).

Our approach builds on a recent analysis by Hin and De Roos (in prep.), who studied the adaptive dynamics of the ontogenetic scaling of maximum ingestion and maintenance metabolism, but implicitly assumed that the attack rate has an identical scaling as the maximum ingestion rate and only used a juvenile-adult trade-off. With these assumptions, Hin and De Roos (in prep.) found strong selection towards an equal scaling of maximum ingestion rate and somatic maintenance requirements with body size. Furthermore, the common exponent of both processes depended on the extent of pre- versus post-maturation growth and on the size-dependency of the mortality rates (Hin and De Roos in prep.). The model we present here is a more general size-structured consumer-resource model that under specified conditions simplifies to the model as studied by Hin and De Roos (in prep.). We show that the identical scaling of attack and maximum ingestion rates as assumed by Hin and De Roos (in prep.) is actually an evolutionary outcome of our more general model. In addition, this outcome is independent of the assumed trade-off. In contrast, the choice of trade-off does influence the evolved outcome of the scaling of energy supply (both maximum ingestion and attack rate) in relation to energy expenditure and we discuss the reason for this. Furthermore, we discuss how these results relate to the variation in the scaling of metabolic rate with body size.

3.2 – Model and method

Model description

We build upon and extend the model as presented by Hin and De Roos (in prep.) who study the evolution of the scaling of maximum ingestion and maintenance metabolism with body size under a juvenile-adult trade-off. We refer the reader to Hin and De Roos (in prep.) for a detailed motivation and description of the modeling approach. All model equations are summarized in table 3.1 and here we will only shortly summarize the model components and highlight the important extensions compared to the model by Hin and De Roos (in prep.).

We use the modeling framework of physiologically structured population models (De Roos 1997; De Roos et al. 1992; Metz and Diekmann 1986). In this framework all assumptions pertain to the details of the individual-level energy budget, which describes the rate of resource ingestion, assimilation, growth and reproduction as a function of body mass s and resource density R. Resource ingestion, $I(R, s)$, follows a type-II functional response of resource density with maximum ingestion rate $M\left(\frac{s}{s_r}\right)^Q$. In this power law function Q is the scaling exponent of maximum ingestion with body mass s and M represents the maximum ingestion rate of an individual with size s_r. In the model of Hin and De Roos (in prep.) the half-saturation constant of the functional response describing resource ingestion is assumed to be independent of body size. Since the half-saturation density in the type-II functional response is equal to the ratio of the maximum ingestion rate and the attack rate, this assumption implies that the attack rate scales in an identical way with body mass as the maximum ingestion rate. Our first modification is to relax this identical scaling assumption in the model by Hin and De Roos (in prep.) by introducing a separate power law scaling function for the attack rate with body mass: $A\left(\frac{s}{s_r}\right)^X$. Similar to the maximum ingestion rate, the scaling of the attack rate is determined by the scaling exponent X, while the constant A sets the attack rate of an individual of mass s_r. The half-saturation density hence equals: $\frac{M}{A}\left(\frac{s}{s_r}\right)^{Q-X}$, which leads to the following expression for the ingestion rate as a function of body size:

$$I(R, s) = \frac{M\left(\frac{s}{s_r}\right)^Q R}{R + \frac{M}{A}\left(\frac{s}{s_r}\right)^{Q-X}}$$

By setting $X = Q$ this expression reduces to the formulation used by Hin and De Roos (in prep.).

Resource assimilation rate equals the product of the ingestion rate and the conversion efficiency σ, which accounts for assimilation efficiency and costs of specific-dynamic action (De Roos and Persson 2013). Biomass production rate is denoted by

Table 3.1 – Model Equations

Equation	Description
$I(R, s) = M\left(\frac{s}{s_r}\right)^Q \frac{R}{R + \frac{M}{A}\left(\frac{s}{s_r}\right)^{Q-X}}$	Resource ingestion
$\Omega(R, s) = \sigma I(R, s) - T\left(\frac{s}{s_r}\right)^P - c_A A\left(\frac{s}{s_r}\right)^X - c_M M\left(\frac{s}{s_r}\right)^Q$	Biomass production
$g(R, s) = \begin{cases} \Omega^+(R, s) & \text{for } s < s_j \\ \kappa(s)\Omega^+(R, s) & \text{for } s \geq s_j \end{cases}$	Growth rate
$b(R, s) = \begin{cases} 0 & \text{for } s < s_j \\ \frac{(1-\kappa(s))\Omega^+(R,s)}{s_b} & \text{for } s \geq s_j \end{cases}$	Fecundity rate
$\kappa(s) = \begin{cases} 1 - 3L(s)^2 + 2L(s)^3 & \text{for } s_j \leq s < s_m \\ 0 & \text{for } s = s_m \end{cases}$	Allocation function
with $L(s) = \frac{s-s_j}{s_m-s_j}$	
$\mu(R, s) = \mu_c - \frac{\Omega^-(R, s)}{s}$	Mortality rate
$G(R) = \delta\,(R_{max} - R)$	Resource growth rate

We use $\Omega^+(R, s)$ to denote $\max(\Omega(R, s), 0)$ and $\Omega^-(R, s)$ means $\min(\Omega(R, s), 0)$

$\Omega(R, s)$ and equals the difference between assimilation rate and the energy expenditure on metabolic maintenance costs. The second extension that we introduce to the model of Hin and De Roos (in prep.) is the inclusion of activity costs and fixed costs for maximum ingestion capacity. We assume that increased foraging activity increases the attack rate, which leads to increased ingestion rate at low resource density. However, increased activity does not come for free, but at the expense of higher metabolic costs. We therefore include in the metabolic costs a component that is proportional to the attack rate function with proportionally constant c_A. Similarly, an increased maximum ingestion rate is assumed to require maintaining and carrying around a larger digestive machinery, which increases metabolic costs. This trade-off implies costs that are fixed (not dependent on the amount of ingested food). We thus include a component in the metabolic costs that is proportional to the maximum ingestion rate with a proportionally constant c_M. As in the original model in Hin and De Roos (in prep.) other metabolic maintenance costs are modeled by $T\left(\frac{s}{s_r}\right)^P$. The biomass

production rate then becomes

$$\Omega(R, s) = \sigma I(R, s) - T\left(\frac{s}{s_r}\right)^P - c_A A\left(\frac{s}{s_r}\right)^X - c_M M\left(\frac{s}{s_r}\right)^Q \tag{3.1}$$

For $c_A = 0$ and $c_M = 0$, in addition to $X = Q$ we retrieve the model of Hin and De Roos (in prep.).

Similar to Hin and De Roos (in prep.) the biomass production, when positive, is entirely used for growth by juvenile individuals ($s < s_j$). Adults ($s \geq s_j$), allocate a size-dependent fraction $\kappa(s)$ to growth and use the remaining fraction $1 - \kappa(s)$ for reproduction. The allocation function $\kappa(s)$ is a sigmoid function that decreases from 1 at $s = s_j$ while reaching zero at $s = s_m$ and hence sets the asymptotic body mass that individuals attain when resource density is sufficient (table 3.1). Individuals suffer from size-independent background mortality μ_c and starvation mortality when ingested resource biomass is insufficient to cover metabolic requirements (*i.e.* $\Omega(R, s) < 0$). The magnitude of the starvation mortality is equal to the mass-specific biomass production when negative (table 3.1). Resource growth follows semi-chemostat dynamics with turn-over rate δ and maximum resource density $R_{\max}$ (table 3.1).

Our ecological system boils down to a single size-structured consumer that feeds on an unstructured food resource. This consumer-resource interaction is the single feedback loop in the model. Ecological theory stipulates that in such a consumer-resource setting the consumer phenotype that can suppress the resource density to the lowest level, will win competition (Tilman 1980). Evolutionary dynamics of the consumer's phenotype will hence converge towards the point that has minimum resource density in equilibrium. In the terminology of adaptive dynamics this point is a continuously stable strategy (Eshel 1983), which is a convergence and evolutionarily stable singular strategy (Geritz et al. 1998).

Model Parameterization

All model parameters are summarized in table 3.2 and we refer to Hin and De Roos (in prep.) for a more thorough description of their derivation. To allow for a direct comparison with the model by Hin and De Roos (in prep.) we adopt their parameter values for $R_{\max}$, δ, M, T, μ_c and σ (table 3.2), which are in turn based upon the parameterization described in De Roos and Persson (2013). Consumer body mass is expressed in grams, while resource biomass density is expressed in milligram per liter (see also De Roos and Persson 2013). The default value for the attack rate constant A is derived from the fact that the half-saturation constant equals the ratio between maximum ingestion rate and attack rate. Hin and De Roos (in prep.) use a value of 0.1 and 3 for the maximum ingestion rate and half-saturation constant, respectively. We hence adopt a default value of 0.033 for the attack rate constant A. The use of gram

Table 3.2 – Model Parameters

Symbol	Units	Value	Description
R_{max}	$mg\ L^{-1}$	30	Maximum resource density
δ	day^{-1}	0.01	Resource renewal rate
Q	–	varies	Maximum ingestion exponent
X	–	varies	Attack rate exponent
P	–	varies	Maintenance exponent
M	$g\ day^{-1}$	0.1	Maximum ingestion constant
T	$g\ day^{-1}$	0.01	Maintenance constant
A	$L\ day^{-1}\ g\ mg^{-1}$	0.033	Attack rate constant
μ_c	day^{-1}	0.0015	Background mortality
σ	–	0.5	Assimilation efficiency
c_A	$mg\ L^{-1}$	0	Constant in attack rate trade-off
c_M	–	0	Constant in maximum ingestion trade-off
s_b	g	0.1	Size at birth
s_j	g	1	Size at maturation
s_r	g	1	Scaling reference size
s_m	g	10	Maximum size

as the unit for consumer body mass and milligram in the unit for resource density, gives the attack rate the dimension of L day^{-1} g mg^{-1}.

In case of an energetic trade-off, the proportionality constant c_A and c_M are chosen based on two considerations. The first is that c_M is a dimensionless constant that should not exceed the value of the assimilation efficiency to guarantee a positive biomass production rate under unlimited food supply. The feasible range for c_M is hence $0.0 - 0.5$ and we adopt a default value of 0.1. The second consideration is that field metabolic rates are estimated to be approximately 2.5 times the resting metabolic rate (Peters 1983). Hence, for an individual of size s_r we can write $c_A A + c_M M + T = 2.5\ T$, which for the default values for A, M and T and $c_M = 0.1$, gives $c_A = 0.15$.

Model Analysis

Model analysis was done with PSPManalysis (De Roos 2016), a software package especially designed for equilibrium and evolutionary analysis of physiologically structured population models. A short description of the methodology of PSPManalysis can be found in Hin and De Roos (in prep.) and more detailed information and references are

given at https://bitbucket.org/amderoos/pspmanalysis. The PSPManalysis package is used to solve the canonical equation of adaptive dynamics (Dieckmann and Law 1996; Durinx et al. 2008). This equation gives an approximate rate of trait change under evolutionary selection and is a function of the selection gradient and a factor, which scales the rate of evolution and includes the population birth rate, the occurrence rate of new mutants and, in case of multiple evolving traits, the mutational covariance matrix. In adaptive dynamics, the selection gradient equals the derivative of mutant invasion fitness in the equilibrium as set by the resident, evaluated at the resident trait value (Geritz et al. 1998). Since we are interested in the qualitative outcome of the evolutionary dynamics, we plot evolutionary trait change as a function of scaled time by setting time equal to 1 when the selection gradient becomes smaller than 10^{-8}. This makes the unit of evolutionary time dependent on the magnitude of the selection gradient, but never changes the qualitative outcome of evolutionary dynamics. We furthermore assume an identical mutation probability for all evolving traits and in case of multiple evolving traits, the mutational covariance matrix is assumed equal to the identity matrix. The PSPManalysis package is also used to continue the population dynamical equilibrium as a function of a model parameter. It furthermore detects evolutionarily singular strategies (ESS) along such an equilibrium curve and classifies these ESSs according to the classification of Geritz et al. (1998). We assessed the non-equilibrium dynamics, such as population cycles, by using numerical integration of the cohort dynamics with the Escalator Boxcar Train method (De Roos 1988; De Roos et al. 1992).

We first study the case of a juvenile-adult trade-off by adopting the size at maturation for the scaling reference size ($s_r = s_j$) and specifying no metabolic costs for activity and maximum ingestion ($c_A = c_M = 0$). Under a juvenile-adult trade-off an increase in the exponents Q, X or P will lead to an increase in maximum ingestion, attack rate and maintenance metabolism, respectively, for adults ($s \geq s_j$) and a concomitant decrease for juveniles ($s < s_j$). In this setting, we study whether the separate scaling for the attack rate influences the results of Hin and De Roos (in prep.). We subsequently adopt an energetic trade-off instead of a juvenile-adult trade-off by setting $s_r = s_m$. With this choice of s_r, changing the scaling exponents Q, X and P will both affect the size-dependency and the overall level of the maximum ingestion rate, the attack rate and maintenance rate, respectively. The overall rates of these processes are also determined by the scaling constants, M, A and T. Selection on the scaling exponents Q, X and P will therefore depend on the values of the scaling constants M, A and T, respectively. To prevent confounding effects of the scaling constants on the evolution of the scaling exponents, we allow the scaling constants to evolve in concert with the associated scaling exponent in case of the maximum ingestion rate and the attack rate. This implies that we explore two-dimensional evolutionary sin-

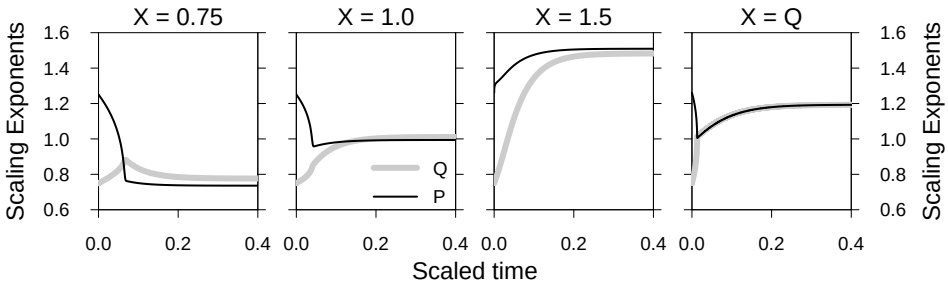

Figure 3.1 – Evolutionary change in scaling exponents of maximum ingestion (Q; gray line) and maintenance (P; black line) as a function of scaled evolutionary time for different values of the attack rate exponent (X; indicated above each panel). In the right-most panel the maximum ingestion and attack rate exponents are equal and the gray line corresponds to both Q and X. Default parameters as shown in table 3.2 are used. The juvenile-adult trade-off is assumed ($s_r = s_j = 1$) and there are no costs of activity or maximum ingestion ($c_A = c_M = 0$).

gular strategies (an evolutionary equilibrium for two traits) containing both a scaling constant (M or A) and a scaling exponent (respectively Q and X). Subsequently we study the dependence of such two-dimensional CSSs on the other scaling exponents.

3.3 – Results

Juvenile-adult trade-off

Hin and De Roos (in prep.) found that under a juvenile-adult trade-off ($s_r = s_j$ and $c_A = c_M = 0$) selection on the scaling exponents of maximum ingestion and maintenance metabolism with body size leads to identical values of these two scaling exponents. We explore here how this result depends on the assumption by Hin and De Roos (in prep.) that the half-saturation constant is independent of body size. This is identical to assuming that the attack rate and maximum ingestion rate scale in an identical fashion with body size ($Q = X$). In figure 3.1 we show the evolutionary dynamics of the maximum ingestion exponent (Q) and the maintenance exponent (P) as a function of scaled time for different values of the attack rate exponent (X). Starting from different initial values, the maximum ingestion and maintenance exponents reach an evolutionary equilibrium (CSS) that depends on the value of the attack rate exponent. Overall, increasing values of the attack rate exponent (X) select for higher values of the maximum ingestion and maintenance exponents (Q and P). For $X = 0.75$ and $X = 1$, the CSS-value of the maximum ingestion exponent (Q) exceeds the CSS-value for the maintenance exponent (P) and vice versa for $X = 1.5$. However, the difference between the CSS-values of the maximum ingestion and maintenance exponents is small compared to the extent both exponents change with changing values

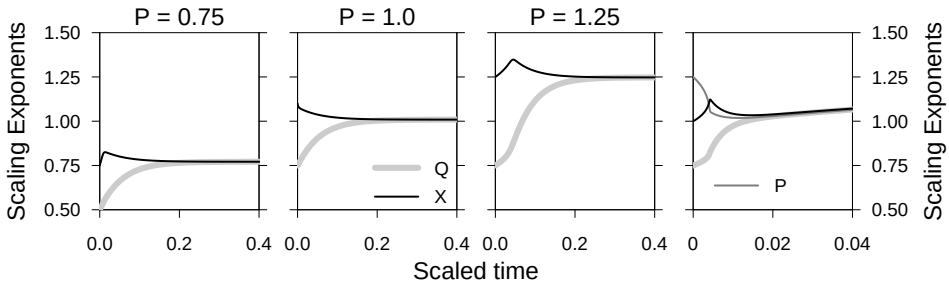

Figure 3.2 – Evolutionary change in the scaling exponents of maximum ingestion (Q; gray line) and attack rate (X; black line) as a function scaled of evolutionary time for different values of the maintenance rate exponent (P; indicated above each panel). Selection leads to convergence of Q and X, irrespective of P. An increased maintenance scaling does increase the evolved scaling exponents of maximum ingestion (Q) and attack rate (X). In the right-most panel all three exponents evolve simultaneously and reach a stable evolutionary equilibrium (CSS). Note that in this panel the x-axis scaling is different from the other panels to better show the transient dynamics. All other parameters as in figure 3.1.

for the attack rate exponent (X). For comparison we also show the case where $X = Q$ (figure 3.1; see also Hin and De Roos in prep.). In this case the evolutionary dynamics consist of two parts. The first part is the rapid convergence of both scaling exponents and in the second part, the selection on the maintenance exponent P reverses and both exponents asymptotically approach a CSS. In this CSS, both exponents are equal to ± 1.19 (Hin and De Roos in prep.). Figure 3.3 (left panel) shows the CSS-values of the maximum ingestion and maintenance exponents (Q and P) as a function of the attack rate exponent (X). At the threshold value of $X = 1.19$ the CSS-values of Q and P intersect each other and simultaneously intersect the diagonal. In this point all three exponents are in evolutionary equilibrium (all selection gradients are equal to zero). In accordance with figure 3.1, the CSS-value of Q exceeds the CSS-value of P for low X-values ($X < 1.19$) and vice versa for large X-values ($X > 1.19$; figure 3.3).

Figure 3.2 shows evolutionary change over scaled time of both the maximum ingestion (Q) and attack rate exponent (X) for different values of the maintenance exponent (P). Independent of the maintenance scaling, the maximum ingestion and attack rate exponents convergence to a CSS-point in which they are equal. The common value of both exponents in this CSS closely follows the maintenance scaling (P), but are only exactly equal to the maintenance exponent at $P \approx 1.19$ (figure 3.3; right panel). At this point, all three scaling exponents are in evolutionary equilibrium (similar to figure 3.3; left panel). For $P < 1.19$ the CSS-values of Q and X are larger than P (above the diagonal), while for $P > 1.19$ they are smaller than P (below the diagonal). Therefore, deviating from the assumption by Hin and De Roos (in prep.)

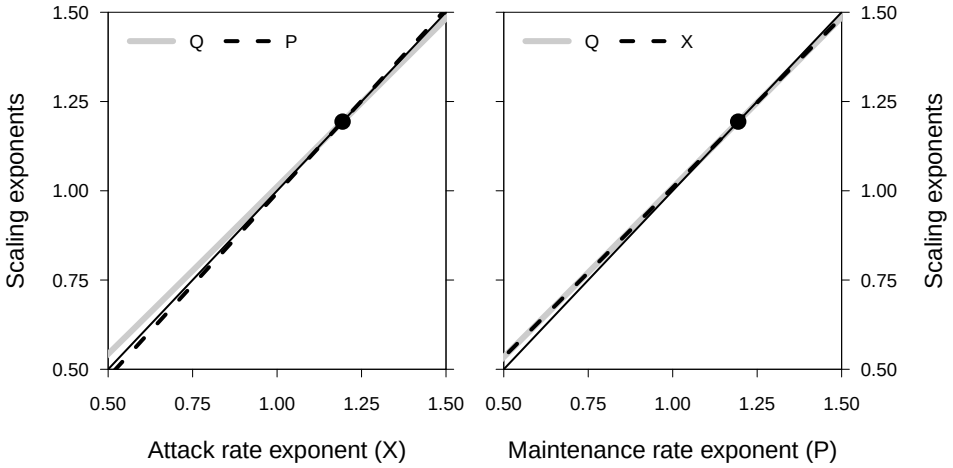

Figure 3.3 – The evolutionary equilibrium (CSS) of Q and P as function of X (left panel), and the evolutionary equilibrium (CSS) of Q and X as a function of P (right panel). The thin black line indicates the diagonal and the black dot is where the diagonal is crossed by the evolutionary equilibrium. At this point selection on the x-axis variable vanishes and all three exponents are at evolutionary equilibrium. A juvenile-adult trade-off is assumed ($s_r = s_j = 1$), and all other parameters are as denoted in table 3.2.

of an identical scaling of attack rate and maximum ingestion rate with body size, changes the evolutionary dynamics of Q and P, but this effect is only small. On the other hand, it appears that $Q = X$ is itself an evolutionary outcome, irrespective of the value of P. The assumption by Hin and De Roos (in prep.) that the scaling exponent of the maximum ingestion rate (Q) and the attack rate (X) are the same, boils down to assuming that they are both in evolutionary equilibrium. As a consequence, when all three exponents evolve (right-most panel in figure 3.2), all the results derived by Hin and De Roos (in prep.) apply. That study furthermore shows how the evolved scaling value of 1.19 changes with patterns of pre- and post-maturation growth and size-specific mortality rates.

Energetic trade-off

As an alternative to the juvenile-adult trade-off that underlies the results by Hin and De Roos (in prep.), we explore a trade-off between energy supply and energy expenditure. Setting c_A and c_M to positive values imposes metabolic costs for increased activity and maximum ingestion capacity. To avoid that the two different trade-offs operate simultaneously, we adopt $s_r = s_m$. Since the reference body size is now an order of magnitude larger ($s_m/s_j = 10$), default values for the scaling constants M, A, and T, which reflect the maximum ingestion, attack and maintenance rate of an individual

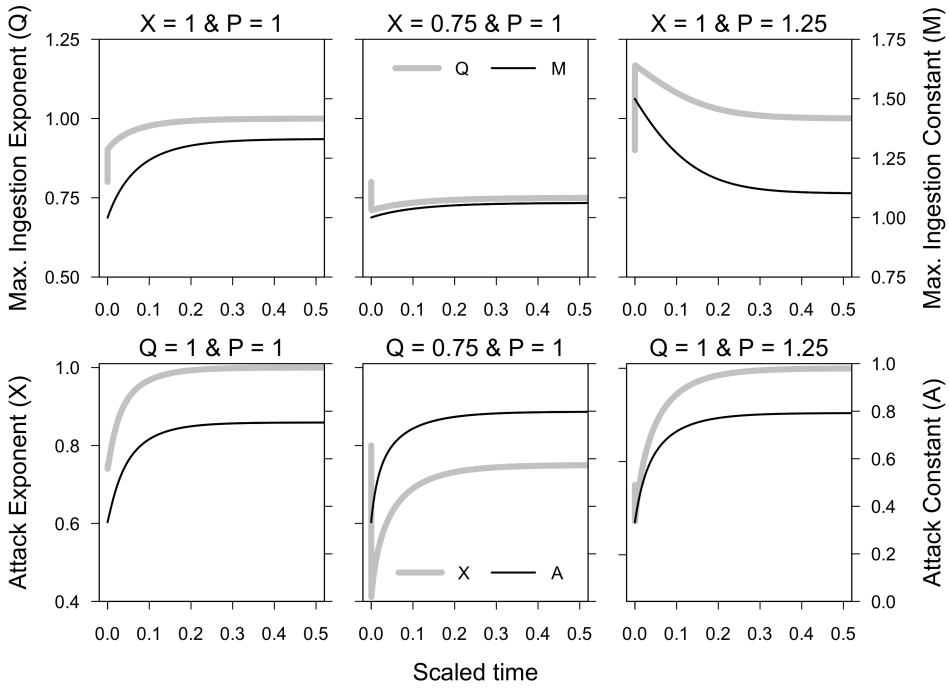

Figure 3.4 – Evolutionary dynamics of the maximum ingestion exponent and constant (Q and M; top panels) and the attack rate exponent and constant (X and A; bottom panels) in case of an energetic trade-off, for different values of the other two exponents (respectively X or Q, and P as indicated above each panel). The scaling exponents related to energy supply (maximum ingestion rate Q or attack rate X) always evolve towards the same value (*i.e.* the fixed value of the other supply related exponent, attack rate X and maximum ingestion rate Q, respectively). This is independent of the value of the maintenance rate exponent (P). The scaling exponents (Q and X) are shown on the left y-axis, while the scaling constants (M and A) are shown on the right y-axis. All parameters as in table 3.2 in addition to $s_r = s_m = 10$, $c_A = 0.15$, $c_M = 0.1$, $M = 1$, $T = 0.1$ and $A = 0.33$.

with reference body size s_r, will be taken 10 times larger as well. This ensures that the change in reference body size from s_j to s_m does not affect the maximum ingestion rate, attack rate and maintenance metabolic rate when the scaling exponents of these processes are one. By adopting $s_r = s_m$, an increase in the scaling exponent Q, X or P will lead to a decrease in the maximum ingestion rate, attack rate and maintenance rate for all individuals in the consumer population. This effect is not equally strong for all differently sized individuals, but increases with the difference between the maximum and the current body size, $s_m - s$, and is zero at $s = s_m$. A trade-off between energy supply and expenditure arises because food intake is determined by the attack rate and the maximum ingestion rate but in addition depends on the resource density

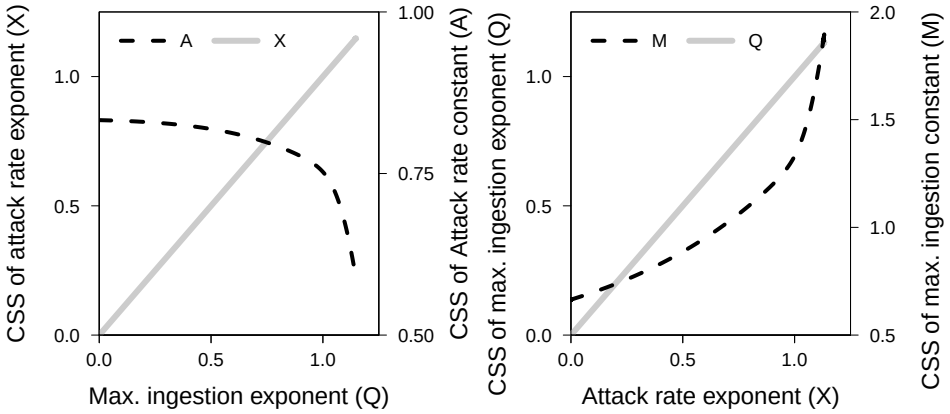

Figure 3.5 – Left panel: evolutionary stable equilibrium values (CSS) of the attack rate scaling exponent (X) and constant (A), as function of the scaling exponent of maximum ingestion rate Q. Right panel: CSS-values of maximum ingestion scaling exponent (Q) and constant (M), as a function of the scaling exponent of attack rate X. In both panels, the evolving scaling exponents (gray lines) are exactly equal to the non-evolving exponent on the x-axis. The scaling exponents (Q and X) are shown on the left y-axis, while the scaling constants (M and A, black dashed lines) are shown on the right y-axis. All parameters as in figure 3.4.

following a type-II functional response. The costs of increased attack rate and/or maximum ingestion capacity, however, have to be paid always, irrespective of resource densities. At low resource density the energy supply is limited by the value of the attack rate. For high resource density, the asymptotic level of the functional response, which is set by the maximum ingestion rate, is the main limiting factor. Increasing the attack rate will increase the limitation by maximum ingestion, and vice versa, increasing the maximum ingestion will lead to a stronger limitation by the attack rate. Increasing both attack rate and maximum ingestion rate will lead to ever increasing rates of energy supply and we will hence refrain from evolving these two parameters simultaneously.

Changing the scaling exponents of attack rate (X) and maximum ingestion (Q) will not only change the size-dependency of the functional response parameters, but also their overall value across the whole size distribution. Selection on the scaling exponents of attack rate (X) and maximum ingestion (Q) is thus confounded by the value of the scaling constants A and M. To avoid such confounding effects we always let the scaling exponents evolve together with their corresponding scaling constants (figure 3.4). Over evolutionary time the value of the evolving scaling exponent related to energy-supply (maximum ingestion Q or attack rate X) always approaches the value of the non-evolving exponent related to energy supply, attack rate X or maximum in-

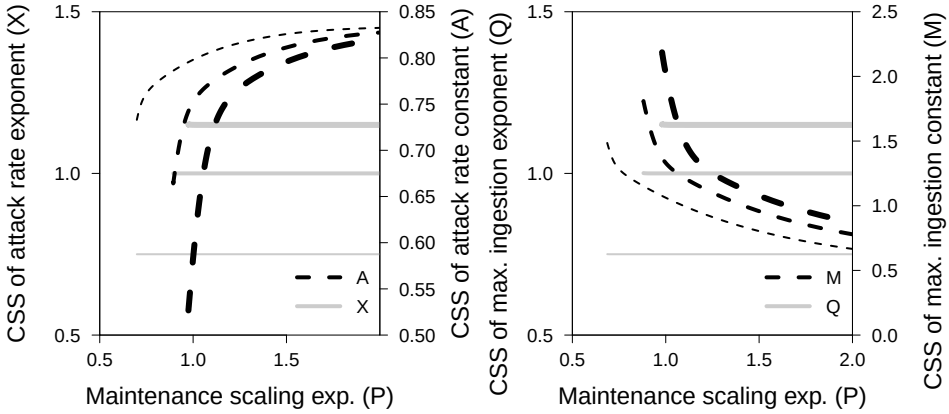

Figure 3.6 – Left panel: evolutionary stable equilibrium values (CSS) of the scaling exponent and constant of attack rate, X and A, respectively, as a function of the scaling exponent of maintenance rate P, for different values of the scaling exponent of maximum ingestion rate: $Q = 0.75$ (thin lines), $Q = 1$ (normal lines) and $Q = 1.15$ (thick lines). Right panel: evolutionary stable equilibrium values (CSS) of the scaling exponent and constant of maximum ingestion rate, Q and M, as a function of the scaling exponent of maintenance rate P, for different values of the scaling exponent of attack rate: $X = 0.75$ (thin lines), $X = 1$ (normal lines) and $X = 1.15$ (thick lines). The evolving exponents (gray lines), are not influenced by the value of the maintenance exponent (P; x-axis). The population goes extinct when the maintenance rate becomes too high (low values of P). Scaling exponents Q and X are shown on the left y-axis, while scaling constants (M and A, black dashed lines) are shown on the right y-axis. All other parameters as in figure 3.4.

gestion Q, respectively. This is independent of the maintenance rate exponent (P; figure 3.4). This result is corroborated by figure 3.5 and figure 3.6, that show the evolutionary equilibrium value of each parameter pair, consisting of the scaling exponent and the constant of the attack rate or the maximum ingestion rate, as a function of either the other, non-evolving energy-supply exponent (maximum ingestion rate exponent Q or attack rate exponent X, respectively; figure 3.5) or of the maintenance rate exponent P (figure 3.6). Indeed, figure 3.5 (left panel) confirms that the evolutionary stable value of the scaling exponent of the attack rate exactly equals the scaling exponent of the maximum ingestion rate, when the latter does not evolve. Vice versa, the evolutionary stable value of the scaling exponent of the maximum ingestion rate is exactly equal to the (non-evolving) attack rate exponent (figure 3.5; right panel). Hence, similar to the result obtained with a juvenile-adult trade-off also in case of a trade-off between energy supply and expenditure we find as evolutionary outcome that $Q = X$. Furthermore, this result is independent of the value of the maintenance rate exponent (P), as changing P has no effect on the evolutionary stable values of

the scaling exponents of attack rate X or the maximum ingestion rate Q (figure 3.6). This result differs from the case of the juvenile-adult trade-off, where the maintenance rate scaling had a strong effect on the evolved body size scaling of maximum ingestion and attack rate (figure 3.3; right panel). However, under a juvenile-adult trade-off both the maximum ingestion and attack rate scaling could evolve, while under an energetic trade-off the evolution of these scaling exponents leads to ever increasing rate of energy supply.

3.4 – Discussion

The model presented here shows that selection on the scaling of metabolic processes with body mass leads to convergence of the scaling exponents of the processes related to energy-supply, independent of the type of trade-off that is considered. These are the maximum ingestion rate, which determines the rate of food ingestion at unlimited food supply, and the attack rate, which describes the rate at which ingestion increases with food density when food density is low. Furthermore, under a juvenile-adult trade-off the scaling of energy supply and energy expenditure (as represented by maintenance metabolism) converge towards the same body mass scaling over evolutionary time. Under an energetic trade-off, however, the evolutionary stable scaling exponents related to energy supply (either maximum ingestion or attack rate scaling) are independent of the scaling exponent of energy expenditure (maintenance metabolism).

The evolutionary outcome of identical maximum ingestion and attack rate scaling exponents, implies that the half-saturation constant of the functional response, which equals the ratio between maximum ingestion and attack rate, is independent of body mass. Consequently, the functional responses of differently sized individuals have the same qualitative shape and only differ from each other by a factor that depends on body size, but not on resource density. The effect of body mass on half-saturation density has been studied on an interspecific level by both Hansen et al. (1997) and Rall et al. (2012), who both concluded that in freshwater and marine invertebrates the half-saturation density is constant with species body mass. However, for species from other ecosystems and metabolic types there is no consistent picture, as half-saturation densities can either increase of decrease with species body mass (Rall et al. 2012). The analysis of Rall et al. (2012) furthermore showed that attack rates and handling times (the inverse of maximum ingestion) show signs of a humped-shaped relationship with body mass. This pattern also arises in the attack rates of several species of fish, when comparing differently sized individuals of the same species (Byström et al. 2004; Hjelm and Persson 2001; Jansen et al. 2003). Attack rates are known to depend on a number of factors, such as prey body size, structural complexity of the habitat, swimming speed and visual accuracy (Rall et al. 2012). These multitude of external and internal

influences can lead to a more complex relationship between attack rate and body size than the power law relation assumed in this study. Further research should point out how external factors shape selection on attack rates and therefore affect the scaling of the half-saturation constant with body size. In our model, fitness relates to the ability of the consumer to suppress and persist on the lowest resource density and we therefore conclude that population-level resource use is an important factor to consider when studying the evolution of attack rates and handling times.

With respect to the scaling exponents of energy supply (attack rate and/or maximum ingestion rate) and energy expenditure, the evolutionary outcomes differ between the different trade-offs investigated. As already found by Hin and De Roos (in prep.), these processes converge towards the same scaling with body size in case of a juvenile-adult trade-off. As a result, the maintenance resource density (MRD), which is the amount of resources required to cover maintenance metabolism, becomes independent of body mass. This precludes the occurrence of starvation and neutralizes size-dependent intraspecific competition (Hin and De Roos in prep.). However, the size-independence of the MRD does not imply that differently sized individuals are equally efficient in converting ingested resource into new biomass. First, larger individuals will have higher foraging rates and hence also higher production rates of new biomass. Second, as the scaling exponents of the energy supply and energy expenditure do not necessarily converge to one (see figure 3.1; right panel), also the mass-specific biomass production rate (MBP) changes with body size. As shown by Hin and De Roos (in prep.), the value of the common scaling exponent is dependent on the size-specific mortality and the extent of pre- and post-maturation growth. The results in this paper show that the outcomes presented by Hin and De Roos (in prep.) are robust against the incorporation of a separate attack rate scaling.

That the scaling exponents of energy supply (either maximum ingestion or attack rate) and energy expenditure evolve to different values in case of an energetic trade-off (figure 3.6) relates to the fact that selection is only stabilizing when a single scaling exponent related to energy supply evolves. Simultaneous evolution in both attack rate and maximum ingestion rate would always lead to a run-away evolutionary process with ever increasing value of the functional response, which is clearly unrealistic. Evolutionary change in one exponent (and associated constant) is thus only stabilizing when the other exponent does not evolve. In addition, with the choice of $s_r = s_m$, an increase in the scaling of maintenance metabolism (P) always leads to a decrease in the maintenance rate for every individual. Hence, selection always favors higher values of P. The energetic trade-off thus provides us with the insight that the scaling exponents of maximum ingestion and attack rate converge over evolutionary time to the same value, irrespective of the choice of the trade-off. Furthermore, it shows that differences in the scaling of energy supply and energy expenditure can only be

understood from the perspective of limited evolvability of two out of the three scaling exponents studied here.

Focusing on the juvenile-adult trade-off we observe that selection on metabolic scaling favors all three scaling exponents to be identical (figure 3.2 and 3.3). Consequently, evolution also favors the scaling of energy supply and energy expenditure to be equal (Hin and De Roos in prep.). This outcome is not in accordance with the MTE, which assumes a value of 0.75 for the scaling exponent of energy supply and an exponent of 1 for energy expenditure. The latter assumption is derived from the logic that each cell (in MTE) requires a fixed maintenance costs (West et al. 2001), which is probably the most parsimonious assumption. Even if the maintenance scaling is fixed, our evolutionary analysis shows that the juvenile-adult trade-off would force the scaling exponents of energy supply (maximum ingestion (Q) and attack rate (X)) to evolve to closely match the linear scaling of maintenance. In DEB theory an increase in energy supply can be achieved by an increase in the surface-area to volume ratio. Many organisms from a wide variety of taxa have indeed evolved adaptations to escape surface-area constraints that occur when growing bigger (Gould 1966; Hirst et al. 2014; Lewis 1976; Okie 2013). The nature and some examples of these adaptations are discussed in Okie (2013), while Hin and De Roos (in prep.) discuss the interpretation of these adaptations in relation to the theoretical result about the convergence of the scaling exponents of energy supply and expenditure over evolutionary time.

How can our approach be related to observed variation in the scaling of metabolic rate with body mass? First of all, it needs to be stressed that the measured scaling of metabolic rate does not directly correspond to any parameter, or combination of parameters in our model. For example, we do not explicitly account for growth overhead costs, which is one of the processes that contributes to the experimentally measured rates of respiration, which are commonly adopted as a measure for metabolic rate (Kooijman 2010). Incorporating such costs of growth in our model would merely rescale the size-at-age curve and does not lead to any qualitative differences in the model outcome. When a fixed proportion of the biomass flow allocated towards growth is spent on the overheads of growth, the energy dissipation rate in our model becomes a linear combination of the growth rate (which scales as the biomass production for juveniles (equation (3.1)) and the maintenance rate. Under a juvenile-adult trade-off, the maximum ingestion and attack rate scaling exponents (Q and X, respectively) become identical to the scaling exponent of the maintenance rate (P) in the evolutionary equilibrium, which would imply that the scaling of the energy dissipation rate for juveniles is equal to the value of this common exponent. As reported by Hin and De Roos (in prep.), this common scaling exponent decreases with increasing juvenile mortality and increasing pre-maturation growth, and decreases with increasing adult mortality and increasing post-maturation growth. In case of the energetic trade-off,

the evolutionary stable scaling exponent of one of the energy supply exponents depends on the value of the other, non-evolving energy supply exponent. Increasing mortality rates of either juveniles or adults do not influence the scaling exponents of energy supply with body size, but only changes the evolution of the constants in these size scalings (results not shown). Changes in the scaling constants might, however, also affect observed variation in metabolic rate when those changes lead to higher growth rates. Because growth rates decrease when body mass increases, an increasing contribution to growth might lead to a decrease in the scaling of metabolic rate. How exactly the scaling constants change the slope of metabolic rate with body mass, and under which conditions, remains a topic for further study. Another source of variation in the metabolic rate under an energetic trade-off stems from the variation in the constrained (non-evolving) scaling exponents that drive evolution in the other scaling exponents. Thus, observed variation in the measured scaling of metabolic rate with body mass will be more straightforward to relate to our results with a juvenile-adult trade-off than when an energetic trade-off constrains the evolutionary process.

Studies on the adaptive consequences of changes in the scaling exponents of energy supply and energy expenditure with body size are rare (but see Hin and De Roos in prep.). Even though the ecological and evolutionary (fitness) consequences of changes in these scaling exponents can be large because of their effect on population and community dynamics. These higher-level consequences can be understood through the concept of ontogenetic asymmetry in energetics (De Roos et al. 2013). This phenomenon occurs when the mass-specific biomass production (MPB) rate is a non-constant function of body size. In other words, individuals that differ in size also have different mass-specific rates at which they produce new biomass. A size-dependent MPB even occurs when all three scaling exponent are equal, but different from one. When one of the three exponents differs from the other two, also the maintenance resource density (MRD) changes with body mass.

Ontogenetic asymmetry in terms of a size-dependent MRD leads to population cycles, with either juvenile- or adult-driven cycles, depending on whether the MRD increases or decreases with body mass (De Roos and Persson 2003; Persson and De Roos 2013; Persson et al. 1998). For example, when the exponent of energy supply is lower than the exponent of energy expenditure (as in descriptions of ontogenetic growth based on DEB or MTE), the MRD increases with body mass. Small individuals can hence persist at lower resource densities than larger individuals. When all individuals compete for a shared resource this type of asymmetry leads to juvenile-driven population cycles (De Roos et al. 2013). Also, ontogenetic asymmetry underlies the phenomenon of biomass overcompensation, which refers to an increase in (stage-specific) biomass density with increasing mortality rates (De Roos et al. 2007). In turn biomass overcompensation is responsible for community-wide effects such as the

emergent Allee effect, facilitation between size-selective predators and alternative stable states (De Roos et al. 2003*b*, 2008*a*; Guill 2009; Van Kooten et al. 2005) . Understanding what drives ontogenetic asymmetry is hence required for understanding their consequences on a population and community level. The present work extends on earlier research by Hin and De Roos (in prep.) that attempts to explain observed patterns of ontogenetic change in energetics from an adaptive point of view. We show that these earlier results are to some extent robust against the introduction into the model of more biological detail, but that model outcomes may differ if a different trade-off is assumed. We conclude that for this approach to be successful, we require a better understanding of the extent to which adaptive evolution can modify the metabolic organization of organisms, and to which extent this process is limited by physiological, physical and developmental constraints.

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Chapter 4

Cannibalism and Intraguild Predation Community Dynamics: Coexistence, Competitive Exclusion and the Loss of Alternative Stable States

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Abstract

Predators often exert strong top-down regulation of prey, but in many systems, juvenile predators must compete with their future prey for a shared resource. In such life-history intraguild predation (LHIGP) systems, prey can therefore also regulate the recruitment and thus population dynamics of their predator via competition. Theory predicts that such stage-structured systems exhibit a wide range of dynamics including alternative stable states. Here we show that cannibalism is an exceedingly common interaction within natural LHIGP systems that determines what coexistence states are possible. Using a modeling approach that simulates a range of ontogenetic niche shift scenarios along a productivity gradient, we demonstrate that only if the predator is competitively dominant can cannibalism promote coexistence by allowing prey to persist. If the prey is competitively dominant, cannibalism instead results in competitive exclusion of the predator and the loss of potential alternative stable states. Further, predator exclusion occurs at low cannibalistic preference relative to empirical estimates and is consistent across LHIGP systems in which the predator undergoes a complete diet shift or diet broadening over ontogeny. Given that prey is frequently competitively dominant in natural systems, our results demonstrate that even weak cannibalism can inhibit predator persistence, prompting exploration of mechanisms that reconcile theory with the common occurrence of such interactions in nature.

4.1 – Introduction

Conspecific individuals that vary in size or stage often differ in their ecological (*i.e.* functional) roles (Werner and Gilliam 1984). For example, in many predatory species, individuals either switch resources or add resources to their diet as they develop (Rudolf and Lafferty 2011; Rudolf et al. 2014; Werner and Gilliam 1984). Predator populations can thus be simultaneously regulated by stage-specific interactions with different species. Furthermore, these shifts in interspecific interactions among stages are generally associated with concurrent shifts in intraspecific interactions (Miller and Rudolf 2011). Accordingly, structured systems exhibit a wider range of dynamics relative to unstructured systems, including alternative stable states (De Roos and Persson 2013; Miller and Rudolf 2011). In particular, which state or dynamics occur depends on the relative strength of different stage-specific interactions. Thus any process, biogenic or anthropogenic, that shifts population size or stage distribution will also alter the relative strength of these interactions, and therefore the capacity of predator-prey systems to transition between alternative stable states. Accordingly, the processes that govern predator-prey coexistence may fundamentally differ for structured versus unstructured predator-prey systems.

The dramatic ecological effects of stage-specific interactions are apparent in a widespread interaction module known as life-history intraguild predation (LHIGP) (Abrams 2011; Hin et al. 2011; Pimm and Rice 1987; Rudolf 2007; Toscano et al. 2016; Walters and Kitchell 2001). LHIGP occurs when juvenile predators compete with their future prey (*i.e.* an intermediate consumer, hereafter 'consumer') for a shared resource (figure 4.1). Thus, LHIGP allows for competitive dynamics in addition to predation. Within LHIGP systems, juvenile and adult predators may feed on separate resources (*i.e.* a complete diet shift: figure 4.1A; Hin et al. 2011; Toscano et al. 2016) or adults may continue to feed on the juvenile resource, thereby broadening their diet over ontogeny (figure 4.1B; Rudolf 2007; Van de Wolfshaar et al. 2006). Due to the opposing forces of competition and predation on different life-history stages, LHIGP systems are predicted to exhibit alternative stable states: when consumers are competitively dominant, strong competition between consumers and juvenile predators can eliminate predator recruitment to the adult stage (*i.e.* juvenile competitive bottleneck), driving predator exclusion (a consumer-resource equilibrium: Hin et al. 2011; Walters and Kitchell 2001). Alternatively, adult predators can regulate consumers, facilitating their own recruitment and persistence (a predator-present equilibrium: Hin et al. 2011; Van de Wolfshaar et al. 2006). Consequently, the long-term trajectory of LHIGP systems is potentially sensitive to predator stage structure: a preponderance of juveniles in LHIGP should free consumers from top-down control leading to predator exclusion, while a preponderance of adults and resulting top-down control should promote predator persistence.

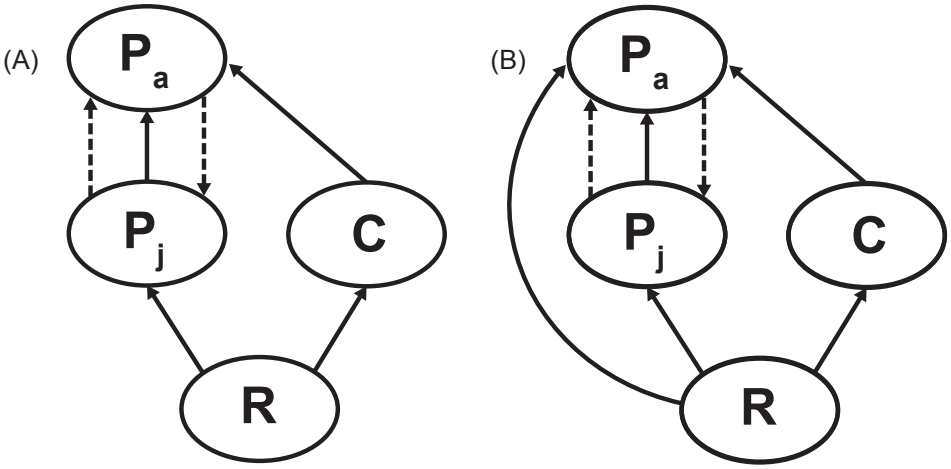

Figure 4.1 – Life-history intraguild predation (LHIGP) with cannibalism: complete ontogenetic diet shift (panel A) and ontogenetic diet broadening (panel B) scenarios. Solid arrows depict feeding relationships while dotted lines depict demographic transitions between predator stages (maturation and reproduction). Adult predators (P_a) feed solely on consumers (C) (complete diet shift: panel A), or feed equally on consumers and the resource (R) (diet broadening: panel B). In both scenarios, juvenile predators (P_j) compete with consumers for the resource and adult predators cannibalize juveniles.

Cannibalism (*i.e.* intraspecific predation) occurs in most animal taxa (Fox 1975; Polis 1981; Polis and Myers 1985) with major ramifications for both population dynamics and size/stage structure (Claessen et al. 2000, 2004; Ohlberger et al. 2012*a*; Persson et al. 2003; Rudolf 2007, 2008). Within LHIGP systems, juvenile predators and consumers must co-occur in order to compete for a common resource, and thus adult predators frequently encounter both conspecific and heterospecific prey (Byström et al. 2013; Rudolf 2007). Furthermore, empirical studies indicate that during such encounters, LHIGP predators often preferentially consume conspecific versus heterospecific prey (figure 4.2). Specifically, in a literature review of 65 experiments in which invertebrate and vertebrate predators were simultaneously offered both conspecific and heterospecific prey (figure 4.2; see caption for details), only 4 experiments failed to measure some degree of cannibalism (*i.e.* Manly's $\alpha = 0$), while 31 experiments demonstrated cannibalistic preference (*i.e.* Manly's $\alpha > 0.5$). These preference estimates come from a diversity of predator taxa (crustaceans, arachnids, insects, fish, amphibians, reptiles), all of which exhibit ontogenetic diet shifts (Werner and Gilliam 1984) and thus presumably engage in LHIGP interactions in nature. The ubiquity of cannibalism and commonness of cannibalistic preference within stage-structured predator-prey systems suggests that cannibalism must be incorporated into current

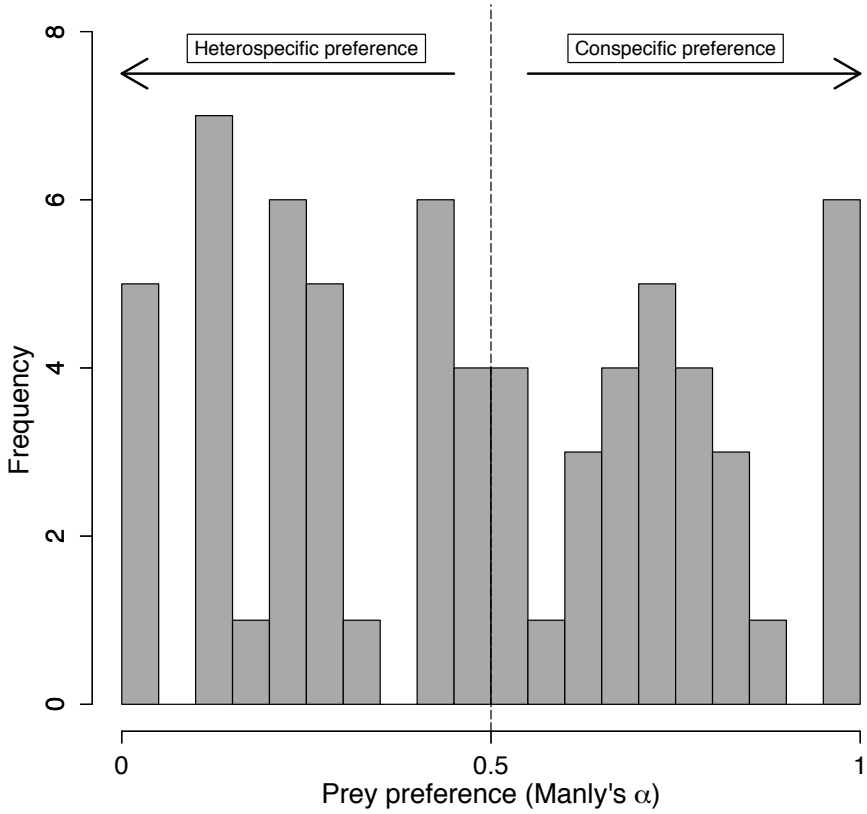

Figure 4.2 – Cannibalistic preference estimates (n = 65; measured as Manly's α, see Chesson (1978) for calculation) from empirical studies (n = 19; references listed in appendix 4.B). Manly's α is a measure of the probability that a conspecific or heterospecifc prey item is selected when both prey types are offered simultaneously. Manly's α scales from 0 to 1 with $\alpha = 0.5$ indicating no preference (marked with vertical dashed line), and $\alpha < 0.5$ and $\alpha > 0.5$ indicating cannibalistic aversion or preference, respectively. Prey depletion was accounted for in calculating Manly's α according to Chesson (1978) in studies in which depletion occurred (as opposed to prey replacement). When Manly's α or data necessary to calculate Manly's α were not reported directly, a plot digitizer (Web Plot Digitizer 3.8) was used to extract relevant data from graphs. Empirical studies were found with a literature search conducted on March 2017 using Google Scholar. Search terms were: "cannibalistic" or "cannibalism" combined with "preference", "choice", "heterospecific", "interspecific", "Manly's" and "Chesson".

LHIGP theory to better understand and predict natural LHIGP dynamics. For example, a recent study using freshwater zooplankton (Toscano et al. 2016) demonstrates a juvenile competitive bottleneck and the alleviation of this bottleneck by adult predators: the precise interactions predicted to drive alternative stable states within LHIGP systems (Toscano et al. 2016). This study further shows that cannibalism modifies

the strength and qualitative outcomes of these interactions (Toscano et al. 2016). For this empirical LHIGP system (Toscano et al. 2016) and many others (figure 4.2), we currently lack theory to predict how the alteration of these short-term interactions by cannibalism might scale up to influence long-term LHIGP dynamics.

Cannibalism could have strong effects on long-term LHIGP dynamics. Within LHIGP systems, predators often feed in a size-dependent manner (Pimm and Rice 1987), becoming cannibalistic at the transition from resource- to prey (*i.e.* consumer)-feeding (Byström et al. 2013; Rudolf 2007). Adult predators thus gain an additional food resource with cannibalism, while juvenile predators experience an additional source of mortality. Such stage-specific effects can have dramatic consequences for the distribution of biomass across stages (De Roos and Persson 2013; De Roos et al. 2007; Reichstein et al. 2015). Cannibalism is therefore an important but currently understudied factor that could determine the dynamics of LHIGP systems and possible coexistence states.

Here, we examine the effects of cannibalism on LHIGP dynamics using models that account for the food-dependence of development and reproduction (De Roos and Persson 2013; De Roos et al. 2007). We explore the effects of cannibalism on LHIGP when adult predators exhibit either: (1) a complete diet shift (juveniles and adults feed on separate food resources: figure 4.1A); or (2) diet broadening over ontogeny (adults continue to feed on the resource of juveniles: figure 4.1B). Both patterns of ontogenetic diet change are common in nature (Rudolf and Lafferty 2011; Werner 1988; Werner and Gilliam 1984), allowing us to assess the generality of potential effects.

We model cannibalism as a single parameter that represents the preference for conspecific versus heterospecific prey. Cannibalistic preference has been estimated empirically for a diversity of predator species (figure 4.2), allowing us to make general predictions for how these cannibalistic predators might influence the long-term dynamics of the LHIGP systems in which they occur. We found that even weak cannibalism can inhibit predator persistence in both LHIGP system variants due a reduction in the total biomass of adult predators and thus the top-down control of competitively-dominant consumers. We further discuss mechanisms that may facilitate predator persistence in LHIGP systems with cannibalism, thus reconciling our findings with the apparently common occurrence of such interactions in nature.

4.2 – Model

Model formulation

We examine the effects of cannibalism on LHIGP dynamics using a stage-structured, bioenergetics modeling approach (De Roos et al. 2007, see also Yodzis and Innes 1992). Using this approach, Hin et al. (2011) analyze a model of LHIGP in which the predator

undergoes a complete ontogenetic diet shift. We build upon this model to explore the effects of cannibalism on both complete diet shift and diet broadening LHIGP scenarios. Two ordinary differential equations (ODEs) keep track of juvenile (P_j) and adult predator (P_a) total biomass densities (mass per unit volume; table 4.1). Despite a simple two-stage population structure, this formulation, under equilibrium conditions, yields predictions identical to that of a physiologically structured population model that explicitly accounts for a continuous population size distribution and mass-specific individual-level physiological rates (De Roos et al. 2007, 2008*b*).

Total biomass densities of unstructured consumer and resource populations, denoted C and R, respectively, are modeled with separate ODEs (table 4.1). In the absence of the consumer and predator, the resource exhibits semi-chemostat growth $\delta\left(R_{max}-R\right)$ with δ and R_{max} as turnover rate and maximum resource biomass, respectively (table 4.1). Resource productivity, δR_{max}, increases linearly with R_{max}, and so we use R_{max} as a proxy for productivity throughout the model analysis (see 'Model parameterization and analysis'). Consumers, juvenile predators and adult predators reduce total resource biomass through feeding (table 4.1). Resource consumption by consumers increases their mass-specific net biomass production

$$\nu_c\left(R\right)=\sigma M_c\frac{R}{H+R}-T_c. \tag{4.1}$$

where σ represents the conversion efficiency of resources consumed with a Type II functional response (M_c and H represent the mass-specific maximum consumption rate of the consumer and half-saturation constant, respectively) balanced by the mass-specific maintenance rate of the consumer, T_c. Thus basic metabolic demands are met before energy is allocated to reproduction. Conversion efficiency, σ, represents the net effects of assimilation efficiency (*i.e.* ingestion lost to feces and urine) and specific dynamic action (*i.e.* thermal energy expenditure due to food processing) (Peters 1983). Consumer biomass is reduced by adult predators (juvenile predators do not feed on the consumer in either LHIGP scenario: figure 4.1) and background mortality, μ_c (table 4.1).

Juvenile and adult predator stages are directly linked by maturation, reproduction and asymmetric cannibalism (juveniles cannot consume adults; figure 4.1). The mass-specific net biomass production of juvenile predators

$$\nu_j\left(R\right)=\sigma M_p\frac{R}{H+R}-T_p. \tag{4.2}$$

also increases with resource consumption with a Type II functional response (M_p represents the mass-specific maximum consumption rate of the predator) balanced by the mass-specific maintenance rate of the predator, T_p. Juvenile predator biomass is reduced by the juvenile mortality rate (ω, subsequently defined eq. 4.5) and altered

by demographic transitions between juvenile and adult predator stages (table 4.1) as follows.

Net biomass production of juveniles $\nu_j(R)$ is spent on juvenile growth which adds biomass to the juvenile stage. Conditional upon survival, growth ultimately leads to maturation and transfer of juvenile biomass to the adult stage. The mass-specific maturation rate equals

$$\gamma\left(\nu_j(R), \omega\right) = \frac{\nu_j(R) - \omega}{1 - z^{1-\omega/\nu_j(R)}} \tag{4.3}$$

and ensures a correct translation between the biomass densities of juvenile and adult predators and a continuously size-structured model under equilibrium conditions from which this stage-structured model is derived (De Roos et al. 2008*b*). Mass-specific maturation increases with net juvenile biomass production and decreases with z, the ratio between predator size at birth and maturation, as well as the juvenile mortality rate, ω. Maturation represents the sole biomass input into the adult predator equation as adults spend all net production on reproduction and are assumed not to grow. Total adult biomass is reduced by predator background mortality (μ_p; table 4.1).

The net biomass production of adults

$$\nu_a\left(R, C, P_j\right) = \sigma M_p \frac{(1-\phi)R + \phi(C + \beta P_j)}{H + (1-\phi)R + \phi(C + \beta P_j)} - T_p. \tag{4.4}$$

increases with consumption of resource, consumer and juvenile predator biomass according to a Type II functional response. ϕ represents the fraction of time the adult predator spends foraging on the resource versus conspecific and heterospecific (*i.e.* consumer) prey: at $\phi = 1$, the adult predator forages solely on prey (*i.e.* complete diet shift with cannibalism depending on β: figure 4.1A), while at $\phi = 0.5$, the adult predator spends equal amounts of time foraging on the resource and prey (*i.e.* diet broadening with cannibalism depending on β: figure 4.1B). β represents the cannibalistic preference of adult predators feeding on juveniles. This parameter scales the preference for conspecific prey relative to heterospecific prey: at $\beta = 1$, adults have no preference for conspecific versus heterospecific prey, while $\beta < 1$ and $\beta > 1$ indicate cannibalistic aversion (*i.e.* heterospecific preference) or cannibalistic preference, respectively. In empirical studies, cannibalistic preference is often measured by Manly's α (Chesson 1978), which scales from 0 to 1 with $\alpha = 0.5$ indicating no cannibalistic preference (as presented in figure 4.2). Thus our $\beta = 1$ corresponds to Manly's $\alpha = 0.5$.

Lastly, cannibalism increases the juvenile mortality rate

$$\omega = M_p \frac{\phi\beta P_a}{H + (1-\phi)R + \phi(C + \beta P_j)} \tag{4.5}$$

which sums the effects of cannibalism and background predator mortality. Juvenile mortality rate reduces juvenile biomass, P_j (table 4.1), as well as the mass-specific

Table 4.1 – Model Equations

Dynamic equation	Description
$\frac{dR}{dt} = \delta\,(R_{max} - R) - M_c \frac{R}{H+R} C - M_p \frac{R}{H+R} P_j - M_p \frac{(1-\phi)R}{H + (1-\phi)R + \phi(C + \beta P_j)} P_a$	Resource biomass
$\frac{dC}{dt} = \nu_c(R)C - M_p \frac{\phi C}{H + (1-\phi)R + \phi(C + \beta P_j)} P_a - \mu_c C$	Consumer biomass
$\frac{dP_j}{dt} = \nu_a(R, C, P_j)P_a + \nu_j(R)P_j - \gamma\left(\nu_j(R), \omega\right) P_j - \omega P_j$	Juvenile pred. biomass
$\frac{dP_a}{dt} = \gamma\left(\nu_j(R), \omega\right) P_j - \mu_p P_a$	Adult pred. biomass

maturation rate, $\gamma(\nu_j(R), \omega)$ (eq. 4.3), since it determines juvenile survival and hence the fraction of newborn biomass that survives to the adult stage.

Model parameterization and analysis

This model was parameterized following Hin et al. 2011, which we refer the reader to for more thorough biological justification of parameter settings. Briefly, this parameterization (see figure 4.3 caption for settings) assumes quarter-power scaling relationships of mass-specific parameters (Brose et al. 2006; Peters 1983; Yodzis and Innes 1992), where adult predators are assumed to be 100 times the mass of consumers. Notably, the predator maximum consumption rate, M_p, is set at 2.5, while the consumer maximum consumption rate, M_c, is set at 10. This parameterization ensures that the consumer is a superior resource competitor in both complete diet shift and diet broadening LHIGP scenarios (Hin et al. 2011). Consumer competitive superiority is likely in nature due to morphological or behavioral tradeoffs associated with the predator ontogenetic diet shift (Toscano et al. 2016; see 'Discussion' for explanation), but we later relax this condition and assume the opposite to examine the effects of cannibalism on both LHIGP scenarios when the predator is the superior resource competitor.

We used MatCont (Dhooge et al. 2003), a MatLab package for numerical bifurcation analysis of ODEs, to calculate equilibrium biomass densities and assess equilibrium stability. We analyze the model scenarios $\phi = 1$ and $\phi = 0.5$, representing a complete ontogenetic diet shift and ontogenetic diet broadening, respectively (figure 4.1). While predators in nature likely occur along a continuum between these two diets, our initial model explorations demonstrated a continuous transition between the dynamics

of these different diet scenarios (not shown here). Therefore, modeling the two separate scenarios (complete diet shift and diet broadening) as we do here captures all possible qualitative dynamics. To examine potential effects of enrichment, we first explore equilibrium dynamics of these two parameterizations over a gradient in resource productivity (*i.e.* R_{max}). This analysis also allows us to compare dynamics to that of classic (*i.e.* unstructured) tri-trophic systems evaluated over gradients in productivity (Oksanen et al. 1981). We then apply numeric continuation methods to these equilibria, evaluated at $R_{max} = 3$, as a function of cannibalistic preference, β, to assess how resource, consumer and predator equilibrium biomasses respond to increasing cannibalism.

We additionally examined equilibrium states over gradients in R_{max} and β in four different LHIGP situations (*i.e.* parameterizations): (1) complete diet shift with the consumer as the superior resource competitor ($M_p = 2.5$); (2) complete diet shift with the predator as the superior resource competitor ($M_p = 5$); (3) diet broadening with the consumer as the superior resource competitor ($M_p = 2.5$); and (4) diet broadening with the predator as the superior resource competitor ($M_p = 5$). These analyses allowed us to explore the effects of cannibalism across a broader spectrum of potential LHIGP systems, notably when consumers are competitively inferior to predators (in contrast to the prior analyses), and extended productivity and cannibalism gradients within each of these four systems.

4.3 – Results

Complete diet shift LHIGP scenario

The dynamics of LHIGP in which the predator undergoes a complete diet shift between stages (without cannibalism) were previously described by Hin et al. (2011). We recapitulate the dynamics here to set the stage for introducing cannibalism to this LHIGP scenario (figure 4.1A).

Starting from low resource productivity (R_{max}), consumers invade the resource-only equilibrium before predators (at productivity threshold $BP_{C\rightarrow R}$) due to their superior competitive ability (figure 4.3A-C). This invasion point marks the start of a consumer-resource equilibrium that is stable (*i.e.* robust to predator invasion) over increasing resource productivity. At higher resource productivity, predator existence becomes possible (at productivity threshold LP_{PCR}) as an alternative stable predator-consumer-resource state (figure 4.3A-C). In this coexistence state, adult predators regulate consumer biomass (figure 4.3), while resource biomass increases with R_{max} (figure 4.3C). Thus coexistence equilibrium dynamics follow that of a traditional top-down food chain (Hin et al. 2011; Oksanen et al. 1981).

The predator stage distribution is dominated by adults in this coexistence state: adult biomass increases with resource productivity while juvenile biomass remains roughly constant (figure 4.3A). This stage-specific response to resource productivity arises because juveniles feed at a higher rate than adults, and thus maturation (*i.e.* biomass transition to the adult stage) occurs at a faster rate than reproduction. This asymmetry forms a reproductive bottleneck behind which adult biomass accumulates (*i.e.* reproduction-regulation), leading to an adult-dominated population structure. Specifically, juveniles feed at a higher rate than adults because their food source is more abundant than that of adults: resource biomass increases with resource productivity (figure 4.3C) because adults regulate competitively-dominant consumers (consumer biomass remains constant with increasing resource productivity: figure 4.3B). Thus, when predators exhibit a complete diet shift in LHIGP systems, reproduction is the limiting life-history process, and the preponderance of adults (and resulting top-down control) is necessary to maintain the coexistence state.

Complete diet shift with cannibalism

The consumer-resource equilibrium remains unchanged by cannibalism (β) due to the absence of predators (figure 4.3D-F). In contrast, the three species (predator-consumer-resource) coexistence equilibrium is highly sensitive to cannibalism (β); when a threshold of cannibalistic preference (LP_{PCR}) is exceeded, this equilibrium is no longer possible (figure 4.3D-F). Given the present model parameterization, this threshold occurs at a cannibalism level of $\beta \approx 0.27$ (*i.e.* heterospecific preference). At values of β beyond LP_{PCR}, the only equilibrium possible is the consumer-resource equilibrium (figure 4.3D-F). Thus moderate levels of cannibalism in the predator can actually prevent predator persistence in the complete diet shift LHIGP scenario, precluding potential alternative stable states.

A shift in the predator stage distribution is the mechanism behind the collapse of the coexistence state: cannibalism reduces total biomass of adult predators, but increases total biomass of juvenile predators (figure 4.3D). This seemingly counterintuitive effect, a reversal in the predator stage distribution, is potentially driven by two processes. First, cannibalism adds an exclusive food resource for adults (conspecific prey) that is not available to juveniles. Thus cannibalism can help free adults from resource limitation, increasing the rate of reproduction relative to maturation. Second, cannibalism induces stage-specific mortality of juvenile predators. Such mortality can alter population stage distribution independent of which life stage is subjected to increased mortality (De Roos et al. 2007). As explained above, the coexistence equilibrium of predators and intermediate consumers is dependent on adult predator-driven top-down control. Accordingly, the reduction in adult predators and increase in com-

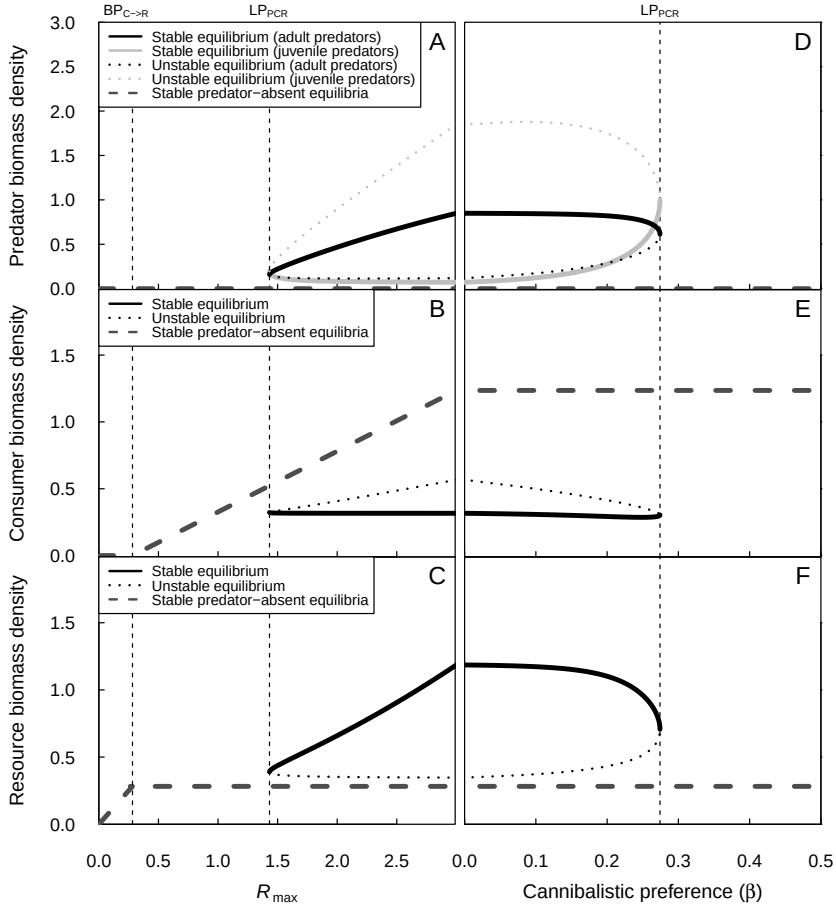

Figure 4.3 – Equilibrium biomasses as a function of resource productivity (R_{max}) when the predator undergoes a complete ontogenetic diet shift (left panels: A-C) and effects of cannibalistic preference (β) on these equilibria (calculated at $R_{max} = 3$; right panels: D-F). The consumer is competitively dominant in resource consumption. Juvenile predator biomass is indicated with gray lines and adult predator biomass with black lines. Solid lines depict stable predator-present equilibria and dotted lines unstable predator-present equilibria. Dark gray dashed lines depict equilibria with zero predator density (i.e. stable resource-only and consumer-resource equilibria). Vertical (dotted) lines mark different productivity (panels A-C) and cannibalistic (panels D-F) thresholds at which a qualitative change in equilibria occurs. In panels A-C, the consumer invasion (branching) point, $BP_{C\rightarrow R}$, marks the productivity threshold at which consumers invade the resource-only equilibrium, while LP_{PCR} (a limit point) marks the lowest productivity at which coexistence is possible. In panels D-F, LP_{PCR} marks the level of cannibalism at which coexistence is no longer possible. Parameter values are as follows: (panels A-C) R_{max} = varied, $\delta = 1$, $\sigma = 0.5$, $M_c = 10$, $M_p = 2.5$, $T_c = 1$, $T_p = 0.3$, $\mu_c = 0.1$, $\mu_p = 0.03$, $H = 1$, $\phi = 1$, $z = 0.01$, $\beta = 0$; (panel D-F) all parameters the same as in panels A-C except $R_{max} = 3$ and β is varied.

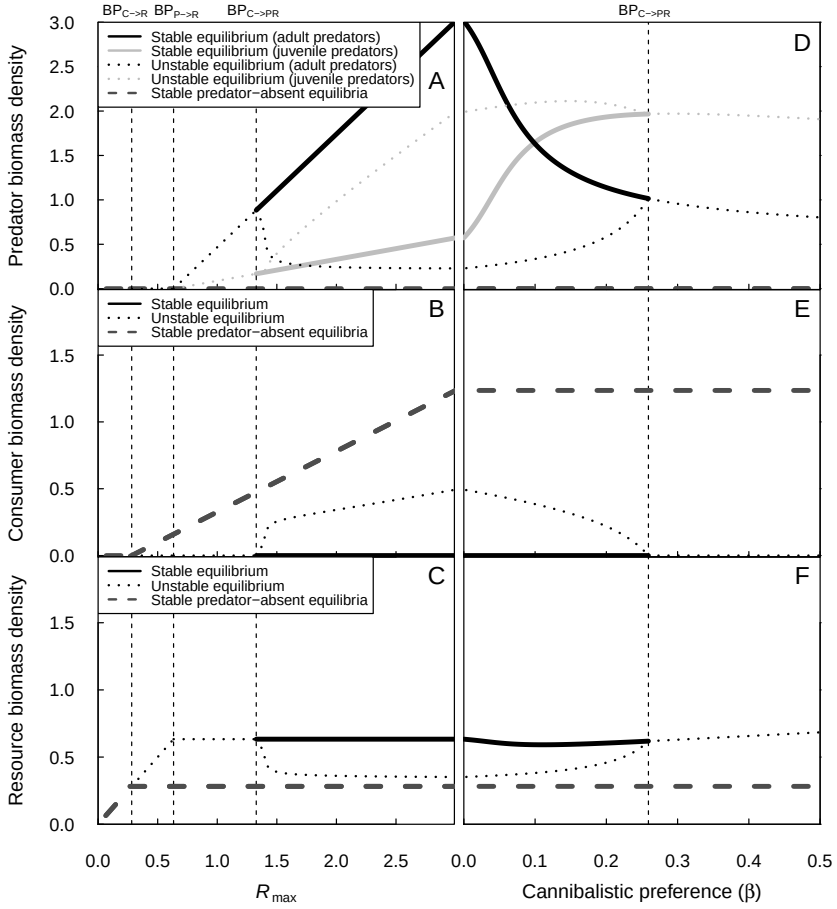

Figure 4.4 – Equilibrium biomasses as a function of resource productivity (R_{max}) when the predator undergoes ontogenetic diet broadening (left panels: A-C) and effects of cannibalistic preference (β) on these equilibria (calculated at $R_{max} = 3$; right panels: D-F). The consumer is competitively dominant in resource consumption. Lines are as in figure 4.3. In panels A-C, the consumer invasion (branching) point, $BP_{C\to R}$, marks the productivity threshold at which consumers can invade the resource-only equilibrium, while $BP_{P\to R}$, the predator invasion (branching) point, marks the productivity threshold at which predators can invade the resource-only equilibrium. A second branching point of the consumer, $BP_{C\to PR}$, marks the productivity threshold at which a stable predator-resource equilibrium becomes possible (as a function of R_{max} in panels A-C) or disappears (as a function of β in panels D-F). Stability of this predator-resource equilibrium branch changes due to the unstable consumer-resource equilibrium that originates at $BP_{C\to PR}$. Parameter settings are as in figure 4.3, except $\phi = 0.5$.

petitively inferior juvenile predators due to cannibalism (*i.e.* a shift from reproduction- to maturation-regulation: De Roos et al. 2007) reduces top-down control, leading to competitive exclusion of the predator and loss of the three species coexistence state.

Additional analyses indicate that juvenile-specific mortality, rather than energy gain for adults, is the primary driver of the effect of cannibalism on predator stage distribution (appendix figure 4.A1A-C, appendix figure 4.A2A-C). Even when energy gain for adult predators from cannibalism is prevented, cannibalism has the same qualitative effect on predator stage distribution (a shift from adult to juvenile biomass dominance) (appendix figure 4.A1A-C). Furthermore, an increase in juvenile-specific mortality alone, in the absence of cannibalism, captures this same qualitative effect (appendix figure 4.A2A-C).Thus it appears that juvenile-specific mortality due to cannibalism acts to shift biomass dominance to the juvenile stage, ultimately eliminating the coexistence equilibrium.

Diet broadening LHIGP scenario

When predators broaden their diet over ontogeny (*i.e.* consume both the intermediate consumer and the resource as adults: figure 4.1B) alternative stable states are again possible, but these states are different than in the complete diet shift scenario (figure 4.1A). In diet broadening, consumer-resource and predator-resource states co-occur over a range of R_{max}. Thus coexistence in the diet broadening scenario when the consumer is competitively dominant is not possible.

Starting at low resource productivity, competitively-superior consumers again invade the resource-only equilibrium before predators (at productivity threshold $BP_{C\rightarrow R}$) leading to a stable consumer-resource equilibrium over increasing resource productivity (figure 4.4A-C). At higher resource productivity, however, predator invasion (at productivity threshold $BP_{P\rightarrow R}$) and then stable existence (at productivity threshold $BP_{C\rightarrow PR}$) becomes possible as an alternative predator-resource state (figure 4.4A-C). While the predator-resource equilibrium branch originates at $BP_{P\rightarrow R}$, it only becomes stable at $BP_{C\rightarrow PR}$ due to an unstable coexistence equilibrium originating at this threshold (figure 4.4A-C). In this alternative stable state, adult and juvenile predators persist solely of resource biomass, regulating resource biomass with increasing resource productivity (figure 4.4C).

As in the complete diet shift scenario, this predator-present state is dominated by adult biomass. Specifically, adult biomass increases at a faster rate than juvenile biomass with increasing resource productivity (figure 4.4A). This occurs because generalist adults, which split their time foraging for resources and prey ($\phi = 0.5$), feed at half the rate of juveniles in the absence of consumers. Accordingly, reproduction occurs at a lower rate than maturation, driving biomass accumulation in the adult stage. Thus, just as in the complete diet shift scenario, the predator-present state in the diet

broadening scenario is reproductively-regulated (dominated by adult biomass) and this condition is necessary to ward off consumer invasion.

Diet broadening with cannibalism

Similar to the complete diet shift LHIGP scenario, cannibalism (β) has no effect on the consumer-resource equilibrium, but quickly destabilizes (at the threshold in cannibalistic preference $BP_{C\rightarrow PR}$: $\beta \approx 0.26$) the predator-present (predator-resource) state (figure 4.4D-F). Thus at values of β beyond $BP_{C\rightarrow PR}$, the only stable state possible is the consumer-resource equilibrium (figure 4.4D-F). Therefore, just as in the complete diet shift LHIGP scenario, low levels of cannibalism (*i.e.* maintaining a heterospecific prey preference) in the diet broadening scenario again prevent predator persistence and thus potential alternative stable states.

The mechanism behind this effect of cannibalism on community structure also involves a shift in the predator stage distribution. Specifically, where adult biomass outweighs juvenile biomass in the absence of cannibalism, cannibalism reverses this asymmetry (figure 4.4D). We show that juvenile-specific mortality, and not addition of an exclusive resource for adults, is again the primary driver of the effect of cannibalism on predator stage distribution (appendix figure 4.A1D-F, appendix figure 4.A2D-F). Thus juvenile-specific mortality due to cannibalism releases adults from resource limitation and shifts biomass dominance to the juvenile stage. Ultimately, this reduction in adult predators and the preponderance of competitively inferior juveniles destabilizes the predator-resource equilibrium (figure 4.4D-F) because predators can no longer resist consumer invasion.

Importance of consumer competitive ability

The aforementioned dynamics occur when consumers are competitively dominant over predators in resource competition. We further show that these same dynamics remain qualitatively unchanged over a wider range of cannibalism (β) and resource productivity (R_{max}) values (figure 4.5A, B). Specifically, predator persistence is limited to low cannibalistic preference and higher resource productivity in both complete diet shift (figure 4.5A) and diet broadening (figure 4.5B) LHIGP scenarios. At high cannibalistic preference and low resource productivity, a consumer-resource state is the only possible equilibrium (figure 4.5A, B) due to competitive exclusion of predators.

In contrast, when the predator is superior in resource competition, a juvenile competitive bottleneck and thus predator exclusion is no longer possible. Instead, a predator-present equilibrium always occurs at low resource productivity and remains robust to cannibalism (figure 4.5C, D). Specifically, in the complete diet shift scenario, the consumer invades the resource-only equilibrium first despite the competitive superiority of the predator (figure 4.5C); this is because adult predators specialize on

consumers and thus predators cannot exist in their absence. The predator, however, invades soon thereafter, leading to coexistence as the only equilibrium state (figure 4.5C). Cannibalism has no effect on qualitative dynamics in this scenario, but does stabilize oscillations (figure 4.5C; note Hopf bifurcation).

In the diet broadening scenario, the predator-resource equilibrium occurs as the only equilibrium state when predators are superior in resource competition in the absence of cannibalism. Interestingly, in contrast to all other scenarios, high cannibalism actually allows consumer invasion into this equilibrium at high resource productivity (*i.e.* three species coexistence: figure 4.5D). Again, this is due to the previously described shift in predator stage distribution due to cannibalism; cannibalism reduces adult biomass and increases juvenile biomass, which in turn permits consumer invasion due to a reduction in top-down control (appendix figure 4.A3). Thus in total, when the predator is the superior resource competitor, cannibalism either has no effect on qualitative equilibria (complete diet shift: figure 4.5C) or promotes coexistence (diet broadening: figure 4.5D).

4.4 – Discussion

A hallmark of structured predator-prey systems is that different predator sizes or stages often engage in different types of species interactions (Rudolf and Lafferty 2011; Werner and Gilliam 1984). Thus influences on predator population stage distribution have the capacity to shift the balance of interactions that shape long-term system dynamics. We present the novel finding that predators can short-circuit their own persistence in LHIGP systems through cannibalism, a nearly ubiquitous interaction in structured predator-prey systems (Byström et al. 2013; Rudolf 2007; Toscano et al. 2016). This occurs via a cannibalism-induced shift in the predator stage distribution from adult to juvenile biomass dominance. The loss of adult predators, solely responsible for regulating consumers, frees competitively-dominant consumers from top-down control, ultimately leading to predator exclusion. Predator extinction via cannibalism is only possible because of the feedback loop between predators and competitors; in a single-species system, cannibalism, a negatively density-dependent process, cannot drive extinction. We further show that this effect hinges upon the competitive superiority of consumers; when predators are competitively superior, cannibalism either has no effect on coexistence (in the complete diet shift scenario) or actually drives coexistence (in diet broadening). Thus while previous work emphasizes the (positive) effects of cannibalism in promoting coexistence (Ohlberger et al. 2012*a*; Rudolf 2007), our study demonstrates a diversity of effects of cannibalism on long-term LHIGP dynamics, depending on system properties.

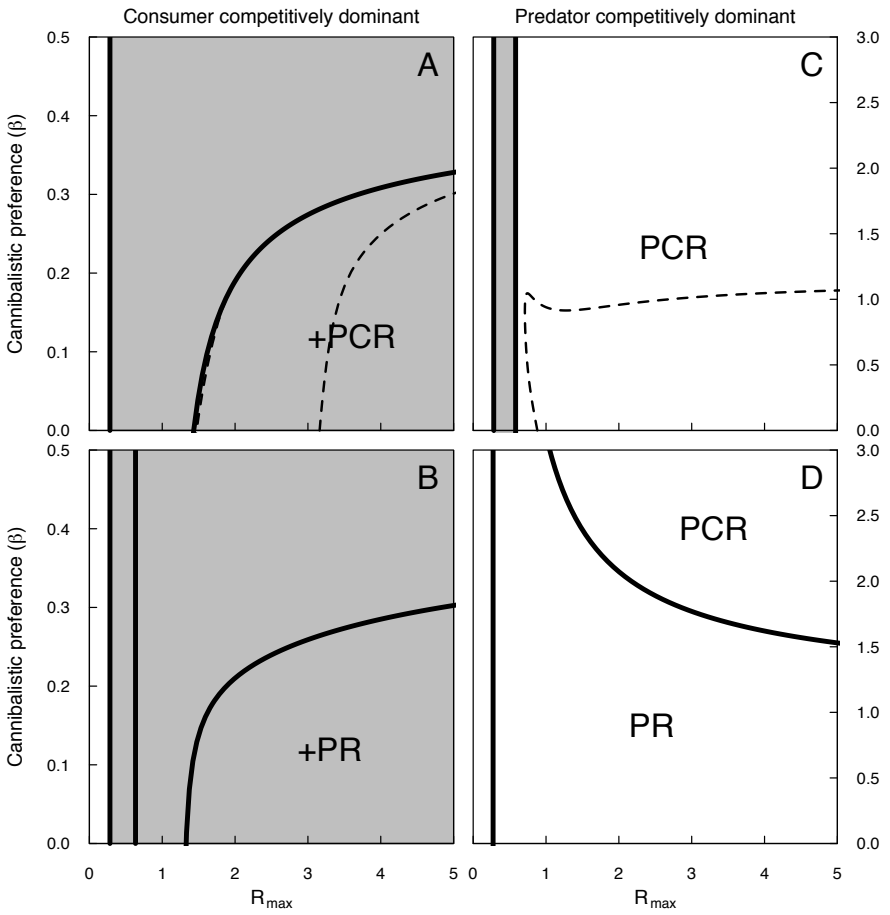

Figure 4.5 – Equilibria dependent on resource productivity (R_{max}) and cannibalistic preference (β) within four life-history intraguild predation (LHIGP) scenarios: (panel A) complete diet shift in predator ($\phi = 1$), consumer as superior resource competitor ($M_p = 2.5$); (panel B) diet broadening in predator ($\phi = 0.5$), consumer as superior resource competitor ($M_p = 2.5$); (panel C) complete diet shift in predator ($\phi = 1$), predator as superior resource competitor ($M_p = 5$); (panel D) diet broadening in predator ($\phi = 0.5$), predator as superior resource competitor ($M_p = 5$). Other parameter settings are as in figure 4.3. Solid lines indicate shifts between different qualitative equilibria. Narrow white region on the left-hand side of all four panels represents the resource-only equilibrium. Gray shading indicates the presence of a consumer-resource (CR) equilibrium. Letters indicate the presence of different predator-present equilibria (PR [predator-resource] or PCR [predator-consumer-resource]). An addition sign (+) preceding letters indicates that these predator-present equilibria occur alternatively to a CR equilibrium. The dashed lines in panels A and C mark the transition from a stable equilibrium point to a stable limit cycle (Hopf bifurcation), with the limit cycle occurring at higher resource productivity.

Ontogenetic asymmetry in LHIGP systems

We show that while the community-level effects of cannibalism can be diverse, they are all driven by a single mechanism: cannibalism (and specifically juvenile predator mortality) switches the predator stage distribution from adult to juvenile biomass dominance. The loss of adult predators (and thus top-down control) either eliminates, destabilizes, or allows consumer invasion into predator-present equilibria. Such asymmetries in biomass distribution arise when the key life-history transitions, development (*i.e.* maturation) and reproduction, differ in their rate of biomass transfer, a phenomenon termed ontogenetic asymmetry (De Roos and Persson 2013; De Roos et al. 2007; Persson and De Roos 2013). Predator populations in LHIGP systems are dominated by adult biomass at equilibrium due to the relatively greater energy intake rate of juveniles compared to adults. Thus maturation occurs at a greater rate than reproduction (*i.e.* reproduction-regulation). Our analyses indicate that cannibalism actually enhances the rate of reproduction relative to maturation by increasing juvenile mortality (*i.e.* a positive effect of mortality: Schröder et al. 2014), reversing the ontogenetic distribution of biomass. Previous analyses support this finding, that mortality can alter population stage distribution independent of which life stage is subjected to increased mortality (De Roos et al. 2007). Such ontogenetic asymmetry is an underappreciated characteristic of structured populations that is likely widespread in nature (De Roos and Persson 2013; Persson and De Roos 2013; Reichstein et al. 2015). Our work thus provides a striking example of the importance of ontogenetic asymmetry for long-term system dynamics, and further suggests that natural LHIGP systems could be more sensitive to changes in predator stage distribution than previously recognized, whether due to cannibalism or other means (*e.g.* human harvesting of adults: Walters and Kitchell 2001). Specifically, any factor that increases juvenile-specific mortality (*e.g.* disease or a size-dependent predator) could elicit similar effects in LHIGP systems without cannibalism.

Cannibalistic preference and coexistence

We found that the degree of cannibalism necessary to inhibit predator persistence in LHIGP is surprisingly low: the loss of predator-present equilibria occurs at a $3:1$ preference for heterospecific versus conspecific prey in both complete diet shift and diet broadening scenarios. While this precise cannibalistic preference is dependent on our model parametrization, the important finding is that adults can short-circuit their own persistence while still exhibiting preference for heterospecific prey. Furthermore, the present parameterization is based on power law scaling of physiological rates with body size and is thus justifiable based on metabolic theory (see Hin et al. 2011, for further explanation). Models based on biomass density, such as the present formulation, allow for a much more realistic parameterization than typical consumer-resource models

based on individual density (Yodzis and Innes 1992). Thus our results are certainly within the realm of biological possibility.

We can use empirical estimates of cannibalistic preference to speculate on whether predator taxa would inhibit their own persistence in LHIGP systems. Our literature review shows that cannibalistic preference varies considerably among a wide range of invertebrate and vertebrate predator taxa, and can rival or even exceed interspecific predation rates (Burgio et al. 2005; Gerber and Echternacht 2000; Rudolf 2008; Yasuda et al. 2001). Byström et al. (2013) for example, found that freshwater fish often exhibit a strong preference for conspecific relative to heterospecific prey. Interestingly, this same study found that fish predators are typically competitively inferior to their prey (intermediate fish consumers: Byström et al. 2013). Our results suggest that in these fish systems, there is a high likelihood of predator exclusion from LHIGP interactions. Thus it appears that the conditions for predator exclusion from LHIGP interactions are met in a number of natural LHIGP systems.

Reconciling theory with nature

Given these empirical patterns of cannibalism, our work thus poses an apparent conundrum: how can LHIGP systems with cannibalism be common in nature (Byström et al. 2013; Rudolf 2007)? The sensitivity of LHIGP to cannibalism is due in part to consumer competitive superiority: any factor that frees consumers from top-down control leads to a juvenile competitive bottleneck that inhibits predator persistence. This assumption (consumers superior to predators in resource competition) is likely upheld in many natural systems due to an ontogenetic tradeoff associated with continuous growth (Byström et al. 2013; Hin et al. 2011; Werner and Gilliam 1984): continuously-growing predators retain the same basic morphology while switching among resource types, and thus juvenile predators may be "burdened" by adult predatory morphology (Werner and Gilliam 1984). In contrast, specialist consumers feed solely on the basal resource over ontogeny and are thus not faced with such a trade-off.

This condition, however, is not necessarily true for species that undergo complete metamorphosis (Werner and Gilliam 1984, but see Toscano et al. 2016). Metamorphosis allows predators to completely "rebuild" their morphology over ontogeny, allowing them to potentially circumvent the same phenotypic trade-off. Thus as an alternative scenario, we present the effects of cannibalism on LHIGP dynamics when predators are competitively superior to consumers in resource competition, showing that the juvenile competitive bottleneck and concomitant loss of predator-present equilibria is no longer possible. Instead, a predator-present equilibrium is the sole equilibrium state in both complete diet shift and diet broadening scenarios. In the complete diet shift scenario, this predator-present state occurs as full (three species) coexistence, while in the diet broadening scenario, this occurs as a predator-resource equilibrium. Nevertheless,

strong cannibalism in the diet broadening scenario actually allows consumer invasion into this predator-resource equilibrium, thus promoting coexistence. Rudolf (2007) demonstrates this same effect of cannibalism in a diet broadening LHIGP model with a fixed (*i.e.* non-food-dependent) maturation rate, showing that this result is robust to model formulation. Thus, when the predator is superior in resource competition, coexistence is a likely outcome of LHIGP with cannibalism. More broadly, consumer versus predator competitive superiority is critical to predicting the long-term outcome of LHIGP interactions, and could depend in part on the details of predator life-history (*e.g.* continuous growth versus metamorphosis).

Other mechanisms could also allow for predator persistence in cannibalistic LHIGP systems when consumers are superior in resource competition, though these require an increase in system complexity beyond the basic LHIGP module. An extraneous (*i.e.* non-predator-driven) increase in consumer mortality, for example, permits predator persistence in both complete diet shift and diet broadening LHIGP scenarios, despite strong cannibalism (Toscano, unpublished results). In this scenario, increased mortality reduces consumer population growth (*i.e.* competitive ability) and thus the ability of consumers to competitively exclude predators. Thus it is possible that natural LHIGP interactions nested within complex food webs are more complicated than suggested by current model formulations, and this additional complexity begets the apparent stability of these interactions in nature.

Empirical ecologists may begin to test the theory we present using multigenerational community-scale experiments. Specifically, to test the effects of cannibalism on LHIGP system dynamics, we suggest using congeneric predators that differ in their degree of cannibalism (e.g copepods: Toscano et al. 2016) nested in otherwise identical LHIGP systems. While different predator species may vary in their precise life-history parameters or feeding rates, our theory is robust to these differences, predicting qualitatively different dynamics in the absence and presence of cannibalism. Specifically, predator persistence should not be possible in the presence of a cannibalistic predator, whereas in the absence of cannibalism, predator-present community states are predicted. Juvenile mortality is the primary process behind the collapse of predator-present stable states, and thus the experimental harvest of juveniles should elicit the same community response as cannibalism while corroborating the stage-dependent mechanism.

Cannibalism is predicted to result in predator-absent LHIGP states, and so the effects of cannibalism could be more difficult to test within intact natural communities. Nevertheless, additional analyses suggest that in LHIGP systems with low cannibalism, increasing consumer mortality (*e.g. via* a removal manipulation) should shift the predator to a more adult-dominated stage distribution (*i.e.* resembling the dynamics of an LHIGP system without cannibalism). Furthermore, consumers should exhibit

complete biomass compensation in response to increased mortality. Such community responses to increased consumer mortality would identify cannibalism as a key regulatory interaction. We emphasize, however, that long-term multigenerational studies of LHIGP dynamics are rare, and any such study would significantly advance our understanding of stage-structured community dynamics.

Conclusions

The present work demonstrates the potential for strong effects of cannibalism on the long-term outcomes of stage-structured species interactions, adding to just a handful of studies that have investigated the effects of cannibalism on complex communities (Ohlberger et al. 2012*a*; Persson et al. 2003; Rudolf 2007; Wahlström et al. 2000). Such a paucity of research attention is surprising, given that cannibalism is nearly ubiquitous in structured populations (Fox 1975; Polis 1981; Polis and Myers 1985). Claessen et al. (2004) suggest this lack of study is because "the complexity of (structured) cannibalistic-population models makes extensions to multi-species systems a daunting task." Nevertheless, we feel this is a task worth taking on, in order to better understand how communities might respond to the full range of biotic interactions in nature.

Acknowledgments

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Appendix 4.A Additional effects of cannibalism on predator equilibria

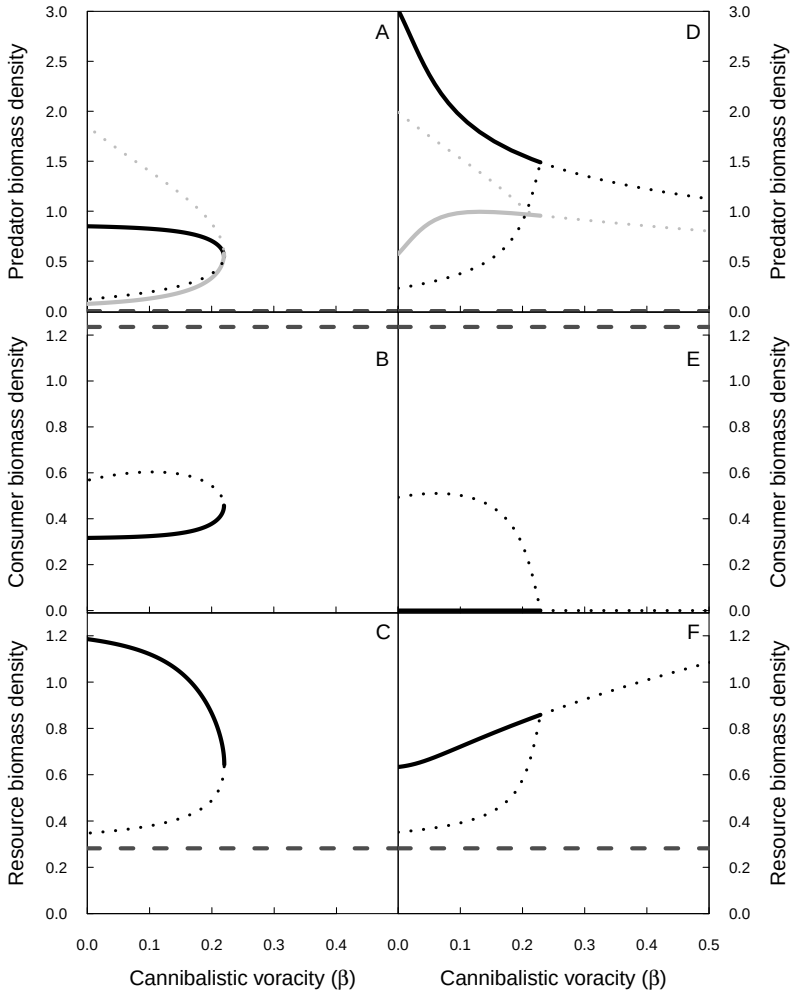

Figure 4.A1 – Effects of cannibalistic preference (β) without energy gain for adults on equilibria when the predator undergoes a complete ontogenetic diet shift (left panels: A-C) or ontogenetic diet broadening (right panels: D-F) (calculated at $R_{max} = 3$). The consumer is competitively dominant in resource consumption. Solid lines depict stable equilibria and dotted lines unstable equilibria. Juvenile predator biomass is indicated with gray lines and adult predator biomass with black lines. Dashed dark gray lines depict stable consumer-resource equilibrium where predators are absent. Parameter values are as follows: (panels A-C) $R_{max} = 3$, $\delta = 1$, $\sigma = 0.5$, $M_c = 10$, $M_p = 2.5$, $T_c = 1$, $T_p = 0.3$, $\mu_c = 0.1$, $\mu_p = 0.03$, $H = 1$, $\phi = 1$, $z = 0.01$, β = varied; (panels D-F) all parameters the same as in panels A-C except $\phi = 0.5$.

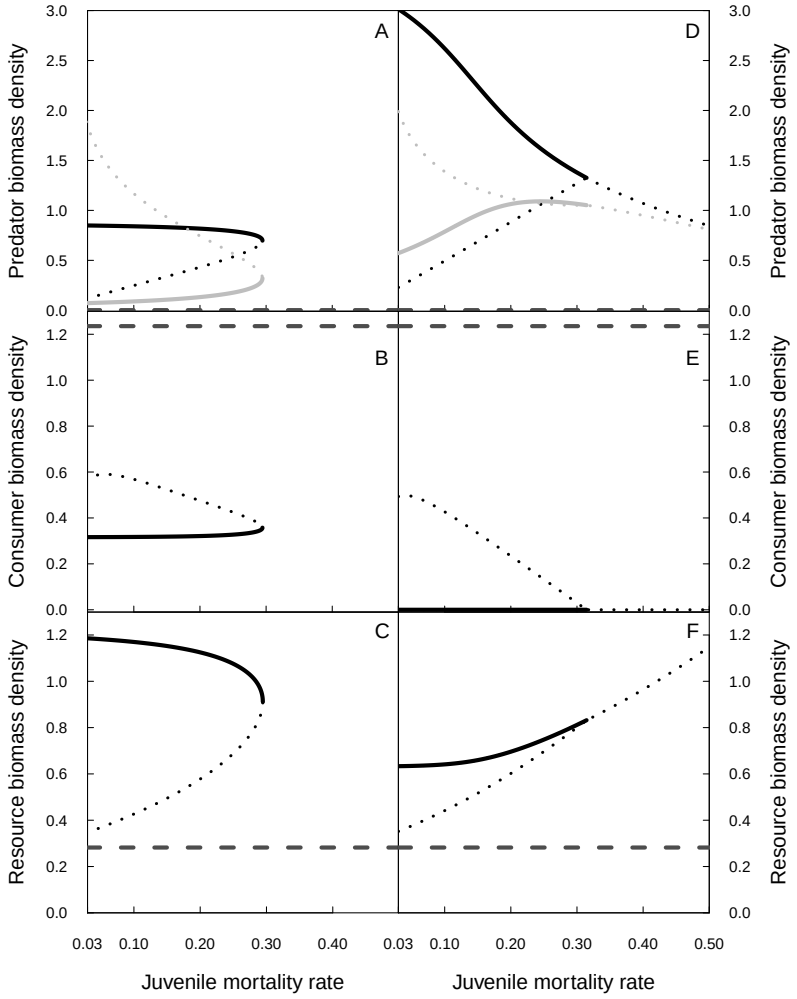

Figure 4.A2 – Effects of juvenile-specific mortality (ω) in the absence of cannibalism on equilibria when the predator undergoes a complete ontogenetic diet shift (left panels: A-C) or ontogenetic diet broadening (right panels: D-F) (calculated at $R_{max} = 3$). The consumer is competitively dominant in resource consumption. Solid lines depict stable equilibria and dotted lines unstable equilibria. Juvenile predator biomass is indicated with gray lines and adult predator biomass with black lines. Dashed dark gray lines depict stable consumer-resource equilibrium where predators are absent. Parameter values are as follows: (panels A-C) $R_{max} = 3$, $\delta = 1$, $\sigma = 0.5$, $M_c = 10$, $M_p = 2.5$, $T_c = 1$, $T_p = 0.3$, $\mu_c = 0.1$, $\mu_p = 0.03$, $H = 1$, $\phi = 1$, $z = 0.01$, $\beta = 0$; (panels D-F) all parameters the same as in panels A-C except $\phi = 0.5$.

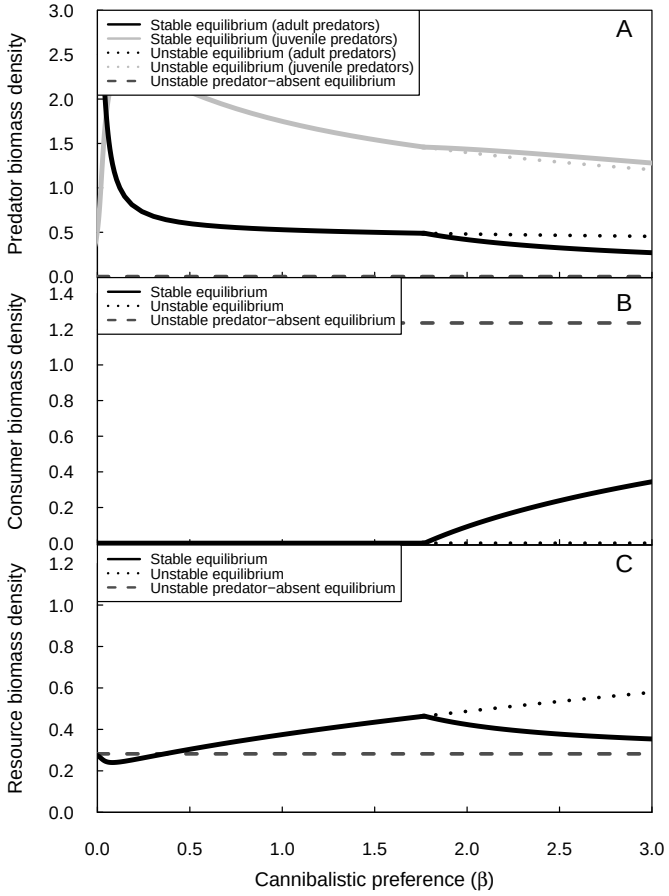

Figure 4.A3 – Consumer invasion into predator-resource equilibrium due to cannibalism (β) when the predator is dominant in resource consumption and undergoes diet broadening over ontogeny. Solid lines depict stable equilibria and dotted lines unstable equilibria. Juvenile predator biomass is indicated with gray lines and adult predator biomass with black lines. Dashed dark gray lines depict (unstable) consumer-resource equilibrium where predators are absent. Parameter values are as follows: $R_{max} = 3$, $\delta = 1$, $\sigma = 0.5$, $M_c = 10$, $M_p = 5$, $T_c = 1$, $T_p = 0.3$, $\mu_c = 0.1$, $\mu_p = 0.03$, $H = 1$, $\phi = 0.5$, $z = 0.01$, β = varied.

Appendix 4.B Studies measuring cannibalistic preference

Preference estimates are shown in figure 4.2 (main text)

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Gagne, I., D. Coderre, and Y. Mauffette. 2002. Egg cannibalism by *Coleomegilla maculata lengi* neonates: preference even in the presence of essential prey. Ecological Entomology 27:285-291.

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Leonardsson, K. 1991. Effects of cannibalism and alternative prey on population dynamics of *Saduria entomon* (*Isopoda*). Ecology 72:1273-1285.

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Park, S. R., J. Y. Jeong, and D. Park. 2005. Cannibalism in the Korean salamander (*Hynobius leechii: Hynobiidae, caudata, amphibia*) larvae. Integrative Biosciences 9:13-18.

Rudolf, V. H. W. 2008. Impact of cannibalism on predator-prey dynamics: size-structured interactions and apparent mutualism. Ecology 89:1650-1660.

Santi, F., G. Burgio, and S. Maini. 2003. Intra-guild predation and cannibalism of *Harmonia axyridis* and *Adalia bipunctata* in choice conditions. Bulletin of Insectology 56:207-210.

Schausberger, P. 1999. Predation preference of *Typhlodromus pyri* and *Kampimodromus aberrans* (*Acari: Phytoseiidae*) when offered con-and heterospecific immature life stages. Experimental & Applied Acarology 23:389-398.

Schausberger, P., and B. A. Croft. 2000. Cannibalism and intraguild predation among phytoseiid mites: are aggressiveness and prey preference related to diet specialization? Experimental and Applied Acarology 24:709-725.

Streams, F. A. 1992. Intrageneric predation by Notonecta (*Hemiptera: Notonectidae*) in the laboratory and in nature. Annals of the Entomological Society of America 85:265-273.

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Chapter 5

Cannibalism Prevents Evolutionary Suicide of Ontogenetic Omnivores in a Life History Intraguild Predation System

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Manuscript in preparation

Abstract

Ontogenetic omnivores are species that change or expand their diet during life. In ontogenetic omnivores without a metamorphosis, genetic correlations can result in a trade-off between resource specialization on early versus late-life resources. We show that such an ontogenetic trade-off has important consequences for persistence and coexistence of ontogenetic omnivores in life-history intraguild predation systems. In these systems, the small omnivores are engaged in resource competition with a specialist consumer species, which is the main prey of large omnivores. In addition, large ontogenetic omnivores are often cannibalistic. Selection on resource specialization under an ontogenetic trade-off leads to evolutionary suicide of the omnivore, but only when levels of cannibalism are low. Evolutionary suicide occurs because directional selection causes increasing resource specialization of one life stage, at the cost of feeding ability in the other life stage. This results in a shift to an alternative community state and extinction of the ontogenetic omnivore. Re-establishment is inhibited by strong competition with consumers in the juvenile phase. Cannibalism stabilizes selection on the ontogenetic trade-off, resulting in persistence of ontogenetic omnivores and coexistence with consumers. Based on these results, we conclude that life-history intraguild predation systems can only persist when levels of cannibalism are not low and (small) ontogenetic omnivores do not suffer from competition with the specialist consumer.

5.1 – Introduction

Ontogenetic or life-history omnivores are species that change or expand their diet during life (Persson 1988; Pimm and Rice 1987). These include organisms with direct development that change resources as a result of body size growth (such as fish and many amphibians), as well as species with indirect development, in which metamorphosis is responsible for the change in diet (*e.g.* holometabolous insects). The majority of all animal species fall within these two categories, with the exception of most birds and mammals (Werner 1988; Werner and Gilliam 1984; Wilbur 1980). The omnipresence of ontogenetic omnivores within the animal kingdom certainly suggests a high evolutionary potential of such a strategy.

The apparent evolutionary success of ontogenetic omnivores seems to be at odds with the insight that ontogenetic omnivores suffer from a trade-off that limits the ability to specialize on different resources (Hjelm et al. 2000, 2003; Robinson et al. 1996; Schluter 1995). Specializing on a specific resource means that body morphology, physiology and behavior (among others) are optimally adjusted to search, capture and process it. Since optimal exploitation of different resources require different sets of phenotypic traits, specialist species are often better adapted to utilize a specific resource than generalists. In ontogenetic omnivores the use of different resources is separated between different life stages. Genetic correlations between life stages are thought to prevent full ecological specialization on each of the resources used in these different life stages (Ebenman 1992; Werner 1988; Werner and Gilliam 1984). Ontogenetic omnivores are hence often considered less efficient foragers on a particular resource than their specialist competitors for that resource (Ebenman 1992; Persson 1988), in particular if they do not have a metamorphosis (Werner and Gilliam 1984). In fact, metamorphosis is hypothesized to have evolved as a mechanism to break these genetic correlations, so that the different life stages can independently specialize on their stage-specific resources (Ebenman 1992; Moran 1994; Ten Brink and De Roos 2017; Werner 1988). Ontogenetic omnivores that do not metamorphose, such as many fish species, have only limited possibility to rearrange their body morphology and physiology along with their change in resource use over ontogeny (Werner 1988; Werner and Gilliam 1984). The use of different resources and the genetic correlations that limit specialization on these different resources creates an ontogenetic trade-off that makes such ontogenetic omnivores vulnerable to being outcompeted by specialist species (Hin et al. 2011; Toscano et al. in press).

Ontogenetic omnivores thus face a trade-off between specializing on resources that are used early versus those that are used late in life (Ebenman 1992; Persson 1988; Werner 1988; Werner and Hall 1977). Within the same species trade-offs in resource specialization have mainly been shown to occur between morphs from dif-

ferent niches, such as benthic and limnetic forms of freshwater fish species (Robinson and Wilson 1996; Robinson et al. 1996; Schluter 1995). An ontogenetic trade-off has been hypothesized to occur between the benthic and piscivorous stages of Eurasian perch (*Perca fluviatilis*), which niches favor different body forms and feeding apparatus (Hjelm et al. 2000; Svanbäck and Eklöv 2002, 2003). However, given the extent of genetic correlations between different life stages (Aguirre et al. 2014; Cheverud et al. 1983; Ebenman 1992; Werner 1988) and the well-studied phenomenon that different resources require different morphologies (Futuyma and Moreno 1988), ontogenetic trade-offs are likely to occur and are probably important in many ontogenetic omnivores that lack metamorphosis.

These ontogenetic trade-offs can have important consequences for persistence and coexistence of ontogenetic omnivores in the context of life-history intraguild predation (LHIGP; Hin et al. 2011; Pimm and Rice 1987; Polis et al. 1989; Toscano et al. in press, 2016). In LHIGP systems, the ontogenetic omnivore (or intraguild predator) competes with a specialist consumer (or intraguild prey) for a shared resource early in life (when small/juvenile), but preys on the consumer later in life (when large/adult). Because of ontogenetic trade-offs the intraguild predator is often considered an inferior resource competitor compared to the consumer (Byström et al. 2013; Holt and Polis 1997; Persson 1988; Werner and Gilliam 1984). In such a case, the consumer potentially limits persistence of the intraguild predator, by suppressing the resource to a level that is insufficient for growth and successful maturation of juvenile intraguild predators (Byström et al. 1998; Hin et al. 2011; Persson and Greenberg 1990; Van de Wolfshaar et al. 2006; Walters and Kitchell 2001). This can occur irrespective of whether the intraguild predator completely changes its diet to exclusive foraging on consumer prey over ontogeny (diet shift LHIGP; Hin et al. 2011; Toscano et al. in press) or merely expands its diet to include consumer prey when large (diet broadening LHIPG; Rudolf 2007; Toscano et al. in press; Van de Wolfshaar et al. 2006). Alternatively, persistence of the intraguild predator can be promoted by adult predators suppressing consumers and thereby increasing resource availability for their juveniles (cultivation hypothesis; Walters and Kitchell 2001). The dominant process in LHIGP systems that ultimately determines the resulting community structure is hence either predation by adult intraguild predators on consumers, or competition between juvenile intraguild predators and consumers, but both processes do not play a major role at the same time (Hin et al. 2011; Persson and De Roos 2012). The selection on feeding specialization in ontogenetic omnivores is hence expected to depend on whether predation or competition is the dominant mode of interaction between the intraguild prey and predator. How these alternative structuring forces impact the evolutionary dynamics of ontogenetic omnivores that are subject to a trade-off between early versus late foraging success is currently unknown.

Cannibalism, or intraspecific predation, further complicates dynamics of LHIGP systems. Cannibalism often is a size-related interaction (Polis 1981), which makes it likely for adult intraguild predators to cannibalize juvenile intraguild predators, in addition to interspecific predation on the consumer (Byström et al. 2013). Also, because of the interconnectedness of the LHIGP food web all species generally occupy the same habitat, which further adds to the likelihood of cannibalism occurring. The common occurrence of cannibalism in LHIGP systems is supported by Byström et al. (2013) who show that 8 out of 11 fish species that shift from feeding on zooplankton or macrobenthic invertebrates to piscivory select conspecific over interspecific piscivorous prey. Theoretical work shows that the effect of cannibalism on persistence of the intraguild predator depends crucially on the competitive hierarchy between intraguild predator and prey (Toscano et al. in press). When the prey is the superior resource competitor, cannibalism in the predator releases top-down control of the prey by the predator, which breaks down the cultivation effect. This substantially limits the possibility for predator persistence (Toscano et al. in press). In contrast, when the predator is the superior resource competitor cannibalism promotes coexistence of intraguild prey and predator because it reduces top-down control of the predator on the resource (Toscano et al. in press). In LHIGP systems the rate of cannibalism is generally linked to the rate of interspecific predation, because both types of predation require the same set of morphological and behavioral adaptations and all species tend to share the same habitat. Therefore, adaption to interspecific predation is likely to also increase rates of cannibalism and, depending on the competitive hierarchy between consumers and intraguild predators, this can hinder predator persistence even further.

Here we study the consequences of an ontogenetic trade-off between early and late foraging success for persistence of an intraguild predator and its coexistence with intraguild prey. We simulate evolutionary dynamics of resource specialization of intraguild predators depending on the level of cannibalism and whether intraguild predators exhibit a diet shift or a diet broadening over ontogeny (Toscano et al. in press). Previous theoretical studies on resource specialization under an ontogenetic trade-off have shown the influence of productivity of early and late life resources and the non-linearity of the trade-off function (Ebenman 1992; Ten Brink and De Roos 2017). Therefore, next to evaluating qualitatively different trade-off forms we also vary productivity of the shared resource, which determines the ratio between consumer and resource abundance. Our findings reveal that non-cannibalistic intraguild predators evolve towards an abrupt threshold in the ecological dynamics beyond which they go extinct. Such evolutionary suicide of the intraguild predator occurs in both diet broadening and diet shift scenarios. Cannibalism, however, can stabilize evolutionary dynamics and prevent evolutionary suicide, irrespective of resource productivity and

the shape of the trade-off function. Cannibalistic ontogenetic omnivores can stably coexist with specialist consumers only when juveniles are specialized on the shared resource and do not suffer from competition. We conclude that the evolutionary success of non-metamorphosing ontogenetic omnivores in a life-history intraguild predation system can be explained by cannibalism and the resource specialization of juveniles.

5.2 – Model and method

Model formulation

We build upon the LHIGP model of Hin et al. (2011) and Toscano et al. (in press), who both use the stage-structured bio-energetics modeling approach as described by De Roos et al. (2007, 2008*b*). This approach is a stage-structured extension of the bio-energetic model presented by Yodzis and Innes (1992), separating the total population biomass into juvenile and adult biomass. Only the stage structure of the intraguild predator is explicitly represented, whereas population stage structure is ignored for the shared resource (R) and the consumer (C) on the grounds that juveniles and adults in these latter two populations do not differ from a dynamical, energetics perspective. In the predator life history we distinguish a juvenile (P_j) and an adult stage (P_a), where adults are assumed to only reproduce and do not grow.

In the original formulation by Hin et al. (2011) both consumers and predators feed according to Holling type II functional responses with a mass-specific maximum ingestion rate (M) and a half-saturation constant (H). The half-saturation constant indicates the resource density at which the functional response equals half the maximum ingestion rate. This parameter equals the ratio of the mass-specific maximum ingestion and the mass-specific attack rate or searching efficiency and therefore is a compound parameter. To perform evolutionary analysis on components of the functional response it is appropriate to choose a formulation that has parameters with a clear interpretation at the individual level (Rueffler et al. 2006*a*). Accordingly, we choose to avoid the formulation with the maximum ingestion rate and half-saturation constant and instead use an attack rate formulation in combination with a handling time constant (Persson et al. 1998; Ten Brink and De Roos 2017). In this formulation the attack rate or searching efficiency represents the amount of space or area that a predator can search for prey per unit of time, which is assumed to scale linearly with the body mass of the individual predator by Hin et al. (2011). Similarly, the handling time equals the inverse of the maximum ingestion rate, where the latter is also assumed to scale linearly with predator body mass (Hin et al. 2011). The handling time thus scales inversely with predator body size and amounts to the time required to handle

or digest one unit resource (or prey) biomass. Because of these scalings with predator body mass, the attack rate of species i feeding on species j will be represented by the mass-specific attack rate, denoted by a_{ij}, while the handling time will be represented by the scalar relating handling time to the inverse of individual body mass, denoted by h_i. The latter is assumed equal across all prey types.

All model equations are summarized in table 5.1. Mass-specific ingestion rate of resource biomass by consumers is hence given by:

$$I_{cr} = \frac{a_{cr}R}{1 + h_c a_{cr} R}$$

and ingested biomass is converted to net-biomass production (ν_c) after multiplication with conversion efficiency σ and subtraction of mass-specific maintenance rate T_c

$$\nu_c = \sigma I_{cr} - T_c$$

Similar to consumers, juvenile intraguild predators solely feed on resource biomass and their mass-specific ingestion and net-biomass production rates are hence given by:

$$I_{jr} = \frac{a_{jr}R}{1 + h_p a_{jr} R}$$

and

$$\nu_j = \sigma I_{jr} - T_p$$

respectively.

Adult intraguild predators feed on resource with attack rate a_{ar} and on consumers with attack rate a_{ac}. Additionally, adult predators are cannibalistic by feeding on juvenile predators. The attack rate for cannibalism is assumed equal to the attack rate for interspecific predation. Following Toscano et al. (in press), the intensity of cannibalism relative to interspecific predation is represented by a scaling factor β. Mass-specific ingestion rate of resource biomass by adult intraguild predators is therefore given by:

$$I_{ar} = \frac{a_{ar}R}{1 + h_p \left(a_{ar}R + a_{ac}\left(C + \beta P_j\right)\right)}$$

Ingestion rates of adults feeding on consumer biomass, (I_{ac}), and for cannibalistic feeding, (I_{aj}) follow similar expressions and are shown in table 5.1. The mass-specific net-biomass production rate of adult predators equals the difference between the sum of the three mass-specific ingestion rates, multiplied by conversion efficiency σ, and the mass-specific maintenance rate for predators, T_p.

$$\nu_a = \sigma \left(I_{ar} + I_{ac} + I_{aj}\right) - T_p$$

Table 5.1 – Model Equations

Description	Equation
Pred. attack rate trade-off	$a_{ac} = \frac{a_p - a_{jr}}{1 + \varepsilon a_{jr}/a_p}$
Consumer ingestion	$I_{cr} = \frac{a_{cr} R}{1 + h_c a_{cr} R}$
Juvenile pred. ingestion	$I_{jr} = \frac{a_{jr} R}{1 + h_p a_{jr} R}$
Adult pred. ingestion on resource	$I_{ar} = \frac{a_{ar} R}{1 + h_p \left(a_{ar} R + a_{ac} \left(C + \beta P_j\right)\right)}$
Adult pred. ingestion on consumer	$I_{ac} = \frac{a_{ac} C}{1 + h_p \left(a_{ar} R + a_{ac} \left(C + \beta P_j\right)\right)}$
Adult pred. ingestion on juvenile pred.	$I_{aj} = \frac{a_{ac} \beta P_j}{1 + h_p \left(a_{ar} R + a_{ac} \left(C + \beta P_j\right)\right)}$
Consumer net-biomass production	$\nu_c = \sigma I_{cr} - T_c$
Juvenile pred. net-biomass production	$\nu_j = \sigma I_{jr} - T_p$
Adult pred. net-biomass production	$\nu_a = \sigma \left(I_{ar} + I_{ac} + I_{aj}\right) - T_p$
Juvenile pred. mortality rate	$D_j = \mu_p + \frac{a_{ac} \beta P_a}{1 + h_p \left(a_{ar} R + a_{ac} \left(C + \beta P_j\right)\right)}$
Juvenile pred. maturation rate	$\gamma\left(\nu_j, D_j\right) = \frac{\nu_j - D_j}{1 - z^{1 - D_j/\nu_j}}$
Resource biomass dynamics	$\frac{dR}{dt} = \delta \left(R_{max} - R\right) - I_{cr} C - I_{jr} P_j - I_{ar} P_a$
Consumer biomass dynamics	$\frac{dC}{dt} = \nu_c C - I_{ac} P_a - \mu_c C$
Juvenile pred. biomass dynamics	$\frac{dP_j}{dt} = \nu_a P_a + \nu_j P_j - \gamma\left(\nu_j, D_j\right) P_j - D_j P_j$
Adult pred. biomass dynamics	$\frac{dP_a}{dt} = \gamma\left(\nu_j, D_j\right) P_j - \mu_p P_a$

Ingestion, production and biomass production rates are mass-specific

To study the evolutionary dynamics of late versus early foraging success we implement a trade-off between the juvenile attack rate on the resource (a_{jr}) and the adult attack rate (a_{ac}) for predation (both interspecific and cannibalism) as follows:

$$a_{ac} = \frac{a_p - a_{jr}}{1 + \varepsilon a_{jr}/a_p} \tag{5.1}$$

with $\varepsilon > -1$. This trade-off is a modified version of the trade-off function used by Ebenman (1992). In eq. 5.1 a_p is the maximum value that the attack rates a_{jr} and a_{ac} can adopt, while ε controls the shape of the trade-off. For $\varepsilon = 0$, the trade-off between the attack rates a_{jr} and a_{ac} is linear, for $\varepsilon < 0$ the trade-off is weak and concave from below, while for $\varepsilon > 0$ the trade-off is strong and convex from below. In order to study both diet shift and diet broadening LHIGP systems the adult attack rate for the resource a_{ar} is not part of the trade-off and can be set independently.

Juvenile and adult predator mortality include the stage-independent background mortality rate μ_p. Juvenile predators furthermore suffer from mortality due to cannibalistic feeding by adult predators, which amounts to:

$$\frac{a_{ac}\beta P_a}{1 + h_p\left(a_{ar}R + a_{ac}\left(C + \beta P_j\right)\right)}$$

Dynamics of resource, consumer, juvenile predator and adult predator biomass are described by four ordinary differential equations (table 5.1). Resource biomass increases following semi-chemostat dynamics (De Roos and Persson 2013) with turnover rate δ and maximum resource density R_{max} and decreases due to ingestion by consumers and juvenile and adult predators. Consumer biomass increases with total consumer net-biomass production, $\nu_c C$ and decreases through total feeding by adult predators, $I_{ac}P_a$ and consumer background mortality $\mu_c C$. Juvenile biomass increases through reproduction, which depends on the net-biomass production of adult predators and equals $\nu_a P_a$. Juvenile predators use their own net-biomass production rate, ν_j, exclusively for somatic growth. Growth increases juvenile biomass and, when positive, growth ultimately leads to maturation of juveniles to the adult stage. The rate of maturation, $\gamma\left(\nu_j, D_j\right)$, hence depends on the net-biomass production, but also on the juvenile mortality rate and the ratio between size at birth and size at maturation, z. The exact functional form of the maturation function is shown in table 5.1 and is derived such that the stage-structured biomass model in equilibrium is identical to a model with a continuous size-structure, as derived in De Roos et al. (2008*b*). The stage-structured biomass model studied hence implicitly accounts for the population size-structure dynamics of both the consumer and the predator population (see De Roos et al. 2008*b*, for details). Adult biomass only increases through maturation as adult predators spend all net-biomass production on reproduction. Adult biomass decreases through background mortality.

Model parameterization

Model parameterization follows Hin et al. (2011), in which a more in-depth motivation about the default parameter values (shown in table 5.2) can be found. To transform the functional response parameters of Hin et al. (2011) to our formulation we use the fact that the handling time equals the inverse of the maximum ingestion rate and the half-saturation constant equals the ratio between maximum ingestion and attack rate. The default parameterization of Hin et al. (2011) is derived from the observed power law scaling of the rates of mass-specific maximum ingestion, mass-specific maintenance and mortality with adult body size raised to the power of -0.25 (see also De Roos and Persson 2013). The constants of these scaling relationships approximately differ one order of magnitude; the mass-specific maximum ingestion equals 10 times the maintenance rate, which in turn equals 10 times the background mortality rate. Since Hin et al. (2011) adopt a value of 1 for the half-saturation constant we adopt $a_{cr} = 10$, $h_c = 0.1$, $T_c = 1$ and $\mu_c = 0.1$. The difference in the mass-specific rates between predators and consumer stems from the difference in their adult body mass. Hin et al. (2011) and Toscano et al. (in press) assume adult predators to be 100 times the mass of consumers. However, because predators grow in body mass 2 orders of magnitude during their life (the newborn-adult size ratio, $z = 0.01$; table 5.2) such a large mass difference would imply that only newborn predators overlap in body size with consumers, which is in contrast to the fact that species competing for a resource often have similar body sizes. We therefore assume a larger size overlap between consumers and juvenile predators and adopt a predator-consumer body mass ratio of 40. This value is close to the geometric average predator-prey body-mass ratio of 42 as reported by Brose et al. (2006). Consequently, the default values for the predator maintenance and mortality rate become $T_p = 0.4$ and $\mu_p = 0.04$, respectively, and for predator handling time $h_p = 1/4$.

The value of a_{ar} controls whether the intraguild predator has a diet shift ($a_{ar} = 0$) or a diet broadening ($a_{ar} > 0$). In addition, a_{ar} together with a_{jr} influence the competitive hierarchy between the intraguild predator and the consumer. This competitive hierarchy is of crucial importance for persistence and coexistence in (life-history) intraguild predation systems (Hin et al. 2011; Holt and Polis 1997; Mylius et al. 2001; Toscano et al. in press). Competitive ability is inversely related to resource density in the population-dynamical equilibrium containing only the resource and the focal population. The species with the lowest equilibrium resource density is hence considered the superior competitor, sensu Tilman (1980). For the intraguild predator we asses the competitive ability in absence of cannibalism. In case of a diet shift ($a_{ar} = 0$) the competitive ability of the intraguild predator is not defined, because predators cannot persist solely on the resource. However, juvenile intraguild predators can still compete with the consumer and to asses the competitive hierarchy between

Table 5.2 – Model Parameters

Description	Intraguild Predators		Consumer	Resource
	Juveniles	Adults		
Attack rate on resource	a_{jr} = varied	a_{ar} = varied	$a_{cr} = 10$	
Attack rate on consumer		a_{ac} = varied		
Cannibalism		β = varied		
Attack rate in trade-off	$a_p = 6$			
Shape of trade-off	$\varepsilon = 0$			
Handling time	$h_p = 0.25$		$h_c = 0.1$	
Maintenance rate	$T_p = 0.4$		$T_c = 1.0$	
Background mortality	$\mu_p = 0.04$		$\mu_c = 0.1$	
Juvenile/adult size ratio	$z = 0.01$			
Conversion efficiency	$\sigma = 0.5$		$\sigma = 0.5$	
Resource productivity				$R_{max} = 3$
Resource turn-over rate				$\delta = 1$

Attack and maintenance parameters are mass-specific. Handling time parameter inversely mass-specific

them requires an individual-level measure of the juvenile predator's competitive ability. For this we use the resource density that is needed for positive growth ($\nu_j > 0$). Juvenile predators are considered superior to consumers when they have a positive net-biomass production rate and can grow in the consumer-resource equilibrium.

Based on the above considerations we distinguish three different cases of competitive hierarchy: *i*) the consumer is superior to the intraguild predator and juveniles cannot grow in the consumer-resource equilibrium, *ii*) the consumer is superior to the intraguild predator, but juvenile predators can grow in the consumer-resource equilibrium and *iii*) the intraguild predator is superior to the consumer. In the last case juveniles are always able to grow in the consumer-resource equilibrium (appendix 5.A). These three cases apply for the diet broadening scenario ($a_{ar} > 0$), while in the diet shift scenario ($a_{ar} = 0$) we can only distinguish the first two cases. Figure 5.1 shows how these three cases of competitive hierarchy correspond to the values that determine the resource feeding of the intraguild predator (a_{ar} and a_{jr}). The derivation of

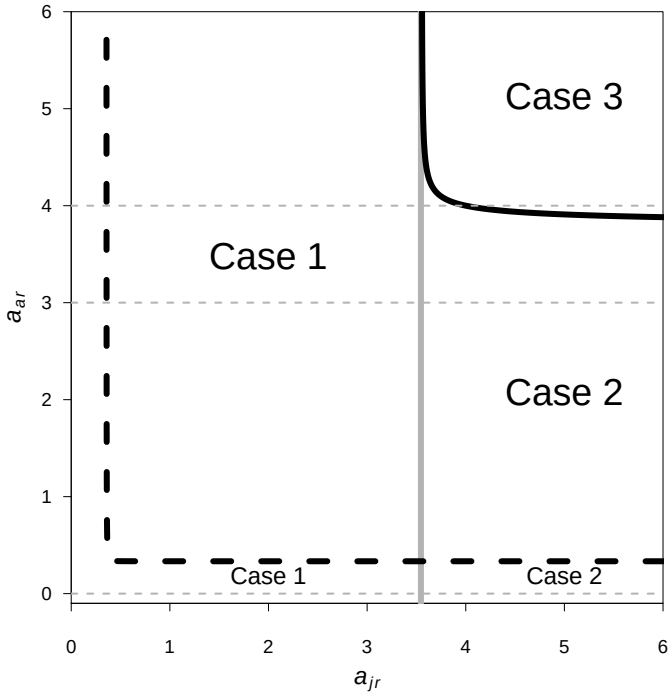

Figure 5.1 – Different cases of competitive hierarchy between consumers and intraguild predators as a function of a_{jr} and a_{ar}. The solid black line indicates the boundary between the overall competitive superiority of consumers vs. intraguild predators, with predators being superior at the upper-right of this line (case 3). The dashed black line indicates the region in which intraguild predators can persist solely on the resource if $R = R_{max}$, with persistence possible at the upper-right of this line. The vertical gray solid line separates the region where juvenile predators are superior to consumers (able to grow in the equilibrium resource density of consumers), with juveniles being superior to the right of this line. In case 1 the consumer is overall superior in resource competition. This also holds for case 2, but with the exception that juveniles are now superior to consumers. Horizontal dashed lines indicate the three different values of a_{ar} that are chosen in the analysis.

the boundaries shown in figure 5.1 is discussed in appendix 5.A. Based on figure 5.1 we choose three values of a_{ar} that, together with changes in a_{jr}, cover all three cases of competitive hierarchy in addition to the diet shift and diet broadening scenarios. For $a_{ar} = 0$ the intraguild predator has a diet shift and juvenile predators are superior to consumers for $a_{jr} > 3.55$. For $a_{ar} = 3$ intraguild predators have a diet broadening and can persist solely on the resource, but consumers are superior resource competitors. Again juvenile intraguild predators are superior to consumers for $a_{jr} > 3.55$. For $a_{ar} = 4$ the intraguild predator also has a diet broadening. Again, the juvenile intraguild predator is superior to the consumer for $a_{jr} > 3.55$ and, in addition, for

$a_{jr} > 4$ the intraguild predator outcompetes the consumer (figure 5.1). Finally, a_p controls the maximum value of a_{jr} and we take $a_p = 6$. By default the strength of the trade-off is set to $\varepsilon = 0$ (linear trade-off), but variation in this parameter is explored.

Model analysis

We used PSPManalysis (https://bitbucket.org/amderoos/pspmanalysis) to study equilibrium and evolutionary dynamics. PSPManalysis is a software package with numerical procedures to perform demographic, bifurcation and evolutionary analysis of physiologically structured population models. More details about the package can be found at https://bitbucket.org/amderoos/pspmanalysis. In addition to PSPManalysis we used MatCont (Dhooge et al. 2003), a Matlab package for numerical bifurcation analysis to asses equilibrium stability.

PSPManalysis uses adaptive dynamics as the framework for evolutionary analysis (Dieckmann and Law 1996; Geritz et al. 1998; Metz et al. 1995). In adaptive dynamics evolutionary change occurs by mutant phenotypes (y') that invade and take over the population-dynamical attractor of the resident phenotype (y). Invasion and replacement is only successful for mutants with phenotypes in the direction of the selection gradient. The selection gradient is sign equivalent with the derivative of the mutant's lifetime reproductive success, $R_0(y, y')$, with respect to the mutant's phenotype and evaluated at $y' = y$: $\left({\partial R_0(y',y)}/{\partial y'}\big|_{y'=y} \right)$ (Durinx et al. 2008; Geritz et al. 1998). Evolutionary change can come to a halt when the selection gradient vanishes. Such an evolutionary singular strategy (ESS) can either be convergence unstable (an evolutionary repeller; ERP), convergence stable and evolutionary stable (a continuously stable strategy; CSS) or convergence stable but evolutionary unstable (an evolutionary branching point; EBP). Convergence stability tells whether gradual evolution moves towards the ESS (convergence stable) or away from the ESS (convergence unstable). Evolutionary stability refers to whether the monomorphic population can evolve towards a dimorphic population, in which the mutant and resident can coexist (evolutionary unstable; see also Geritz et al. 1998). PSPManalysis calculates the selection gradient numerically and detects and classifies evolutionary equilibria according to the above classification.

Our primary interest lies with the two values of a_{ar} where consumers outcompete intraguild predators, $a_{ar} = 3$ and $a_{ar} = 0$ (figure 5.1), which, respectively, correspond to the diet broadening and diet shift scenarios as studied by Toscano et al. (in press). Next we adopt $a_{ar} = 4$, in which case intraguild predators can outcompete consumers for $a_{jr} > 4$. We analyze the equilibrium and evolutionary dynamics as a function of a_{jr} in case of no cannibalism ($\beta = 0$) and with cannibalism ($\beta = 1$). We furthermore check robustness of the results with respect to a continuous change in the level of cannibalism (β), system productivity (R_{max}) and the strength of the trade-off (ε).

5.3 – Results

In our three species community we can have four different types of equilibria: a resource only equilibrium (R-equilibrium), a consumer-resource equilibrium (CR-equilibrium), a predator-resource equilibrium (PR-equilibrium) and a predator-consumer-resource-equilibrium (PCR-equilibrium). In the following we will use the above abbreviations to refer to the different types of equilibria. Except for very low values of resource productivity ($R_{max} < 0.2820$), the R-equilibrium is unstable because it can always be invaded by consumers. We will not pay further attention to this equilibrium. The PCR-equilibrium is also referred to as the coexistence equilibrium, or coexistence state. With the term 'stability' we refer to the stability of the population dynamical equilibrium and stable equilibria are those that are (locally) stable against invasion or extinction of a certain species (all eigenvalues have a real part less then zero). Limit cycles only occur in a small region of parameter space and their amplitude is insignificant, for which reason we will not consider them further below.

Diet broadening without cannibalism ($a_{ar} = 3$, $\beta = 0$)
For $a_{ar} = 3$ the intraguild predator exhibits a broadening of its diet throughout ontogeny and can persist on the resource alone. However, the intraguild predator is an inferior resource competitor compared to the consumer, irrespective of the value of a_{jr} (figure 5.1, case 1 or 2). Without cannibalism ($\beta = 0$) there is no stable coexistence between the intraguild predator and the consumer as a function of a_{jr} (figure 5.2; Toscano et al. in press). When the ontogenetic trade-off of the intraguild predator is directed towards late foraging success (low a_{jr} and consequently high a_{ac}), predator persistence is not possible and only the CR-equilibrium is stable. Although a PR-equilibrium is possible for low a_{jr}-values it is unstable due to the low food availability for juvenile predators. Food limitation in the juvenile stage leads to low adult biomass and, consequently, low predation pressure of adult predators on consumers (figure 5.2, left panels). Consumers can therefore invade this PR-equilibrium and outcompete the predator. An increase in a_{jr} changes the size distribution of the predator towards adult dominated. This initially increases predation pressure that invading consumers would experience, despite the decrease in predatory attack rate a_{ac} associated with the increase in a_{jr} (equation 5.1). In addition, the increase in a_{jr} leads to a decrease in equilibrium resource density and these processes stabilize the PR-equilibrium against invasion of the consumer. Initially, the stable PR-equilibrium co-occurs with a stable CR-equilibrium as an alternative stable state. A further increase in a_{jr} destabilizes the CR-equilibrium, as the higher a_{jr}-value allows predators to invade it. This makes the PR-equilibrium the only stable equilibrium state. At $a_{jr} \approx 6$, the adult predation rate on consumers reaches zero and consumers and intraguild predators only

interact through competition for the shared resource. Since consumers are superior competitors, the CR-equilibrium becomes the only stable outcome here.

In the PR-equilibrium, selection is always positive on a_{jr} and negative on a_{ac} (arrows along the predator equilibrium in figure 5.2; top left panel). Due to the lack of consumers in this state and the absence of cannibalism, predators derive no benefits from retaining a predatory morphology. Therefore, intraguild predators become increasingly specialized as resource foragers by increasing a_{jr}. Eventually this destabilizes the PR-equilibrium and allows consumers to invade and outcompete the intraguild predator. Selection on resource specialization of juveniles ultimately leads to the extinction of the intraguild predator (evolutionary suicide).

Diet broadening with cannibalism ($a_{ar} = 3$, $\beta = 1$)
When intraguild predators are cannibalistic the properties of the ecological equilibria are different (figure 5.2; right panels). The PR-equilibrium now becomes stable against invasion by the consumer at a value of a_{jr} that is higher than the value at which the CR-equilibrium destabilizes and can be invaded by the predator. The latter point has remained at the same a_{jr}-value compared to the non-cannibalistic situation. In between these two threshold values of a_{jr} a stable PCR-equilibrium occurs. Although adult predators benefit from feeding on the consumer in this coexistence state, selection on a_{jr} is still positive. This leads to a lower per capita predation rate on consumers, but total mortality of consumers still increases due to an increase in adult predator density. Along with an increase in a_{jr} the PCR-equilibrium changes into a PR-equilibrium when consumers go extinct. In this PR-equilibrium selection on a_{jr} reaches an evolutionary endpoint (arrows along predator equilibrium in figure 5.2; top right panel). As adult predators cannibalize juveniles, they benefit from maintaining a predatory morphology and the intraguild predator therefore does not completely specialize towards the highest possible resource foraging ability of juvenile predators. The stabilizing selection on a_{jr} as a result of cannibalism prevents the evolutionary suicide of the intraguild predator.

Diet shift without cannibalism ($a_{ar} = 0$, $\beta = 0$)
Equilibrium properties of LHIGP systems with a full ontogenetic diet shift have been described before by (Hin et al. 2011) and (Toscano et al. in press). Because adult predators do not feed on the resource ($a_{ar} = 0$; complete diet shift, see figure 5.1), reproduction, and therefore predator persistence, is only possible in the presence of consumers. Figure 5.3 shows the ecological equilibria as a function of resource specialization, parameterized by a_{jr} with concomitant changes in a_{ac} following the trade-off relation in equation 5.1. At the two extremes of this trade-off ($a_{jr} \approx 0$ or $a_{jr} \approx 6$) the intraguild predator cannot persist and there is only a stable CR-

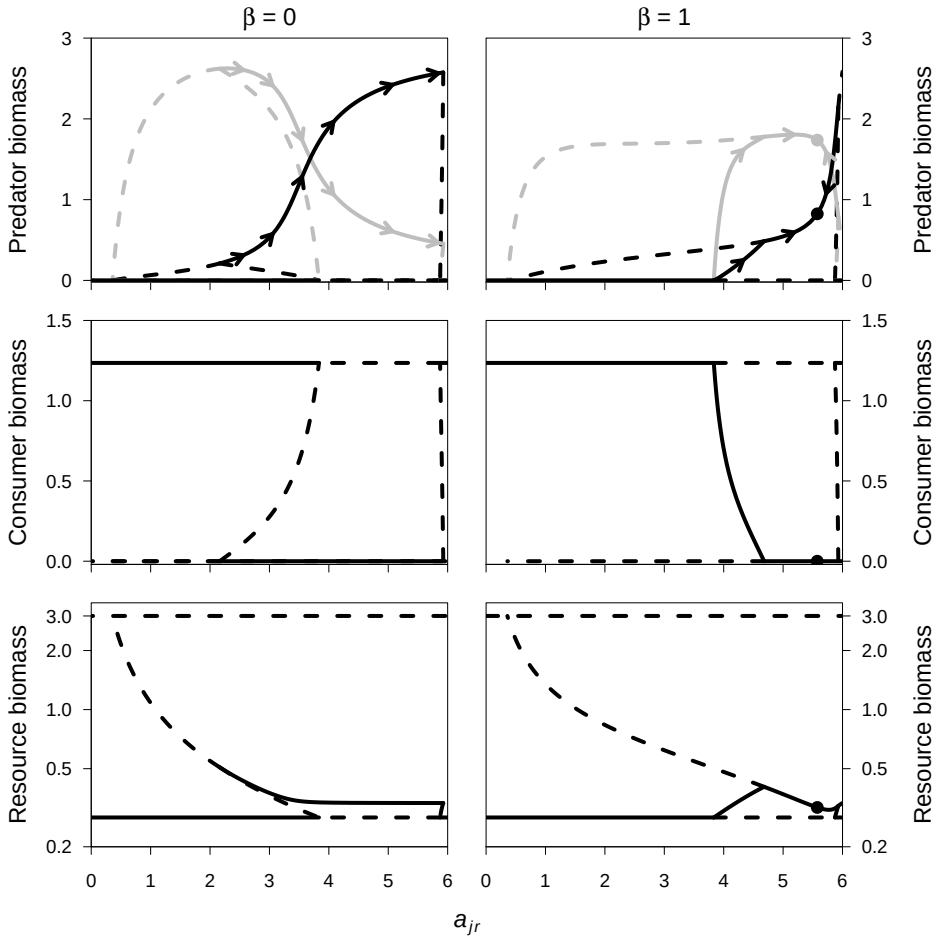

Figure 5.2 – Evolutionary dynamics of resource specialization (plotted as a function of a_{jr}, which is negatively related to a_{ac} through the trade-off in equation (5.1)) leads to evolutionary suicide of the intraguild predator when cannibalism is absent ($\beta = 0$; left panels) and to a stable evolutionary equilibrium when cannibalism is present ($\beta = 1$; right panels). Adult intraguild predators are able to feed on the shared resource (diet broadening scenario: $a_{ar} = 3$). All other parameters have default values (table 5.2). Solid (dotted) lines depict stable (unstable) equilibria of the ecological dynamics. Direction of selection on a_{jr} in the stable equilibrium with intraguild predators is indicated with arrowheads. Filled dots indicate evolutionary endpoints for a_{jr}, which are convergence and evolutionary stable. Black and gray curves in top panels represent adult and juvenile predator biomass, respectively.

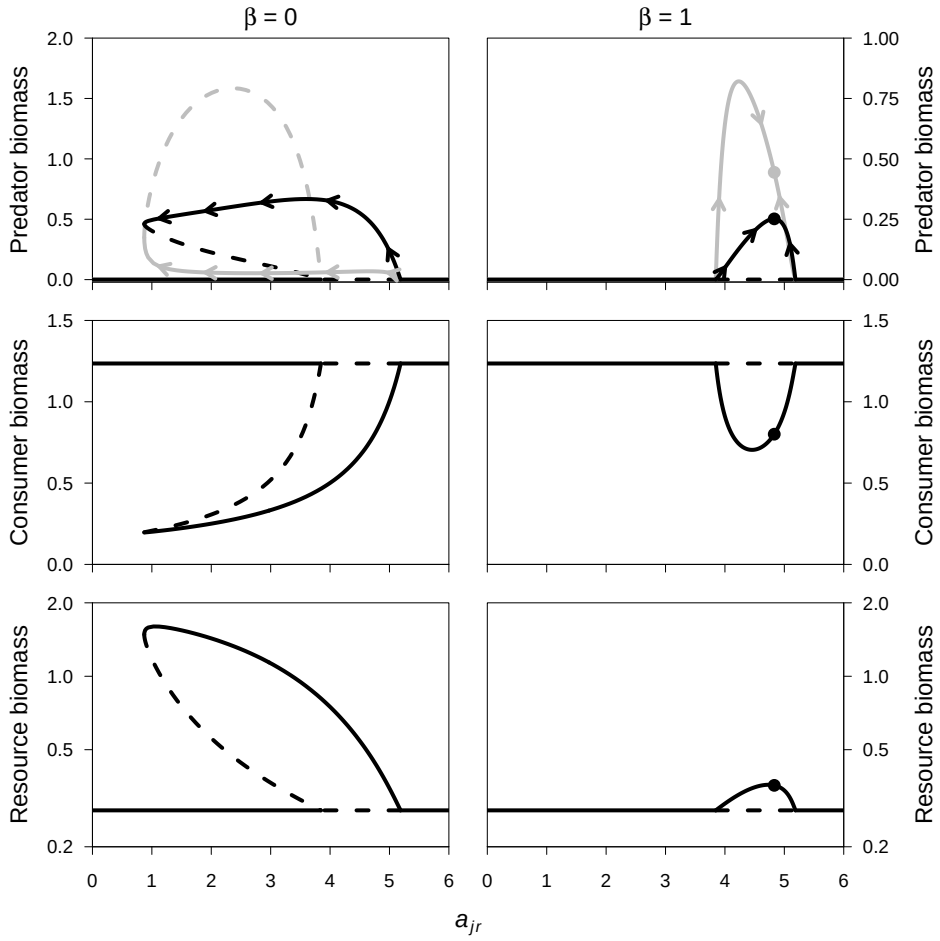

Figure 5.3 – Evolutionary dynamics of resource specialization (a_{jr} with concomitant change in a_{ac} following the trade-off in equation (5.1)) in the diet shift scenario (adult intraguild predators do not feed on the shared resource: $a_{ar} = 0$) leads to evolutionary suicide of the predator when cannibalism is absent ($\beta = 0$; left panels) and to a stable evolutionary equilibrium when cannibalism is present ($\beta = 1$; right panels). All other parameters have default values (table 5.2). All lines and symbols as in figure 5.2. Note the difference in y-axis scaling for predator biomass between left and right panels.

equilibrium. Persistence is possible when adult predators suppress consumers, thereby increasing resource availability for juvenile predators (figure 5.3; Hin et al. 2011; Walters and Kitchell 2001). This coexistence state is stable from $a_{jr} \approx 1$ until $a_{jr} \approx 5$, but co-occurs as an alternative stable state next to a stable CR-equilibrium for part of this parameter range. The CR-equilibrium can be invaded by predators when juveniles specialize on the resource (a_{jr} is high), which allows for positive juvenile predator growth for resource densities as present in the CR-equilibrium. However, for very high values of a_{jr} ($a_{jr} \leq 5$) invasion of the CR-equilibrium by predators is no longer possible, as adults no longer feed on consumers ($a_{ac} \approx 0$).

In the coexistence state, the suppression of consumers by adult predators leads to a high resource biomass density at equilibrium. Consequently, there is strong competition in the adult stage and little competition among juveniles. Maturation rates are high and reproduction rates are low, resulting in a low juvenile-adult biomass ratio. The fierce competition between adults favors a higher adult predation efficiency, at the expense of juvenile foraging ability. Thus, selection leads to lower values of a_{jr} and higher values of a_{ac} (arrows along PCR-equilibrium in figure 5.3; top left panel). Ultimately, this drives the predator population beyond the ecological threshold (fold bifurcation) that marks the minimum level of a_{jr} for which predator persistence is possible. Also in the case of a complete diet shift of the intraguild predator we therefore observe evolutionary suicide as a consequence of resource specialization in a non-cannibalistic LHIGP system, but remarkably enough now through selection towards adult as opposed to juvenile resource specialization.

Diet shift with cannibalism ($a_{ar} = 0$, $\beta = 1$)

Cannibalism among intraguild predators that exhibit a diet shift during ontogeny prohibits the occurrence of a PCR-equilibrium and a CR-equilibrium as alternative stable states (Toscano et al. in press). This is revealed by figure 5.3 (right panels), which shows the ecological equilibria as function of resource specialization. The range of a_{jr}-values for which the CR-equilibrium is unstable against invasion by predators remains identical to the case without cannibalism. However, persistence of the predator is not possible for a_{jr}-values outside this range. Cannibalism thus greatly diminishes the possibility of ecological persistence for the intraguild predator (Toscano et al. in press). Similar to the diet broadening scenario, cannibalism does stabilize selection on the resource specialization trade-off. From an evolutionary point of view cannibalism promotes predator persistence, by preventing the evolutionary suicide that results from strong selection towards adult specialization.

For values of a_{jr} below its evolutionary equilibrium value (indicated with the dot in figure 5.3, right panels) there is positive selection on a_{jr} in case of cannibalism, where in the non-cannibalistic case the selection gradient for similar values of a_{jr} is negative.

This change in the direction of selection with cannibalism can be explained by the change in predator size-structure that cannibalism induces. Cannibalism disrupts the top-down control of adult predators on consumers by shifting their foraging efforts toward juvenile predators (Toscano et al. in press). Consumer density is therefore higher and resource density lower than in the absence of cannibalism. The decrease in resource density as a consequence of cannibalism is so substantial that competition is most severe in the juvenile as opposed to the adult stage. As a consequence of cannibalism, the predator population therefore changes from being reproduction regulated, where competition is most severe in the adult stage, towards maturation regulated, where competition is most severe in the juvenile stage (De Roos and Persson 2013; De Roos et al. 2007). In a maturation regulated predator population, selection will tend to decrease juvenile competition through an increase in juvenile performance.

The effect of resource productivity (R_{max}) and trade-off shape (ε) in a diet broadening scenario ($a_{ar} = 3$)

The consequences of changes in resource productivity (R_{max}) and the shape of the trade-off (ε) on the model outcomes for the diet broadening scenario ($a_{ar} = 3$) are summarized in figure 5.4. Earlier studies have shown that an increase in resource productivity in a LHIGP system translates into an increased ecological persistence of the intraguild predator (Hin et al. 2011; Toscano et al. in press). In the diet broadening scenario this translates into an increase in the parameter range, for which a stable PR-equilibrium is possible (figure 5.4). Qualitatively, however, the parameter ranges of resource specialization and cannibalism that lead to different model outcomes do not change with changes in productivity (R_{max}) and trade-off shape (ε) (figure 5.4). Weak trade-offs (low ε-values) and high productivity decrease the threshold level of cannibalism (β) below which evolutionary suicide occurs and thus increase the region where selection on a_{jr} is stabilizing. For high levels of cannibalism the predator persists in a PCR-equilibrium, as the consumer can invade the PR-equilibrium. Also in this equilibrium a stable evolutionary endpoint of a_{jr} is possible (figure 5.4). Irrespective of resource productivity or trade-off shape, resource specialization of the predator is always directed towards juvenile specialization on the resource, at the expense of adult specialization on consumers/juveniles.

The effect of resource productivity (R_{max}) and trade-off shape (ε) in a diet shift scenario ($a_{ar} = 0$)

For the diet shift scenario ($a_{ar} = 0$), the model outcomes as presented in figure 5.3 depend on resource productivity (R_{max}) and trade-off shape (ε), but implications of the results remain the same. Quantitatively, weak trade-offs (low ε-values) and higher resource productivity increase the region of resource specialization for which

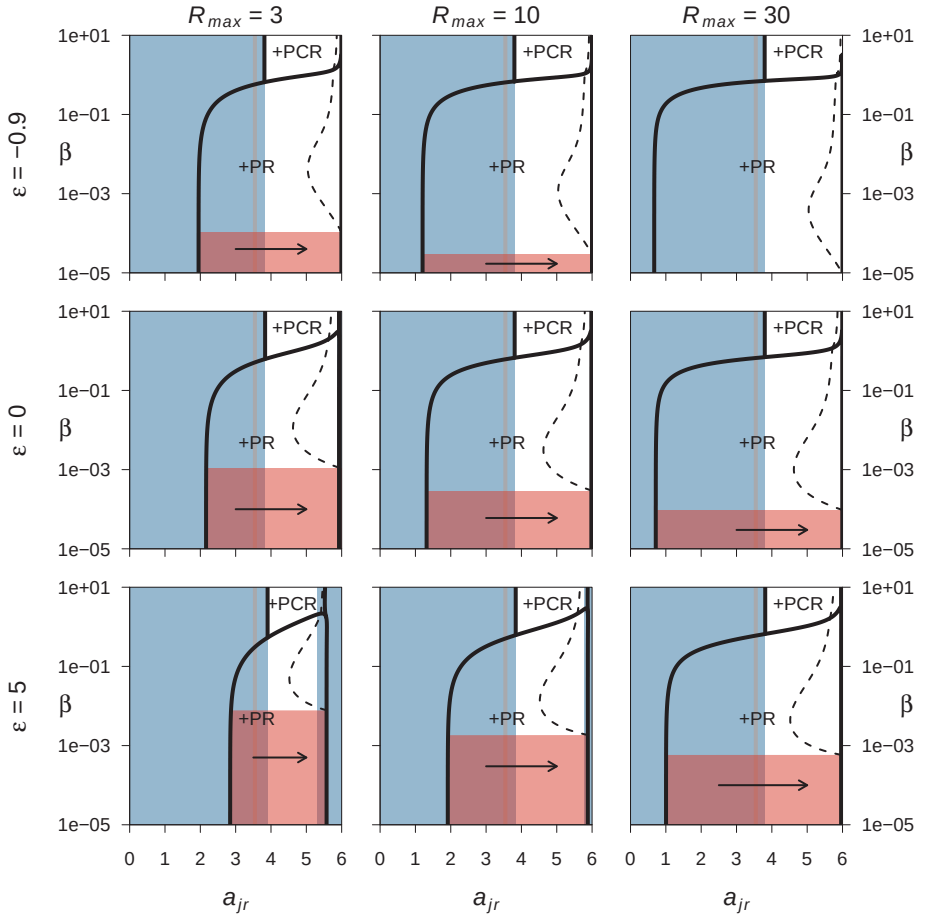

Figure 5.4 – Increasing productivity and decreasing trade-off strength decrease the regions of evolutionary suicide in the diet broadening scenario ($a_{ar} = 3$). Each panel shows the possible stable equilibria of the ecological dynamics as a function of a_{jr} and β, with a_{ac} related to a_{jr} following the trade-off in equation (5.1). The thick black lines indicate boundaries between parameter combinations for which different types of ecological equilibria with predators occur, with the text labels in between these boundaries indicating the type of equilibrium that occurs (+PR = PR-equilibrium, +PCR = predator-consumer-resource equilibrium). The blue shaded parameter regions indicate the presence of a CR-equilibrium that is stable against predator invasion. The dashed lines show the evolutionary equilibrium value of a_{jr}. All evolutionary equilibria are convergence and evolutionary stable (CSSs). In the red shaded regions, the evolutionary equilibrium for a_{jr} either does not exist or is located outside the region in which the ecological equilibrium is stable. Hence, the red shading indicates the parameter range for which selection on a_{jr} leads to evolutionary suicide. Horizontal arrows indicate the direction of selection in the region of evolutionary suicide. Outside the red shaded region the direction of selection is not indicated, but always points towards the evolutionary equilibrium (dashed line). To the right of the vertical gray lines the juvenile intraguild predators can grow at the resource density as set by consumers ($a_{jr} \approx 3.55$). For high values of β the predator persists in coexistence with the consumer and selection on a_{jr} is stabilizing.

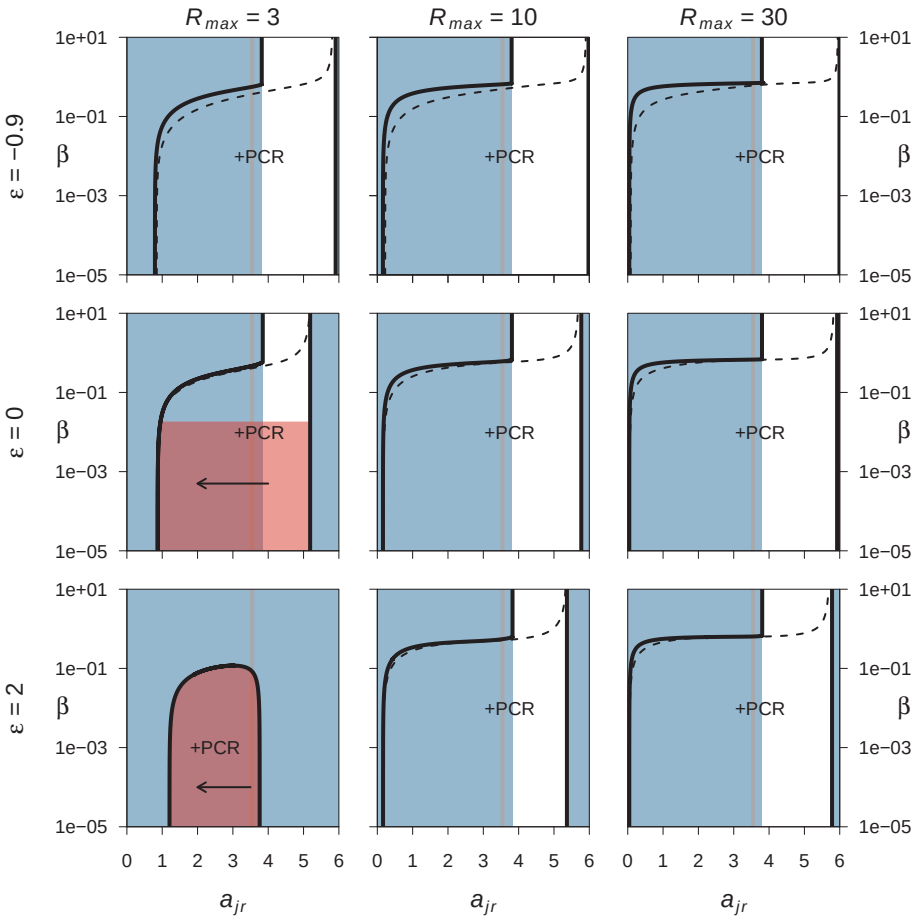

Figure 5.5 – In the diet shift scenario ($a_{ar} = 0$) the occurrence of evolutionary suicide depends on resource productivity (R_{max}) and trade-off strength (ε). For default parameter values ($R_{max} = 3$ & $\varepsilon = 0$), evolutionary suicide is, strictly speaking, only possible for low levels of cannibalism (on the y-axis) because there is no evolutionary equilibrium for a_{jr} (indicated by the dashed line) within the parameter range for which the ecological equilibrium with predators is stable (see also figure 5.3). For either a higher productivity or a weaker trade-off, the evolutionary equilibrium is within the parameter range with a stable ecological PCR-equilibrium, but located in close proximity to the persistence boundary of the predator (black solid lines) when cannibalism levels are low. When there is an overlapping stable CR-equilibrium (indicated by blue shading), any perturbation to the system can induce a shift to this alternative stable state and thus extinction of the predator. Only for higher levels of cannibalism the evolutionary equilibrium of a_{jr} is further away from the persistence boundary of the predator, in an area for which there is no overlapping stable CR-equilibrium. For a strong trade-off and low resource productivity the alternative CR-equilibrium is stable, independent of the level of cannibalism or the value of a_{jr}. All symbols and colors as in figure 5.4.

a stable PCR-equilibrium exists and decrease the parameter regions for which the CR-equilibrium is stable against predator invasion, in particular for higher values of a_{jr} (figure 5.5). Beside these quantitative changes there are two qualitative differences. In case of a low resource productivity and a strong trade-off ($R_{max} = 3$ and $\varepsilon = 2$) the CR-equilibrium is stable for all values of the resource specializations (a_{jr} on the x-axis). The predator can persist in the PCR-equilibrium that occurs as alternative stable state next to the CR-equilibrium. However, in this PCR-equilibrium there is no stable evolutionary endpoint and negative selection on resource specialization will lead to extinction of the predator (evolutionary suicide). Another noteworthy difference is the absence of evolutionary suicide in the strict sense for values of resource productivity and trade-off shape other than $R_{max} = 3$ and $\varepsilon \leq 0$. Evolutionary suicide technically only occurs when very close to the ecological threshold of predator persistence (the solid black lines in figure 5.5) the selection on a_{jr} is in the direction of this threshold. While this is the case for $R_{max} = 3$ with $\varepsilon = 0$ and $R_{max} = 3$ with $\varepsilon = 2$, for all other cases an evolutionary equilibrium does occur, albeit for a value of a_{jr} close to the ecological threshold of predator persistence (figure 5.5). Therefore, for the value of a_{jr} corresponding to the ecological threshold of predator persistence, the direction of selection on a_{jr} points away from this threshold and evolutionary suicide does, strictly speaking, not occur. However, the evolutionary endpoint occurs in an area of trait space that is very close to the predator's threshold of persistence and any perturbation in resource productivity or large mutational step can push the predator population over this ecological threshold towards extinction.

Diet broadening with superior intraguild predators ($a_{ar} = 4$)

For $a_{ar} = 4$ the intraguild predator is superior in resource competition to the consumer for $a_{jr} > 4$ (appendix 5.A). The evolutionary outcomes for this case are comparable to the diet broadening scenario with $a_{ar} = 3$ (figure 5.2) and we therefore only highlight the main differences. For $a_{ar} = 4$, the PR-equilibrium can no longer be invaded by consumers at high a_{jr}, as was the case for $a_{ar} = 3$ (figure 5.6), and is thus stable. Without cannibalism ($\beta = 0$) positive selection on a_{jr} does not lead to evolutionary suicide and the resource specialization of juveniles becomes constraint by the value of a_p (figure 5.6; left panels). With cannibalism ($\beta = 1$) there is again stabilizing selection for a_{jr}-values in the PR-equilibrium (figure 5.6; right panels), which leads to an evolutionary equilibrium for high resource specialization of juveniles. Even higher values of cannibalism allow for the invasion of consumers in the PR-equilibrium and, consequently, lead to a stable PCR-equilibrium (figure 5.B1; Toscano et al. in press). Also in this PCR-equilibrium selection on resource specialization is stabilized at high

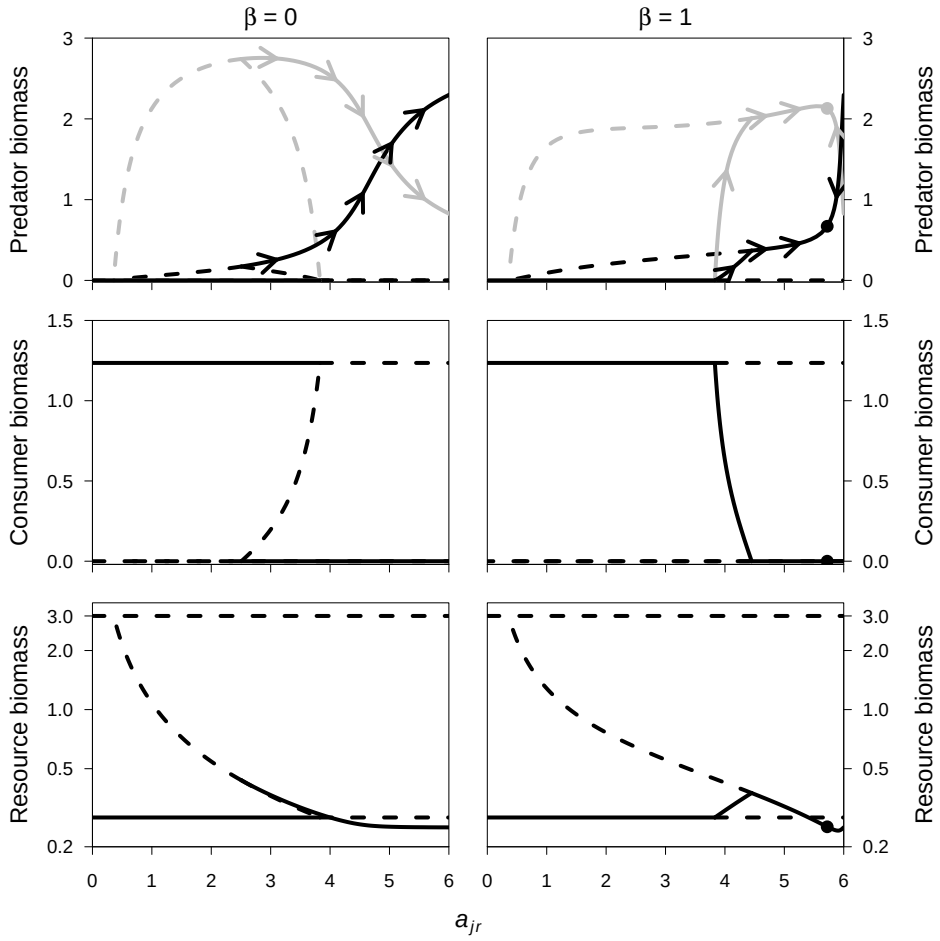

Figure 5.6 – Evolutionary dynamics of resource specialization (plotted as a function of a_{jr}, which is negatively related to a_{ac} through the trade-off in equation (5.1)) for $a_{ar} = 4$ with no cannibalism ($\beta = 0$; left panels) and with cannibalism ($\beta = 1$; right panels). For $a_{jr} > 4$, the intraguild predator becomes a better resource competitor compared to the consumer (when $\beta = 0$). Selection on the resource specialization trade-off (equation (5.1)) leads to increasing values of a_{jr}, and a decrease in a_{ar}. In case of cannibalism (right panels), selection is stabilized at high values of a_{jr}. All other parameters have default values (table 5.2). Lines and symbols as in figure 5.2.

juvenile specialization on the resource. Appendix figure 5.B1 shows furthermore that these results are qualitatively independent of the trade-off shape (ε) and resource productivity (R_{max}).

5.4 – Discussion

We study the evolutionary and ecological dynamics of a potentially cannibalistic, ontogenetic omnivore in a life-history intraguild predation system, in which the ontogenetic omnivore (or intraguild predator) competes as a juvenile with a consumer species (or intraguild prey) that becomes its prey later in life (Abrams 2011; Hin et al. 2011; Pimm and Rice 1987; Polis et al. 1989; Van de Wolfshaar et al. 2006). We show that such ontogenetic omnivores (or intraguild predators) when subject to a trade-off in resource specialization between early- and late-life resources can only stably persist over evolutionary time when adult intraguild predators cannibalize juvenile predators. Without cannibalism, selection either increases resource specialization of juveniles, which negatively affects the feeding ability of adults, or selection favors (predatory) resource specialization of adults at the cost of poor performance of juveniles on the resource that is shared with the consumer. The direction of selection depends on whether adult predators are able to feed on the resource, which distinguishes a diet broadening scenario from a diet shift scenario and determines whether predators persist alone (diet broadening) or in coexistence with consumers (diet shift). In both scenarios, directional selection leads to evolutionary suicide of the intraguild predator because it forces the predator population into an area of trait space where its ecological persistence is compromised. Cannibalism prevents evolutionary suicide by stabilizing selection on the resource specialization trade-off. This happens because cannibalism creates a benefit to keep a predatory morphology for adult predators (in the diet broadening scenario) or because cannibalism changes the population regulation of the predator such that resource specialization of juveniles becomes important (in the diet shift scenario).

Our results potentially have broad ecological and evolutionary significance, as the majority of all animal species are ontogenetic or life-history omnivores that undergo an ontogenetic niche shift either because of ontogenetic growth (direct development) or as a result of metamorphosis (indirect development) (Persson 1988; Pimm and Rice 1987; Werner 1988; Werner and Gilliam 1984; Wilbur 1980). In ontogenetic omnivores without a metamorphosis, the morphological and behavioral adaptations that are related to feeding on the different resources are controlled by a single genetic architecture or are subject to genetic correlations (Ebenman 1992; Marshall and Morgan 2011; Schluter et al. 1991; Werner 1988). When the different resources require different optimal phenotypes this results in opposing selection pressures between the life stages that feed on different resources (Ebenman 1992; Hjelm et al. 2000, 2003; Robinson et al. 1996; Schluter 1995; Schluter et al. 1991; Werner 1988). Such an ontogenetic trade-off makes persistence of ontogenetic omnivores vulnerable to competition with specialist species, raising the question of how ontogenetic omnivory can be such a widespread strategy.

Persistence of intraguild predators

An ontogenetic trade-off in feeding efficiency was originally proposed as the reason for why intraguild predators are inferior in resource competition compared to their specialist prey (Persson 1988; Werner and Gilliam 1984). Moreover, the competitive superiority of prey was put forward as one of the requirements that enabled coexistence between intraguild predators and prey in the absence of size-specific interactions (Diehl and Feißel 2000; Holt and Polis 1997). The assumption of competitive dominance of the prey is likely to hold in many, but certainly not all systems (Vance-Chalcraft et al. 2007). However, when intraguild predation is the result of an ontogenetic niche shift (life-history omnivory/intraguild predation; Abrams 2011; Hin et al. 2011; Rudolf 2007; Toscano et al. in press; Van de Wolfshaar et al. 2006, *this study*) the predictions about persistence and coexistence in such systems become more complicated and depend on a number of factors. We integrate these predictions in table 5.4, and distinguish between *i*) the extent of diet change during ontogeny of the intraguild predator, *ii*) the three cases of competitive hierarchy between consumers and predators and *iii*) the level of cannibalism. Table 5.4 describes the opportunity for persistence and potential coexistence on an ecological time scale by denoting the stable population dynamical equilibria that occur. In addition, table 5.4 also provides an overview of the evolutionary dynamics that result from selection on the ontogenetic trade-off in resource specialization.

The assumption that consumers are competitively superior to all predators diminishes the ecological scope for persistence of predators and coexistence with consumers (Hin et al. 2011; Toscano et al. in press). For no or low levels of cannibalism, the superiority of consumers leads to alternative stable states between an equilibrium with predators present (either PR or PCR-equilibrium, depending on the extent of diet change of the intraguild predator) and a CR-equilibrium (table 5.4; Hin et al. 2011; Toscano et al. in press). With higher levels of cannibalism the competitive superiority of consumers is even more detrimental as the equilibrium, in which predators are present, disappears completely and only a CR-equilibrium remains. Cannibalism hence disrupts ecological persistence of intraguild predators in LHIGP systems, in which consumers are superior to juvenile predators, a result that was pointed out by Toscano et al. (in press). Even in cases that non-cannibalistic omnivores in LHIGP systems are predicted to persist ecologically (Hin et al. 2011; Toscano et al. in press), we show here that their potential to persist over evolutionary time-scales is threatened by selection on the ontogenetic trade-off in feeding ability on the different resources. In these cases, cannibalism enhances persistence on evolutionary timescales if intraguild predators can evolve such that the competitive hierarchy changes from consumers being superior towards juvenile predators being superior or even predators being overall superior. The incorporation of evolutionary dynamics into models of LHIGP systems

thus leaves us to conclude that these systems can only persist stably when levels of cannibalism (compared to interspecific predation) are not low and when (juvenile) intraguild predators are competitively superior to consumers.

The occurrence of cannibalism and the competitive hierarchy between juvenile intraguild predators and consumers in LHIGP systems were reviewed for a number of freshwater fish species by Byström et al. (2013). They concluded that large ontogenetic omnivores preferentially select conspecifics over interspecific prey (high values of β in our study) and that consumer species are more efficient zooplankton foragers than juvenile ontogenetic omnivores. The latter conclusion was based on attack rate measurements from separate feeding experiments (Byström et al. 2013). The high cannibalistic preference as observed by Byström et al. (2013) is in accordance with the requirements for predator persistence in LHIGP system that we pose here. However, it is difficult to draw conclusions about competitive inferiority of juvenile ontogenetic omnivores relative to their prey species based on a higher attack rate alone. To be able to compare the results of Byström et al. (2013) to our framework, information on other processes that contribute to competitive ability are required. These processes include handling times, maintenance metabolism and conversion efficiency. The crucial experimental test would be to study whether juvenile ontogenetic omnivores can successfully grow and mature under resource conditions as set by the specialist consumer species they compete with for resources. Besides the role of exploitative competition, other interspecific interactions such as interference or predation can also play an important role in determining to what extent juvenile ontogenetic omnivores suffer from competition with specialist consumers under natural conditions.

Table 5.4 (opposite page) – Theoretical predictions of persistence of (cannibalistic) ontogenetic omnivores and coexistence with specialist consumers in life-history intraguild predation systems. The table distinguishes between 3 types of competitive hierarchy: *i*) consumers are superior, *ii*) juvenile predators are superior to consumers, but adult predators are not and *iii*) both juvenile and adult predators are superior to consumers; 3 levels of cannibalism: *i*) no or low cannibalism, *ii*) medium cannibalism and *iii*) high cannibalism; and whether the intraguild predator undergoes a diet broadening or diet shift over ontogeny (top vs bottom part of the table, respectively). In case of a diet shift predators cannot be competitively superior to consumers since adult predators do not feed on the resource. For each combination of competitive hierarchy, level of cannibalism and ontogenetic diet change the table presents the possible ecological equilibria and the evolutionary process. Evolution acts either to increase resource specialization of juveniles on the shared resource, or of adults on the consumer as studied with the trade-off in equation (5.1). CR; consumer-resource equilibrium; PR: predator-resource equilibrium; PCR: predator-consumer-resource equilibrium; ASS: alternative stable states.

Diet broadening			Competitive hierarchy		
			Consumer > all predators	Juvenile predators > consumers	All predators > consumers
Level of cannibalism	No / low	Ecological	ASS: PR/CR[1]	PR	PR[1]
		Evolutionary	Suicide	Suicide	Persistence[a]
	Medium	Ecological	ASS: PC/CR or only CR[1,b]	PR	PR[1]
		Evolutionary	*Evolutionary transient to juvenile predators > consumers*	Persistence	Persistence
	High	Ecological	Only CR[1]	PCR	PCR[1]
		Evolutionary	No persistence	Persistence	Persistence
Diet shift					
Level of cannibalism	No / low	Ecological	ASS: PCR/CR[1,2]	PCR[1,2]	
		Evolutionary	Suicide	Suicide	
	Medium	Ecological	ASS: PCR/CR or only CR[1,c]	PCR[1]	
		Evolutionary	Persistence	*Evolutionary transient to consumers > all predators*	
	High	Ecological	Only CR[1]	PCR[1]	
		Evolutionary	No persistence	Persistence	

[1] Toscano et al. in press [2] Hin et al. 2011 [a] Evolution toward maximum juvenile specialization and loss of ontogenetic omnivorous life history
[b] Changes from PR/CR to only CR with increasing cannibalism [c] Changes from PCR/CR to only CR with increasing cannibalism

A number of multi-generation laboratory experiments on LHIGP systems studied the factors that determine persistence and coexistence after invasion of *Poecilia reticulata* (Common Guppies; the intraguild predator) into food-limited populations of *Heterandria formosa* (Least Killifish; the intraguild prey) (Reichstein et al. 2013; Schröder et al. 2009*a*). Schröder et al. (2009*a*) showed that in this system invasion success of *P. reticulata* depends on the invader body size: small, juvenile *P. reticulata* do not manage to invade *H. formosa* populations, whereas large *P. reticulata* can successfully invade and after invasion drive *H. formosa* to extinction. Stable, long-term coexistence between *P. reticulata* and *H. formosa* was rarely observed by Schröder et al. (2009*a*), a result that corresponds to theoretical predictions of LHIGP systems if the intraguild predator has a diet broadening during ontogeny (Toscano et al. in press; Van de Wolfshaar et al. 2006, *this study*). The *P. reticulata* used in the experiment of Schröder et al. (2009*a*) were derived from a high predation (HP) environment. By using *P. reticulata* from a low predation (LP) environment, Reichstein et al. (in prep.) showed that weaker predation increases the possibility for coexistence, although only in the absence of spatial refuges. Invasion success still depended on invaders body size in the experiment by Reichstein et al. (in prep.) and coexistence was especially enhanced for the large invaders. Theory on LHIGP can accommodate for this result in case the *P. reticulata* from low predation environments have an enhanced competitive ability and adult/large *P. reticulata* are cannibalistic (Toscano et al. in press, *this study*). The former is indeed the case according the Reichstein et al. (in prep.). Cannibalism also occurs in *P. reticulata* (Reichstein et al. in prep.) and, based on the theory developed here and in Toscano et al. (in press), we hypothesize that the level of cannibalism in each replicate experiment might be the decisive factor that determines whether coexistence or prey extinction results.

Implications for the occurrence of cannibalism in nature

It is recognized that cannibalism is a common interaction in terrestrial and aquatic food webs (Fox 1975; Polis 1981; Polis et al. 1989; Smith and Reay 1991). This holds especially for systems with substantial body size growth, such as the life-history intraguild predation systems studied here. Our results show that non-cannibalistic LHIGP systems do not persist on evolutionary timescales because of the evolutionary suicide of non-cannibalistic intraguild predators as a result of selection on the ontogenetic trade-off in resource specialization. This mechanism thus provides an explanation for the common occurrence of cannibalism in such systems. However, since already small amount of cannibalism (relative to interspecific predation) can stabilize selection on the ontogenetic trade-off (figure 5.4 and figure 5.5), systems that might seem non-cannibalistic could potentially still be persisting because of the stabilizing effect that

results from a small degree of cannibalistic feeding. Furthermore, the incorporation of a small degree of cannibalism can lead to small changes in the population dynamics, but have a large qualitative effect on the evolutionary outcome. This study adds to the idea that cannibalism can have multiple, diverse effects on ecological and evolutionary dynamics of natural populations (Claessen et al. 2000, 2004; Dercole 2003; Getto et al. 2005; Ohlberger et al. 2012*a*; Polis 1981; Rudolf 2007; Smith and Reay 1991) and the true impact of cannibalism is therefore studied best in a specific context when a considerable amount of system-specific detail is available.

Evolution of resource specialization over ontogeny

Evolution of resource specialization has mainly been studied at an interspecifc level, addressing the question under which circumstances species evolve to be specialist or generalist (Futuyma and Moreno 1988; Levins 1962, 1963; Nurmi and Parvinen 2008, 2013; Rueffler et al. 2006*b*, 2007; Wilson and Turelli 1986). A central result from these studies is that under a weak trade-off (or a convex fitness set) generalist should evolve, while under a strong trade-off (or concave fitness set) specialists should evolve (Levins 1962; Ma and Levin 2006; Mazancourt and Dieckmann 2004; Rueffler et al. 2006*b*). However, frequency- and density-dependence have been shown to complicate this picture (Ma and Levin 2006; Mazancourt and Dieckmann 2004; Rueffler et al. 2006*b*, 2007). Resource specialization across ontogeny is much less studied (but see Ebenman 1992 and Ten Brink and De Roos 2017 for a theoretical treatment and German et al. 2014; Hjelm et al. 2000, 2003; Schluter et al. 1991 for empirical work). Ebenman (1992) finds that for strong trade-offs (corresponding to a large difference between juvenile and adult niche) selection favors juvenile specialization, at the expense of adult performance. For a weak trade-off (when niche differences are small) specialization does not occur and an intermediate phenotype evolves. In the study of Ebenman (1992) adult specialization only occurs for weak trade-offs and when the productivity of the adult niche is low in comparison with the juvenile niche. Ten Brink and De Roos (2017) show that ontogenetic niche shifts only evolve when the adult habitat is sufficiently productive and when this does not hamper juvenile performance in the original habitat. Selection even favors maintaining high juvenile growth rates with adults being maladapted to their resource (Ten Brink and De Roos 2017). Both Ebenman (1992) and Ten Brink and De Roos (2017) hence show that juvenile performance is more important than adult performance. Our study is in accordance with this view, since we find that most evolutionary stable outcomes are those with high resource specialization of juvenile intraguild predators (high a_{jr}). Adult specialization is only observed in the diet shift scenario and always occurs in combination with a cultivation effect that nullifies competition in the juvenile stage (figure 5.2 and figure 5.4).

However, in all of these cases there is an imminent risk of extinction for the predator, because the evolutionary equilibrium occurs close to the ecological persistence boundary and there exists an alternative stable CR-equilibrium.

Evolutionary suicide in a community context

Evolutionary suicide or Darwinian extinction is a process in which a population adapts in a way that compromises its own persistence (Ferrière and Legendre 2013; Parvinen 2005, 2016; Rankin and López-Sepulcre 2005; Webb 2003). It has been observed in a diversity of ecological models, but receives only little attention from empirical workers (but see Fiegna and Velicer 2003; Rankin and López-Sepulcre 2005). Parvinen (2005, 2016) distinguishes deterministic evolutionary suicide, in which selection pushes the population across a population dynamical threshold or bifurcation (*e.g.* Ferrière and Legendre 2013; Gyllenberg and Parvinen 2001; Parvinen 2005; Parvinen and Dieckmann 2013), from demographically stochastic evolutionary suicide, where selection reduces population size and this increases the likelihood of extinction due to demographic stochasticity (*e.g.* Matsuda and Abrams 1994). It was shown by Gyllenberg and Parvinen (2001) that deterministic evolutionary suicide can only occur when the population dynamical bifurcation is a discontinuous transition towards extinction (see Webb 2003 or Parvinen 2005 for an overview of such population dynamical bifurcations). A common example of evolutionary suicide is when the population evolves across a saddle-node (or fold) bifurcation towards extinction (Dercole 2003; Ferrière and Legendre 2013; Gyllenberg and Parvinen 2001; Parvinen 2005). Such a bifurcation also occurs in the diet shift scenario of the model studied here, where for low levels of cannibalism two equilibria collide and disappear with decreasing juvenile resource specialization (figure 5.3, left panels). Evolutionary suicide in the diet broadening scenario operates through a different mechanism. In this case, adaptation of the predator drives the system across a continuous transition in population dynamics, namely the invasion boundary of the consumer. Immigration of consumers then leads to an abrupt shift in ecological dynamics (attractor switching) and extinction of the predator. The latter possibility for evolutionary suicide arises because we study evolutionary dynamics of a species (the intraguild predator) in a community context, therefore allowing for alternative community attractors. Since most studies on evolutionary suicide study species in isolation, or merely accompanied by a resource, we postulate that evolutionary suicide might be much more common than currently acknowledged when evolutionary dynamics are studied in a community context.

Conclusions

Size-specific interactions prevail in natural communities and many ontogenetic omnivores are likely involved in a mixed predation/competition interaction with a specialist consumer species (Olson et al. 1995; Persson 1988; Toscano et al. 2016; Wilbur 1988). We extend the current body of theory on persistence and coexistence in these systems by studying evolutionary dynamics under an ontogenetic trade-off in feeding ability between early and late resources. We show that evolutionary suicide limits persistence of a non-cannibalistic intraguild predator, but cannibalism can lead to ecological and evolutionary stable persistence if juveniles can overcome the negative effects of competition with consumers. Our analysis shows that the requirements of species for persistence on ecological and evolutionary time scales differ and advocates for considering both processes simultaneously.

Acknowledgments

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Appendix 5.A Motivation for attack rate parameters

Similar to Hin et al. (2011) we evaluate for which values of a_{ar} and a_{jr} the predator is superior in resource competition compared to the consumer. Based on this we select three value of a_{ar} that distinguish between three qualitatively different cases.

The competitive abilities of the consumer and the predator are quantified by the resource density in the consumer-resource (CR) and predator-resource (PR) equilibrium (Tilman 1980), respectively, provided such an equilibrium exists. This is denoted by R_c^* in case of the consumer-resource equilibrium and R_p^* for the predator-resource equilibrium. The expression for R_c^* follows from solving dC/dt for R, while setting $P_a = 0$, and is given by

$$R_c^* = \frac{T_c + \mu_c}{(a_{cr}\,(\sigma - h_c\,(T_c + \mu_c))}$$

Next, we evaluate for which combinations of a_{jr} and a_{ar} the predator can persist in case $R = R_c^*$ and $C = 0$ and $\beta = 0$. The condition for predator persistence in a predator-resource equilibrium is $R_0(R_p^*) = 1$, where $R_0(R)$ is the expected lifetime reproduction of a single predator individual and given by De Roos and Persson (2013).

$$R_0(R) = \frac{\nu_a(R)}{\mu_p} z^{D_j/\nu_j(R)-1}$$

To assess the competitive ability of the predator in relation to the consumer we evaluate $R_0(R_c^*) = 1$. This results in the black solid curve that is a function of a_{jr} and a_{ar} (figure 5.1 main text). To the upper-right of this curve predators outcompete consumers ($R_p^* < R_c^*$), while at the other side consumers outcompete predators: ($R_c^* < R_p^*$). We refer to this type of competitive hierarchy as the overall superiority or inferiority of consumers and predators. Furthermore, we assess the persistence boundary for the predator as a function of a_{jr} and a_{ar} in case $R = R_{max}$ by evaluating $R_0(R_{max}) = 1$. This results in the black dashed curve in figure 5.1 (main text). At the bottom-left of this line predators cannot persist solely on the resource.

Besides the overall competitive hierarchy of consumers and intraguild predators, we also distinguish the competitive hierarchy between consumers and juvenile predators alone. To this end we propose that juvenile intraguild predators are competitively superior to consumers when they can grow in the resource density in the consumer-resource equilibrium: $\nu_j(R_c^*) > 0$. Evaluating this expression for the default parameters in table 5.2 leads to the gray solid line in figure 5.1 (main text).

Based on the different regions of competitive ability as shown in figure 5.1 (main text) we choose three values of a_{ar} that, together with changes in a_{jr}, cover all the qualitative competitive hierarchies between consumers and intraguild predators. For $a_{ar} = 0$, adult intraguild predators do not feed on the resource and the overall competitive ability of the predator is not defined. Juvenile predators can grow in the

resource equilibrium as set by consumers and are competitively superior to consumers for $a_{jr} > 3.55$, while for lower values consumers are competitively superior to juvenile predators. This case corresponds to the diet shift scenario as studied by Toscano et al. (in press). For $a_{ar} = 3$, intraguild predators can persist on the resource but they are overall competitively inferior compared to consumers irrespective of a_{jr}. Again, for $a_{jr} > 3.55$ juvenile predators are superior to consumers. Case 3: $a_{ar} = 4$, intraguild predators can persist on the resource and they are overall competitively superior to consumers for $a_{jr} > 4$. Already for $a_{jr} > 3.55$ juvenile predators are superior to consumers. The three different values are indicated in figure 5.1 (main text).

Appendix 5.B Supplementary figure

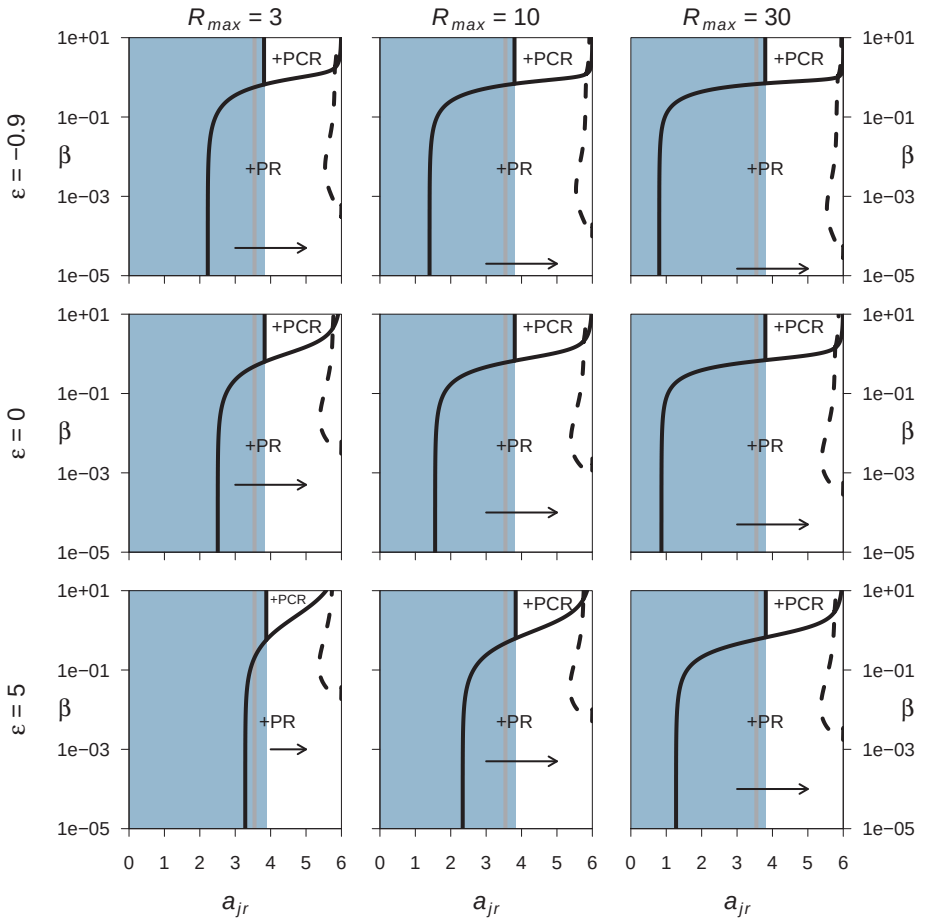

Figure 5.B1 – For $a_{ar} = 4$ intraguild predators can become overall superior in resource competition provided that a_{jr} is sufficiently large, in which case selection on the resource specialization trade-off (equation (5.1)) no longer leads to evolutionary suicide for low levels of cannibalism (β). There is still positive selection on a_{jr} (as indicated by the horizontal arrows), but predators persist in a stable PR-equilibrium at $a_{jr} = a_p = 6$ and, following equation (5.1), $a_{ac} = 0$. Higher levels of cannibalism lead to stabilizing selection on a_{jr}, as indicated by the dashed lines that show the evolutionary equilibria of a_{jr}. All evolutionary equilibria are convergence and evolutionary stable (CSSs). Even higher levels of cannibalism lead to predators persisting in coexistence with consumers. Different panels show the result of changes in productivity (R_{max}) and trade-off shape (ε). The thick black lines indicate boundaries between parameter combinations for which different types of ecological equilibria with predators occur, with the text labels in between these boundaries indicating the type of equilibrium that includes predators (+PR = PR-equilibrium, +PCR = predator-consumer-resource equilibrium). Right to the vertical gray lines the juvenile intraguild predators can grow in the resource density as set by consumers ($a_{jr} \approx 3.55$). The blue shaded parameter regions indicate the presence of a CR-equilibrium that is stable against predator invasion.

Chapter 6

Fisheries-Induced Evolution in Cannibalism Promotes Collapses of Fish Populations

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Manuscript in preparation

Abstract

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

6.1 – Introduction

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

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

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

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

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

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

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

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

6.2 – Model and method

Model description

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

The ingested food is assimilated with efficiency σ_{a}, which gives the assimilated energy $E_{\text{a}}(w) = \sigma_{\text{a}} I(w)$. Maintenance costs $E_{\text{m}}(w)$ are modeled with a power law function of body size:

$$E_{\text{m}}(w) = m_1 w^{m_2} \tag{6.6}$$

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

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

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

$$b(w) = \begin{cases} s_e \sigma_{\text{r}} (1 - \kappa) E_g(w) {w_{\text{b}}}^{-1} & \text{if } w \geq w_{\text{f}} \\ 0 & \text{otherwise} \end{cases} \tag{6.8}$$

where w_{b} is the size at birth, σ_{r} is the reproductive efficiency and s_{e} is the egg survival.

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

$$\mu_{\text{e}}(w) = \sum_{\text{i}=1}^{3} p_{\text{i}} N_v \left(l(w)|\mu_{\text{i}}^{\text{v}}, \sigma_{\text{i}}^{\text{v}}\right) \int_0^{\infty} \frac{\upsilon(y) N_c \left(l(y)|\mu_{\text{i}}^{\text{c}}, \sigma_{\text{i}}{}^{\text{c}}\right)}{1 + h(y)\gamma_{\text{tot}}(y)} n(y)\, \mathrm{d}y \tag{6.9}$$

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

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

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

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

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

Total mortality rate is then given by:

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

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

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

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

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

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

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

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

Model parameterization

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

Parameters used in the cannibalistic interaction kernel are derived from stomach content data described in Amundsen (1994); Finstad et al. (2001); Hammar (1998); Malmquist et al. (1992) and kindly provided by P. A. Amundsen and P. Byström (*pers.*

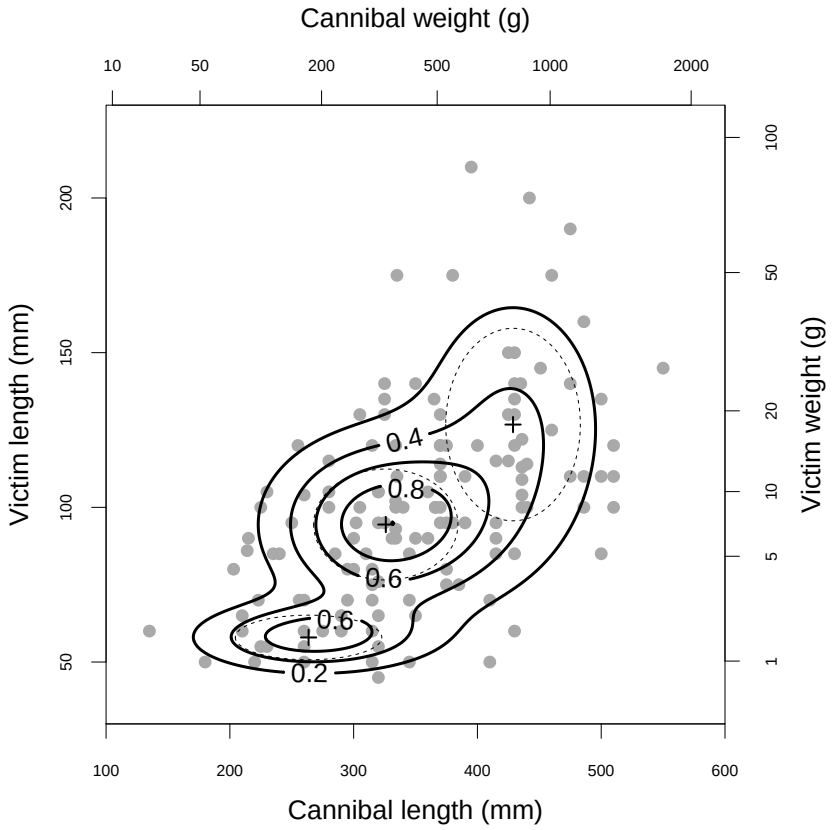

FIGURE 6.1 – Observed relationship between cannibal length (mm) and victim length (mm) for Arctic char (*Salvelinus alpinus*). Data from Malmquist et al. (1992), Hammar (1998), Finstad et al. (2001) and Amundsen (1994) and kindly provided by P. A. Amundsen and P. Byström. The '+'-symbols and dashed contour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

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

Table 6.1 – Model Parameters

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

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Table 6.1 – *Continued from previous page*

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

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

Evolutionary dynamics

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

Model analysis

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

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

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

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

6.3 – Results

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

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

Figure 6.2 (bottom panels) show that increasing the rate of cannibalism leads to lower fisheries yield as a function of fishing mortality. However, this result is dependent on the minimum size threshold for fishing (w_h). For $w_h = 10$ (figure 6.2; left panels), the highest yield is achieved when the cannibalistic rate is nearly zero ($\beta = 0.0001$) and the yield decreases with increasing β, irrespective of the fisheries-induced mortality (μ_h). For $w_h = 100$ (figure 6.2; right panels) the yield is always highest for $\beta = 0.2$, intermediate for $\beta = 1$ and $\beta = 0.0001$ and lowest for $\beta = 5$. Population extinction only occurs in case the minimum size threshold is below the size at maturation ($w_f < w_h$; figure 6.2 left panels). For a high minimum size threshold the increased fisheries-induced mortality is applied only to large individuals that have

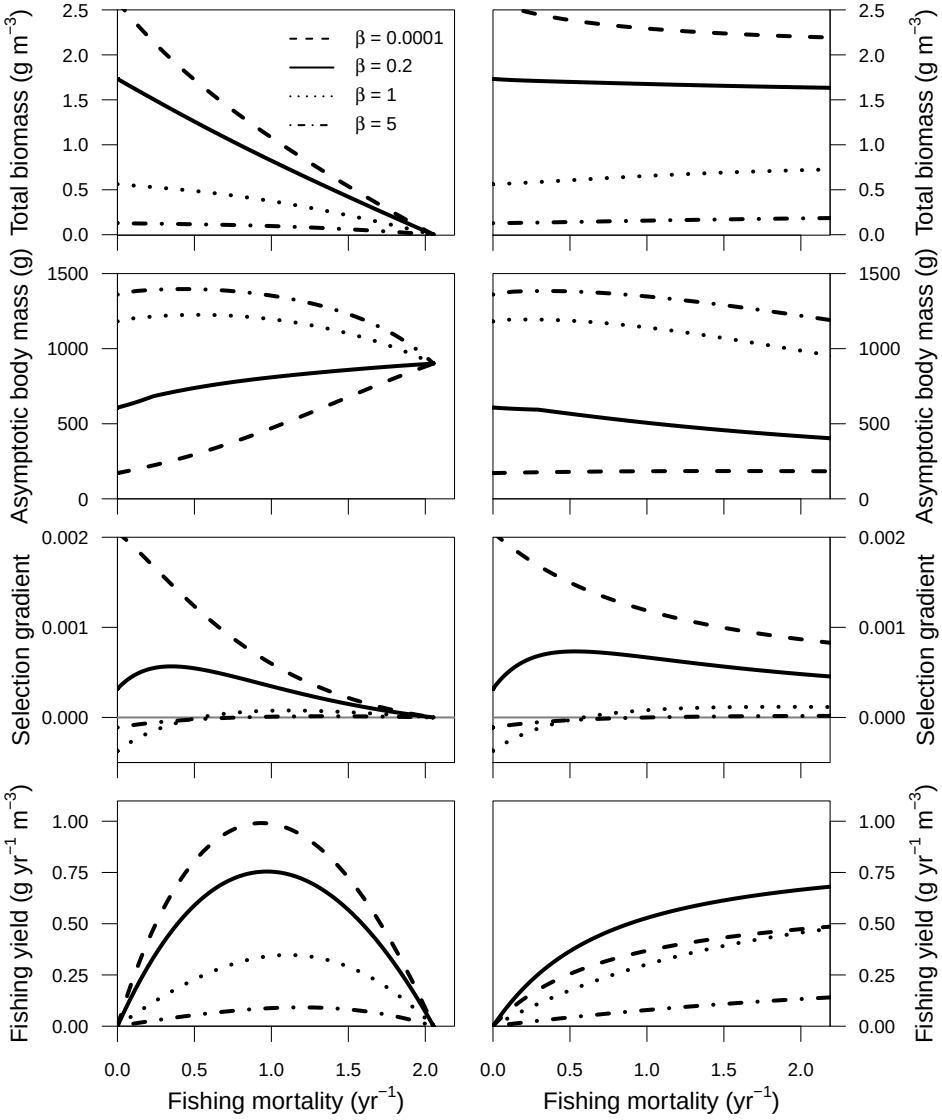

Figure 6.2 – Population equilibrium response to increasing fishing mortality (μ_h; on x-axis) mainly targeting individuals above 10 g (w_h = 10 g; left panels) or mainly targeting individuals above 100 g (w_h = 100 g; right panels). Different lines represent different levels of cannibalism (see plot legend in top left panel). Top row panels show total population biomass in g m^{-3}, second row panels show the asymptotic body mass in g, third row panels show selection gradient on the cannibalistic rate constant (β) and bottom row panels show fishing yield in g yr^{-1}m^{-3}. All other parameters are at their default value (table 6.1)

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

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

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

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

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

Figure 6.4 explores the population response to increased fishing mortality by comparing three scenarios: *i*) fixed values of size at maturation and cannibalism (left panels figure 6.4), *ii*) only evolution in the size at maturation with fixed values of the cannibalistic rate (middle panels figure 6.4), *iii*) evolution in both the size at maturation and the cannibalistic rate (right panels figure 6.4). The fixed parameters are in all cases

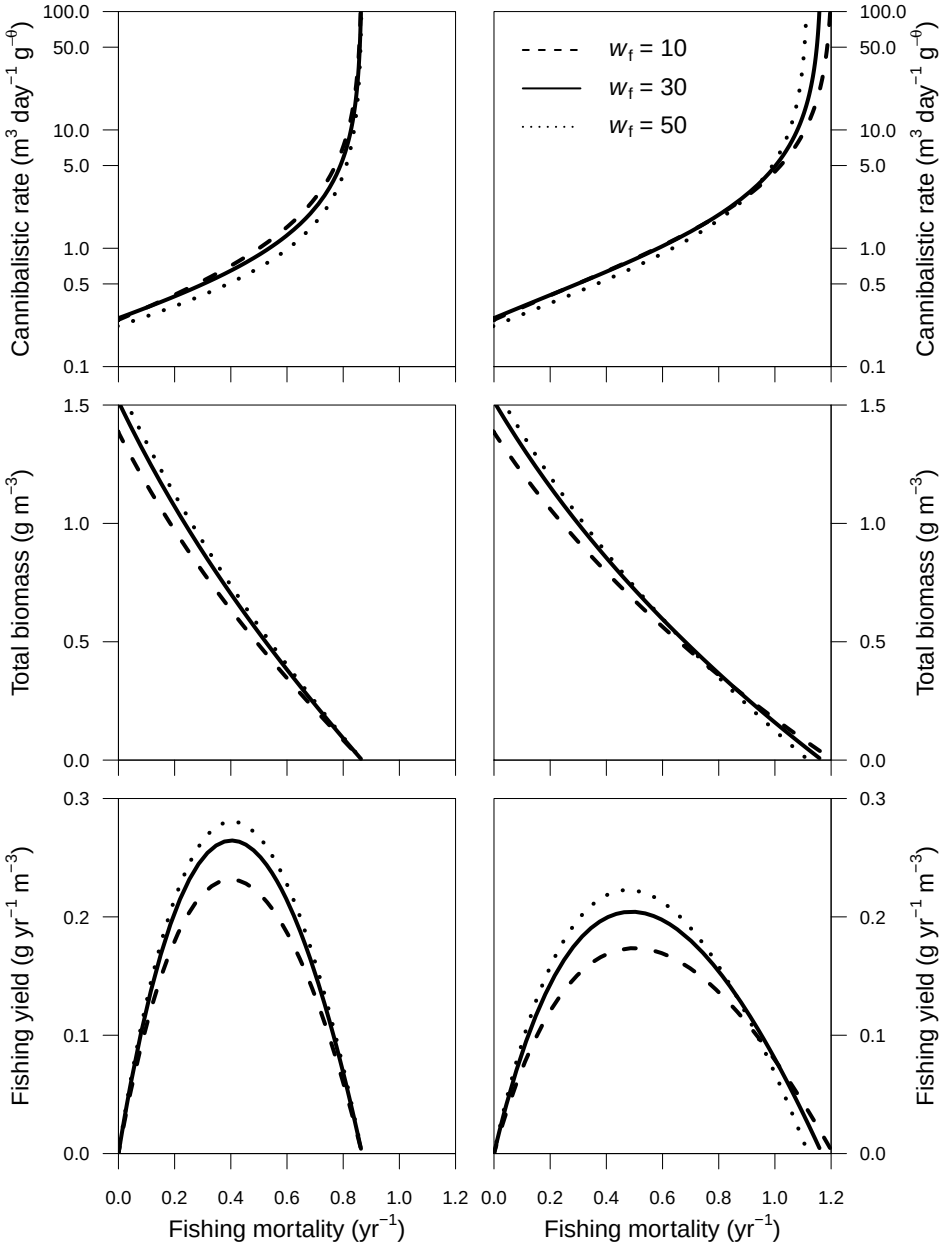

Figure 6.3 – Increasing fishing mortality (μ_h; on x-axis) leads to an increase in the evolutionary equilibrium of the cannibalistic rate constant (β), shown in top panels. Middle panels show total population biomass density and bottom panels show the fishing yield. In the left panels fishing mainly targets individuals above 10 g (w_h = 10 g) and in the right panels fished individuals are mainly above 100 g (w_h = 100 g). The different lines indicate different values for the size at maturation (w_f), shown in the legend in the top right panel.

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

6.4 – Discussion

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

Increasing fishing mortality changes the stabilizing selection on the cannibalistic rate that occurs at no or low exploitation levels into positive directional selection. This change in selection drives the rapid increase in cannibalistic rate and the concomitant decrease in population biomass. The stabilizing selection on cannibalism at low fishing mortality is a consequence of the trade-off between the gain of cannibalism

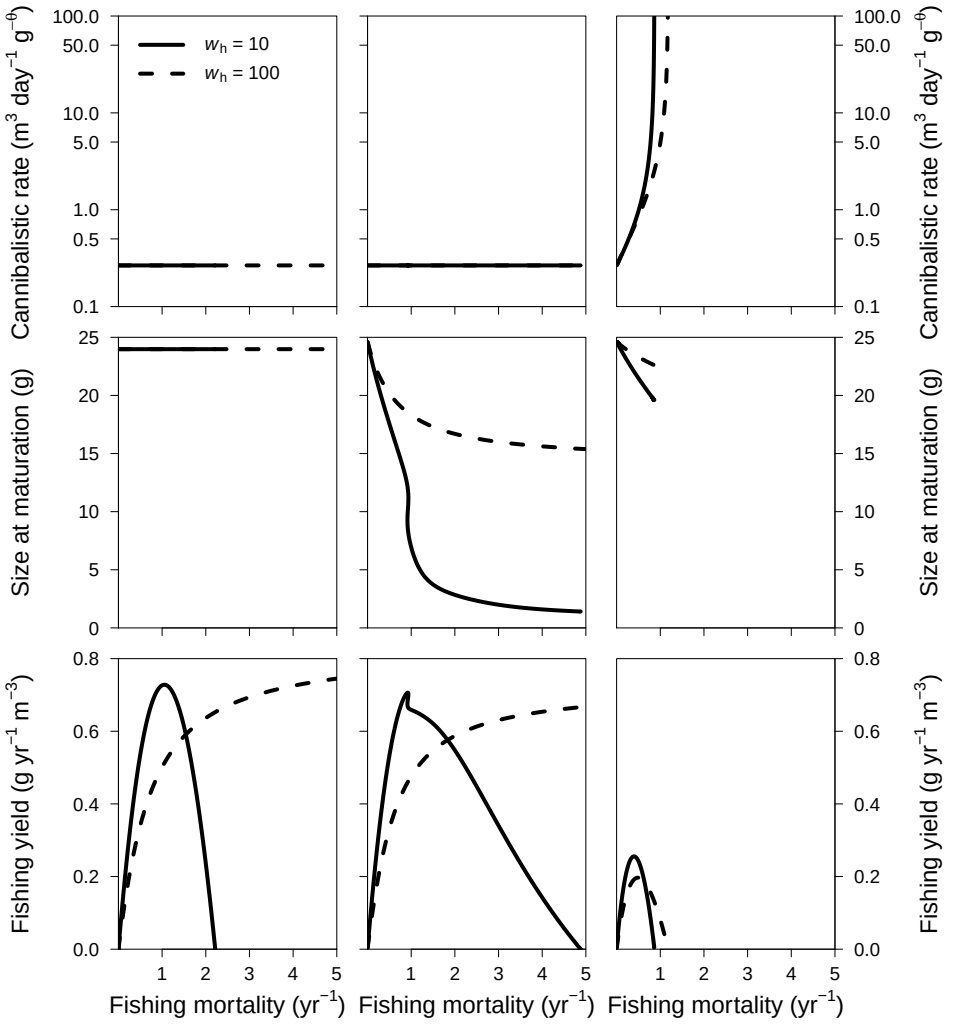

Figure 6.4 – Cannibalism evolution nullifies the evolutionary rescue effect of evolution in the maturation size threshold. Left panels show the response to increasing fishing mortality (μ_h; on the x-axis) for a fixed size at maturation and a fixed cannibalistic rate. Both parameters are fixed at their evolutionary equilibrium value for $\mu_h = 0.0$. Middle panels show the response in case of evolution only in the size at maturation with β fixed at its evolutionary equilibrium value at $\mu_h = 0.0$. Right panels show the response of joined evolution in the size at maturation and the cannibalism rate constant β. All other parameters are at their default value (table 6.1). For $w_h = 10$ there is an evolutionary rescue effect of the evolution in maturation size that delays the population extinction point to higher μ_h-values (compare solid lines in left and middle panels). This effect is nullified in case cannibalism evolution also occurs (solid lines in middle and right panels). For $w_h = 100$ (dashed lines), there is no population extinction, so there cannot be an evolutionary rescue effect. Cannibalism evolution also in this case leads to population extinction at low fishing mortality (dashed line in right panels)

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

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

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

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

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

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

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

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

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

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

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Chapter 7

General Discussion

Vincent Hin

7.1 – Ontogenetic asymmetry

Ecological theory based on structured population models has revealed an important role of ontogenetic development for the dynamics of populations and communities (De Roos and Persson 2013). Ontogenetic development of organisms includes processes such as individual body size growth (ontogenetic growth) and development of maturity. Ontogenetic development is a major life-history process in many, if not all species, and can substantially affect the type and the strength of the interaction between an individual organism and its environment. Examples of such changes in ecological interactions during ontogeny are: diet changes of individuals during ontogeny, differences in predation rates between newborn and fully grown individuals, or the ability of large individuals to better withstand resource scarcity than small individuals. Most of the changes in ecological interactions that arise from ontogenetic development have consequences for population and community dynamics (reviewed in De Roos and Persson 2013).

However, there are conditions under which ontogenetic development, through its impact on ecological interactions of individual organisms, does *not* affect population and community dynamics. These are the conditions referred to as *ontogenetic symmetry*. In case of ontogenetic symmetry, *i*) the mass-specific rate of resource ingestion, *ii*) the mass-specific rate of biomass production (MBP) and *iii*) the individual mortality rate do not change during ontogenetic development (De Roos et al. 2013). The resource ingestion rate determines an individual's impact on the environment. The rate of biomass production describes the productivity of an individual that can be used for either growth (in juveniles), or reproduction (in adults), and is therefore an important component of fitness. Any deviation from the above three conditions leads to *ontogenetic asymmetry*. In the event of ontogenetic asymmetry, ontogenetic development will lead to a change in the interaction between an organism and its environment in a way that affects population and community dynamics (De Roos et al. 2013).

Most likely, ontogenetic asymmetry plays an important role in all populations. The predominance of ontogenetic asymmetry can be derived from both individual-level observations and inferred from dynamics of natural and experimental systems. On the individual level, ontogenetic asymmetry arises from the non-linear relationships of resource ingestion and maintenance metabolism with body size. Maintenance metabolism tends to be proportional to body mass, while resource ingestion rates increase sub-linearly with size (Kooijman 2010). Consequently, adults (or large individuals) are less productive on a mass-specific basis, compared to juveniles (or small individuals). In population dynamical equilibrium, ontogenetic asymmetry in terms of low productivity for adult individuals creates an energetic bottleneck in the adult life stage (De Roos et al. 2007). Any increase in mortality then relaxes competition

among adults more than among juveniles and this leads to a disproportional increase in the productivity of adult individuals, with a concomitant increase in reproduction rate. As a result, recruitment to the juvenile stage increases and leads to an increase in juvenile biomass. The positive response of juvenile biomass to increasing mortality is a type of biomass overcompensation (De Roos et al. 2007). The phenomenon of biomass overcompensation has been observed in experimental and natural systems and is indicative of ontogenetic asymmetry in the energetics of individuals. Next to biomass overcompensation, another population-level expression of ontogenetic asymmetry are cyclic population dynamics. That many natural populations appear to show such oscillatory dynamics is an indication that ontogenetic asymmetry is likely driving the dynamics of these populations.

Overall, ontogenetic asymmetry appears to be common in natural populations (Schröder et al. 2014) and there is a good understanding about the connection between ontogenetic asymmetry and the dynamics of populations and communities (De Roos and Persson 2013). This thesis takes a next step and studies the eco-evolutionary implications of ontogenetic asymmetry. The general question is whether and how natural selection can explain the occurrence of ontogenetic asymmetry. Also, what are the evolutionary consequences of ontogenetic asymmetry for the long-term persistence of species? The previous chapters contribute in various ways to answering these questions. In this final chapter, I will summarize these results and discuss them in the context of the evolutionary origin and consequences of ontogenetic asymmetry.

7.2 – The origins of ontogenetic asymmetry

Competitive asymmetry

Chapter 2 and 3 study whether evolution can lead to ontogenetic asymmetry in consumer individuals that compete for a single food resource. The consumer energetics are described by a net-production Dynamic Energy Budget (DEB) model, with body-mass scaling exponents for energy supply (represented by maximum ingestion rate of the functional response) and energy expenditure (represented by maintenance metabolic rate). These scaling exponents determine the strength and direction of ontogenetic asymmetry. Furthermore, because all individuals feed on a shared resource, if ontogenetic asymmetry occurs, it translates into competitive asymmetry between consumer individuals of different sizes. A competitive difference between two differently-sized individuals can mean two things (illustrated in figure 7.1). Firstly, it can mean that a superior competitor can eat more per unit of body mass (it has a higher mass-specific resource ingestion rate) and, in addition, spends only little energy on maintenance (it has a lower mass-specific maintenance rate), compared to an inferior competitor. As a consequence, the superior competitor produces more biomass per unit of body mass

(it has a higher mass-specific biomass production (MBP) rate), and also requires less resources to cover its maintenance metabolism (it has a lower maintenance resource density; MRD), in relation to the inferior competitor (left panels figure 7.1). Secondly, a competitive difference can also exist when the superior competitor can eat more per unit of body mass, but also spends more energy on maintenance, than the inferior competitor. In this case, the superior competitor still produces more biomass per unit of body mass compared to the inferior competitor (has a higher MBP), but the dependence on resources for maintenance is identical to that of the inferior competitor (equal MRD; right panels figure 7.1).

Evolution of competitive asymmetry

Chapter 2 shows that evolution of ontogenetic asymmetry will only lead to the second type of competitive asymmetry (right panels figure 7.1). Therefore, the evolved outcome of ontogenetic asymmetry implies that the maintenance resource density (MRD) does not change with consumer body size. Competitive asymmetry only arises through differences in the mass-specific biomass production (MBP). Both an increase in juvenile mortality and an increase in the extent of pre-maturation growth, favor evolution towards a higher juvenile mass-specific biomass production. This adaptive response increases juvenile growth rates. Conversely, an increase in adult mortality and an increase in the extent of post-maturation growth, favor evolution towards a higher adult mass-specific biomass production. This increases growth and reproduction of adults. With this evolved form of ontogenetic asymmetry, population cycles, that are readily induced by changes in the maintenance resource density with body size (Persson et al. 1998), do not occur. Biomass overcompensation only occurs when the scaling exponents respond adaptively to increasing mortality. There is no biomass overcompensation for equal and fixed scaling exponents (chapter 2).

The evolved form of ontogenetic asymmetry (size-independent MRD and size-dependent MBP; right panels figure 7.1) is robust to the incorporation of an additional scaling exponent of energy supply (chapter 3). In chapter 3, the body-mass scaling of energy supply is controlled by two processes that can scale with body mass: the maximum ingestion rate (that was also considered in chapter 2) and the attack rate. Attack rates and maximum ingestion rates represent different processes that both control resource ingestion rates by altering the shape of the functional response. It is therefore important to consider the effect of both these processes on the evolution of ontogenetic asymmetry. With a total of three scaling exponents, the maintenance resource density and the mass-specific biomass production rate can change in more diverse ways with body size than with only two exponents. Nonetheless, the same type of competitive asymmetry evolves. Also with three scaling exponents, selection

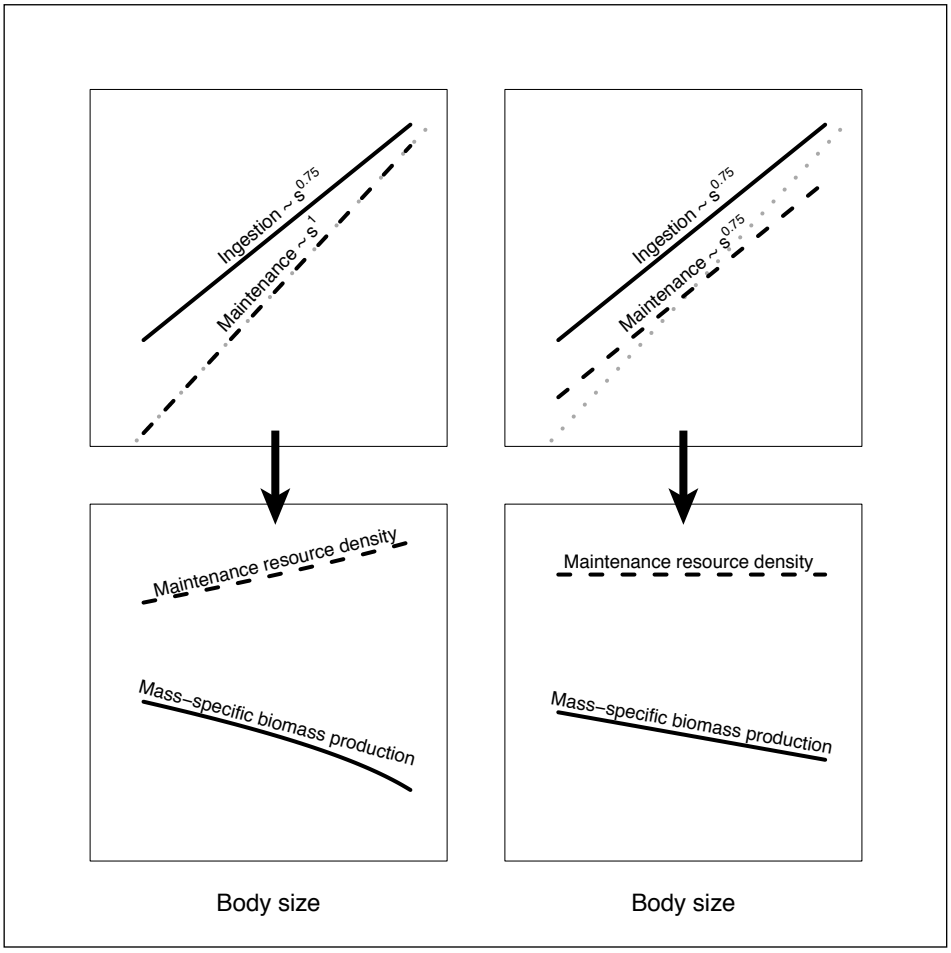

Figure 7.1 – The body-mass scalings of resource ingestion and maintenance rate (top panels) determine the body-mass scalings of the mass-specific biomass production (MBP) and the maintenance resource density (MRD). Biomass production is used for growth and reproduction and is therefore an important component of fitness. The MRD is the resource level required to cover the costs of maintenance metabolism. For resource levels below the MRD, individuals face increased mortality due to starvation. A steeper scaling of maintenance compared to ingestion (top left; as occurs in theories for ontogenetic growth Kooijman 2010; West et al. 2001), causes the mass-specific biomass production to decrease with body size, while the maintenance resource density increases with size (bottom left). In such a setting, small individuals are competitively superior compared to larger individuals, both through a lower MRD and a higher MBP. Selection leads to equal body-mass scalings of maintenance and ingestion rates (top right). This causes a decrease in the mass-specific biomass production when the scaling exponents are below one (0.75 in this case), but a constant maintenance resource density (bottom right). In this case, small individuals are only competitively superior because they have a higher MBP. Dotted gray lines in top panels indicate linear scaling (scaling exponent equals one).

leads to a size-independent maintenance resource density and a size-dependent mass-specific biomass production rate (figure 7.1; right panels).

The evolved type of ontogenetic asymmetry (figure 7.1; right panels) only comes about when all the size-dependent metabolic processes (*i.e.* maximum ingestion rate, attack rate and maintenance rate) scale in an identical, but non-linear way with body mass. The size-dependent changes in the mass-specific biomass production can explain variation in the ontogenetic scaling of metabolic rate between different species of teleost fish (chapter 2), possibly through changes in life-history processes that contribute to metabolic rate, such as growth rates. However, the evolved size independency of the maintenance resource density does not match very well with observations (chapter 2). The maintenance resource density seems to be an increasing function of body mass for at least some populations of fish (Byström and Andersson 2005; Hjelm and Persson 2001; Lefébure et al. 2014; Persson and De Roos 2006). In Daphnia, ingestion scales with a lower exponent than maintenance requirement (Gurney et al. 1990; McCauley et al. 1990). This leads to an increase in the maintenance resource density with body size. Also, the lack of biomass overcompensation from the evolved type of ontogenetic asymmetry (chapter 2), is at odds with empirical observations that biomass overcompensation does occur (Cameron and Benton 2004; Ohlberger et al. 2011; Reichstein et al. 2015; Schröder et al. 2009*b*, 2014, 2015). Summarizing, the approach adopted in chapters 2 and 3 does not contain a full story on how ontogenetic asymmetry as observed in nature evolves. It does provide a useful baseline for exploring how different types of trade-offs and more ecological complexity can lead to the evolution of the type of ontogenetic asymmetry that is readily observed in nature.

Trade-offs

Trade-offs are an insurmountable aspect of life and crucial for the outcome of evolutionary processes (Roff 1992; Zera and Harshman 2001). Simultaneously, it is notoriously hard to discover which trade-off constrains evolutionary processes most. Therefore, in many cases a certain trade-off is assumed, with limited prior knowledge about the importance of this trade-off in living systems. In chapters 2 and 3 this is not any different. It was assumed that a juvenile-adult trade-off constrains the evolution of ontogenetic asymmetry, by parameterizing the scaling constant on the body size at maturation. An increase in the scaling exponent of a metabolic process (*i.e.* maintenance rate or maximum ingestion rate), then leads to an increase in the rate of this process for adult individuals, and to a concomitant decrease for juveniles. The empirical evidence of a juvenile-adult trade-off is limited. However, such a trade-off can still be of use for studying selection on metabolic scaling. Studying the evolution of

metabolic scaling under a juvenile-adult trade-off translates into studying the relative importance of these different life stages, and their associated life-history processes (growth vs. reproduction), in determining the evolutionary process.

The use of multiple trade-offs helps to get an idea about the robustness of the obtained results. Besides the juvenile-adult trade-off, evolutionary dynamics of metabolic scaling were also studied with an energetic trade-off (chapter 3). Under this trade-off, increasing the processes related to energy supply (maximum ingestion rate and attack rate) also leads to increasing metabolic costs. Furthermore, the energetic trade-off does not constrain evolution of the maintenance rate exponent, which is therefore assumed to be fixed. Evolution of both scaling constants and scaling exponents under the energetic trade-off, reveals that the processes related to energy supply evolve towards the same scaling with body mass (chapter 3). Therefore, the energetic trade-off confirms the result that was already obtained under the juvenile-adult trade-off; optimally, attack rate and maximum ingestion rate should scale in the same way with body mass. Any evolved ontogenetic asymmetry cannot be expected to occur from scaling differences of these processes. However, the energetic trade-off allows evolution in only a single scaling exponent related to energy supply (either the maximum ingestion rate exponent or the attack rate exponent). If these processes evolve simultaneously, this leads to run-away selection towards ever increasing rates of resource ingestion (chapter 3). Therefore, the energetic trade-off cannot address the question whether evolution leads to ontogenetic asymmetry that is derived from a difference between energy supply (maximum ingestion and attack rate) and energy expenditure (maintenance metabolism).

The juvenile-adult trade-off and the energetic trade-off each have their applications and limitations. An interesting step for further research would be to combine these two trade-offs. This will allow one to separate selection that operates on the scaling exponents, from the selection that operates on the scaling constants (through the energetic trade-off). It furthermore permits the simultaneous evolution of one scaling exponent of energy supply with the scaling exponent of maintenance. Potentially, such an exercise could explain to which extent the evolution of a size-dependent mass-specific biomass production rate under a juvenile-adult trade-off, is driven by selection on the size-independent consequences that are part of changes in these scaling exponents. Because both trade-offs separately show evolution towards ontogenetic symmetry, although each within the restrictions of the parameters that can evolve, combining these trade-offs is unlikely to lead to the evolution of ontogenetic asymmetry.

Box 7.1

Increasing ecological complexity has the potential to lead to ontogenetic asymmetry when the scaling of maintenance rate is fixed at one. The model as studied in chapter 2 is adapted to include size-dependent cannibalism and size-dependent interference competition. Cannibalistic voracity of adults (β) increases mortality rates for juveniles, which becomes:

$$\mu_c + \mu_j + M\left(\frac{s}{s_r}\right)^Q \frac{\beta A}{R + \beta J + H} \tag{7.1}$$

Cannibalism furthermore increases adult resource intake, $I_A(R, J)$, which becomes:

$$I_A(R, J) = M\left(\frac{s}{s_r}\right)^Q \frac{R + \beta J}{R + \beta J + H}$$

Interference of adults negatively affects juvenile resource intake. This is modeled by an interference scalar ε. Juvenile resource ingestion becomes:

$$I_J(R, A) = M\left(\frac{s}{s_r}\right)^Q \frac{R}{R + \varepsilon A + H}$$

These size-dependent ecological interactions lead to a decrease in the evolutionary equilibrium (CSS) of the maximum ingestion scaling exponent (Q), when the maintenance exponent is constraint at one ($P = 1$). In figure 7.2, the effect of cannibalistic mortality (last term in equation 7.1), is compared with the effect of increasing juvenile mortality μ_j. Comparing left and right panels in figure 7.2, it can be concluded that the effect of interference is less pronounced than the effect of mortality.

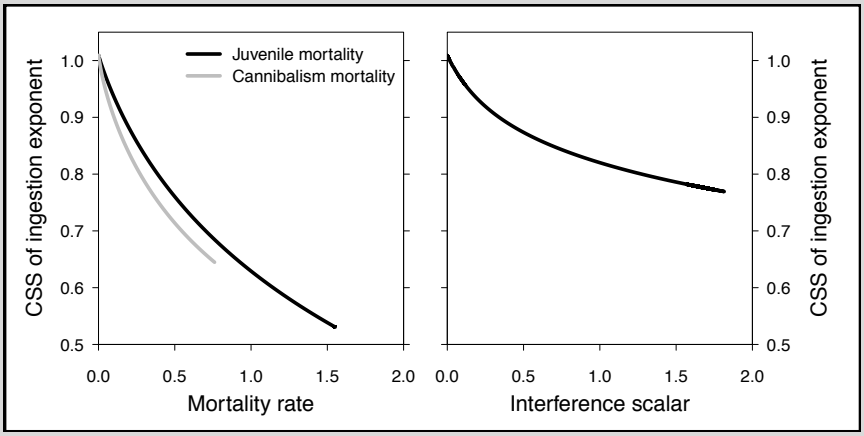

Figure 7.2 – The effect of cannibalism and interference on the evolution of the maximum ingestion exponent. Model is adapted from the model in chapter 2 (see table 2.1)

Conclusions and perspectives

Concluding, ontogenetic asymmetry in the basic ecological setting of chapters 2 and 3 probably arises from constraints on metabolic processes. Of the three different scaling processes that are explored in chapters 2 and 3 (attack rate, maximum ingestion rate and maintenance metabolism), the maintenance rate scaling is probably most constraint. A constant mass-specific maintenance rate is used in both DEB and the Metabolic Theory of Ecology (MTE) and arises from the idea that each cell (in MTE), or unit of structure (in DEB theory), requires a fixed amount of energy for maintenance (Hou et al. 2008; Kooijman 2010; West et al. 2001).

The results of chapters 2 and 3 show that when the maintenance is fixed at one, the scaling of energy supply (maximum ingestion rate and attack rate) evolves to closely match this linear maintenance scaling. This close match represents only a slight deviation from ontogenetic symmetry. Further research could study the factors that lead to an increase of this deviation. Examples are increasing juvenile mortality, size-dependent cannibalism or interference competition. In combination with a fixed maintenance scaling, such asymmetries through size-dependent ecological conditions could lead to asymmetries in the metabolic scaling processes of organisms. Preliminary explorations show that these factors can give rise to ontogenetic asymmetry in terms of a size-dependent maintenance resource density (box 7.1). In such a way, ontogenetic asymmetry evolves as a consequence of more complex ecological interactions.

7.3 – Eco-evolutionary dynamics of ontogenetic asymmetry in more complex communities

Ontogenetic asymmetry and intraguild predation

Ontogenetic asymmetry in intraguild predation (IGP) systems can arise through different ecological processes. In intraguild predation systems, an intraguild predator and an intraguild prey (or intermediate consumer) also compete for a basal resource (Polis et al. 1989). In many IGP systems, intraguild predation results from an ontogenetic diet change in the intraguild predator. The intraguild predator switches from feeding on the resource as a juvenile, to preying on consumers and, potentially, juvenile predators (cannibalism) as an adult. This interaction is referred to as life-history intraguild predation (LHIGP; Hin et al. 2011; Pimm and Rice 1987; Van de Wolfshaar et al. 2006). Competition between predators and prey is therefore restricted to the juvenile life stage of the intraguild predator. Both the ontogenetic diet change and cannibalism induce ontogenetic asymmetry in the intraguild predator. The ontogenetic diet change leads to ontogenetic asymmetry through a shift in the mass-specific resource ingestion rate at maturation. This shifts also affects the mass-specific biomass production rate. Cannibalism induces ontogenetic asymmetry because it leads to higher resource inges-

tion and production rates for adult predators and increased mortality rates of juvenile predators.

Ontogenetic asymmetry from different sources (*e.g.* cannibalism or ontogenetic diet shifts) manifests itself either as a maturation bottleneck or as a reproduction bottleneck. In population dynamical equilibrium, ontogenetic asymmetry in the intraguild predator leads to asymmetry in the productivity of the different predator life stages (De Roos and Persson 2013; De Roos et al. 2007). A high density of consumers, or high rates of cannibalism, lead to a net source of biomass in the adult stage and a net sink of biomass in the juvenile stage. Consequently, in such a population, biomass turn-over rate is regulated most by maturation (maturation bottleneck). Alternatively, low consumer density and high resource density lead to a net source of biomass in the juvenile stage, and a net loss of biomass in the adult stage. Hence, biomass turn-over in such a population is regulated most by reproduction (reproduction bottleneck; De Roos et al. 2007). The type of bottleneck largely determines the size distribution. High juvenile/adult biomass ratio is indicative of a maturation bottleneck and low juvenile/adult ratio points to a reproduction bottleneck (De Roos et al. 2007).

In fact, in absence of cannibalism there are only two possibilities for ontogenetic symmetry to occur in this system. In population dynamical equilibrium, ontogenetic symmetry occurs when there is no energetic bottleneck in the life history of the intraguild predator. For this to happen, the population maturation rate in biomass must equal the population reproduction rate in biomass (De Roos et al. 2007), which implies that both resource ingestion rates and mortality rates are equal for juveniles and adults. The first possibility for this, is when consumer and resource biomass densities are equal. This possibility for ontogenetic symmetry represents an exceptional case that cannot be predetermined, but depends on how resource and consumer densities equilibrate. The second possibility is when adult predators can still feed on the resource, because they undergo a partial diet shift (diet broadening), instead of a complete diet shift. With a diet broadening, ontogenetic symmetry arises when consumers are absent from the population dynamical equilibrium. Furthermore, adult and juvenile predators must feed with equal efficiency on the resource. This possibility only exists in the model of chapter 5, when juvenile and adult attack rates on the resource are equal.

Persistence of life history intraguild predators depends on ontogenetic asymmetry

Theoretical work on LHIGP systems shows that intraguild predators generally only persist in a reproduction regulated state, because the maturation bottleneck leads to predator exclusion (Hin et al. 2011; Van de Wolfshaar et al. 2006; Van Leeuwen et al. 2013). An ontogenetic diet shift in the intraguild predator generally leads to

two types of stable community equilibria. Predators only persist in one of these two stable states and in this state, predator biomass turn-over is generally regulated by reproduction. Due to a strong maturation bottleneck, predators cannot invade the alternative community state.

Chapter 4 shows that cannibalism in LHIGP is detrimental for the persistence of the predator, because it changes the reproduction bottleneck into a maturation bottleneck. Cannibalism in the intraguild predator has two effects: it increases biomass production in the adult stage and it increases mortality in the juvenile stage. Both these effects increase the maturation bottleneck. This results in the disappearance of the stable ecological equilibrium in which predators can persist (figure 4.3). The increased juvenile mortality is the main effect of cannibalism that disrupts predator persistence (chapter 4).

In cannibalistic LHIGP systems, intraguild predators can only persist when they can overcome the maturation bottleneck. This is for example the case when juvenile predators are superior in resource competition compared to consumers. In such a situation, cannibalism even promotes coexistence between intraguild predators and prey, because it diminishes the competitive effects of predators on consumers (chapter 4). These results show the importance of the strength and direction of ontogenetic asymmetry for the persistence of species in complex ecological communities.

Evolution of ontogenetic asymmetry in intraguild predators

Selection on resource specialization can alter the degree and direction of ontogenetic asymmetry. As argued in chapter 5, ontogenetic omnivores often face an ontogenetic trade-off in resource specialization, due to genetic constraints between different life stages. As a consequence, increased juvenile specialization on the resource leads to adults being less adapted to inter- and intraspecific predation. Vice versa, increased adult predation rates diminish foraging efficiency of juveniles on the resource. In terms of ontogenetic asymmetry, increasing juvenile specialization increases mass-specific ingestion and biomass production rates of juveniles and decreases mass-specific ingestion and biomass production rates of adults. This reinforces the reproduction bottleneck. Alternatively, increasing adult specialization strengthens the maturation bottleneck, through an increase in adult mass-specific ingestion and biomass production, and a concomitant decrease of those rates for juveniles. But does selection lead to more or less ontogenetic asymmetry in life-history intraguild predators?

If selection tends to decrease ontogenetic asymmetry, adult specialization should evolve when intraguild predators persist in a reproduction-regulated population. Conversely, juvenile specialization should evolve when intraguild predators persist in a maturation-regulated population. Because the opportunity for persistence in a maturation-regulated population is limited, we mainly expect to see evolving adult

specialization. In non-cannibalistic predators, this indeed occurs for the diet shift scenario, where intraguild predators coexist with consumers and are reproduction regulated (low juvenile/adult biomass ratio; chapter 5). However, in the diet broadening scenario, consumers are excluded from the stable community equilibrium. In absence of cannibalism, adults only feed on the resource. Therefore, there are no benefits of increasing adult specialization on consumers and no costs of increasing juvenile specialization on the resource. The community dynamics hence determine the effectiveness of the ontogenetic trade-off in constraining evolutionary change. Consequently, selection favors juvenile specialization.

Due to eco-evolutionary feedbacks, the processes that drive selection are not necessarily equal to what selection ends up achieving. Evolution can select for more ontogenetic *symmetry*, but when the ecological feedback loop changes fast enough, selection can result in more ontogenetic *asymmetry*. This happens because the indirect effect through the ecological feedback loop is opposite to the direct effect of selection (Richard 2014; Richard et al. 2015). In the diet shift scenario, selection increases adult specialization, which potentially releases the reproduction bottleneck. However, the community equilibrium responds by a decrease in consumer biomass and an increase in resource biomass. This response increases the reproduction bottleneck and counters the direct effect of selection. The combined result of the direct effect of selection (the decrease in juvenile attack rate on the resource and a concomitant increase in adult attack rate for predation) and the indirect effect of selection (decreasing consumer biomass and increasing resource biomass), is a relatively small change in ontogenetic asymmetry (juvenile/adult biomass ratio stays approximately constant). Ultimately, selection towards increasing adult specialization results in evolutionary suicide of the intraguild predator. Evolutionary suicide is probably the most convincing example of how the outcome of evolution is disconnected from the processes that drive evolutionary change (Ferrière and Legendre 2013).

To answer the question whether selection leads to more or less ontogenetic asymmetry in non-cannibalistic LHIGP systems hinges on two contingencies. First, the ontogenetic trade-off must be fully operational. In the diet broadening scenario when consumers are absent, stabilizing selection against poor adult feeding ability is missing. In the diet shift scenario, the ontogenetic trade-off is functional. In this case, selection is directed towards less ontogenetic asymmetry. However, the ecological feedback loop prevents selection from decreasing the level of ontogenetic asymmetry.

Although the direction of selection coincides with a decrease in the reproduction bottleneck, we cannot conclude that ontogenetic asymmetry is the main determinant of the strength and direction of selection on the ontogenetic trade-off. Moreover, the cannibalistic case provides an example where selection is disconnected from the type of ontogenetic asymmetry. Cannibalism changes the reproduction bottleneck to a

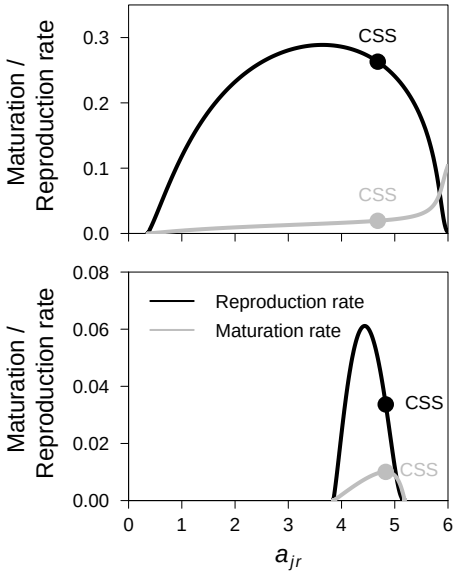

FIGURE 7.3 – Population-level biomass maturation and reproduction rates as a function of resource specialization (a_{jr}) of juvenile intraguild predators, as studied in chapter 5. Top panels are for the diet broadening scenario ($a_{\text{ar}} = 3$) and bottom panel for the diet shift scenario ($a_{\text{ar}} = 0$). All parameters as in table 5.2, in addition to $\beta = 1$. The dot indicates the position of the continuously stable strategy (CSS) for the resource specialization trade-off (equation 5.1). Each panel only shows the stable equilibrium in which the predator persists.

maturation bottleneck (chapter 4) and stabilizes selection on the ontogenetic trade-off (chapter 5). If selection on the ontogenetic trade-off corresponds directly to the type of ontogenetic asymmetry, the evolutionary equilibrium would correspond to the transition between maturation versus reproduction regulation. Figure 7.3 shows that this is not the case. Instead, selection is stabilized in a maturation regulated population. Possibly, evolution towards maturation regulation occurs because of the high benefits of cannibalism in a maturation regulated population, which is characterized by a high juvenile/adult biomass ratio.

Conclusions and perspective

Concluding, compared to the basic ecological setting as studied in chapters 2 and 3, ontogenetic asymmetry more readily evolves in the more complex ecological community of life-history intraguild predation (chapters 4 and 5). The direction of ontogenetic asymmetry has a considerable impact on persistence of intraguild predators and coexistence with intraguild prey. Ontogenetic asymmetry can drive evolutionary dynamics, but evolution does not necessarily act to minimize the extent of ontogenetic asymme-

try. This is because the indirect response of selection through the ecological feedback loop can counter the evolution of ontogenetic symmetry and because more (complex) ecological interactions (such as cannibalism) can induce evolution towards ontogenetic asymmetry. Therefore, a certain amount of ecological complexity (as in the nature and number of ecological feedbacks) seems a prerequisite for the evolution of ontogenetic asymmetry.

The evolution of cannibalism establishes a novel ecological interaction and, as such, provides a route to increased ecological complexity in simple communities. Cannibalism is an inherently size-dependent interaction (Claessen et al. 2004; Polis 1981) and therefore a good candidate to lead to the evolution of ontogenetic asymmetry in simple ecological communities (box 7.1). Furthermore, cannibalism can inhibit persistence of intraguild predators on ecological timescales (chapter 4), but also stabilize evolutionary dynamics and prevent evolutionary suicide (chapter 5). Cannibalism readily evolves in ecological models if there are not explicit disadvantages to cannibalistic feeding (Getto et al. 2005). Chapter 6 shows that cannibalism also evolves when cannibalistic feeding brings associated costs through increased mortality rates. Moreover, mortality from other sources, such as fisheries-induced mortality, promotes the evolution of cannibalism, by discounting the expected remaining lifetime of cannibals.

7.4 – Remaining questions and future directions

The above discussion shows that different levels of ecological complexity can result in different outcomes for the evolution of ontogenetic asymmetry. In a simple ecological system ontogenetic symmetry evolves, but more ecological complexity (multiple feedbacks in the ecological dynamics) can lead to the evolution of ontogenetic asymmetry. However, the influence of the nature and number of ecological feedbacks on the evolution of ontogenetic asymmetry is still very implicit. Future research can contribute to a more thorough understanding about the level of ecological complexity that is needed for ontogenetic asymmetry to evolve. Another aim is to get a more complete understanding of the conditions, under which selection leads to an increase or a decrease in ontogenetic asymmetry. Furthermore, if ontogenetic asymmetry evolves in response to increased ecological complexity, the obvious question arises how ecological complexity originates.

The most important prerequisite for studying the above questions, is a good understanding of the trade-offs and constraints that mark the boundaries of the evolutionary process. Currently, there is a focus on the variation in metabolic scaling with body mass and the possible adaptive consequences of this variation (Glazier 2005; Hirst et al. 2014). Unfortunately, there is little knowledge of the limits of this variation or about which trade-offs are most important. This knowledge will be helpful for making

further progress in understanding eco-evolutionary effects of ontogenetic asymmetry on populations and communities.

With more detailed information on the constraints and trade-offs that limit the evolution of ontogenetic asymmetry, the approach adopted in this thesis is promising for making further progress. In this thesis, complex ecological interactions are combined with evolutionary processes that shape individual-level dynamic energy budgets. This combination has not been used often (see *e.g.* Kooi and Van der Meer 2010; Troost et al. 2005), but has the potential to connect the evolution of metabolic organization and life histories to the complexities of ecological reality.

Box 7.1 shows that cannibalism can induce an evolutionary response in the scaling of ingestion rate with body mass. In turn, cannibalism readily evolves in ecological models (Getto et al. 2005), even if there are associated costs to cannibalistic feeding (chapter 6). An interesting avenue for future research is to study the coevolution between cannibalism and ontogenetic asymmetry. Is cannibalism evolution dependent on levels of ontogenetic asymmetry? And how is cannibalism influencing the selection on ontogenetic asymmetry?

Complexity does not only occur in ecological interactions. The metabolic organization of individuals is also highly complex. Although the DEB models used in this thesis are based on mass and energy conservation, they are simplified versions of metabolic organization, because they only consider a single metabolic compound (biomass). According to DEB theory (Kooijman 2010), heterotrophic organisms have at least two types of compounds, namely structural volume and energy reserves. However, the effect of adding explicit reserve dynamics only matters when considering variable food supply, as the reserve density equilibrates under constant food supply. The assumption of a constant food environment holds for most results in this thesis, with the exception of the adult-driven cohort cycles in chapter 2 (figure 2.1). Under variable food environments, periods of resource scarcity and starvation events become important. These events can be expected to considerably impact the evolution of the scaling of maintenance and ingestion with body size. Therefore, a full understanding of the origins of ontogenetic asymmetry is not complete without studying the effects of variable food supply on DEB models with explicit reserve dynamics.

Ultimately, the complexity of ecological systems with multiple feedbacks, should be integrated with the complexity of metabolic organization with multiple metabolic compounds, to answer questions about how complex life forms and ecosystems have coevolved, and how they are maintained.

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Summary

Ontogenesis: an eco-evolutionary perspective on life history complexity
In all organisms, ontogenetic development represents an essential life-history process that has major impacts on the interaction between an organism and its ecological environment. Ontogenetic development can be regarded as the collection of changes in the state of an individual that occur during its life, in terms of changes in size, shape, physiology, maturity status, or behavior. Ontogenetic development changes many ecological processes. For example, when organisms grow considerably during life, or undergo metamorphosis, small and large individuals often consume different types of food or live in different habitats. As such, ontogenetic development has consequences for both the type of ecological interactions (*e.g.* absence or presence of predation or competition) and the strength of ecological interactions (*e.g.* rates of predation or competitive ability). In turn, changes in ecological interactions during ontogenetic development have major implications for the dynamics of natural populations and communities.

However, there are conditions under which ontogenetic development, through its impact on the ecological interactions of individual organisms, does *not* affect the behavior of populations and communities. These are the conditions of *ontogenetic symmetry*. Ontogenetic symmetry describes how the strength of ecological interactions between an organism and its ecological environment, changes as the ontogenetic development of the organism unfolds. In case of ontogenetic symmetry, the change in ecological interaction strength happens in exact parity with the ontogenetic development of the organism. This creates a type of ecological *symmetry* between individuals that are at different stages of ontogenetic development. In case of a deviation from *ontogenetic symmetry*, the ecological interaction strength changes either faster, or slower, compared to the ontogenetic development of the individual. This is referred to as *ontogenetic asymmetry*. In the event of ontogenetic asymmetry, ontogenetic development will lead to a change in the ecological interaction strength of an organism, in a way that affects population and community dynamics.

The consequences of ontogenetic asymmetry for dynamics of natural populations and communities are well described, both in a theoretical and an empirical context. Furthermore, there are numerous indications that ontogenetic asymmetry pertains to most, if not all populations. However, the evolutionary aspects of ontogenetic asymmetry have not been studied. This thesis takes this step and focuses on the evolutionary origins of ontogenetic asymmetry. For this purpose, mathematical models are used that combine an accurate description of life-history processes (*i.e.* ontogenetic development, reproduction and mortality), with ecological interactions between different populations. The general question of this thesis, is whether and how evolution through natural selection will lead to ontogenetic asymmetry.

Chapter 2 and 3 describe the evolution of ontogenetic asymmetry in a simplified ecological system of a consumer species that lives of a single type of food (*i.e.* resource). Consumer individuals take up and assimilate food to meet the costs of metabolism. On top of that, they can invest energy in growth (both juveniles and adults are assumed to grow) and reproduction (only in case of adults). Because all consumer individuals compete for the single resource, ontogenetic asymmetry leads to a difference in competitive ability between individuals at different stages of ontogenetic development. A good competitor can take up and assimilate resources fast and also requires little energy for maintenance. Therefore, good competitors can spend a lot of energy on growth and reproduction, and this increases their fitness. A poor competitor has a low rate of resource uptake and high maintenance costs. When poor competitors have too little energy for maintenance, their mortality risk increases (starvation) and this leads to low fitness. Through a trade-off it is assumed that a good competitive ability in the juvenile phase, leads to poor competitive ability in the adult phase, and vice versa.

In chapter 2 and 3 it is shown that in this simplified setting, evolution of ontogenetic asymmetry neutralizes strong competitive differences. With the evolved type of ontogenetic asymmetry, individuals at different stages of development (*e.g.* juveniles versus adults), all require the same amount of food to meet their maintenance costs. Consequently, consumer individuals never suffer from starvation. However, differences in competitive ability do arise through differences in growth and reproduction rates. When either the juvenile phase of the life cycle, or juvenile mortality is increased, selection increases juvenile fitness (*i.e.* juvenile growth), at the expense of adult fitness (*i.e.* adult growth and reproduction). Vice versa, an extension of the adult phase of the life cycle, or increased adult mortality, leads to higher adult fitness, and lower juvenile fitness. However, this adaptive response is such that it does not lead to starvation in any part of the life cycle.

The evolved type of ontogenetic asymmetry does not match well with observations from nature. In many natural populations, individuals require different resource levels

to cover their maintenance metabolism. Accordingly, strong competition between individuals in different life stages can induce starvation events. Concluding, the simple ecological setting as studied in chapter 2 and 3 does not explain the type of ontogenetic asymmetry that is observed in nature.

In chapter 4 and 5 it is studied whether the more complex ecological setting of life-history intraguild predation gives rise to the evolution of ontogenetic asymmetry. Intraguild predation describes the mixed predation/competition interaction between a predator and a prey species. Juvenile predators compete with the prey for a shared food source, while adult predators feed on the prey and, in addition, can cannibalize juvenile predators. The shift in diet from resource feeding to predation, implies a change in the type of ecological interaction and this leads to ontogenetic asymmetry. Cannibalism is another source of ontogenetic asymmetry, because it provides a food source for adult predators and leads to higher mortality for juveniles. Taking together the effects of cannibalism and diet shifts can lead to two types of ontogenetic asymmetry in the predator population when it is in equilibrium (*i.e.* population density does not change over time). Either the predator population becomes maturation-regulated, characterized by low juvenile growth rates and high juvenile mortality. Or the population becomes reproduction-regulated, characterized by low adult reproduction and high adult mortality. These two types are separated by ontogenetic symmetry, in which the predator population is neither reproduction, nor maturation regulated.

In chapter 4 it is shown that cannibalism is detrimental for the persistence of the intraguild predator, because it changes the ontogenetic asymmetry from reproduction-regulation into maturation-regulation. In case of maturation-regulation, competition of juvenile predators with consumers becomes too severe for stable predator persistence. Therefore, cannibalism leads to ecological extinction of predators by changing the type of ontogenetic asymmetry.

Chapter 5 describes the evolution of ontogenetic asymmetry in the intraguild predator, dependent on the level of cannibalism. In chapter 5 it is assumed that predators can evolve to increase resource feeding rates of juveniles (which decreases maturation regulation), or increase predation rates of adults (which decreases reproduction regulation). An ontogenetic trade-off between the life stages prevents simultaneous increase in resource feeding and predation rates. In absence of cannibalism, selection on this ontogenetic trade-off leads to an increase in specialization of one life stage, at the expense of feeding performance in the other life stage. Ultimately, increasing one type of specialization causes a shift in the community dynamics to a state in which predators can no longer persist. Consequently, selection on the ontogenetic trade-off in absence of cannibalism leads to evolutionary suicide of the intraguild predator. Cannibalism, however, prevents evolutionary suicide by stabilizing the selection on the ontogenetic trade-off in resource specialization.

In the more complex ecological setting of intraguild predation, ontogenetic asymmetry is also determined by the densities of consumers and resources. Selection on ontogenetic asymmetry leads to an ecological feedback on consumer and resource density. This feedback acts in opposite direction to the forces that drive selection (*i.e.* the amount and direction of ontogenetic asymmetry). Consequently, selection can act to decrease ontogenetic asymmetry, but due to the feedback in the ecological dynamics, selection might not be successful in doing so, or instead, even lead to more ontogenetic asymmetry. Furthermore, cannibalism can induce selection towards ontogenetic asymmetry, because the fitness benefits of cannibalism are greater when the population is in a maturation-regulated state. This is because juvenile density is high in such a state.

Concluding, in intraguild predation systems, the ecological persistence of predators depends crucially on the direction of ontogenetic asymmetry (chapter 4). Furthermore, selection of ontogenetic asymmetry can have unanticipated effects (evolutionary suicide; chapter 5). Increased ecological complexity through cannibalism can stabilize evolutionary dynamics and lead to ontogenetic asymmetry (chapter 5). Comparing these outcomes with the results described in chapter 2 and 3, shows that a certain amount of ecological complexity (as in the number and nature of ecological feedback loops) seems a prerequisite for the evolution of ontogenetic asymmetry.

The evolution of cannibalism can establish a novel ecological interaction and, as such, provides a route to increased ecological complexity in simple communities. Furthermore, cannibalism can inhibit persistence of intraguild predators on ecological timescales (chapter 4), but cannibalism can also stabilize evolutionary dynamics and prevent evolutionary suicide (chapter 5). It is therefore important to understand the conditions that inhibit or promote the evolution of cannibalism. Chapter 6 addresses this topic in the more applied and practical context of fisheries-induced evolution. A model for the population dynamics of cannibalistic Arctic char (*Salvelinus alpinus*), shows that fisheries-induced mortality promotes the evolution of cannibalism. Under low rates of mortality, cannibalism evolution is stabilized by the mortality costs associated with cannibalistic feeding. However, fisheries-induced mortality changes the stabilizing selection into positive directional selection to ever increasing rates of cannibalism. This leads to a double effect of mortality on the population. The fisheries-induced mortality decreases population biomass directly, but also selects for even higher rates of cannibalism, which further reduces population density.

Overall, this thesis combines complex ecological interactions with evolutionary processes that shape individual life histories. This combination has not been used often, but has the potential to provide insights on how complex life forms and ecosystems have coevolved and how they are maintained.

Samenvatting

Ontogenese: de ecologie en evolutie van complexe levensontwikkeling
Ontogenetische ontwikkeling is een essentieel proces in het leven van alle organismen en bepaalt in belangrijke mate de interactie tussen organismen en hun ecologische omgeving. Ontogenetische ontwikkeling kan worden beschouwd als de verzameling van veranderingen in afmeting, vorm, fysiologie, levensfase en gedrag, die plaatsvinden gedurende het leven van een organisme. Deze vorm van ontwikkeling is van grote invloed op allerlei ecologische processen. Zo leidt bijvoorbeeld groei in lichaamsgrootte of metamorfose vaak tot veranderingen in het dieet of habitat van het organisme. Op deze manier beïnvloedt ontogenetische ontwikkeling zowel de aard van de ecologische interactie (zoals de aanwezigheid van predatoren of concurrenten), als de sterkte van de ecologische interactie (de predatiedruk of de sterkte van competitie). Veranderingen in ecologische interacties als gevolg van ontogenetische ontwikkeling hebben grote gevolgen voor de dynamiek van natuurlijke populaties en levensgemeenschappen.

Er zijn echter bepaalde omstandigheden waarbij ontogenetische ontwikkeling, ondanks haar invloed op ecologische interacties van individuele organismen, geen effect heeft op de dynamiek van natuurlijke populaties en levensgemeenschappen. Onder zulke omstandigheden verkeert de populatie in een toestand van ontogenetische symmetrie. In het geval van ontogenetische symmetrie is de verandering in de sterkte van de ecologische interactie precies parallel aan de ontogenetische ontwikkeling van het organisme. Op deze manier ontstaat er een ecologische symmetrie tussen individuen die in verschillende stadia van hun ontogenetische ontwikkeling verkeren. Bij een afwijking van ontogenetische symmetrie neemt de sterkte van de ecologische interactie sneller toe of af, vergeleken met de ontogenetische ontwikkeling van het organisme. Dit wordt ook wel ontogenetische asymmetrie genoemd. Bij ontogenetische asymmetrie zorgt de ontogenetische ontwikkeling dus voor een verandering in de sterkte van de ecologische interactie, op een manier die van invloed is op de dynamiek van natuurlijke populaties en levensgemeenschappen.

Er zijn tal van aanwijzingen dat ontogenetische asymmetrie geldt voor de meeste, zo niet alle, populaties, en de gevolgen van ontogenetische asymmetrie op de dynamiek van populaties en ecosystemen worden tegenwoordig goed begrepen. De evolutionaire aspecten van ontogenetische asymmetrie zijn echter minder goed onderzocht. Het onderzoek in dit proefschrift richt zich daarom op de evolutionaire oorsprong en gevolgen van ontogenetische asymmetrie. Hiertoe worden wiskundige modellen gebruikt die een beschrijving van verschillende levensprocessen (bijv. ontogenetische ontwikkeling, reproductie en mortaliteit), combineren met een beschrijving van de ecologische interacties tussen verschillende populaties. De overkoepelende vraag van dit proefschrift is of, en op welke manier, evolutie door middel van natuurlijke selectie leidt tot ontogenetische asymmetrie.

In hoofdstuk 2 en 3 wordt de evolutie van ontogenetische asymmetrie in een gesimplificeerd ecosysteem onderzocht. Dit systeem bestaat uit een heterotrofe consument, die zich voedt met een enkele voedselbron. Consumenten gebruiken de energie van de voedselbron voor hun basale metabolisme. Bovenop de energetische kosten van het metabolisme investeren consumenten energie in lichaamsgroei (zowel juveniele als adulte consumenten kunnen groeien) en reproductie (alleen in het geval van adulten). Omdat alle consumenten afhankelijk zijn van dezelfde voedselbron, leidt ontogenetische asymmetrie tot concurrentieverschillen tussen individuen die in verschillende stadia van hun ontogenetische ontwikkeling verkeren. Een sterke concurrent kan snel voedsel opnemen en is weinig energie kwijt aan het basale metabolisme. Hierdoor kan een sterke concurrent veel energie besteden aan groei en reproductie, hetgeen de biologische fitness verhoogt. Een minder sterke concurrent neemt voedsel langzaam op en besteedt veel energie aan het basale metabolisme. Hierdoor kan een mindere concurrent weinig energie besteden aan groei en reproductie. Tevens kan te weinig energie zorgen voor een verhoogde sterftekans, wanneer er niet aan de kosten van het basale metabolisme wordt voldaan. Op deze manier hebben zwakke concurrenten een lage biologische fitness. Er wordt verder aangenomen dat er een trade-off bestaat tussen de concurrentiekracht in de juveniele fase en die in de adulte fase. Hierdoor leidt een verhoging van de concurrentiekracht in de juveniele fase, tot een verlaging van de concurrentiekracht in de adulte fase, en andersom.

In hoofdstuk 2 en 3 wordt beschreven dat in deze gesimplificeerde ecologische setting de evolutie van ontogenetische asymmetrie de sterke concurrentieverschillen in de populatie neutraliseert. Het type ontogenetische asymmetrie dat hierbij ontstaat zorgt ervoor dat individuen in verschillende fase van ontogenetische ontwikkeling (zoals juvenielen en adulten) dezelfde hoeveelheid voedsel nodig hebben voor hun basale metabolisme. Daardoor treedt er geen verhoogde mortaliteit op als gevolg van voedseltekort. Concurrentieverschillen zullen echter blijven bestaan door verschillen in groei- en reproductiesnelheid. Wanneer ofwel de lengte van de juveniele fase,

ofwel de juveniele mortaliteit verhoogd wordt, verhoogt selectie de juveniele fitness (juveniele groeisnelheid), ten koste van de adulte fitness (adulte groei- en reproductiesnelheid). Andersom zal een verlenging van de adulte fase, of een verhoging van de mortaliteit onder adulten, leiden tot selectie voor verhoogde adulte fitness, ten koste van de juveniele fitness. Deze evolutionaire respons leidt echter in geen enkel deel van de levenscyclus tot extra mortaliteit als gevolg van voedseltekort.

Dit geëvolueerde type van ontogenetische asymmetrie komt echter niet goed overeen met observaties uit de natuur. In veel natuurlijke populaties verschillen individuen uit verschillende levensstadia in de hoeveelheid voedsel die ze nodig hebben voor hun metabolisme. In zulke populaties zorgt competitie tussen individuen uit verschillende levensstadia voor verhoogde mortaliteit. De simpele ecologische setting, zoals beschreven in hoofdstuk 2 en 3, kan dus niet de ontogenetische asymmetrie van natuurlijke populaties goed verklaren.

In hoofdstuk 4 en 5 wordt onderzocht of ontogenetische asymmetrie evolueert in de complexere ecologische setting van leeftijdsafhankelijke omnivorie. Er is sprake van omnivorie wanneer de predator, naast het prederen op de prooi, ook concurreert met de prooi om dezelfde voedselbron. Bij leeftijdsafhankelijke omnivorie beperkt deze competitie zich tot de juveniele levensfase van de predator, terwijl de predatie alleen plaatsvindt in de adulte levensfase van de predator. Daarnaast prederen adulte predatoren ook op hun eigen juvenielen (kannibalisme). De transitie van competitie (als juveniel) naar predatie (als adult) gedurende het leven van de predator, impliceert een verandering in de ecologische interactie en dit leidt tot ontogenetische asymmetrie. Ontogenetische asymmetrie ontstaat ook door kannibalisme van adulte predatoren, omdat het kannibalisme zowel een voedselbron voor adulten vormt als zorgt voor verhoogde mortaliteit onder juvenielen. Wanneer de populatie in evenwicht is (d.w.z. de populatiedichtheid verandert niet door de tijd) kunnen dieetverandering en kannibalisme zorgen voor twee soorten ontogenetische asymmetrie. De predatorpopulatie wordt ofwel gereguleerd door maturatie, met een lage juveniele groeisnelheid en hoge juveniele mortaliteit, ofwel gereguleerd door reproductie, met een lage reproductiesnelheid en hoge adulte mortaliteit. Ontogenetische symmetrie begrenst deze twee manieren van populatieregulatie, waarbij de predatorpopulatie noch door reproductie, noch door maturatie wordt gereguleerd.

In hoofdstuk 4 wordt beschreven dat kannibalisme nadelig is voor het voortbestaan van de predator, omdat het zorgt voor een transitie van reproductie- naar maturatieregulatie. In het geval van maturatieregulatie is de competitie tussen juveniele predatoren en prooien te sterk om het voortbestaan van de populatie veilig te stellen. Kannibalisme leidt dus tot het plaatselijk uitsterven van de predator door een verandering in het type ontogenetische asymmetrie.

In hoofdstuk 5 wordt de evolutie van ontogenetische asymmetrie bij de (omnivore) predator bestudeerd, afhankelijk van het niveau van kannibalisme. In hoofdstuk 5 wordt aangenomen dat evolutie, ofwel de concurrentiekracht van juveniele predatoren verhoogt (dit verlaagt de maturatieregulatie), ofwel de predatiedruk van adulte predatoren verhoogt (dit verlaagt de reproductieregulatie). Een trade-off tussen de verschillende levensfasen verhindert echter dat beide processen gelijktijdig toenemen. Wanneer er geen sprake is van kannibalisme, zal selectie op deze ontogenetische trade-off zorgen voor de specialisatie van een enkel levensstadium (ofwel juvenielen ofwel adulten specialiseren zich op hun voedselbron). Dit gaat ten koste van de mate van specialisatie binnen het andere levensstadium (respectievelijk het adulte of juveniele stadium). Uiteindelijk leidt dit tot een verandering in het ecologische evenwicht en tot het uitsterven van de predator. Selectie op de ontogenetische trade-off zorgt dus voor evolutionaire suïcide van de predator. Kannibalisme kan dit echter voorkomen door de selectie op de ontogenetische trade-off te stabiliseren.

In de complexere ecologische setting van leeftijdsafhankelijke omnivorie wordt de ontogenetische asymmetrie bepaald door de dichtheid van prooien en voedselbron. Selectie op ontogenetische asymmetrie zorgt via een ecologisch terugkoppelingsmechanisme voor veranderingen in de dichtheid aan prooien en voedselbron. De ecologische terugkoppeling werkt echter in tegengestelde richting ten opzichte van de selectiedruk. Hierdoor kan selectie die erop gericht is om de ontogenetische asymmetrie te verminderen, via de ecologische terugkoppeling leiden tot een toename van ontogenetische asymmetrie. Daarnaast kan kannibalisme leiden tot selectie voor ontogenetische asymmetrie, omdat de fitnessopbrengsten van kannibalisme groter zijn in een maturatie-gereguleerde populatie. Dit komt doordat in dit geval de juveniele dichtheid hoog is.

Samengevat, in systemen met leeftijdsafhankelijke omnivorie is het ecologische voortbestaan van predatoren afhankelijk van het type ontogenetische asymmetrie (hoofdstuk 4). Verder leidt selectie op ontogenetische asymmetrie tot onverwachte effecten (evolutionaire suïcide; hoofdstuk 5). Een toename van ecologische complexiteit, door de aanwezigheid van kannibalisme, stabiliseert de evolutionaire dynamiek en leidt tot ontogenetische asymmetrie (hoofdstuk 5). Indien men deze resultaten vergelijkt met de resultaten van hoofdstuk 2 en 3, dan lijkt een bepaalde mate van ecologische complexiteit (d.w.z. het aantal ecologische terugkoppelingen) een voorwaarde voor de evolutie van ontogenetische asymmetrie.

De evolutie van kannibalisme kan leiden tot een nieuwe ecologische interactie, en op deze manier bijdragen aan een toename van complexiteit in simpele ecologische gemeenschappen. Kannibalisme verhindert het voortbestaan van omnivoren op ecologische tijdschaal (hoofdstuk 5), maar stabiliseert ook de evolutionaire dynamiek en het voorkomt evolutionaire suïcide (hoofdstuk 5). Daarom is het belangrijk om

te begrijpen welke omstandigheden de evolutie van kannibalisme remmen of juist bevorderen. Hoofdstuk 6 bestudeert dit onderwerp in de toegepaste context van visserij-geïnduceerde evolutie. Een model voor de populatiedynamiek van kannibalistische trekzalm (*Salvelinus alpinus*) laat zien dat visserij-geïnduceerde mortaliteit de evolutie van kannibalisme bevordert. Bij een lage mortaliteit wordt de evolutie van kannibalisme gestabiliseerd door de negatieve gevolgen van kannibalisme. Echter, bij een verhoging van de visserijdruk verandert deze stabiliserende selectie naar directionele selectie voor toenemende kannibalistische predatiedruk. Dit zorgt voor een tweeledig effect van mortaliteit. De visserij-geïnduceerde mortaliteit verlaagt direct de populatiedichtheid, maar selecteert tevens voor hogere predatiedruk door kannibalisme, wat zorgt voor een verdere afname van de populatiedichtheid.

In dit proefschrift wordt de bestudering van complexe ecologische interacties gecombineerd met de bestudering van evolutionaire processen die de levensontwikkeling van soorten bepalen. Deze benadering wordt nog niet veel gebruikt, maar levert mogelijk belangrijke inzichten op over hoe complexe levensvormen en ecosystemen zijn geëvolueerd en hoe deze blijven voortbestaan.

Author Contributions

2 **Evolution of Size-Dependent Intraspecific Competition Yields Paradoxical Predictions on the Scaling of Metabolism with Body Size**
Vincent Hin and André M. de Roos
VH and AMdR designed the research. VH analyzed the model and wrote first version of manuscript. VH and AMdR contributed to later versions of manuscript.

3 **Evolution of Metabolic Scaling**
Vincent Hin and André M. de Roos
VH and AMdR designed the research. VH analyzed the model and wrote first version of manuscript. VH and AMdR contributed to later versions of manuscript.

4 **Cannibalism and Intraguild Predation Community Dynamics: Coexistence, Competitive Exclusion and the Loss of Alternative Stable States.**
Benjamin J. Toscano, Vincent Hin and Volker H. W. Rudolf
BJT and VHWR designed the research. VH and BJT analyzed the model. BJT wrote first version of manuscript. BJT, VH and VHWR contributed to later versions of manuscript

5 **Cannibalism Prevents Evolutionary Suicide of Ontogenetic Omnivores in a Life History Intraguild Predation System**
Vincent Hin and André M. de Roos
VH designed the research, analyzed the model and wrote first version of manuscript. VH and AMdR contributed to later versions of manuscript.

6 **Fisheries-Induced Evolution in Cannibalism Promotes Collapses of Fish Populations**
Vincent Hin, André M. de Roos and Ulf Dieckmann
VH and UD designed the research. VH analyzed the model and wrote first version of manuscript. VH and AMdR contributed to later versions of manuscript.

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