The role of dispersal in life history and population dynamics: an experimental and theoretical approach

A thesis submitted for the degree of Doctor of Philosophy

by

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Declaration

I declare that this thesis was composed by myself and that the work contained herein is my own except where explicitly stated in the text. This work has not been submitted for any degree or professional qualification except as specified.

Jacques Deere, Trinity Term 2015

Abstract

The role of dispersal in life history and population dynamics: an experimental and theoretical approach – D.Phil. thesis by Jacques A. Deere

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Understanding the evolution and maintenance of phenotypic and genetic variation within populations is a key challenge in population biology. Discrete phenotypic variation such as alternative reproductive strategies and dispersal strategies are extreme forms of this. To understand phenotypic variations, such as male dimorphisms and dispersal expression, requires investigating the costs and benefits of these different phenotypes. In this thesis I do so. First, at the individual level, I determine trade-offs between life-history traits and phenotypic expression. The influence of male morph expression is assessed by determining whether male morph survival is frequency-dependent. Costs of dispersal expression were assessed by comparing individuals that expressed the dispersal phenotype during their ontogeny with individuals that did not. Second, at the population level, I specifically investigate costs of phenotype expression to the natal population when dispersers fail to disperse. To determine any demographic costs to natal populations, I used structured integral population models to calculate population biology quantities, which I compared between populations that produced dispersers that fail to disperse and populations that produced no dispersers. I show that expressing a dispersal morph is costly to life-history traits and skews the male morph

ratio, indicating that these two conditional strategies interact during ontogeny. This questions whether current models explaining single conditional strategies, such as the environmental threshold model, should consider interactions between different conditional strategies. In natal populations where dispersal is expressed, but dispersers fail to disperse, populations suffer reduced fitness and this demographic cost is enhanced in stochastic environments. These results do not include benefits of successful dispersal or other costs such as inbreeding. However, they do provide a cost of dispersal expression which indicates what the benefits of dispersal would need to be for dispersal to evolve. One aspect that the results do not inform on is possible eco-evolutionary dynamics in populations. Future work should look to incorporate eco-evolutionary feedback, within a metapopulation structure, to identify the maintenance and evolution of male dimorphism and dispersal.

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Author contributions

Chapter 1

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Chapter 2

Authors: Jacques A. Deere (JAD) and Isabel M. Smallegange (IMS)

JAD and IMS conceived the research question. JAD conducted the experiments, the analyses and wrote the manuscript. IMS gave general guidance with advice on the experimental design and statistical analyses. All co-authors contributed to the interpretation of the results and commented on the manuscript.

Chapter 3

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JAD and IMS conceived the research question. JAD conducted the experiments, the analyses and wrote the manuscript. IMS and TC gave general guidance and advice on the statistical analyses. All co-authors contributed to the interpretation of the results and commented on the manuscript.

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JAD, IMS and TC conceived the research question. JAD conducted the analyses and wrote the manuscript. IMS and TC advised on the construction of the model which is an extension of a similar model by IMS (IMS provided the MATLAB code for translation and extension into the statistical programming language R by JAD). IMS, SC and TC gave general guidance and advice on the statistical analyses. All co-authors contributed to the interpretation of the results and commented on the manuscript.

Chapter 5

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JAD and IMS conceived the research question. JAD conducted the analyses and wrote the manuscript. IMS advised on the construction of the model and provided MATLAB code of a similar model for translation and extension into the statistical programming language R by JAD. IMS and TC gave general guidance and advice on the statistical analyses. All co-authors contributed to the interpretation of the results and commented on the manuscript.

Chapter 6

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Chapter 1

Introduction

It has long been recognised that life-history plays an integral role in biology and in understanding the diversity and complexity of organisms that we see (Stearns 1992; Roff 2002). Individuals vary in their life-history; this variation is caused by individual characteristics and environmental effects including abiotic variation and interspecific and intraspecific competition. Selection can act on any phenotypic variation. Whether it generates an evolutionary response requires that at least some of it is genetic, then evolution can lead to the existence of individual characteristics such as polymorphisms (Stearns 1992; Andersson 1994; Roff 1996). Strong male-male competition, for example, can lead to alternative reproductive phenotypes (ARPs) (Gross 1996) whereas spatio-temporal variation in environmental conditions contributes to the evolution of distinct dispersal strategies/morphologies (Clobert et al. 2001; Clobert et al. 2009; Bonte et al. 2012; Cotto et al. 2014). The challenge is to determine how these complex life-histories (e.g. ARPs, dispersal), and possibly also their interactions, affect population dynamics. For example, evolution of the expression of alternative phenotypes affects population structure and dynamics (Piou and Prevost 2013; Smallegange and Deere 2014). Dispersal, in turn, changes the structure, and therefore dynamics, of populations (Stenseth and Lidicker 1992) and there is increasingly more evidence showing that dispersal affects selection on other life-history traits (Stearns 1992; Clobert et al. 2001; Roff 2002; Stevens et al. 2012). Understanding these effects is important because increasing anthropogenic climate change continues to impact population distribution and persistence, and to fully understand how environmental variation affects population dynamics ultimately requires linking individual-level and population-level processes.

The use of structured population models allows individual characteristics to be incorporated as drivers of change in population size and structure (Leslie 1945;

Lefkovitch 1965). For example, work on Soay sheep (*Ovis aries*) (Coulson et al. 2001; Ozgul et al. 2009) and on the gray wolf (*Canis lupus*) (Coulson et al. 2011) has characterised phenotypic and population responses to climate change by including individual variation in size, age, sex and genotype into structured models. These studies show that linking the individual and population levels is crucial for understanding what drives trait and population dynamics. One type of structured model, the integral projection model (IPM; Easterling et al. 2000), has become one of the preferred tools to study how individual and population level processes are linked (Coulson 2012; Merow et al. 2014). The model can be used to predict a number of population biology parameters but can also be used to pose hypotheses that can be tested with additional data (Coulson 2012). For example if a hypothesis states that, for a given species, environmental change will reduce average survival rates, the effect can be simulated by reducing the value of the mean intercept in the survival function of the model. The predictions from the model can then be compared to experimental or subsequent observation data to see if model predictions match actual population responses. Including individual variation, therefore, can improve the predictive performance of population dynamic models.

In this thesis I contribute to and expand on this work and provide some general insights into the effects that phenotypic polymorphisms, dispersal and ARPs, have on life-history and consequently population dynamics. In my work I use a well-studied mite model system, the bulb mite (*Rhizoglyphus robini*). This species has an interesting life cycle in that it has a distinct facultative juvenile dispersal stage, and adult males are one of two alternative reproductive phenotypes. This mite system is therefore ideal to test hypotheses on the effects of dispersal and ARPs on population responses and can also inform on whether these two very different life history characteristics might

interact to influence population responses. In Chapters 2 and 3 I will focus on unravelling the drivers of and links between dispersal and ARPs; specifically the role of frequency-dependence in determining survival in alternative reproductive phenotypes, and the influence of dispersal on the life-history in alternative reproductive phenotypes. In Chapters 4 and 5 I develop and parameterise integral projection models to investigate the effects of dispersal on local population dynamics in both deterministic and stochastic environments. I will now first briefly discuss the topic of dispersal, which forms the main focus of the thesis. I will then also discuss the modelling approach I will take and explain why I chose this approach over alternative ones. Finally, I will introduce each chapter in more detail before explaining the structure of the thesis.

Dispersal

The movement of organisms can be defined as the "change in spatial location of the whole individual in time" and is driven by processes acting across multiple spatial and temporal scales (Nathan et al. 2008). Research on movement is extensive and resulted in four movement research frameworks being developed (reviewed in Nathan et al. 2008): 1) the biomechanical framework focuses on individual's physical machinery (e.g. mechanics, physiology); 2) the optimality framework focuses on the relative efficiency of different strategies to optimize fitness (e.g. reproduction, survival) over evolutionary and ecological time scales; 3) the cognitive framework focuses on mechanisms by which an individual motile organism makes movement-related decisions (mechanisms are at the cellular level); 4) the random framework where the focus is on simple phenomenological descriptions of movement paths and null models (these are related to theories, e.g. random walk and diffusion). However, given these four frameworks, a general unifying framework was lacking. A conceptual framework

for movement ecology that integrated the four frameworks was then developed, based on focal individuals (Nathan et al. 2008). More recently, a framework integrating movement ecology with biodiversity research has also been developed (Jeltsch et al. 2013). In the above unifying frameworks movement is not restricted to one type but can take many forms and has consequences for individuals, populations and communities (Nathan et al. 2008; Jeltsch et al. 2013; Barton et al. 2015). The three most common forms of movement are dispersal, migration and foraging (Clobert et al. 2012; Jeltsch et al. 2013). Each of these movement types can be affected by a number of different factors with each movement type potentially impacting different levels of biodiversity at different spatiotemporal scales (Jeltsch et al. 2013). Given the complexity surrounding movement types, in this thesis I will focus on dispersal as the form of movement only.

The meaning of dispersal, as with the other forms of movement, is often dependent on the investigator, discipline or taxa studied (Clobert et al. 2001; Nathan et al. 2008). Because many different definitions of dispersal exist, Clobert et al. (2001) developed an operational definition of two types of dispersal; natal dispersal and breeding dispersal. Natal dispersal is defined as "the movement between the natal area or social group and the area or social group where breeding first takes place". Breeding dispersal is defined as "the movement between two successive breeding areas or social groups". However, Clobert et al. (2001) do acknowledge that the distinction of their types of movement and other types of movement may not always be distinct, with the potential of dispersal interacting with other forms of movement. As such the definition is open to modification and one such modification generally employed is defined as "any movement of individuals or propagules with potential for gene flow across space" (Ronce 2007; Bonte et al. 2012). In order to maintain a generally employed definition I therefore use the aforementioned definition of dispersal by Ronce (2007) in this thesis.

Dispersal plays an important role in the response of populations and species to global climate change (Kokko and Lopez-Sepulcre 2006; Berg et al. 2010; Clobert et al. 2012; Travis et al. 2013; Tesson and Edelaar 2013), habitat fragmentation (Kokko and Lopez-Sepulcre 2006; Clobert et al. 2012; Tesson & Edelaar 2013) and species invasions (Kokko and Lopez-Sepulcre 2006; Berg et al. 2010; Clobert et al. 2012; Tesson and Edelaar 2013). Changes in climate conditions can cause changes in dispersal behaviour thus affecting the speed of range expansions, while habitat fragmentation can shape individual strategies in dispersal and associated traits (e.g. dispersal rates or distance) which can affect metapopulation dynamics. With species invasions, reduced predation pressure on invasive species compared to native species can result in higher competitive ability, growth and reproduction for invasives. Additionally, dispersing individuals have distinct phenotypes which can result in extreme or subtle specializations of dispersal traits (Clobert et al. 2004; Clobert et al. 2009; Bonte et al. 2012). Development of these phenotypes may come at a cost resulting in complex trade-offs with other traits not involved in dispersal (Bonte et al. 2012). Given this, the dispersal process is considered complex and multiple factors are at play that can influence dispersal phenotypes, rates of dispersal and the evolution of dispersal in different ways. It is therefore essential to disentangle, and quantify, how complex life histories (e.g. ARPs), environmental change, habitat fragmentation and species invasions interact with dispersal (and related trade-offs); a key process in a number of disciplines such as landscape ecology, spatial population dynamics and metapopulation biology (Clobert et al. 2012).

This thesis will contribute to our understanding of how dispersal and environmental variability influence the fluctuations of populations with complex life

histories. Furthermore, this work can contribute to developing a unified theory of dispersal which is currently lacking (Clobert et al. 2004; Bonte et al. 2012).

Modelling population fluctuations

There are different approaches to understand the fluctuations of populations where individual characteristics (e.g. reproduction or mortality) are dependent on its state (e.g. individual size). The relationship between individual life histories and population-level processes can be modelled directly from observational life-history data (phenomenologically) or mechanistically from life-history mechanisms (de Roos 2008; de Roos and Persson 2013). Phenomenological models use phenomenological relationships (e.g. regression models) to describe how demographic rates depend on individual characteristics; in mechanistic models life history processes are described by, for example, an energy budget model on growth and reproduction (de Roos and Persson 2013). Importantly, a model can be appropriate under some circumstances but not others. Examples of population models that describe individual life histories using phenomenological regression models are Matrix Population Models (MPM) and Integral Projection Models (IPMs). Mechanistic models include Ordinary – and Delayed Differential Equation (ODE and DDE) models, Individual Based Models (IBMs) or Physiologically Structured Population Models (PSPMs) (Metz and Dieckmann 1986; Tuljapurkar and Caswell 1997). I will briefly explain each of these types of models and then explain my choice of models: IPMs.

Ordinary Differential Equation (ODE) and Delayed Differential Equation (DDE) models

In ODE models, the description of population fluctuations is in continuous time and the population is characterized into discrete stages (individual state/phenotypic character is a discrete variable, e.g. juveniles and adults). A DDE is basically an ODE where time delay terms are included. Time delays account for the time required for individuals to move through the stages and can represent incubation periods, movement delays or gestation periods (Tuljapurkar and Caswell 1997). The population is described by a vector of stage abundances, with the dynamics described by an ODE incorporating a time delay. DDE models are advantageous in that they are parameterized with instantaneous rates of mortality, reproduction and development; and given the time delay development rate can be studied as a dynamically varying quantity (Tuljapurkar and Caswell 1997). These models are useful as there is evidence for delay effects in population fluctuations of various species (e.g. Turchin 1990; Turchin and Taylor 1993; Jaenike 2002). There are also disadvantages to the DDE model. When faced with discrete transition data, parametrization is difficult, which means that when individuals are divided into discrete classes population fluctuations may not be best explained with DDE models (Tuljapurkar and Caswell 1997). DDE models also assume that all individuals within a certain class have identical demographic rates. Furthermore, without efficient numerical algorithms to solve the delayed differential equations, DDE models can be difficult to simulate numerically and analyse analytically (Tuljapurkar and Caswell 1997).

Individual based models (IBMs)

IBMs allow researchers to study the emergent population level properties from the adaptive behaviour of individuals and in turn how the population affects individuals (Grimm et al. 2006). IBMs are generally based on a mechanistic representation of individual life history trajectories and aim to capture variation amongst individuals (i.e. by capturing the unique experiences of each individual). The actions of single individuals are tracked through time along with changes in the individual's biotic or abiotic environment, processes on the level of the population as a whole are then inferred by the combined contributions from the individuals (de Roos and Persson 2013). As such there is a strong link to individual life-history. Another aspect of all IBMs is that demographic stochasticity is considered to be an intrinsic property of the model (DeAngelis and Mooij 2005). However, because all individuals are tracked, IBMs are more complex in structure and more difficult to analyse and understand than analytical models (Grimm et al. 2006).

Physiologically Structured Population Models (PSPMs)

PSPMs start from the same principles as IBMs where the dynamics of a population are described by the physiology and behaviour of individual organisms, along with changes in the environment, on a continuous time-basis (de Roos 2008; Nisbet et al. 2015). PSPMs then make some assumptions; populations are considered to be large (effectively infinite) and all individuals that are in the same state respond in the same way to any given environment (Nisbet et al. 2015). These models are time dependent and so the state of the system depends on the state of the previous time and the conditions of that previous time. The link to individual life-history in these models are stronger than in either MPMs or IPMs (see below); additionally they can take into

account population feedback on individual development which MPMs are unable to do (de Roos et al. 2003; de Roos 2008). However, the assumption made by this type of model regarding individuals means that individual *variability* in development (i.e. differences between individuals in the *same* state) is not included. PSPMs can be parameter heavy as a large amount of individual level information is required (e.g. ingestion rate, assimilation rate, respiration rate) in addition to trait information such as body size or body weight. In addition, the analysis of PSPMs requires more complicated methodology than many other models, but this is being addressed with the development of software to aid modellers (de Roos 2014). Furthermore, unlike IPMs or MPMs, the continuous-time nature of these models mean they are not comparable to data collected in discrete time and so are difficult to link to many experimental and field data.

Matrix population models (MPM)

MPMs are discrete-time models and the primary assumption of this framework is that individuals occupy discrete classes or stages (most often age, size or stage of development) (Caswell 2001) as opposed to the continuous state in IPMs. In brief, the state of the population (the number of individuals within each stage class) is projected forward by one time step by a population projection matrix. The entries of the projection matrix are (derived from) stage-specific vital rates of survival, growth, reproduction and development which determine how many individuals in a certain stage will appear in the next time step (Tuljapurkar and Caswell 1997). The advantages of these matrices are that they are easy to construct from life history data (where individuals are followed over time and individual state is recorded in discrete time intervals), they are easy to simulate with simple matrix multiplication, and are relatively easy to analyze (Tuljapurkar and Caswell 1997; Caswell 2001). A disadvantage of this

method is that variation between individuals within each stage is ignored and the artificial separation into stages could introduce discretization errors of individuals and in turn potentially affect demographic predictions (Tuljapurkar and Caswell 1997; de Roos 2008). MPMs are suitable when life-cycles have distinct stages, however often continuous traits are more effective (i.e. individuals are in reality classified by a continuously varying trait, e.g. body size) and then this framework may not best explain the potential dynamics.

Integral projection model (IPM)

IPMs can be used to simultaneously track the dynamics of continuous phenotypic character distributions through time along with fluctuations in population size and structure (Easterling et al. 2000). This means that both individual and population-level responses can be jointly investigated within a single model (Easterling et al. 2000; Coulson 2012). Because IPMs are built from character demography functions which summarize individual data on survival, growth and reproduction they can be used to understand how changes in quantitative characters, life history and population dynamics are linked (Smallegange and Coulson 2013). The demographic functions are estimated using phenomenological regression models that best fit the demographic data, allowing vital rates to be estimated at any value of the continuous phenotypic character (Merow et al. 2014). As such, IPMs are easy to parameterize and require fewer parameters than Matrix Population Models (MPMs) fitted with the same data (Merow et al. 2014). Parameterising IPMs using observational data also accounts phenomenologically for unexplained variability in vital rates among individuals of the same stage (de Roos 2008). Additionally, IPMs require relatively straightforward mathematical techniques from matrix calculus and there is an existing toolbox available

to analyze IPMs (toolbox initially developed to analyse MPMs) (Coulson et al. 2010, 2012). It is worth noting that a standard IPM cannot account for any population feedback, however feedback can be included using density-dependent IPMs. Here transition rates vary as a function of population density where density can be included as a term in the statistical demographic functions. As with all models there are also limitations to IPMs. Because IPMs are phenomenological models they lack a mechanistic representation of the biological processes that give rise to observed demographic change (de Roos and Persson 2013). Furthermore, IPM predictions are only as good as the parametric assumptions and inferred transitions from vital rate regressions (Merow et al. 2014). There is also the possibility that artefacts (e.g. population extinction) can be introduced by the time discretisation in IPMs; a different time step could avoid this (Smallegange et al., in prep.).

I will use IPMs as the tool to address my questions regarding the influence that dispersal has on natal populations, I do so for two reasons. The first is that I will be using sampled/census data collected at discrete intervals and it is often easier and more practical to model time discretely (field and laboratory data are frequently collected at regular time intervals, e.g. yearly or seasonally). Second, the character state (or phenotypic character) that I will be using, that influences individual characteristics, is body size and is a continuous variable. I use this trait as body size is considered to be an important individual trait in many species influencing many key life-history processes and ecological interactions that an individual is exposed to (de Roos et al. 2003).

Frequency dependence in a complex life-history (Chapter 2)

Alternative reproductive phenotypes (ARPs) are typically found in species where male-male competition over access to females is intense (Shuster and Wade 2003; Oliveira et al. 2008) and are evident across a range of different taxa (Taborsky et al. 2008). Different evolutionary models have been suggested to explain the evolution and maintenance of ARPs but empirical evidence suggests that the conditional strategy model, where male morph expression depends on both genetic and environmental factors, best describes ARPs (Gross 1996; Kurdziel and Knowles 2002; Tomkins and Hazel 2007; Oliveira et al. 2008; Lukasik 2010; Buoro et al. 2012). The conditional strategy model that is currently favoured to explain the evolution and maintenance of ARPs is the environmental threshold (ET) model (Tomkins and Hazel 2007). The model states that there are genetic differences between individuals in the threshold at which development switches from one phenotype to the other. The threshold is under polygenic control and is sensitive to a cue (e.g. body size). Every male in the population can in principle develop into either phenotype, but whether or not a male does depends on whether or not it reaches a critical (size) threshold during ontogeny. Those that reach the threshold develop into so-called fighters, which are large and have morphological structures that are used as weapons in male-male contests. Those males that do not reach the threshold develop into sneakers, which are smaller and do not have weapons. Sneakers were long considered to make the best of a bad job, but recent research shows that sneakers potentially have their own fitness merits (Lee 2005; Reichard et al. 2007). This means that condition-dependent selection on its own is not enough to maintain a conditionally determined male dimorphism. Frequency-dependence is one mechanism that can play a role in the maintenance of male dimorphisms in single populations (Gross 1996), yet the role of frequency-dependent selection in the maintenance of an

environmentally determined threshold trait has not yet been explored empirically or theoretically (Tomkins and Hazel 2007).

In Chapter 2 I empirically determine if frequency-dependence is a mechanism that maintains male dimorphism, an environmentally determined threshold trait, in my study system, the bulb mite. To this end, I applied an experimental approach where I manipulated the fighter-scrambler ratio to assess daily survival of both male morphs. This was done in two environment scenarios thereby manipulating the environmental cue; the first was in an environment with a rich diet and the second in an environment with a poor diet, diet was chosen as diet quality dictates body size during development.

Individual costs of dispersal (Chapter 3)

Dispersal is a strategy that can increase individual fitness in a heterogeneous (spatially and temporally) landscape as it allows an organism to move to a different, usually more favourable, habitat. The variability in expected fitness between different habitat patches is a main determinant in the evolution of dispersal (Bowler & Benton 2005). Various dispersal strategies have evolved, ranging from unconditional dispersal strategies, where individuals disperse at constant rates regardless of environmental factors, to more complex conditional dispersal strategies, where dispersal expression is highly sensitive to local conditions such as habitat type or population size (McPeek & Holt 1992). Within these strategies, dispersal itself is achieved in a number of different ways: through an alteration in behaviour (Li & Margolies 1994), physiology (Hanski & Saccheri 2006) or life history (Hanski et al. 2006). This means that a multitude of phenotypic traits are related to dispersal capability (see Clobert et al. 2009). However, all forms of dispersal have associated costs and different trade-offs between dispersal capability and fitness exist (see review by Bowler & Benton 2005).

For a long time, costs of dispersal associated with emigration and immigration were considered the main dispersal-related drivers of population dynamics. However, dispersal is now considered a multi-phase (pre-departure, transfer and settlement) life-history process with each phase having an associated cost to individuals. Taking an individual based perspective allows the costs at each stage to be identified and incorporates individual variation in the dispersal process (Clobert et al. 2009, Bonte et al. 2012). This is important not only to understand the evolution of dispersal, but also how dispersal expression contributes to population level processes. Any other life-history traits that influence reproduction and survival will also influence the dynamics of populations, and there is a strong focus on unravelling the trade-offs between dispersal and reproduction and/or survival (Rankin and Burchsted 1992; Zera and Deno 1997; Zera and Harshman 2001; Hanski et al. 2006; Bonte et al. 2012). In invertebrates there has been an overwhelming focus on dispersal via flight with studies on dispersal by other means lacking (see review by Bonte et al. 2012). This may prove significant as the physiological costs involved in developing dispersal morphology will vary among dispersal modes. For example, the cost of investing in wings to disperse will differ substantially to costs of investing in other dispersal modes such as phoresy. The development of wings requires huge investment in flight muscles and in the actual process of flight. In a species that disperses by phoresy, as in the case of the bulb mite, the physiological investment is in a distinct non-feeding morphological stage during development, and there will be less physiological investment while moving between habitats. Therefore, any co-variation amongst physiological traits and dispersal differ depending on dispersal mode. Similarly, the nature of the trade-offs between dispersal and life-history traits likely depend on the mode of dispersal, with additional interactions with complex life-histories (such as ARPs) influencing these trade-offs. The

over-representation of dispersal via flight, and under-representation of other dispersal modes, hinders our ability to fully understand these effects. This highlights the need to investigate trade-offs between dispersal and life-history traits in more species with varying modes of dispersal.

In Chapter 3 I investigate the possible trade-offs between dispersal by phoresy, an understudied mode of dispersal, and life-history traits in a system with a complex life-history. As in Chapter 2 I applied an experimental approach. I isolated individuals and monitored their growth, survival and reproduction to compare the life-history trajectories of dispersers and non-dispersers.

Disperser costs to the natal population (Chapter 4)

Dispersal of an individual bears costs at one or all of the three phases of dispersal; pre-departure, transfer and settlement phase. By dispersing to a new environment, these costs are potentially outweighed by the benefits of being in the new environment. This has been shown both empirically and theoretically in a number of studies (reviewed in Bonte et al. 2012). However, individuals may not always be able to disperse. For example, if dispersal is probabilistic and requires a host (e.g. phoresy), successful dispersal away from the natal habitat is dependent on the frequency and predictability of the host. If individuals are unable to disperse after investing in dispersal (at the pre-departure stage) these costs are not outweighed by benefits in the natal environment (Bonte et al. 2012). Currently, it is not clear to what extent the fitness costs of these unsuccessful dispersers will affect the natal population and possible dispersal trade-offs as to date these effects have not been addressed. Therefore, to accurately understand how the different properties of dispersal affect fluctuations of

populations and metapopulations, the effects of unsuccessful dispersers on natal populations need to be accounted for.

Integral projection models (IPMs) use individual life-history data to investigate changes in character distributions (Coulson et al. 2010, Smallegange and Coulson 2013). Because both the dynamics of population size and of a phenotypic character are tracked within IPMs, this allows for the joint investigation of individual and population level dynamics (Easterling et al. 2000, Coulson et al. 2012). From an IPM, a number of population biology quantities such as the population growth rate can be calculated, which can further be analysed for their sensitivity to perturbation of the underlying, individual-level parameters, such as dispersal probability, using perturbation analyses. Therefore, using IPMs is one way in which we can assess the costs of unsuccessful dispersers on natal populations.

In Chapter 4 I aim to determine how individuals that invest in the dispersal stage and don't disperse, influence the natal population. I do so by constructing two size- and stage-structured IPMs; one for a population that includes (unsuccessful) dispersers and one that does not. I use a stage structured approach as I want to assess how the dispersal stage may influence population structure and growth. Body size is also used (as opposed to say age) as size is considered to be an important individual trait in many species that influences key life-history processes; this is also true in the study system I use. I then compare predicted population biology quantities between the two populations. I also increased the proportion of unsuccessful dispersers and conducted perturbation analyses to determine if there was a shift in selection pressure on the population growth rate from non-disperser individuals in the population to dispersers.

Combined effects of dispersal and environmental variation (Chapter 5)

Understanding how environmental variation influences population dynamics is key to population persistence and changes in selection pressure on processes such as dispersal, especially as populations can respond to a combination of environmental properties (e.g. Ripa and Lundberg 1996; Petchey 2000; Fontaine and Gonzalez 2005; Tuljapurkar et al. 2003; Marshall and Burgress 2014). To characterise environmental variation, studies often look to define variation in terms of its temporal autocorrelation value, which, for convenience, is also defined as noise colour (high positive values are red, a zero value is white and high negative values are blue). These studies have highlighted the strong influence that environmental variation can have on population viability and extinction (Petchey et al. 1997; Heino et al. 2000; Schwager et al. 2006), life-history trajectories (Laakso et al. 2003; Smallegange et al. 2014) and dispersal (McPeek and Holt 1992; Travis and Dytham 1998; Travis 2001). Further studies have shown that frequency of the occurrence of the same environmental state is an additional environmental property that can, in combination with environmental variance (autocorrelation), influence life-history traits and demographic rates (Marshall and Burgess 2014; Tuljapurkar et al. 2003; Smallegange et al. 2014). However, how the interaction of a process such as dispersal and environmental variation affects population characteristics, like population growth rate, is not known.

In Chapter 5 I investigate the combined effect of noise colour, frequency of occurrence of specific environmental states and an increasing proportion of dispersers (that don't disperse) on the stochastic growth rate. I do so by implementing a stochastic version of the integral projection model developed for dispersers in chapter 4, where the stochastic model is parameterised for a good- and bad environment.

Concluding remarks

Following the research chapters (Chapter 2 – 5) is a final discussion chapter (Chapter 6). In this chapter I take the findings and discuss how this work relates to the environmental threshold model and its role in explaining conditional strategies, and how natal populations influence dispersal rate in metapopulations. Each chapter is written as a stand-alone manuscript. However, I include figures and tables within the text and do not collate them at the end of the chapter. Additionally, chapters have their own supporting information section and references; however the introduction and discussion have a joint reference list that is at the end of the thesis. I have included two papers in the appendix of the thesis on which I am a co-author. The first paper is on the response of life-history variables to environmental change and the second on the eco-evolutionary interactions of selection on a secondary sexual trait. I refer to both papers throughout the thesis.

Chapter 2

Does frequency-dependence determine male morph survival in the bulb mite *Rhizoglyphus robini*?

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Does frequency-dependence determine male morph survival in the bulb mite *Rhizoglyphus robini*?

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Abstract Alternative reproductive phenotypes (ARPs) represent discrete morphological variation within a single sex; as such ARPs are an excellent study system to investigate the maintenance of phenotypic variation. ARPs are traditionally modelled as a mixture of pure strategies or as a conditional strategy. Most male dimorphisms are controlled by a conditional strategy, where males develop into a particular phenotype as a result of their condition which allows them to reach a certain threshold. Individuals that are unable to reach the threshold of a conditional strategy are considered to 'make the best of a bad job'; however, these individuals can have their own fitness merits. Given these fitness merits, condition-dependent selection alone is not sufficient to maintain a conditionally determined male dimorphism and other mechanisms, most likely frequency-dependent selection, are required. We studied in an experiment, the male dimorphic bulb mite *Rhizoglyphus robini*—where males are fighters that can kill other males or benign scramblers—to assess the strength of frequency-dependent survival in a high and low-quality environment. We found that male survival was frequency-dependent in the high-quality environment but not the low-quality environment. In the high-quality environment the survival curves of the two morphs crossed but the direction of frequency-dependence was opposite to what theory predicts.

Keywords Alternative reproductive tactics · Male dimorphic mite · Frequency-dependence · Threshold trait · Polymorphism

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Introduction

One of the biggest challenges in evolutionary ecology is to understand how phenotypic variation is maintained in populations. An excellent system to investigate the maintenance of phenotypic variation is alternative reproductive phenotypes (ARPs), which are defined as discrete morphological variation within a single sex, typically males (Oliveira et al. 2008). In males, ARPs are often characterised by the absence or presence of sexually dimorphic structures which are used as weapons in male–male competition (e.g. horns in the dung beetle *Onthophagus taurus* (Hunt and Simmons 2001); thickened hind legs in some mite species (Tomkins et al. 2004; Radwan 2009)). Traditionally, ARPs were modelled as a mixture of pure strategies or as a conditional strategy (Gross 1996; Shuster and Wade 2003). Pure strategies are genetically different alternative phenotypes, which are maintained within a population by frequency-dependent selection. However, most male dimorphisms are controlled by a conditional strategy (Oliveira et al. 2008). In a conditional strategy, males develop into a particular phenotype as a result of their condition such as body size: only if males are large can they develop the morphological structures that they use as weapons in male–male competition. Early models of the conditional strategy assumed that condition-dependent selection maintains male morph coexistence (Gross 1996; Maynard Smith 1982). Later models, however, showed that frequency- and condition-dependent selection can act together to maintain a male dimorphism that is controlled by a conditional strategy (Repka and Gross 1995). The model that is currently at the forefront as a means to understand the maintenance and evolution of the conditional strategy is the environmental threshold (ET) model. The ET model assumes that there are genetic differences between individuals in the switch point position, or threshold, at which development switches from one phenotype to the other. The switch point is assumed to be under polygenic control that is itself sensitive to a cue (e.g. body size and hormone levels) that reliably correlates with the status of the environment. Assessing this 'environmental' cue against the genetically specified threshold of sensitivity at a specific sensitive period during an individual's development elicits an all or-none response resulting in the expression of one ARP or another (Hazel et al. 2004).

Whereas individuals that reach the threshold are able to develop into fighters, those that do not reach the threshold during development are called sneakers, and these have traditionally been considered to 'make the best of a bad job' (Maynard Smith 1982). However, sneakers can have their own fitness merits. In aquatic environments, for example, females may benefit from sneakers and at times actively seek to mate with them (Reichard et al. 2007). In terrestrial environments, parents of some species appear to play a mixed reproductive strategy as they actively alter environmental conditions (by varying the amount of resources available during ontogeny) to produce sons of both morphs (Alcock et al. 1977; Tomkins et al. 2001; Kotiaho et al. 2003; Oliveira and Schlindwein 2010). Furthermore, even though alternative phenotypes can vary in mean payoff, payoff can vary substantially within ARPs and can overlap between ARPs, which rules out the notion that one ARP is necessarily 'worse' than another (Lee 2005). If sneakers have their own fitness merits and do not make the best of a bad job, this means that condition-dependent selection alone is not enough to maintain a conditionally determined male dimorphism. Instead, other mechanisms, with frequency-dependent selection being the most likely candidate mechanism (Repka and Gross 1995), also play a role in maintaining such a male dimorphism. Yet the role of frequency-dependent selection in the maintenance of an environmentally determined threshold trait has not yet been explored empirically (Tomkins and Hazel 2007).

Here we explored if frequency-dependence plays a role in the maintenance of the male dimorphism in the bulb mite *Rhizoglyphus robini* Claparède. Male bulb mites are either fighters, which have a thickened third pair of legs with which they can kill other mites (Radwan et al. 2000), or scramblers (sneakers) which do not have these thickened legs. Bulb mites are a good system for this investigation because scramblers do not display typical low-quality characteristics. Firstly, although scramblers emerge from final instars that are on average smaller than those that fighters emerge from (Smallegange 2011), after maturation, scrambler adults can increase in size to be larger than fighter adults (Smallegange unpublished data, see Online Resource 1). This suggests that scramblers are not making the best of a bad job. Secondly, scramblers can displace fighters from females during mating (Smallegange et al. 2012). Whether or not males develop into a fighter depends on whether they reach a critical size threshold during ontogeny (larger individuals develop into fighters) (Smallegange 2011). This threshold, in turn, is genetically determined (Smallegange and Wilson unpublished data; Radwan 2003). To our knowledge, it is still unclear why the two male morphs coexist (Radwan 2009). Radwan and Klimas (2001) investigated the mating success of scramblers and fighters under rich food conditions, but found no evidence that mating success of either morph is frequency-dependent. Here, we investigate if the survival rates of fighters and scramblers are frequency-dependent. We assume that a reduction in survival has a detrimental effect on an individual's fitness and we hypothesize that frequency dependence can maintain this male dimorphism if the survival functions of both morphs cross with changes in male morph frequency (Fig. 1: note that survival of only one morph needs to be frequency-dependent to maintain this male dimorphism). We investigate whether survival depends on male morph frequency for two different environmental scenarios. In the first scenario, experimental populations are put on a rich diet and in the second, populations are on a poor diet; both diets have a 1:1 sex ratio. We investigate frequency-dependent survival under these two scenarios to assess if the shape of frequency dependence varies across environments.

Materials and methods

Experimental set-up

All bulb mites used were from the Imperial College London stock cultures which are maintained as detailed in Smallegange (2011). In the experiment we tested the effects of two different environments and the effect of fighter frequency on fighter and scrambler survival probability over time. The two environments were: (1) high quality (HQ) food diet and (2) low quality (LQ) food diet. The LQ diet was *ad lib* access to filter paper and the HQ diet was *ad lib* access to yeast. Yeast is rich in protein, whereas filter paper contains only cellulose on which the mites feed. These two food types represent extremes in terms of food quality and for this reason are commonly used in life table studies to assess effects of food quality on growth and development of mites of the family Acaridae (Gerson et al. 1983, 1991; Radwan 2001; Smallegange and Coulson 2011). Experiments were run over 20 days. Fighter frequency was manipulated by varying the fighter-to-scrambler ratio as follows: 1:15, 3:13, 5:11, 7:9, 9:7, 11:5, 13:3 and 15:1. The total number of males was therefore always 16. Additionally, to keep a 1:1 sex ratio 16 females were included for each fighter-to-scrambler ratio manipulation.

Treatment combinations were replicated three times, resulting in a total of 2 environments (diet quality) $\times 8$ fighter frequencies $\times 3$ replicates $= 48$ trials (Fig. 2). Due to

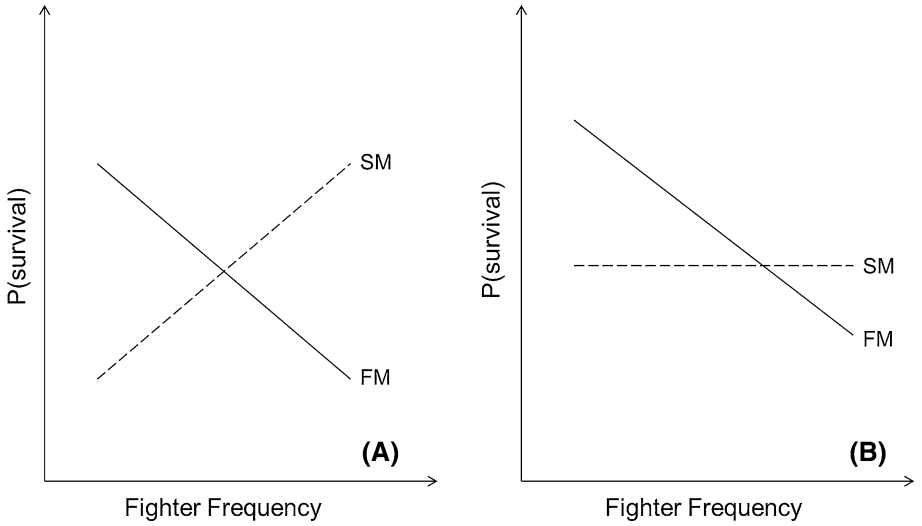

Fig. 1 Predictions for frequency-dependence. Frequency-dependence occurs when the functions of the alternative morphs cross with changes in frequency; this can be the case when both (**a**) or one morph (**b**) is frequency dependent. (**a**) As fighter frequency increases fighter survival probability (*black line*) decreases while simultaneously scrambler survival probability (*dashed line*) increases, in this case there is frequency-dependence in both morphs. (**b**) As fighter frequency increases fighter survival probability (*black line*) decreases and scrambler survival probability (*dashed line*) does not change, in this case there is frequency-dependence in only one morph (fighter). In both cases the functions for fighters and scramblers can be reversed and both predictions can still apply

logistic reasons, trials could not be performed in a randomised order and were run as two sets. The first set of trials run comprised the HQ treatment and the second set of trials the LQ treatment. We do not expect the order to influence the results as our stock cultures have been kept under constant temperature and humidity conditions since 2008 and as such we expect the demography of the stock cultures to be stable.

One to two days prior to running each of the two treatments, 1,400 adult mites were taken randomly from the stock cultures and grouped into glass tubes (100 mites per tube) (h × ∅: 5 × 2.5 cm) by sex and morph. These tubes are referred to as the stock tubes. Mites in the stock tubes were given *ad lib* access to yeast or filter paper to standardise satiation levels. Populations that were used in the experiment (see below) were formed using mites from these stock tubes. The stock tubes were also used to replenish any mites that died during the experiment to keep the number of fighters, scramblers and females as constant as possible. However, it turned out that there were not enough replacement females and fighters for the LQ treatment so that at day 15 and 18 respectively, replacement mites had to be taken from the stock cultures.

Experimental populations were formed by putting the appropriate number and type of mites from the stock tubes into a glass tube (h × ∅: 5 × 2.5 cm). These are referred to as the population tubes and each tube contained one population. Population and stock tubes had a plaster of Paris substrate and powdered charcoal base to avoid desiccation of the mites. Tubes were kept in a Sanyo MIR-153 incubator at 24 °C and >70 % relative humidity. Yeast and filter paper was replaced daily. Tubes were kept moist by adding two drops of water per day (approx. 150 µl water per day).

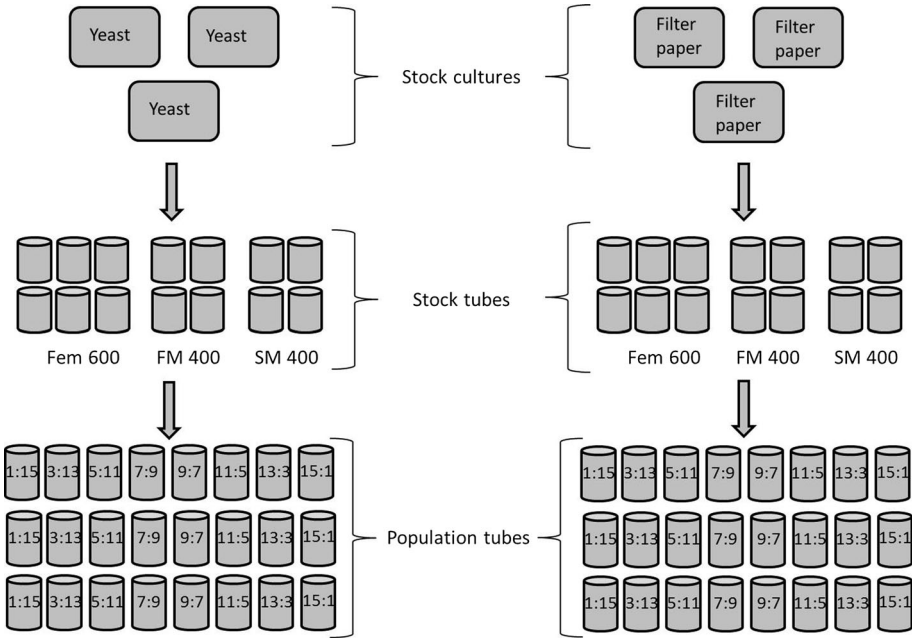

Fig. 2 Experimental setup for the high quality (yeast) and low quality (filter paper) diet treatments. Individuals were taken randomly from the yeast or filter paper stock cultures and separated by sex and morph (Fem—female, FM—fighter male, SM—scrambler male) into stock tubes. Numbers indicate total mites per sex and morph for all stock tubes combined (100 mites per stock tube). Stock tubes were used to setup population tubes with varying fighter-to-scrambler ratios (e.g. 1:15 indicates 1 fighter and 15 scramblers). Each population tube contained 16 males and 16 females, keeping the sex ratio constant at 1:1. Stock tubes were used to replenish mites that had died in the population tubes during the experiment. See text for details

Table 1 Slope parameter estimates (ê) from the minimal model with upper (97.5 %) and lower (2.5 %) confidence intervals for both treatments for both morphs (FM—fighter male; SM—scrambler male). For the minimal model the explanatory variable was day with trial included as a random factor (see text for details of minimal model estimates)

Treatment*	Estimate	2.5 %	97.5 %
High quality diet			
SM survival^{A}	−0.126	−0.17	−0.085
FM survival^{B}	−0.224	−0.272	−0.18
Low quality diet			
SM survival^{A}	−0.062	−0.088	−0.0369
FM survival^{A}	−0.049	−0.0785	−0.02

* Letters denote significant differences within each treatment, $P < 0.05$

The number of surviving scramblers, fighters and females in each population tube was assessed daily over a period of 20 days for both treatments. To count the number of surviving mites, all individuals of each population were removed from the population tube and placed on a petri dish (without a plaster of Paris base) under a 10× by 18× magnification Wild Heerbrugg stereomicroscope (Leica Microsystems, Wetzlar, Germany). Any

dead individuals were replaced with fresh individuals from the stock tubes after which all mites were returned to their population tube.

At day 6 and 12, each population was transferred to a fresh tube as juveniles that were born during the experiment were about to mature as adults, which would alter the adult morph and sex ratio (during the course of the experiment eggs were laid and egg-to-adult time can be as short as 10 days (Smallegange and Coulson 2011)).

Statistical analysis

To analyse how fighter and scrambler survival varied with fighter frequency and age (length of time since the start of the experiment) we analysed the effect of fighter frequency, male morph, day and their two-way interaction on the daily male survival probability (proportion of surviving mites per day) using generalised linear mixed-effects models (GLMMs) with binomial errors (because survival is calculated as a proportion). We used GLMMs because repeated measures of survival were measured for each trial over the course of the experiment. Trial (48 in total) was included as a random factor and treated as a repeated measure, while day (i.e. day of the experiment) was included as a continuous variable. Fighter frequency was initially included as a fixed factor. However, none of the analyses revealed a decline or increase in the proportion of mites surviving with increasing or decreasing fighter frequency. Therefore fighter frequency was included as a continuous variable. Male morph was included as a categorical variable. This was done in order to test for an interaction between male morph and fighter frequency which would indicate if the survival functions of fighters and scramblers cross. These analyses were conducted for each treatment separately. Because all fighter mites from the stock tubes with filter paper were used prior to the end of the 20 day period of the LQ treatment, only the first 18 days were used in the analysis.

For each treatment analysis, a model simplification procedure was followed. The full model was fitted, after which the least significant term (from the two-way interaction downwards) was removed if the removal resulted in an insignificant increase in deviance. Significance of simplified models was assessed by performing a likelihood ratio test. The model simplification process was repeated until the null model contained only significant terms (level $p < 0.05$). The likelihood ratio tests are reported in the results section where appropriate, as well as parameter estimates (ê) of the explanatory variables of the minimal models; the parameter estimates are the coefficients in the linear regression model and represent the relationship between an explanatory variable or interaction and the response variable. All analyses were performed in R (version 2.13.1) with models fitted by maximum likelihood in the 'lme4' package (R Development Core Team 2011).

Results

Model simplification

Model simplification was applied to both the HQ and LQ treatments. For the HQ treatment the interaction of fighter frequency and day was removed with no significant difference between the full model and the final simplified model observed (likelihood ratio test: HQ $\chi^2 = 0.096$, Δdf 1, $P = 0.76$). In the case of the LQ treatment, there was no significant difference between the full model and the reduced model when the interaction of fighter frequency and day was removed (likelihood ratio test: LQ $\chi^2 = 2.456$, Δdf 1, $P = 0.12$).

When simplified further by removing the interaction of fighter frequency and male morph, there was again no significant difference between the reduced model and final simplified model (likelihood ratio test: LQ $\chi^2 = 0.520$, Δdf 1, $P = 0.47$).

Male survival probability

There was a significant interaction effect of fighter frequency and male morph on male survival in the HQ but not the LQ diet treatment (HQ: $\hat{e} = -0.084 \pm 0.038$ (s.e.), $z = -2.190$, $P = 0.029$; LQ: $\hat{e} = 0.021 \pm 0.029$ (s.e.), $z = 0.718$, $P = 0.47$). When comparing survival functions, in the HQ treatment, the interaction effect and crossing survival functions of the male morphs suggests frequency dependence; fighter survival probability increased with increasing fighter frequency and scrambler survival decreased with increasing fighter frequency (Fig. 3a). In the LQ treatment, however, there was a lack of interaction between fighter frequency and male morph; in addition the functions did not cross suggesting there is no frequency dependence (Fig. 3b).

Day, however, did have a significant effect in both the HQ and LQ treatments with survival probability of both fighters and scramblers decreasing over time (HQ: $\hat{e} = -0.178 \pm 0.016$ (s.e.), $z = -11.157$, $P < 0.001$; LQ: $\hat{e} = -0.057 \pm 0.010$ (s.e.), $z = -5.775$, $P < 0.001$) (Fig. 4; Table 1).

Discussion

Frequency-dependence is an important mechanism that can maintain variation in both genotypes and phenotypes (Ayala and Campbell 1974; Adler et al. 2007). This occurs when the fitness of a genotype (or phenotype) relative to the alternative genotype(s) varies with the frequency of that genotype in the population (Ayala and Campbell 1974). Frequency-dependence has been well documented; playing a role in biological phenomena such as species coexistence (Adler et al. 2007); the maintenance of genetic diversity (Ayala and Campbell 1974) and heterozygote advantage (Clarke 1964). In this study we investigated frequency-dependent survival under different environmental scenarios (high and low quality diet treatment) by altering the fighter-to-scrambler ratio over time and focusing on how survival probabilities of adult male bulb mites varied with fighter frequency.

A previous study looking at frequency-dependent mating success in the bulb mite under favourable environmental conditions (Radwan and Klimas 2001) revealed no evidence for frequency-dependence. Our test of whether frequency-dependence in male morph survival can maintain this male dimorphism revealed significance of fighter frequency on male morph survival, but only in the HQ diet treatment. However, on closer inspection of the crossing survival functions of the male morphs in the HQ treatment (Fig. 3a), from which we infer that frequency-dependence occurs, the effect on survival is very low and the slopes of the functions were shallow; suggesting the strength of frequency-dependence is weak. In addition, there is a similar change in slopes when comparing the decrease in scrambler survival and the increase in fighter survival. However, the change seen in survival of both morphs does not conform to the classic idea of frequency-dependence (Fig. 1) suggesting that other mechanisms are at play. We suggest that the apparent effect of fighter frequency on male survival is a result of the low numbers of individuals used in allocating the various frequencies; specifically, low number of individuals at low frequencies. The proportional effect of one individual of a certain morph dying/surviving when there is only one individual of that morph in a population is higher than when there

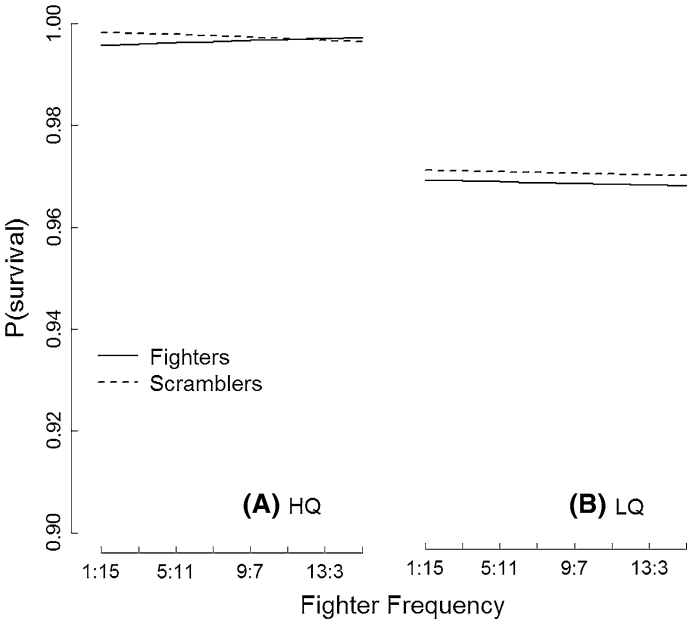

Fig. 3 The probability of survival (survival functions) for fighters (*solid line*) and scramblers (*dashed line*) with increasing fighter frequency for (**a**) high quality diet (HQ) and (**b**) low quality diet (LQ) treatments (see text for details of model estimates). Lines are the predicted survival probability using the parameter estimates ($\hat{e}$) from the minimal model where the explanatory variables were male morph, fighter frequency and day with trial included as a random factor

are more individuals of that morph; this may be driving the change in survival with change in fighter frequency and explain why this change in morph survival does not follow the classic idea of frequency-dependence (Fig. 1). Our findings also differ to those of Radwan and Klimas (2001) in that males had a higher survival probability (with changing fighter frequency) in both treatments. Radwan and Klimas (2001) suggest that the higher mortality in males, in their study, is due to mortality caused by male fights. We did not observe any male killing behaviour in our experiment which would suggest that mortality due to male killing behaviour was not present and could explain the higher male survival probabilities we observe and may influence the presence and/or strength of the frequency-dependence we found. This being said, we did not specifically test for fighting behaviour in this study and so cannot definitively say there was no male killing behaviour. Nonetheless, in the study by Smallegange et al. (2012) where they observed fighting behaviour in the male bulb mite no actual killing behaviour was observed during the course of the experiment which would support our findings.

Whether or not frequency-dependent survival plays a role in the maintenance of the bulb mite male dimorphisms depends on several things. First, if the fitnesses of the alternative morphs are very similar, then the required strength of frequency-dependence for the maintenance of the male dimorphism is weak. Specifically, recent theoretical work has shown that the strength of stabilizing mechanisms (such as density-dependence or frequency-dependence (Gross 1996) depends on the degree of fitness equivalence (Adler et al. 2007; Chesson 2000). This means that in our case, if the different morphs have similar survival probabilities, then coexistence can be maintained by a near-zero slope of the

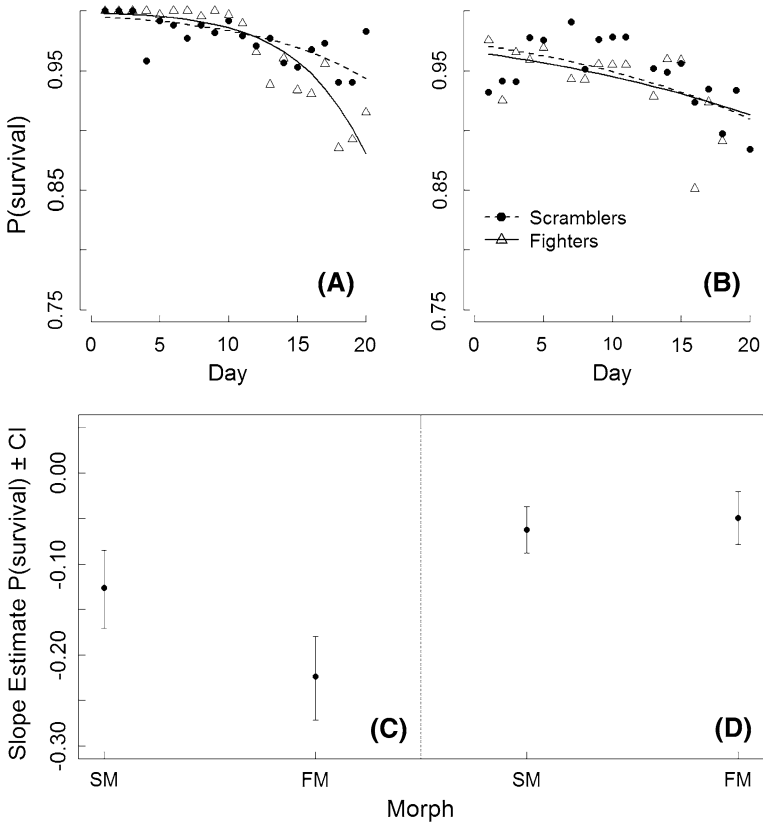

Fig. 4 The survival probability over the course of 20 days for (**a**) high quality diet (HQ) and (**b**) low quality diet (LQ) treatments. Survival probabilities are shown for: scramblers (*filled circles* and *dashed lines*) and fighters (*open triangles* and *solid lines*). Lines are the predicted survival probability using the parameter estimates (ê) from the model where the explanatory variable was day with trial included as a random factor. Panels (**c**) and (**d**) show the slope parameter estimates (ê) from the minimal model with upper and lower confidence intervals for high quality diet (HQ) (**c**) and low quality diet (LQ) (**d**) treatments for both morphs (see text for details of minimal model estimates)

relationship between fitness and relative frequency of each morph in the population (Adler et al. 2007). We indeed found that the slopes of the survival functions, in the HQ treatment at least, were shallow. However, if the differences in survival probability between the different morphs would have been large, then strong stabilizing mechanisms would have been needed to overcome the large fitness differences (Adler et al. 2007; Chesson 2000). In that case fitness functions would have been characterised by a negative slope of the relationship between fitness and relative frequency of each morph in the population (Adler et al. 2007). Secondly, frequency-dependence may be acting through juveniles and not adults. Łukasik (2010) found, in a study on soil mites *Sancassania berlesei*, that fighter males kill conspecific juveniles. This could lead to a change in adult morph frequency and thus influence fitness-tradeoffs between male morphologies, which in turn, could affect the evolution of conditional strategies. If this was indeed the case, then any such effects would not be seen in our current study. Finally, if there really is no frequency-dependence this would suggest that ARP expression in the bulb mite is not maintained in the population by

frequency-dependence. Given the importance of frequency-dependence in a wide range of biological phenomena (Ayala and Campbell 1974; Gray and McKinnon 2007; Adler et al. 2007; Takakura et al. 2009), it is surprising that most studies have not found empirical evidence for frequency-dependence in ARPs (Tomkins and Hazel 2007) (e.g. Radwan and Klimas 2001; Simmons et al. 2004; Cox and Calsbeek 2010). The limited empirical evidence for frequency-dependence may be because frequency-dependence is trait specific so that lack of support for frequency-dependence in a specific trait may not indicate a general lack of frequency-dependence in a species. One such exception, providing support for frequency-dependence, is a study on Chinook salmon (*Oncorhynchus tshawytscha*) (Berejikian et al. 2010) which showed support for frequency-dependent selection in spawning success in alternative male phenotypes. They showed that there was a significant positive correlation between the frequency of younger males (that adopted a sneaking approach) and average adult male reproductive success (adult males were larger and older that dominated courting opportunities and monopolized access to females).

We furthermore found a gradual decrease in survival probability over time, which is comparable to previous findings of adult mortality over time in male bulb mites (Radwan and Bogacz 2000; Deere and Smallegange unpublished data). The mean survival probability declined over time despite the fact that we added 'fresh' individuals from the stock tubes to replace dead mites (these fresh mites were not old; old mites can be identified by a non-smooth and wrinkled appearance of the cuticle and were not selected). This would explain the slightly higher survival probabilities we found compared to the study by Radwan and Bogacz (2000). Furthermore, Radwan and Bogacz (2000) showed that over the first 20 days fighters and scramblers have similar survival probabilities but after 20 days fighter male survival reduces significantly more than scrambler survival probability. This indicates that any significant differences in survival of the male morphs due to day only starts occurring after the first 20 days, given this if there would be any changes in male survival due to fighter frequency this would most likely be identified during this period. Interestingly, in our study in the HQ treatment, where there was a significant effect of fighter frequency on male survival, we see that fighter survival declines more rapidly than scrambler survival over the 20 days (Fig. 4a, c; Table 1). This would suggest that difference in the morph slopes may be due to an interaction of the weak effect of fighter frequency and the effect of day. However we did not find a significant interaction effect of fighter frequency and day on morph survival for the HQ treatment, so it is more likely that there is another driving force causing this change.

In conclusion, our results indicate that the strength of frequency-dependent survival only needs to be weak to maintain this male dimorphism, which may explain why other studies have failed to document frequency-dependence acting in the bulb mite (Radwan and Klimas 2001). In addition, further studies should focus on other traits and include juvenile life stages to be able to unequivocally confirm whether or not frequency-dependence plays a role in the maintenance of male morph expression in the bulb mite.

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Chapter 3

Life-history consequences of the facultative expression of a dispersal life stage in the phoretic bulb mite (*Rhizoglyphus robini*)

Running head

Dispersal effects on life-history

Title

Life-history consequences of the facultative expression of a dispersal life stage in the phoretic bulb mite (*Rhizoglyphus robini*)

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Abstract

Life history traits play an important role in population dynamics and correlate, both positively and negatively, with dispersal in a wide range of taxa. Most invertebrate studies on trade-offs between life history traits and dispersal have focused on dispersal via flight, yet much less is known about how life history trade-offs influence species that disperse by other means. In this study, we identify the effects of dispersal on life history traits in the male dimorphic bulb mite (*Rhizoglyphus robini*), a species that has a juvenile life stage (deutonymph) that disperses by phoresy. Males are either fighters (which kill other mites) or scramblers (which are defenceless). We test, in both sexes and in both male morphs, the effect of dispersal expression on age at maturity, size at maturity and, in females, lifetime reproductive success. We show that in both sexes, life history traits do not respond uniformly to expression of the dispersal stage, suggesting a complex response that depends on individual life-history. Remarkably, dispersing males developed only into the more competitive, fighter morph and never into the scrambler morph. This suggests that alternative, male reproductive strategies and dispersal should not be viewed in isolation but considered concurrently. This work provides a better understanding of life-history evolution in species with complex life cycles, where there is a trade-off between dispersal capability and life-history traits.

Key words

Alternative reproductive tactics, male morph, trade-off, complex life-cycle

Introduction

Life history traits, such as age at maturity and size at maturity, play a crucial role in population dynamics as they directly influence reproduction and survival. Importantly, their evolution is constrained by trade-offs (Stearns 1992; Roff 2002; Ronce and Clobert 2012). Dispersal effects have been shown to correlate with life history traits, both positively and negatively, in a wide range of taxa (Stearns 1992; Roff 2002; Stacey and Koenig 1990; Zera and Harshman 2001; Clobert et al. 2009; Stevens et al. 2012; Le Galliard et al. 2012; but see Ronce and Clobert 2012). Identifying which life-history traits co-vary with dispersal-related traits will allow a better understanding of the evolutionary dynamics of dispersal (Stevens et al. 2012; Clobert et al. 2001).

The literature on life history trade-offs and dispersal is especially abundant for terrestrial invertebrates, with a strong focus on fecundity and survival as the traits of interest. Most invertebrate studies have focused on dispersal via flight, with varying results in wing-monomorphic and wing-polymorphic species (Zera and Harshman 2001; Clobert et al. 2001 and references therein; Rankin and Burchsted 1992; Zera and Denno 1997). Studies on wing-monomorphic species have shown that fecundity is higher in dispersive than in sedentary individuals (Rankin and Burchsted 1992; Min et al. 2004; Hanski et al. 2006). It has been suggested that this outcome is due to physiological effects: in the Glanville fritillary butterfly *Melitaea cinxia* (Hanski et al. 2006) for example, high metabolic performance resulted in high dispersal and oviposition rates whereas increased juvenile hormone levels enhanced reproduction after long-duration flight in the grasshopper *Melanoplus sanguinipes* (Min et al. 2004). In contrast, the majority of studies on wing-polymorphic species has reported negative correlations between dispersal capability and fecundity (Bonte et al. 2012) (but note that several

exceptions exist: see review by Zera and Denno 1997). Few studies exist that examine which trade-offs are involved when dispersal occurs via means other than flight such as phoresy, ballooning, or walking, and their results vary (Li and Margolies 1994; Yano and Takafuji 2002; Friedenberg 2003). For example, in the two-spotted spider mite (*Tetranychus urticae*), no trade-off was found between dispersal by ballooning and fecundity (Li and Margolies 1994), whereas fecundity was reduced if mites dispersed by walking (Yano and Takafuji 2002). This suggests that different dispersal methods within the same species correlate differently with the same life history traits. Furthermore, if trade-offs of investing in dispersal involve growth, individuals may respond by compensating for any lost growth. This compensation for lost growth during development is known as compensatory growth and occurs across many taxa (Arendt 1997; Metcalfe and Monaghan 2001; Hector and Nakagawa 2012). Additionally, compensatory growth may also come at a cost to fitness components (e.g. survival and reproductive output) (Hector and Nakagawa 2012) which may, in turn, affect population dynamics (Metcalfe and Monaghan 2001). Any compensatory growth could thereby compound the effect of investing in dispersal. Insights from all these studies can be summarised into the following points. Firstly, life history traits can relate both positively and negatively to dispersal. Secondly, the type of association can depend not only on the species but also on the method of dispersal. Finally, there are very few studies on trade-offs between non-flight dispersal and life-history traits. These observations highlight the need to investigate dispersal-induced life history trade-offs further, both in a wider range of taxa and of dispersal-related traits.

Here we aim to investigate if trade-offs exist between dispersal and life history traits in the bulb mite (*Rhizoglyphus robini*, Acaridae), a species that disperses by phoresy. Phoresy is an interspecific relationship in which one species is carried by

another for the purposes of dispersal of the first species (Houck & Oconnor 1991). Dispersal occurs when juveniles develop into the (facultative) deutonymph stage in response to unfavourable environmental conditions. The development of this dispersal stage requires energetic investment and in other taxa energetic investment in dispersal morphology has been shown to be costly (Bonte et al. 2012). When juveniles do develop into this stage, a sucker plate on their ventral side allows them to attach to invertebrate hosts, such as beetles. The bulb mite is also male dimorphic; males are either fighters, which kill other mites with a thickened third pair of legs, or scramblers, which do not have this modification and are defenceless (Radwan et al. 2000). In an experiment we test for both sexes and both male morphs, the effect of the dispersal stage on the following life-history traits: age at maturity, size at maturity and, in females, lifetime reproductive success. We do this by comparing individuals that did not develop into a deutonymph (non-dispersers) with individuals that did develop into a deutonymph (dispersers). Furthermore, we tested whether individuals compensate for lost growth associated with deutonymph development, for example by increasing their growth rate in a subsequent life stage. We conclude by discussing possible consequences of dispersal expression on these life-history traits and highlight how these consequences may affect the evolution of life-history in dispersal capable species.

Methods

Study species

The bulb mite is a cosmopolitan pest species with a broad host range (Diaz et al. 2000). Its life cycle consists of six life stages: egg, larva, protonymph, deutonymph (non-feeding and facultative), tritonymph and adult (Fig. 3.1). Deutonymph expression

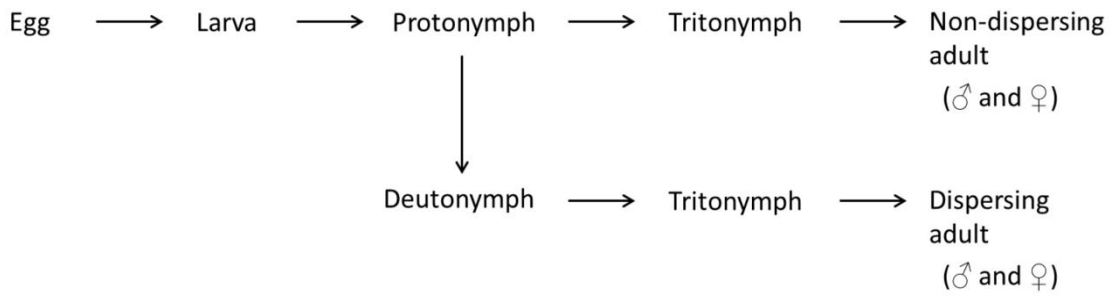

Figure 3.1. Life cycle of the bulb mite. The life cycle has six life stages; the deutonymph stage is the facultative dispersal stage that develops under unfavourable conditions.

depends on environmental conditions including temperature, humidity, food quality or a combination thereof (see review by Diaz et al. 2000) and increases as environments deteriorate. Longevity and generation time of both sexes is dependent on temperature and food quality. Longevity can be as short as 14.3 days (fed on garlic at 35°C) or as long as 73 days (fed on peanuts at 27°C) (Diaz et al. 2000), while generation time can be as short as 12 days when fed yeast at 24°C (Smallegange 2011a) or as long as 56.33 days (fed on garlic at 16°C) (Diaz et al. 2000). Reproduction is strictly sexual (Diaz et al. 2000).

Data collection

Data were collected between August 2012 and May 2013. The mites were taken from stock cultures that were collected in December 2010 from private flower bulb storage rooms (with permission from Koppert Biological Systems) in the Netherlands. No local or government authority was involved and collection and transport of the mites was in line with Dutch law on the use of animals in ecological studies. The stock cultures were maintained on oats, which produce viable populations and induce

deutonymph expression (personal observation). The cultures were maintained as described in (Smallegange 2011a). However, in our experiment only a very small proportion (3 from 100 individuals isolated as eggs) expressed the deutonymph stage. Consequently, we were unable to obtain enough individuals to statistically analyse all of the life stages (from egg to deutonymph to adult). Therefore, we isolated deutonymphs from the stock cultures and reared them individually, under the same conditions as those reared from eggs (which were non-dispersers). To ensure that there was no difference in life history responses between non-dispersers that were taken out of the stock cultures as eggs and dispersers that were taken out of the stock cultures as deutonymphs, we isolated individual protonymphs (n = 50) at the same time as we isolated deutonymphs from the stock cultures (see *Life history data of dispersers* for more details) and followed their growth and survival until death; none of these individuals developed into the deutonymph stage. Comparing the demographic trajectory of these protonymphs with those of individuals that were individually reared from the egg stage onwards (see *Life history data of non-dispersers*), revealed no significant effect of the life stage at which non-disperser individuals were extracted from the stock culture (i.e. egg vs protonymph) on growth and survival of non-dispersers (Table. S1). Therefore we assumed that any differences in life history traits between dispersers and non-dispersers were due to deutonymph expression and not data collection. As a result, non-dispersers were reared from the egg stage and dispersers were isolated and reared from the deutonymph stage. Based on the assumption that the demographic trajectories of deutonymphs are the same as those of non-dispersers, we used the egg, larvae and protonymph data of the non-dispersers for the equivalent stages of dispersers. Life history data were then used to calculate growth rate (mm per day), lifetime reproductive success and survival for each individual. Individuals that had developed from a

deutonymph stage were coded as 1 (deutonymph stage present) and those that had developed without this stage were coded as 0 (deutonymph stage absent).

Life history data of non-dispersers

Twenty seven females were isolated from the stock cultures over three consecutive, replicate time periods: 15 in the first period, 6 in the second and 6 in the third. These females were allowed to lay eggs for 3-8 days. From the 27 females a total of 132 eggs were collected over the three replicates time periods: replicate one, 51 eggs collected over 7 days; replicate two, 54 eggs collected over 8 days; and replicate three, 27 eggs collected over 3 days. Eggs were collected daily from each female, and individually isolated into single tubes with ad libitum oats. The tubes were kept in an unlit incubator at 24°C and >70% relative humidity. Until maturation, each individual was photographed daily using a Lumenera Infinity 3.1 camera (Lumenera Corporation, Ottawa, 22 Ontario, Canada) connected to a Meiji 20 EMZ-8TRD (10-45x) stereomicroscope and its length (without mouthparts) measured to the nearest 0.001 mm using Infinity Analyze Imaging Software (Lumenera Corp.). Henceforth body length is referred to as size. Some individuals died before reaching maturity (replicate time period 1, $n = 25$; replicate time period 2, $n = 33$; replicate time period 3, $n = 14$). Mature females were mated with randomly chosen virgin males (from either morph), which were placed in the same tube for the duration of the female's lifetime. Eggs were counted daily until the female died.

Life history data of dispersers

For the egg-protonymph comparison (see *Data collection*), deutonymphs and protonymphs were individually isolated from the same stock cultures within the same replicate time periods. For the first two replicates, each over the course of 5 days, 26 deutonymphs and 25 protonymphs were isolated (day 1-4, 5 deutonymphs and 5 protonymphs per day; day 5, 6 deutonymphs and 5 protonymphs). Thirty deutonymphs were isolated over 5 days in the third replicate (6 individuals per day). The isolated individuals were maintained under the same conditions, and monitored in the same way, as for non-dispersers. Seventy-two of the 132 individuals died before reaching maturity (replicate time period 1, $n = 33$; replicate time period 2, $n = 20$; replicate time period 3, $n = 19$).

Statistical analyses

Firstly, for dispersers only, we tested the effect of time spent in the deutonymph stage (deutonymph duration), deutonymph size, their interaction, and sex on size at maturity using a generalised linear mixed effects model (GLMM) with a Gaussian distribution where replicate time period was included as a random term. Secondly, for all individuals, we assessed the effects of sex, deutonymph expression, age at maturity and all interactions on size at maturity using a GLMM with a Gaussian distribution and included replicate time period as a random term. To assess whether dispersers compensate for reduced growth as a result of developing into a deutonymph, we tested for each sex, whether total growth (mm) and the standardised growth rate (d^{-1}; non-dispersers, growth per day per protonymph size; dispersers, growth per day per deutonymph size) during the tritonymph stage differed between non-dispersers and

dispersers. For both analyses, and for each sex separately, we used a GLMM with a Gaussian distribution; deutonymph expression was the explanatory variable and replicate time period was included as a random effect. We predict that if dispersers compensate for reduced growth, total growth for dispersers and non-dispersers during the tritonymph stage would be the similar, and the standardised growth rate during the tritonymph stage in dispersers would be higher than that in non-dispersers. Thirdly, we used a two-way ANOVA to investigate the effects of deutonymph expression and sex and their interaction on age at maturity. Fourthly, we used a GLMM with a Gaussian distribution to test the effects of deutonymph expression, longevity, and their two-way interaction, on female lifetime reproductive success, with replicate time period included as the random term. Finally, determine whether the sex ratio, and the ratio of male fighters to male scramblers differed between dispersers and non-dispersers, we tested for equality of proportions by conducting a 2-sample test using the function *prop.test.* All of the statistical analyses were conducted in R (version 3.0.2) (R Core Team 2013).

A model simplification procedure was used in all of the statistical analyses. After fitting the full model, the least significant term from the highest order interaction downwards was identified and removed if the removal resulted in an insignificant increase in deviance (significance was assessed using likelihood ratio tests). The model assumptions of Gaussian errors and homoscedasticity were confirmed by visual inspection of the probability plots and error structures. Parameter estimates (ê) of the explanatory variables in the minimal models are reported in the results section; the parameter estimates were coefficients in the linear regression model, and represent the relationship between an explanatory variable (or an interaction between several explanatory variables) and the response variable. Where standard errors are indicated they are the standard errors of the parameter estimates and are calculated as the standard

deviation of the sampling distribution of the parameter estimate. Where linear regression models were implemented, confidence intervals are calculated for model parameter estimates and are calculated as 1.96 (the critical value for a 95% confidence interval) multiplied by the standard error of the relevant parameter estimate, which are then added or subtracted to the parameter estimate. In the case of an ANOVA, confidence intervals are calculated as 1.96 multiplied by the standard error of the data, which are then added or subtracted to the mean. These are 95% confidence interval and indicates the estimated range of values which is likely to include a parameter estimate or an estimated mean.

Results

Effect of deutonymph expression on sex ratio and male morph expression

The sex ratio and the ratio of fighters to scramblers differed between dispersers and non-dispersers. The sex ratio of non-dispersers was not significantly different from 50:50 at 44:56 (χ^2 = 2.353, *df* = 1, *P* = 0.125). The fighter-to-scrambler ratio within non-dispersing males significantly differed from 50:50 at 65:35 (χ^2 = 7.042, *df* = 1, *P* = 0.008). In dispersing individuals, the sex ratio of 32:68 was female-biased, and significantly different from 50:50 (χ^2 = 7.118, *df* = 1, *P* = 0.008). However, the most striking finding was that no single dispersing male developed into a scrambler, as the fighter-to-scrambler ratio in dispersing males was 100:0 (n=11). Consequently we did not obtain any data on dispersing scramblers, and all subsequent analyses that compared the life history traits of dispersers and non-dispersers only included data on fighters and females.

Effect of deutonymph size and duration on size at maturity of dispersers

Neither deutonymph size (ê = 1.581±1.001 (s.e.), t = 1.578, P = 0.126), deutonymph duration (ê = -0.0004±0.001 (s.e.), t = -0.421, P = 0.677) significantly affected the size of dispersers at maturity. However, sex did have a significant effect on size at maturity as dispersing males (0.512mm) matured at a smaller size than dispersing females (0.627mm) (ê = -0.115±0.026 (s.e.), t = -4.378, $P < 0.001$).

Effect of deutonymph expression on size at maturity

Size at maturity was not significantly affected by the three-way interaction between age, sex and deutonymph expression, or by the two-way interactions between age and sex, age and deutonymph expression or sex and deutonymph expression. Age at maturity also had no significant effect on size at maturity (ê = -0.0005±0.001 (s.e.), t = -0.461, P = 0.646). Sex (ê = -0.130±0.014 (s.e.), t = -9.114, $P < 0.001$) and deutonymph expression (ê = 0.080±0.015 (s.e.), t = 5.376, $P < 0.001$) did significantly affect size at maturity. Females were larger at maturity (mean ± s.e.: 0.712 ± 0.011) than males (0.582 ± 0.014), and non-dispersers matured at a larger size (females, 0.712 ± 0.011; males, 0.570 ± 0.007) than dispersers (females, 0.627 ± 0.014; males, 0.513 ± 0.019).

Compensatory growth and deutonymph expression

Total growth and standardised growth were not significantly different between disperser tritonymphs (that developed from deutonymphs) and non-disperser

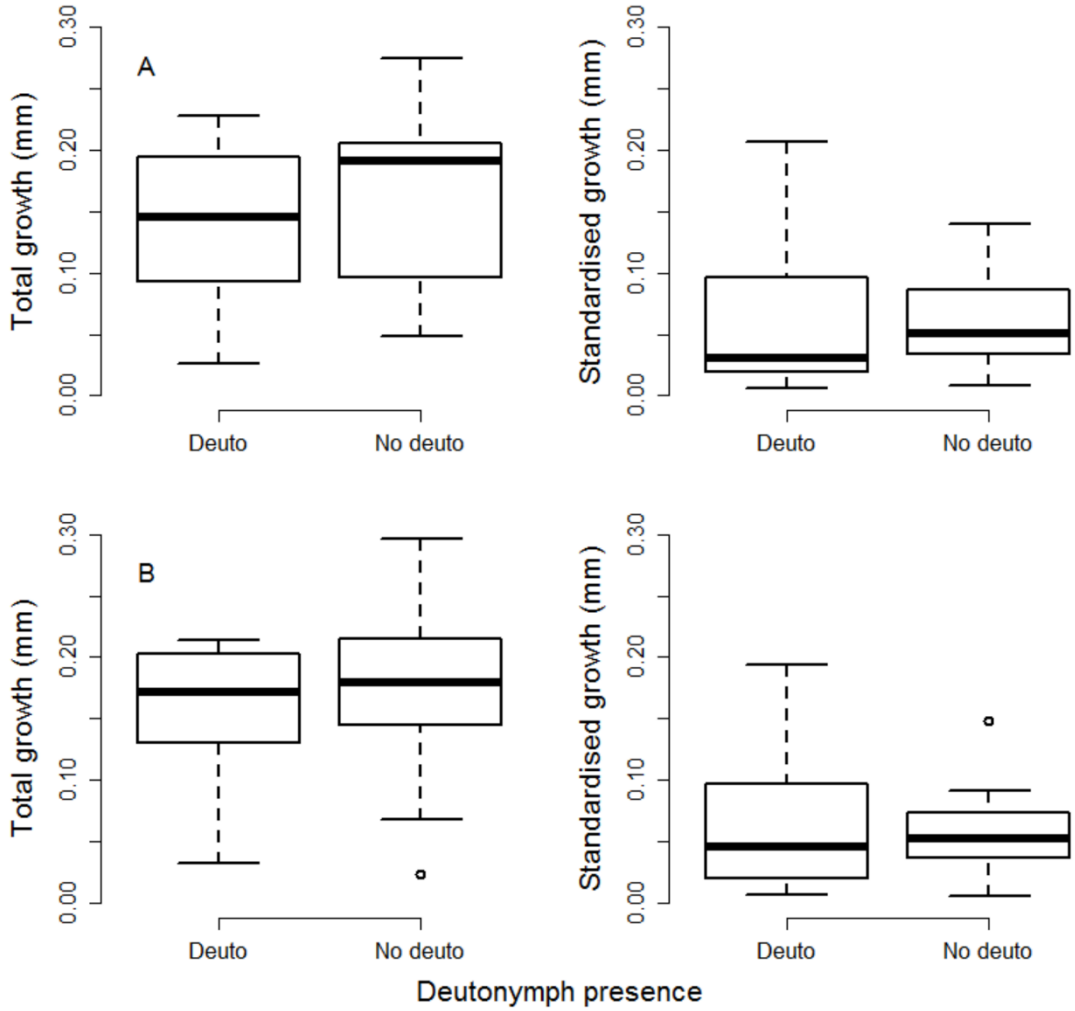

Figure 3.2. Compensatory growth. Total growth (mm) and standardised growth (mm per day per tritonymph length) during the tritonymph stage, as a function of deutonymph presence (Deuto) or deutonymph absence (No Deuto) during development in females (A) and fighter males (B). Boxes represent upper and lower quartile ranges, middle bands are medians and whiskers represent the extremes. Outliers are shown as points.

tritonymphs (that developed from protonymphs) in females (total growth: $\hat{e}$ = 0.029 ± 0.021 (s.e.), t = 1.383, P = 0.176; standardised growth: $\hat{e}$ = 0.026 ± 0.021 (s.e.), t = 0.920, P = 0.364) (Fig. 3.2A) or males (total growth: $\hat{e}$ = 0.015±0.025 (s.e.), t = 0.613, P = 0.546; standardised growth: $\hat{e}$ = -0.001±0.033 (s.e.), t = -0.037, P = 0.971) (Fig. 3.2B).

Effect of deutonymph expression on age at maturity

Age at maturity was not significantly affected by the interaction between sex and deutonymph expression ($F_{(1,93)}$ = 0.580, P = 0.448) or sex ($F_{(1,93)}$ = 3.626, P = 0.059). Deutonymph expression, however, did have a significant effect on age at maturity ($F_{(1,93)}$ = 39.317, P < 0.001). A post hoc Tukey test showed that dispersing females and males matured at a significantly later age than non-dispersing males and females (P < 0.05) (Fig. 3.3).

Effect of deutonymph expression on female lifetime egg production

There was a significant effect of the interaction between deutonymph expression and longevity on female lifetime egg production (ê = -0.276±0.081 (s.e.), t = -3.413, P < 0.005) . Female lifetime egg production increased with female longevity, but the slope of this relationship was steeper for non-dispersing females than it was for dispersing females (Fig. 3.4). As a result, lifetime egg production between dispersing and non-dispersing females did not differ when female longevity is low (<40 days) (confidence intervals overlap); although very young disperser females (at 10 days) do show a trend of having higher lifetime reproductive success than non-dispersing females. However, as female longevity increased above 40 days the confidence intervals of the curves did not overlap, indicating that at this point lifetime egg production differed; non-dispersing females had a higher lifetime egg production than did dispersing females (Fig. 3.4).

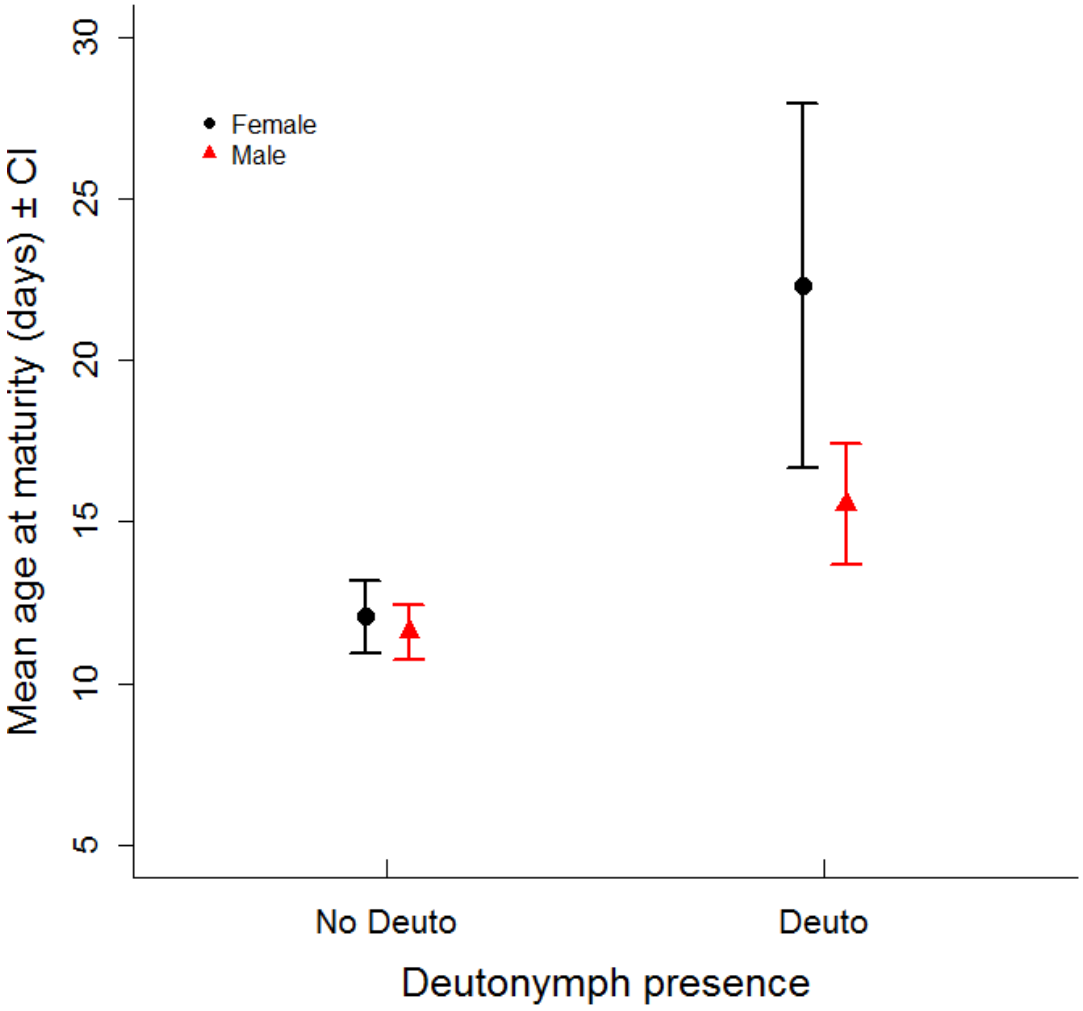

Figure 3.3. Age at maturity. Age at maturity (mean ± CI) for dispersers (Deuto) and non-dispersers (No Deuto), in females (black points and lines) and fighter males (red triangles and lines). CI's are calculated as 1.96 multiplied by the standard error of the data.

Discussion

Variation in life-history traits in dispersing invertebrates has been well documented, but studies have been skewed towards a few taxa with a strong focus on dispersal by flight. However, it is not necessarily the case that dispersal other than by

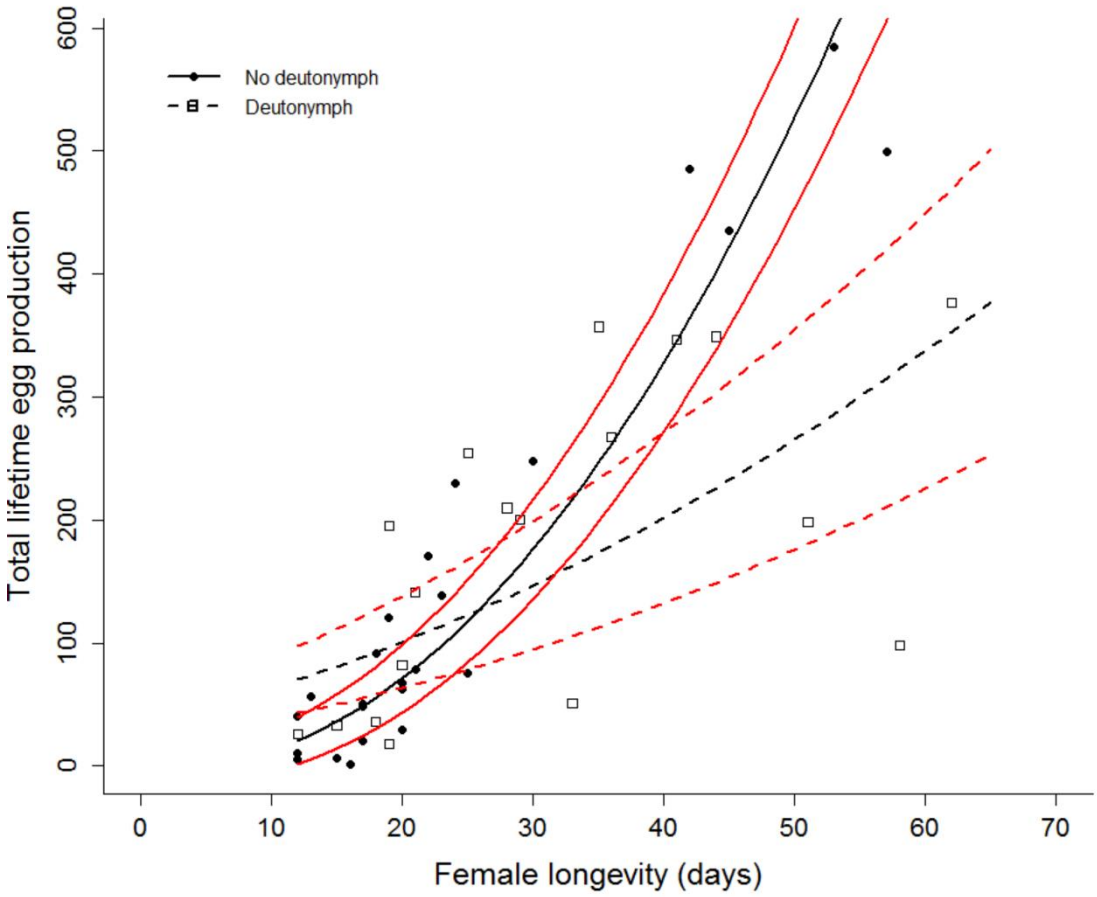

Figure 3.4. Female lifetime egg production. Lifetime egg production as a function of female longevity for dispersers (open squares and dashed lines) and non-dispersers (solid points and solid line). Confidence intervals (CI's) are 95% CI's calculated as 1.96 multiplied by the standard error of the model parameters and are shown in red; dispersers (dashed red lines), non-dispersers (solid red lines).

flight has the same life history consequences. In insects for example, larger individuals can fly and disperse further than smaller individuals which have smaller energy stores (Bowler and Benton 2005; Stevens et al. 2014). Dispersal by phoresy, however, may eliminate the need for large energy stores and has been suggested as a means to compensate for the disadvantages of small size during long-distance migration (Houck and Oconnor 1991). Here we investigated how dispersal affects life-history traits in a

Table 3.1. Summary of the effects of deutonymph expression. "Yes" indicates a cost to life-history traits or change to sex or male morph ratio. Disperser response informs of the type of change (if any); e.g. "Mature smaller" indicates dispersers mature at a smaller size than non-dispersers.

Trait	Cost/Change	Disperser response (vs. non-dispersers)
Males		
Size at maturity	Yes	Mature smaller
Age at maturity	No	No difference
Females		
Size at maturity	Yes	Mature smaller
Age at maturity	Yes	Mature later
Lifetime egg production	Yes	Lower egg production
Sex ratio	Yes	Female biased
Male morph ratio	Yes	Only fighters (no scramblers)

mite species that disperses as a deutonymph by phoresy; our results reveal that deutonymph expression incurs several life history costs to bulb mites (Table 3.1).

Firstly, both male and female dispersers did not compensate for lost growth during the deutonymph stage and matured at a smaller size than non-dispersing individuals. It turned out, however, that the length of time that individuals were a deutonymph did not influence their size at maturity, suggesting that any costs associated with being a deutonymph do not accumulate to negatively affect size at maturity. Neither was deutonymph size a predictor of size at which an individual matured. Secondly, male and female dispersers matured later than non-dispersers; this is not entirely surprising as is expected to naturally extend the age to maturity suggesting that male and females do not compensate for time spent in the additional life stage. Thirdly,

as in many other taxa (Zera and Denno 1997), we found that the expression of this costly dispersal morphology also trades off against investment into reproductive success as, in the case of longer-lived females (longevity > 40 days), non-dispersers produced more offspring in their lifetime than did dispersers of the same age. In many species, a trade-off between current and future reproduction exists (Stearns 1992). Here, this could mean that female dispersers have a higher reproductive output earlier in adulthood than non-dispersers. However, the data collected here cannot be used to test this hypothesis as we have only investigated total lifetime egg production of dispersing and non-dispersing females (Fig. 3.4) and did not analyse reproduction patterns over the lifetime of female dispersers and non-dispersers. From these results it is clear that both sexes are unable to compensate for the energetic investment in producing a deutonymph (inferred from the fact that both sexes show reduced size at maturity and females suffer reduced lifetime egg production).

We also found a clear difference in the sex ratio and male morph ratio between dispersers and non-dispersers: dispersers have a female-biased sex ratio and, more significantly, no scramblers developed from a deutonymph (see also Smallegange and Coulson 2011 for the same result). Since the sex ratio in the bulb mite is genetically determined (Gerson et al. 1983), ecological factors such as differential survival, probably caused the sex ratio to deviate from 50:50. For example, males might have a lower survival rate than females if they have been a deutonymph, or, although less likely, male deutonymphs on a developmental path to become a scrambler do not survive to adulthood. A more probably explanation for why male deutonymphs only develop into fighters in our study is that their weapons, i.e. their fighter legs, can be a useful tool when arriving in a new environment. In addition to fending off other males when competing for females, fighters can also use their legs to kill and consume other

(con- or heterospecific) mites (e.g. Lukasik 2010) or to defend themselves from predatory mites (Iza Lesna, personal communication). Recent work (Leigh and Smallegange 2014) has revealed that male morph determination in the bulb mite is more complex than originally thought (Radwan 1995); Leigh and Smallegange (2014) suggest that early male ontogeny, as well as environmental quality, may play a role in morph determination. Unravelling why, so far, all male deutonymphs in our study only develop into fighters adds to this complexity. Fighter expression is environmentally but also in part genetically determined (Smallegange 2011b). Given this genetic influence, it remains to be investigated to what extent successful colonisation of a new environment affects the evolution and coexistence of fighters and scramblers if the founder males of a colonising population are all fighters.

Our results show that there is a negative correlation between life-history traits and dispersal and, in males, is male morph specific which is in line with the increasing view that dispersal and life history patterns are interrelated in a complex manner (Stevens et al. 2014). Life history trajectories are strongly influenced by environmental change (Smallegange et al. 2014) and how changes in the environment influence life history patterns in relation to dispersal is another challenge remaining to be addressed. Only by investigating a wide variety of taxa and dispersal modes across a broad range of environmental conditions can we gain a better understanding of the biology and evolution of species life-histories.

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Supporting Information

Table S1. Growth and survival in post-protonymph stages

Table S1. Comparison of post-protonymph life stages (tritonymph and adult) between non-dispersers that were collected as eggs or protonymphs.

		Estimate	**Std. error**	***t*-value**	***z*-value**	***p*-value**
Growth*	Tritonymph	-0.0019	0.0043	-0.45	NA	>0.05
	Adult	-0.0093	0.0058	-1.6	NA	>0.05
Survival†	Tritonymph	0.001	0.2099	NA	0.005	0.996
	Adult	-0.1349	0.2093	NA	-0.6044	0.519

*Analysed using linear mixed effects model with a Gaussian error structure

†Analysed using generalized mixed effects model with a binomial error structure

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Chapter 4

Demographic consequences of unsuccessful dispersal in a phoretic mite: natal population costs

Running head

Cost of unsuccessful dispersal on natal populations

Title

Demographic consequences of unsuccessful dispersal in a phoretic mite: natal population costs

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Abstract

Individuals disperse from their natal habitat into a new one to increase individual fitness. The (potential) fitness benefits accrued in the new habitat have to be traded off against costs associated with dispersal. Most studies primarily focus on costs at settlement and the effects on the settlement populations. However, whether there is a cost of dispersal to the natal populations is unclear. Here, we use an Integrated Projection model (IPM) to assess the costs of developing into a disperser on the natal population dynamics in the bulb mite (*Rhizoglyphus robini*). Bulb mites develop into a (facultative) dispersal (deutonymph) stage during development. We found that deutonymph expression reduces individual growth and survival, which carries over to affect the population as generation time is increased and lifetime reproductive success and population growth rate are reduced. The population growth rate was most sensitive to perturbation of non-disperser reproduction, but, at high(er) proportions of deutonymphs, it was most sensitive to disperser reproduction. Dispersal costs to natal populations are rarely considered, but our findings suggest that these costs cannot be ignored if we are to fully understand the consequences of dispersal within a meta-population context that includes both natal and settlement populations.

Key words

Dispersal, Integral Projection Model, bulb mite *Rhizoglyphus robini*, perturbation analysis

Introduction

The importance of dispersal on the ecology of wild populations can be substantial (see Clobert et al. 2001). The role of dispersal into and out of populations impacts numbers, sex ratios, age structure, social dynamics and genetic structure of the population and so, as with reproduction and mortality, can be a crucial demographic process (Stenseth and Lidicker 1992). With the dispersal process comes associated costs that are traded off against (potential) fitness benefits that are accrued in the new habitat at both the individual and (meta-) population level (see review by Bowler and Benton 2005; and Bonte et al. 2012). Dispersal is therefore a strategy that increases individual fitness in a heterogeneous (spatial and temporal) landscape by the process of moving the organism into a new environment, whereby variability in expected fitness between different habitat patches drives the evolution of dispersal (Bowler and Benton 2005). Initially, dispersal was assumed to be genetically determined and insensitive to environmental conditions (Clobert et al. 2004). However, we now know that dispersal can be conditional upon a variety of traits (e.g. age, stage) (McPeek and Holt 1992; Clobert et al. 2004; Bowler and Benton 2005).

Dispersal is viewed as a multi-phase life-history process that comprises traits related to departure (initiating the eventual movement), transfer (the movement itself) and settlement (completion of the movement phase) (Bonte et al. 2012). Each stage can have an associated cost; hence identifying the costs associated with each stage provides an insight into where the costs of dispersal lie (is it immediate or delayed; are there changes in selection on aspects of dispersal such as density-dependence?) (Bonte et al. 2012). Here, we focus on the costs of the pre-departure stage. Specifically, we focus on

the expression of a morphologically distinct, facultative dispersal stage during development.

With the expression of a dispersal stage during development are associated costs, such as energetic investment and delayed maturation. In addition, these costs will not be outweighed by any benefits in the natal environment if the individual is not successful at dispersing from its habitat (Bonte et al. 2012). Here we define individuals that remain in the natal habitat after investing in dispersal morphology as unsuccessful dispersers. The costs suffered by these unsuccessful dispersers likely carry over to negatively affect the natal population. To what extent fitness costs of unsuccessful dispersers (e.g. reduced lifetime reproductive success; Deere et al., in prep, Chapter 3) affect natal populations is, however, unclear. It is therefore important to identify these costs as they will affect the trade-offs that determine the dispersal strategy, especially when individual costs carry over to affect population level quantities (e.g. growth rate, generation time). These population level effects alter the population dynamics and can, in turn, affect population persistence and dispersal rates.

One way in which we can assess the effects of unsuccessful dispersal on natal population dynamics is by using Integral Projection Models (IPMs) (Easterling et al. 2000; Ellner and Rees 2006; Rees and Ellner 2009; Coulson et al. 2010). IPMs have become a powerful tool that can be used to simultaneously examine the ecological and evolutionary dynamics of a population (Smallegange and Coulson 2013). Together with the dynamics of population size, the dynamics of continuous phenotypic character distributions can be tracked through time using IPMs, which means that both individual and population-level dynamics can be jointly investigated within a single model (Easterling et al. 2000; Coulson 2012).

Here we use IPMs to assess what effect unsuccessful dispersal of individuals has on the natal population fluctuations in the bulb mite (*Rhizoglyphus robini,* Claparède). This species is an ideal study system as its life cycle contains a facultative, juvenile dispersal morph: the (non-feeding) deutonymph (Fig. 4.1). Dispersal occurs via phoretic association with an arthropod host and attachment to the host is through the use of a sucker plate that is unique to the deutonymph stage (Diaz et al. 2000). Deutonymph expression depends on environmental conditions including temperature, humidity, food quality or a combination thereof; with increased proportions of deutonymph under unfavourable conditions (see review by Diaz et al. 2000). In order to assess the effect of deutonymph expression, we construct a size- and stage-structured IPM that tracks the changes in the distribution of body size of females through time (Fig. 4.1). Female life history data are used to parameterise the functions that build the IPM, with the functions describing the association between a continuous character (here body length, which is our measure of body size) and individual survival, development, recruitment and inheritance rates (Easterling et al. 2000; Coulson 2012). From the constructed IPM, we calculate a number of population biology parameters: generation time, lifetime reproductive success (R_0), mean body size of each life stage and the population growth rate (λ_0). By constructing IPMs from mites that passed through the deutonymph stage versus those that did not, a comparison of the population growth rates of these models can be made which gives an estimate of the demographic cost of unsuccessful dispersal on the natal population. We then conduct perturbation analyses, with increasing proportions of deutonymphs, to investigate whether a shift in selection pressure on the sensitivity of population growth rate from non-dispersers to dispersers occurs (Benton and Grant 1999). We expect that in all cases where the dispersal stage is present, there

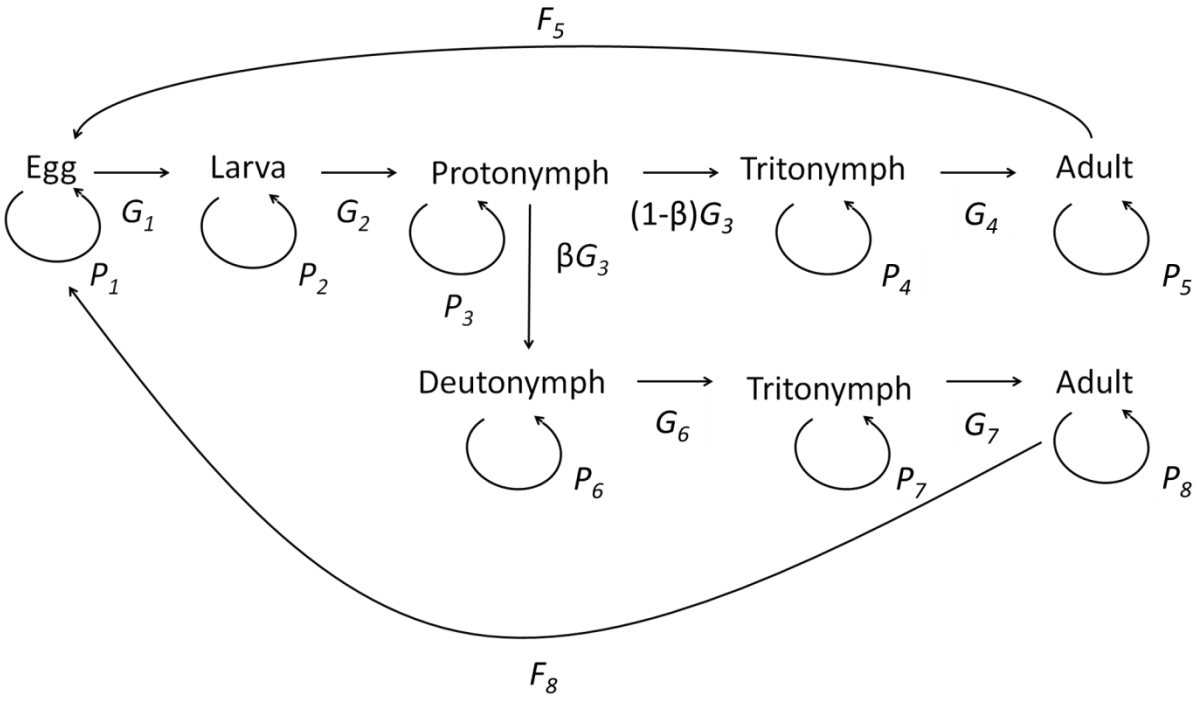

Figure 4.1. Life cycle of the bulb mite indicating the six life stages. From the life cycle we calculated survival (*P*) and fecundity (*F*) rate and the probability of surviving and growing into the next stage (*G*). The deutonymph stage is the facultative dispersal stage.

will be a reduction in fitness that will become greater with increasing proportions of the dispersal stage.

Methods

Data collection

Life-history data from Deere et al. in prep (Chapter 3) were used to parameterize all IPM's. Briefly, bulb mites were reared on ad lib access to oats and data on the expression of the deutonymph stage, female juvenile growth and survival as well as adult growth, survival, egg production and the relationship between size of the mother

and her offspring were collected. An oats diet ensured viable populations but at the same time induced the expression of deutonymphs. Data were collected between August 2011 and May 2013.

Integral projection model

We constructed two size- and stage- structured integral projection models; a non-dispersal IPM and a dispersal IPM. The IPMs consists of statistical character demography functions that describe how the body size z distribution at time t is influenced by survival, growth, reproduction and inheritance, resulting in a new body size distribution at time t+1. From day t to t+1 a female that survives can either stay in the current life stage or move to the next life stage; in both cases the individual grows from size z to z'. Only adult females produce eggs. These changes in the IPM are captured by statistical, character demography functions that relate body size z at each time t to: (1) the survival probability at time t+1, $S(z,s,t)$; (2) the transition probability that females develop into the next stage s at time t+1, $P(z,s,t)$; (3a) the increase in body size among survivors of size z that stay in stage s at time t+1, $G(z'|z,s,t)$; (3b) the increase in body size among survivors of size z that have moved to stage s+1 at time t+1, $G(z'|z,s+1,t)$; (4) the number of eggs produced by individuals of size z at time t+1 (assuming a pre-breeding census), $R(z,s,t)$; and (5) the size of eggs, z', produced by individuals of size z at time t+1, $D(z'|z,s,t)$. Two of the functions, (3) and (5), also describe how the variance in size at time t+1 is affected by size at time t. In the non-dispersal IPM the number of stages is five: egg, larva, protonymph, tritonymph and adult (Fig. 4.1). The number of stages in the dispersal IPM is six which accounts for the deutonymph stage: egg, larva, protonymph, deutonymph, tritonymph and adult (Fig.

4.1). However, we expect dispersers to have different character-demography functions for post-deutonymph life stages than non-dispersers. Character-demography functions of tritonymphs and adults are therefore split into two sets: one set for individuals that have not gone through the deutonymph stage and one set for individuals that have (Fig. 4.1). Furthermore, as the dispersal IPM will have disperser and non-disperser individuals the number of juvenile stages will differ depending on the individual (disperser or non-disperser). As such, the dispersal IPM comprises a number of equations to deal with the change in number of life stages as well as post-deutonymph dispersing and non-dispersing individuals (Table 4.1). These equations calculate the number of females in each stage *s* at time *t* which is described by *n(z,s,t)*, with all the equations taking a similar form. For example, if we want to calculate the number of dispersing female tritonymphs (stage 7 in Fig. 4.1) that develop into dispersing adults (stage 8 Fig. 4.1) the equation is written as follows (Eq. 1.8, Table 4.1):

$$n(z',8,t+1) = \int G(z' \mid z,8,t)P(8 \mid z,7,t)S(z,8,t)n(z,7,t)dz$$

The equations vary slightly depending on the life stages concerned; this is shown in Table 4.1. To generate a non-dispersal IPM (no dispersal stage so non-dispersers have five life stages), we exclude all disperser functions (Eq. 2; Table 4.1). Since only adults reproduce, the *R(z,s,t)* and *D(z'|z,s,t)* functions are zero for all non-adult stages in both IPMs.

Equation (1) provides a description of the dynamics of body size *z* (which is a continuous trait) for a population consisting of dispersers and non-dispersers; Eq. (2) provides a description of a population of only non-dispersers (Table 4.1). Predicted

Table 4.1. The Integral Projection Model (IPM) is a combination of the equations which generates an approximated matrix. Equations are constructed from the five statistical demography functions: 1) Survival *S(z,s,t)*; 2) Transition *P(s|z,s,t)*; 3) Growth *G(z'|z,s,t)*; 4) Reproduction *R(z,s,t)* and 5) Inheritance *D(z'|z,s,t)* (see text for details). The equations calculate the number of females in each stage *s* at time *t* which is described by *n(z,s,t)* with the *R(z,s,t)* and *D(z'|z,s,t)* functions zero for all non-adult stages as only adults reproduce. Ω_s is a closed interval indicating the size domain of stage *s*.

	Life stage Equation	Description
Dispersal IPM		
1.1	$n(z',1,t+1) = \int D(z' \mid z,5,t)R(z,5,t)n(z,5,t)dz + \int D(z' \mid z,8,t)R(z,8,t)n(z,8,t)dz,$	Egg production by non-dispersal and dispersal adults
1.2	$\left.\begin{aligned} n(z',s+1,t+1) &= \int_{\Omega_s} G(z' \mid z,s+1,t)P(s+1 \mid z,s,t)S(z,s+1,t)n(z,s,t)dz \\ n(z',s,t+1) &= \int_{\Omega_s} G(z' \mid z,s,t)P(s \mid z,s,t)S(z,s,t)n(z,s,t)dz \end{aligned}\right\} 1 \le s \le 2$	Eggs and Larvae developing into the next stage and staying in the same stage
1.3	$n(z',4,t+1) = \int G(z' \mid z,4,t)P(4 \mid z,3,t)S(z,4,t)n(z,3,t)dz$	Non-dispersal Tritonymphs developing from Protonymphs
1.4	$n(z',6,t+1) = \int G(z' \mid z,6,t)P(6 \mid z,3,t)S(z,6,t)n(z,3,t)dz$	Deutonymphs developing from a Protonymphs

Table 4.1 (cont.)

	Life stage Equation	Description
(Dispersal IPM cont.)		
1.5	$n(z',3,t+1)=\int G(z'\mid z,3,t)P(3\mid z,3,t)S(z,3,t)n(z,3,t)dz$	Non-dispersal Protonymphs staying Protonymphs
1.6	$n(z',6,t+1)=\int G(z'\mid z,6,t)P(6\mid z,6,t)S(z,6,t)n(z,6,t)dz$	Deutonymphs staying Deutonymphs
1.7	$n(z',7,t+1)=\int G(z'\mid z,7,t)P(7\mid z,6,t)S(z,7,t)n(z,6,t)dz$	Dispersal Tritonymphs developing from Deutonymphs
1.8	$n(z',8,t+1)=\int G(z'\mid z,8,t)P(8\mid z,7,t)S(z,8,t)n(z,7,t)dz$	Dispersal adults developing from dispersal Tritonymphs
1.9	$n(z',7,t+1)=\int G(z'\mid z,7,t)P(7\mid z,7,t)S(z,7,t)n(z,7,t)dz$	Dispersal Tritonymph staying Tritonymphs
1.10	$n(z',5,t+1)=\int G(z'\mid z,5,t)P(5\mid z,5-1,t)S(z,5-1,t)n(z,5-1,t)dz+\int G(z'\mid z,5,t)S(z,5,t)n(z,5,t)dz$	Non-dispersal adults developing from non-dispersal Tritonymphs and surviving non-dispersal adults
1.11	$n(z',8,t+1)=\int G(z'\mid z,8,t)P(8\mid z,8-1,t)S(z,8-1,t)n(z,8-1,t)dz+\int G(z'\mid z,8,t)S(z,8,t)n(z,8,t)dz$	Dispersal adults developing from dispersal Tritonymphs and surviving dispersal adults

Table 4.1 (cont.)

	Life stage Equation		Description
Non-dispersal IPM			
2.1	$n(z',1,t+1)=\int_{\Omega_s} D(z'\mid z,s,t)R(z,s,t)n(z,s,t)dz,$	$s=5$	Egg production adults
2.2	$n(z',s+1,t+1)=\int_{\Omega_s} G(z'\mid z,s+1,t)P(s+1\mid z,s,t)S(z,s,t)n(z,s,t)dz$ $n(z',s,t+1)=\int_{\Omega_s} G(z'\mid z,s,t)P(s\mid z,s,t)S(z,s,t)n(z,s,t)dz$	$1\le s\le 4$	Egg and all juvenile stages: staying in the current stage and developing into next stage
2.3	$n(z',5,t+1)=\int_{\Omega_{s-1}} G(z'\mid z,s,t)P(s\mid z,s-1,t)S(z,s-1,t)n(z,s-1,t)dz+$ $\int_{\Omega_s} G(z\mid z,s,t)S(z,s,t)n(z,s,t)dz,$	$s=5$	Adults developing from Tritonymphs and surviving adults

values from these equations are calculated to create a discrete approximation of the IPM. This is done by dividing the size domain of each stage into very small-width discrete bins ('mesh points'). A number of different bin sizes were used and results compared, this was done as an increase in the number of mesh points increases the numerical accuracy of the approximation (Ellner and Rees 2006). The body size domain of each stage was eventually divided into 50 size bins as a higher number of bins did not produce notably different results. Transition rates for the midpoint of two adjacent mesh points were estimated for each stage class. A number of population biology parameters were subsequently calculated: the asymptotic population growth rate (λ_0), generation time (T) and lifetime reproductive success (R_0). In the non-dispersal IPM, the final matrix size was 250X250 (50 bins x 5 stages = 250 mesh points); whereas in the dispersal IPM the final matrix size was 400X400 (50 bins x 8 stages = 400 mesh points). The dispersal IPM takes into account the different number of life stages of dispersers and non-dispersers as well two sets of tritonymph and adult life stages (tritonymphs and adults without dispersal stage, and tritonymphs and adults with dispersal stage) into a single IPM, hence there are eight stages in the final matrix and not six (see above; Fig. 4.1). The population growth rate was calculated as the dominant eigenvalue from the matrix approximation of each IPM. Lifetime reproductive success was calculated from the l_x and m_x schedules, where l_x is the survivorship function (probability of surviving from birth to age class x) and m_x is the maternity function that describes reproduction (expressed as female offspring per female) (Stearns 1992; Caswell 2001). We calculated generation time as T=log(R_0/log(λ_0) (Coale 1972; Caswell 2001). As constructed here, the IPM can only inform on the above mentioned population biology quantities, and on the stable stage distribution of the population, given the parameterised fundamental functions. Because the IPM is density-independent and is constructed from linear fundamental functions and can only predict

population increase or decrease, it cannot inform on population dynamics such as stability, oscillations or chaos.

Parameter estimation

We estimated the parameters using statistical models for five character-demography functions: the survival function (1), transition function (2), growth function (3), reproduction function (4) and inheritance function (5) (Fig. 4.2). For non-disperser functions, parameters were estimated following the method used by Smallegange et al. (2014) (see also supporting information for details). In the case of dispersers we again estimated the survival, growth, fertility and inheritance rates. The rates for eggs and larvae were the same as for the non-dispersers (Fig. 4.2). However, in the disperser IPM, protonymphs can also develop into a deutonymph and we estimated the probability that a protonymph develops into a deutonymph, tritonymph or stays as a protonymph using a multinomial logisitic (which generated the three transition probabilities). For the multinomial logistic the linear predictor was body length at time *t*, the response variable was stage at time *t*+1 and the reference level was set as the protonymph stage. This gives the probability of developing into a tritonymph, a deutonymph or remaining as a protonymph as a function of individual size. As such, the regression coefficients $\beta\mu$ are the log of the ratio of the two probabilities of developing into a tritonymph or deutonymph over staying in the protonymph stage (the reference level/choice). For example, if $\beta\mu$ represents the effect of μ (size), we expect that for one unit change in μ, the relative risk of developing into a tritonymph over staying a protonymph will increase by $\exp(\beta\mu)$. The multinomial logistic analyses were performed in R (version 3.0.2) using the 'mlogit' package (R Development Core Team 2013). All other parameter estimates

were calculated in the same way, and using the same analyses, as those for non-dispersing individuals.

In all statistical analyses a model simplification procedure was used. The full model was fitted, after which the least significant term from the highest order interaction downwards was identified and removed if the removal resulted in an insignificant increase in deviance. Significance of simplified models was assessed by performing a likelihood ratio test. The likelihood ratio (Λ) is calculated as $\Lambda = 2(LL_f - LL_c)/(p_f - p_c)$; where LL_i is the log-likelihood of the full model ($i = f$) or constant-only model ($i = c$) and p_i is the number of estimable parameters in the full and constant-only model. The likelihood ratio is X_ν^2 distributed, where ν is the difference in number of estimable parameters (in this case $\nu = 1$). The random factor was never removed during model simplification. Model assumptions on Gaussian errors and homoscedacity were confirmed by inspection of probability plots and error structures. All analyses were performed in R (version 3.0.2) with models fitted by maximum likelihood in the 'lme4' package (R Development Core Team 2013).

Dispersal expression

To assess the effect of an increase in the proportion of deutonymphs in the population on the population biology parameters (asymptotic growth rate (λ_0), generation time (T) and lifetime reproductive success (R_0)), the transition probabilities of developing from a protonymph to a deutonymph (deutonymph expression) were increased in the IPM from 0.1% to 100% in 10% intervals. For each interval, the population biology parameters (λ_0, T, R_0) were then recalculated to assess the effects of this increase; the influence of these effects will partly be due to the change in population structure (i.e. proportion of the population that

become deutonymphs) and costs of the deutonymph stage (e.g. smaller size, increased age at maturity).

In addition, in order to determine if changes in life history variables were indeed due to the effects of the deutonymph stage and not just due to the inclusion of an additional life stage, two dispersal IPMs (i.e. IPMs including a dispersal stage) with increasing proportion of deutonymphs were constructed. The first was constructed with demography functions parameterised from dispersing and non-dispersing individuals and the second from only non-dispersing individuals (see above for details of non-disperser parameters). A difference between the population biology values calculated from the two IPMs (difference-value) would indicate a negative effect on the life history variables due to pre-departure costs of the deutonymph stage (e.g. smaller size, increased age at maturity). This approach provides similar information to that of a life table response experiment (Caswell 2001).

Perturbation analysis

The perturbation analysis consisted of three steps; 1) perturbing each function parameter independently by a small amount and examining how much each matrix element changed by, 2) perturbing each matrix element independently and examining how much population growth rate (λ_0) changed by and 3) multiplying step one and two together and taking the sum over all matrix elements to generate the final perturbation values.

In step one, all parameters were perturbed upwards; positive values were multiplied by 1.001 and negative values by 0.999. By altering the parameter values of each function, within the IPM we alter the transition rates. However, we not only have to know how the transition rates are affected by altering the function parameters but also how altering each transition rate affects the population biology parameters. If we focus on one parameter, say

asymptotic growth rate (λ_0), and see how this changes when parameters are altered as mentioned above, this requires a number of considerations. Firstly, consider an arbitrary function parameter to be ω and, by changing this parameter whilst holding all other model parameters at their original value, any change in λ_0 as a result of changing ω is a partial derivative: $\frac{\partial\lambda}{\partial\omega}$. However, when we change a function parameter in the model we change many transition rates a_{ij} within the matrix approximation of the IPM. So in order to calculate $\frac{\partial\lambda}{\partial\omega}$ we need to know not only how altering ω changes each element in the matrix but also how changing each of these matrix elements changes λ_0. This is step two, and we can write the effect of altering a single element, a_{ij} (transition rates), in the matrix on λ_0 as a partial derivative $\frac{\partial\lambda}{\partial a_{ij}}$ and the effect of altering ω on a single matrix element as $\frac{\partial a_{ij}}{\partial\omega}$.

Finally, in the third step, we multiply these two effects together and take the sum over all matrix elements (Caswell 2001):

$$\frac{\partial\lambda}{\partial\omega} = \sum_{ij} \frac{\partial\lambda}{\partial a_{ij}} \frac{\partial a_{ij}}{\partial\omega} \qquad \text{(Eq. 4.1)}$$

This will generate the effect of perturbing function parameters on the population biology parameter in question.

Results

Model performance

We compared the population biology values derived from the IPMs to those derived from the data. Generally there was a good match between the population biology values for the dispersal and non-dispersal IPM of the mite populations at equilibrium (Table 4.2). The

Table 4.2. Population biology values generated from the IPM (dispersal IPM: Deutonymphs, Predicted; non-dispersal IPM: No Deutonymphs, Predicted) and from raw data (dispersers: Deutonymphs, Observed; non-dispersers: No Deutonymphs, Observed). Values generated were: population growth rate (λ_0); generation time (T); lifetime reproductive success (R_0); mean (Z) and variance (σ^2) of body size in μm (E – eggs, L – larva, P – protonymph, D - deutonymph, TD – dispersal tritonymphs, TP – non-dispersal tritonymphs, AD – dispersal adults, AP – non-dispersal adults). NA – Not Available.

	Deutonymphs		No Deutonymphs	
Quantity	Predicted	Observed	Predicted	Observed
λ_0	1.26	NA	1.30	NA
R_0	56.74	66.24	76.19	66.18
T	17.54	14.24	16.70	14.21
Z_E	0.163	0.176	0.164	0.176
Z_L	0.242	0.256	0.219	0.256
Z_P	0.346	0.383	0.309	0.383
Z_D	0.314	0.299	NA	NA
Z_{TP}	0.478	0.561	0.428	0.561
Z_{TD}	0.400	0.464	NA	NA
Z_{AP}	0.788	0.848	0.767	0.848
Z_{AD}	0.685	0.766	NA	NA
σ^2_E	0.00014	0.00069	0.00019	0.00069
σ^2_L	0.00065	0.00120	0.00116	0.00120
σ^2_P	0.00311	0.00192	0.00205	0.00192
σ^2_D	0.00428	0.00045	NA	NA
σ^2_{TP}	0.00586	0.00550	0.00612	0.00550
σ^2_{TD}	0.01370	0.01300	NA	NA
σ^2_{AP}	0.00608	0.00686	0.00824	0.00686
σ^2_{AD}	0.01160	0.00371	NA	NA

observed variance (σ^2) in body size of each life stage is calculated as the average of the squared differences from the mean. In the case of the predicted values, σ^2 is calculated as the expected size, with respect to the normalized stable size distribution, subtracted by the square of the mean size (Z).

Effects on character demography functions

After parameterising the character demography functions, the predicted estimates of the character-demography functions differed between dispersers and non-dispersers at the adult and tritonymph stage (Fig. 4.2). Additionally, the deutonymph transition rate resulted in a logit function with a significant quadratic term (body size^2) (quadratic logit) (Fig. 4.2). Typically, however, transition rates between life stages in acarid mites are best described by a linear logit function (Ozgul et al. 2012; Smallegange et al. 2014). As such, to be able to generalise our results to other species we ran additional analyses but with the deutonymph transition rate fitted using a linear logit function (linear logit) (Fig. S1). In addition, the deutonymph function with a quadratic logit may contribute to a large extent to the approximation of the IPM and so a deutonymph transition rate with a linear logit would allow a comparison between the two (see below). The two transition rates differed in rates for smaller individuals. The quadratic deutonymph transition rates for small individuals were low, reducing to almost zero, for intermediate sizes and then increasing sharply to high rates for larger individuals. In the case of the linear deutonymph transition rates, small individuals had transition rates very close to zero which increased with individual size to very high rates for large individuals (Fig. S1).

Population level effects

Asymptotic population growth rate (λ_0) and mean lifetime reproductive success (R_0) are lower and generation time (T) higher in populations with dispersers than in populations without dispersers (Table 4.2). The quantities λ_0 and R_0 are lower because dispersers have

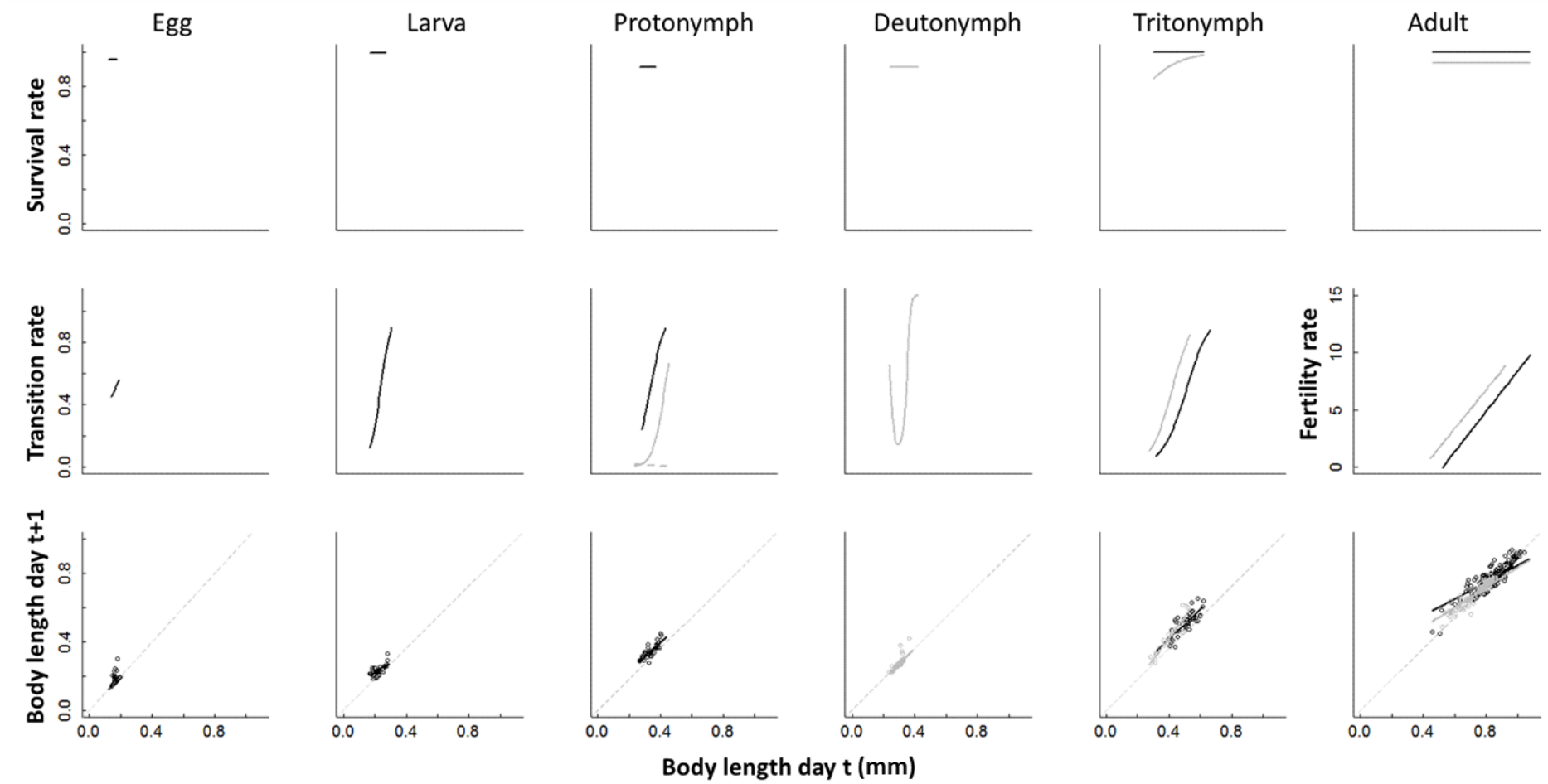
Egg
Larva
Protonymph
Deutonymph
Tritonymph
Adult
Survival rate
Transition rate
Fertility rate
Body length day t+1
Body length day t (mm)
0.0
0.4
0.8
0
5
10
15

Figure 4.2. Character demography functions of each of the six life stages showing the relationship between body length and growth, survival, transition rate (moving to next life stage), fertility rate and mean growth rate (when staying in same life stage). Functions are used to parameterise the dispersal IPM (grey lines and points) and the non-dispersal IPM (black lines and points). For the protonymph stage in the dispersal IPM, the transition function is a multinomial logistic and so has three probabilities. These probabilities are indicated by the solid grey line (probability of remaining in the protonymph stage), the dashed grey line (transition rate to the deutonymph stage), and the solid black line (transition rate to the tritonymph stage). The inheritance function and growth when growing into the next life stage for both IPMs are not shown (inheritance: egg size was not dependent on maternal size so mean and variance of egg size at t+1 are constant; growth: growth between size t and t+1 is described by growth rate of stage s+1 as in this figure). Points are raw data and lines are predictions from regressions. The size of the smallest and largest individual observed within each life stage determined the minimum and maximum size of the size domain of each stage.

lower adult survival rate and lower total adult fertility rate compared to non-dispersers (the size range of non-disperser adults producing eggs is higher than that of disperser adults). The combination of the reduced fertility and survival, along with the additional life stage during disperser development results in an increase in generation time (T).When increasing the proportion of deutonymphs in populations with dispersers, λ_0 and R_0 are reduced further and there is a simultaneous increase in T. Changes in population biology values with increasing proportion of deutonymphs are likely due to the increase in maturation time as a result of the extra time spent in the deutonymph life stage. In order to show that the changes we see in λ_0, R_0 and T are indeed due to costs of investing in the deutonymph stage, and not due the extra time spent in the deutonymph life stage, we compared the difference-value of the two dispersal IPMs. The two IPMs have the same number of life stages and so any difference in population biology values would indicate that change is due to costs of investing in the dispersal stage (Fig. 4.3).

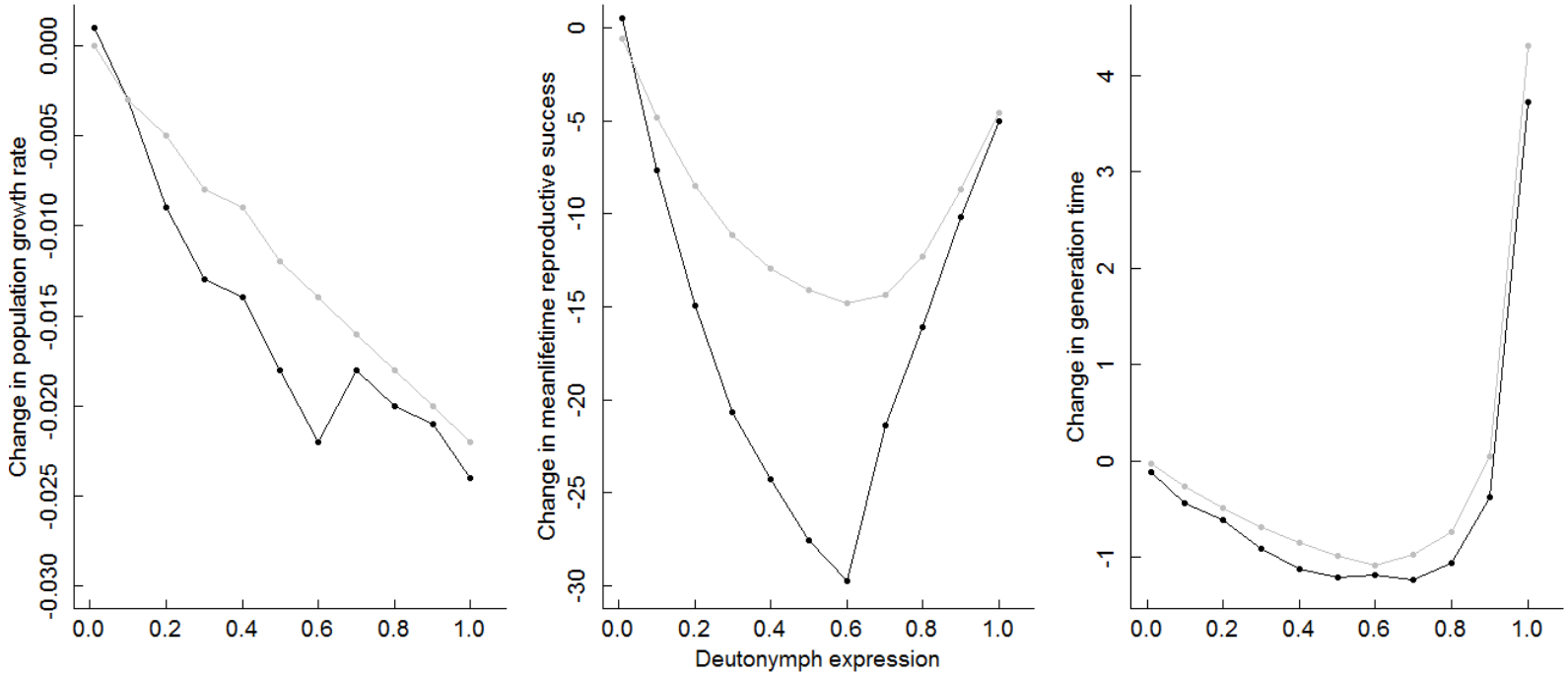

Figure 4.3. Difference (Δ) in population growth rate (λ_0), mean lifetime reproductive success (R_0) and generation time (T, days) with increasing deutonymph expression from (0.01% - 100% in 10% increments). The change is the difference between population biology values generated by an IPM including the deutonymph stage and only non-disperser function parameters (i.e. no cost of deutnoymph stage and increase in life stages) and that of an IPM with the deutonymph stage and function parameters from dispersers (i.e. cost of deutonymph stage and increase in life stages). Black lines and points based on a quadratic logit deutonymph transition function in the IPM, grey lines and points based on a linear logit deutonymph transition function in the IPM (see text for details).

Population biology values differed between the two dispersal IPMs with the difference-value increasing negatively with increased proportion of deutonymphs. However, after deutonymphs reach proportions of 70% there is an opposite response in the difference for the R_0 and T values where the difference-values start increasing, but not for λ_0, between the two IPMs. Furthermore, the difference in T approaches zero by deutonymph proportions of 90% and then increases positively at 100%. The same differences in population biology values between the two dispersal IPMs were calculated with IPMs which had a linear deutonymph transition function. This was done to test whether the results we found were not due to the non-linearity of the deutonymph transition function and how it affected the approximation of the IPM; an affect which may be specific to our study system. Results for the linear deutonymph transition function were similar with the exception of the change in R_0 which, although significant, was not as strong (Fig. 4.3).

Changes in mean body size of the population were limited to tritonymphs and adults (Table 4.2). Between dispersal and non-dispersal IPMs, there were larger non-dispersers in the dispersal IPM compared to individuals in the non-dispersal IPM. Within the dispersal IPM, there were differences in mean size between tritonymphs and adults of non-dispersers and dispersers. Dispersers were smaller in both cases.

Perturbation analysis

After multiplying the partial derivative matrices and summing the matrix elements we can examine how perturbing the function parameters in the model influence λ_0. Of all character-demography functions, two were most influential to λ_0: adult fertility rate and offspring inheritance of non-dispersers. Of each of these

functions, perturbation of the intercept of the offspring inheritance function and the intercept and slope of the reproduction function had the largest positive effect when the proportion of deutonymphs is low (<1%) (Fig. S2). When increasing the proportion of deutonymphs, adult fertility parameters and offspring inheritance parameters of non-dispersers were again influential to λ_0 sensitivity (the same parameters as above again had the largest positive effect) but this effect, although positive, steadily declined when the proportion of deutonymphs increased. However, with an increase in proportion of deutonymphs, sensitivity of λ_0 to the adult fertility parameters (intercept and slope) of dispersers increased, with the positive effect steadily increasing and becoming more positive than non-dispersers. In addition, perturbed parameters of the deutonymph transition function steadily had an increasing negative effect on the sensitivity of λ_0 as the proportion of deutonymphs increased (Fig. S2). The relative changes of adult fertility parameters and offspring inheritance parameters with increasing proportion of deutonymphs resulted in a shift of sensitivity of population growth rate from non-disperser parameters to disperser parameters (crossing of the dashed diagonal line, Fig. 4.4).

For IPMs with a linear logit deutonymph transition function (see *Effects on character demography functions*) we found the same results except that, with increasing proportion of deutonymphs, λ_0 was not sensitive to perturbation of the deutonymph transition parameters (Fig. S3). Importantly there was still a relative change in sensitivity of λ_0 to the adult fertility parameters, although less pronounced, with increasing proportion of deutonymphs resulting in a shift of sensitivity of λ_0 from non-disperser functions to disperser functions (Fig. 4.4).

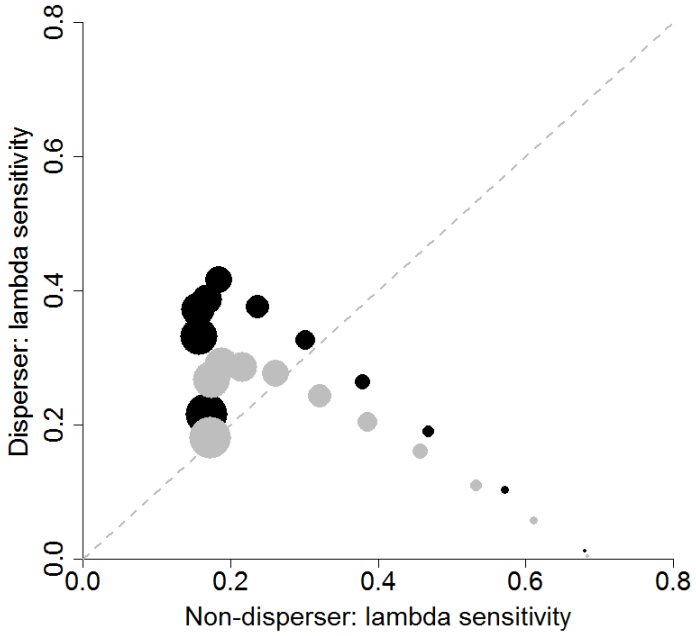

Figure 4.4. Shift in sensitivity, of population growth rate to parameters of the fertility rate function, from non-disperser functions to disperser functions. Circles are highest sensitivity values of population growth rate to fertility parameters of dispersers as a function of the highest sensitivity values of population growth rate to fertility parameters of non-dispersers. Size of the circles indicate increased deutonymph expression (0.01% - 100% in 10% increments). Black circles indicate bulb mite specific values (quadratic logit deutonymph transition function in the IPM), grey circles indicate values that are indicative of general species (linear logit deutonymph transition function in the IPM) (see text for details).

Discussion

Studies of the consequences of dispersal usually investigate the role of dispersal at the point of settlement (e.g. patch colonisation, Hanski 1999; Bowler and Benton 2005) and how dispersal can influence the meta- population (Bowne and Bowers 2004; Lecomte et al. 2004) and community dynamics (Loreau et al. 2003; Gouhier et al. 2010;

Steiner et al. 2011). However, as far as we know, this is the first study to investigate effects of the presence of unsuccessful dispersers on the natal population. Costs of unsuccessful dispersers will affect the trade-offs that determine the dispersal strategy when individual costs carry over to affect population level quantities. Here we used IPMs to capture the cost of investing in a dispersal stage to the natal population when dispersers do not disperse. In a previous study it has been shown that investment in the deutonymph stage resulted in costs to several life history traits of individual bulb mites; dispersing individuals were smaller, matured later and females had lower lifetime reproductive success (Deere et al. in prep, Chapter 3). These effects to post-deutonymph life-stages suggests that the costs to individuals that are unable to disperse successfully are immediate in this species.

Given that deutonymph expression is costly, one would expect these costs to be reflected at the population level. This is indeed what we found. When looking at mean body size in the population, dispersers were on average smaller as a tritonymph and as an adult than non-dispersers (Table 4.2). However, when comparing tritonymph and adult non-dispersers from a population with dispersers to one without dispersers, non-dispersers were larger in the population with dispersing individuals. This is driven by population structure and the development rate of deutonymphs from protonymphs. Only protonymphs within a certain size range develop into deutonymphs; large protonymphs do not develop into deutonymphs (Protonymph: min. length = 0.234µm, max. length = 0.455µm; Deutonymph: min. length = 0.237µm, max. length = 0.390µm). This affects the average tritonymph (and ultimately adult) body size for non-dispersers as fewer, smaller non-dispersing individuals will develop into tritonymphs if there are deutonymphs in the population. There is also an effect of increased generation time

because of the additional deutonymph life stage; this, and the fact that dispersers have lower growth rate, survival and fertility ultimately results in a reduction in the number of recruits to the population (R_0), with recruits having lower fertility. This can be inferred by comparing population biology values of the two IPMs that both had the same number of life stages but parameterised with different life history functions (one using disperser functions and one using non-disperser functions). The difference-value calculated indicates that the negative effect on the population biology values are not solely due to the addition of a life-stage during development but also due to the costs incurred by individuals that invest in the deutonymph stage during development which are reflected in the vital rates of the IPM. Importantly, these effects are seen at a low proportion of dispersal expression; with the proportion of deutonymphs at <1%, there is a reduction of population growth rate and mean lifetime reproductive success. This suggests that, when the environment is not in a poor condition, populations incur a cost if dispersal morph expression is (partly) probabilistic.

When increasing the proportion of deutonymphs in the population, the consequences for the different population biology values are enhanced. An increase in the proportion of deutonymphs increased the cost (difference-value), however when the proportions reach ~60% the shape of the graphs that characterise the difference in population biology values change (Fig. 4.3). We attribute this to population heterogeneity. In a heterogeneous population, the mean trait value of concern can be qualitatively different from the values, of the same trait, that make up the subpopulations that comprise the heterogeneous population. This trait dynamic in a heterogeneous population has been shown before (e.g. mortality rates in human populations, Vaupel 2010). The dispersal IPMs constructed can be considered as a

heterogeneous population in that adults fall into two sub-groups: one composed only of non-dispersers and one composed only of dispersers. Each group would contribute differently to the mean population biology values depending on their frequency in the IPM. As the proportion of deutonymphs increases the number of disperser adults increase and non-disperser adults decrease. This, in turn, changes the contributions of each adult group to the population biology values. The mean value of the whole population will then follow a trajectory similar to the value of the sub-group which has the highest number of adults. In all cases it must be noted that the IPMs generated here can only make predictions on population growth and structure (e.g. stable stage distribution) but cannot inform on population dynamics (e.g. stability/chaos). What could also not be explored with these IPMs are how costs of unsuccessful dispersal feeds back to affect individual size and deutonymph initiation. For example, the separate IPMs generated for all proportions of dispersers only gives an indication of the structure of the population given the values of the parameters of the different demographic functions. If dispersal in our study was density-dependent (and not determined by environmental quality), then potential feedbacks of the costs of unsuccessful dispersal can be included. This could be done using density-dependent IPMs where density is included as a term in the fundamental functions such that vital rates dependent on population density.

To determine the relative importance of different life history stages on population growth rate, we altered the parameter estimates of the character-demography functions and their variance. Specifically we focused on how sensitive the population growth rate was to perturbations of the parameter estimates. Population growth rate was most sensitive to the parameters of the adult reproduction and inheritance functions.

Importantly, sensitivity changed with increasing proportion of deutonymphs. At low proportions of deutonymphs the population growth rate is most sensitive to non-disperser reproduction parameters and as proportions of deutonymphs increase there is a shift to disperser reproduction parameters in the sensitivity of population growth rate. Sensitivities can inform on the selection pressure on an organism's life history (Benton and Grant 1999). In short, selection on a vital rate occurs when there is a change in a vital rate with a concurrent change in fitness, with the selection proportional to the change in fitness (Benton and Grant 1999). As such, the shift we see is a shift in the selection pressure from reproduction in non-dispersers to reproduction in dispersers. An alternative cause of the shift could be due to the increase in disperser individuals when the proportion of deutonymphs increases. However, as proportions of deutonymphs reaches values above 60%, sensitivity shifts back to non-disperser reproduction. Indeed, in addition to the sensitivity shift in reproduction, sensitivity of population growth to the parameters of the deutonymph transition function was increasingly negative when the proportion of deutonymphs increased. The increase in proportion of deutonymphs not only increases the number of dispersing individuals but also changes the stable stage distribution. Therefore, it seems that the combination of a changing stable stage distribution (for dispersers and non-dispersers) and the change in number of dispersers is most likely the cause of the shift back to non-disperser reproduction. Consequently, the proportion of deutonymphs not only influences the size of the shift in the selection pressure but the direction of the shift as well. Our study shows changes in natal population biology values and changes in the sensitivity of population growth rate when dispersers are present in low and high numbers in the population. Variation in disperser numbers that remain in the natal population, to the extent we investigated, is plausible

as dispersal in the bulb mite is via phoretic associations with arthropod hosts (Diaz et al. 2000). This host dependence can bring about the variation in disperser numbers when host species fluctuate in presence and density.

Our ability to generalise our findings to other species, with similar life histories to the bulb mite, depends largely on how similar our character demography functions are to those of other species. As the deutonymph transition function in our model (henceforth "bulb mite IPM") has an atypical hump-shape (Fig. 4.2), in order to have a realistic alternative for the transition function we removed the quadratic term of the function and re-ran the analysis (henceforth "general IPM") (Fig. S1). The analysis of the "general IPM" resulted in population biology values similar to the "bulb mite IPM". By altering the shape of the deutonymph transition rate in the IPM we see a similar change in life history responses and population biology values when compared to the "bulb mite IPM". The similarity of the results, therefore, allow generalisation to other species with similar life histories. Most models for dispersal have focused on either how resident populations are affected by dispersal rate (or distance) and ultimately metapopulation dynamics (Clobert et al. 2001; Hanski 2001); or the evolution of dispersal (McPeek and Holt 1992; Holt and McPeek 1996; Doebeli and Ruxton 1997; Clobert et al. 2001; King and Roff 2010). In both instances a number of different models have been used to address these issue (e.g. matrix population models, individual based models, differential equation models) (McPeek and Holt 1992; Doebeli and Ruxton 1997; Clobert et al, 2001; King and Roff 2010; Strevens and Bonsall 2011; Travis et al. 2012). This study focuses on the effects on the natal population only; we see this as the first step of the effects of unsuccessful dispersal. To the best of our knowledge, this is also the first time that such natal population costs have been

quantified, neither using IPMs nor other population models. The model presented here could form a basis for the structure of populations within a metapopulation model. The model is structured in a way that dispersal rates will be calculated from only disperser individuals in the natal populations while also accounting for unsuccessful dispersers that remain in the natal habitat.

Changes in population biology values will ultimately alter the dynamics of populations over time, whereby the magnitude of this change in dynamics depends on the level of dispersal expression (i.e. the distribution of the dispersal phenotype). Locally, dispersal has been shown to not only ensure persistence in a population, by increasing mean population density, but can also change the population dynamics of persistent populations (Coffman et al. 2001; Ives et al. 2004; Lecomte et al. 2004). Furthermore, dispersal success has been shown to be influenced by the interaction of habitat suitability and habitat fragmentation (O'Sullivan et al. 2014). Here we suggest that dispersing individuals that fail to disperse may affect the population dynamics of persistent natal populations, especially in fragmented landscapes. Consequently, how the dynamics of natal populations are affected could change the connectivity of a group of habitat patches which has important implications for how connectivity is measured in natural populations. In a meta-population context, the contribution of dispersal to population persistence and synchrony will then be two-fold: 1) contribution of dispersers that disperse away from their natal population (Bowler and Benton 2005) and 2) contribution of dispersers that fail to disperse and remain in their natal population (this study). The effect we see on natal populations may not necessarily be restricted to dispersal. Other forms of movement with distinct phenotypes, such as partial migration, may experience similar population level effects. Partial migration occurs when

individuals within a population differ in their migratory behaviour (within-population migratory dimorphism); a population contains both migratory and sedentary individuals (Adriaensen and Dhondt 1990; Kaitala et al. 1993; Brodersen et al. 2008; Chapman et al. 2011). Often, migratory and sedentary individuals differ in traits other than migratory behaviour (e.g. size and dominance). These differences can lead to differences in vital rates which ultimately effect the population. Given this, it is feasible that individuals that initiate migration but do not migrate will ultimately affect the natal population. Individuals that are large often outcompete smaller individuals or survive better in harsh overwintering conditions than smaller individuals, with the smaller individuals migrating (Chapman et al. 2011). If these individuals fail to migrate, however, there may be a similar effect, as in our study, on the population.

When compared to how the contribution of dispersal is currently included in metapopulation models the new, combined, contribution of dispersal will change how individuals move between populations. The evolution of dispersal can depend on metapopulation conditions (Bonte and de la Pena 2009; Bonte et al. 2010; Cotto et al. 2014) and given the importance of understanding the multiple effects of dispersal rates (Hanski 2001; Abbott 2011), it is key that this combined contribution of dispersal is incorporated into future metapopulation models.

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Supporting Information

Supporting methods Parameter estimation and parameterisation of IPM

Figure S1 Comparison of the two deutonymph transition rates

Figure S2 Perturbation analysis with a quadratic term included in the deutonymph transition function

Figure S3 Perturbation analysis with a linear term and no quadratic term included in the deutonymph transition function

Supporting methods

The first section describes the methods used to parameterise the functions for non-dispersers (following Smallegange et al. 2014); we also give the parameter estimates for all the character-demography functions for the dispersal and non-dispersal IPMs. After this are four figures. Figure S1 shows the deutonymph transition rate used in the dispersal IPM. Figure S2 and S3 show the sensitivity analysis for population growth rate for an IPM with a deutonymph transition rate including the quadratic term and an IPM with a deutonymph transition function excluding the quadratic term respectively.

Parameter estimation: non-dispersers

Function parameters were estimated using the following statistical models were used: (1) Survival - generalised linear mixed model (GLMMs) with binomial error structure; (2) Transition rates – GLMM with binomial error structure; (3) Growth – GLMM with a Gaussian error structure; (4) Fertility rate (Reproduction) – GLMM with a Gaussian error structure; (5) Inheritance kernel – generalised linear model (GLM) with a Gaussian error structure. In all cases body length and body length squared were linear predictors and, with the exception of the inheritance function where we fitted a GLM, mite identity was included as a random factor. The response variables for the five functions were: (1) Survival – from time t to time t+1 (this is binary and set as 0 or 1), (2) Transition - probability of growing to the next stage at time t+1, $\gamma_{s,t+1}$ (see below), (3) Growth - mean and variance in body size at time t+1, (4) Fertility - the number of eggs produced at time t+1, and (5) Inheritance - mean and variance in size of eggs

produced at time t+1 by each individual at time t. In the case of eggs their size at time t+1 equalled their size at time t as eggs do not increase in size.

During data collection it was not always possible to locate each individual every day. As such, the days where an individual was not seen but was still alive (i.e. observed alive the next day), body length was estimated (not including these observations would result in an underestimation of the survival function). The missing values were filled in by using the Gompertz function to estimate female body length at age a (Smallegange et al. 2014):

$$z_a = z_\infty e^{-e^{-k(a-a_0)}} \qquad \text{(Eq. S1)}$$

where Z_a is body length (mm) at age a (days), Z_∞ is the mean maximum length (mm, at $a = \infty$), k is the instantaneous growth rate at age a_0 , and a_0 is the inflection point of the curve and the age at which absolute growth rate begins to decline.

In the case of function (2), we used duration in the life stage as an indicator of the transitioning to the next stage (Smallegange et al. 2014); i.e. time spent in the current stage depends on the probability of growing to the next stage (Caswell 2001). Therefore, $\gamma_{s,t+1}$ is given by $\gamma_{s,t+1} = 1/d_{s,t}$, where $d_{s,t}$ is the number of days that an individual still has to spend in stage s. This means that d_s equals the total duration of stage s on the first day that a female is in stage s, and that when an individual develops from stage s into stage s+1 at time t+1, d_s=1 so that $\gamma_{s,t+1}$ =1.

For function (3) and (5) the minimal model, which generated the predictors of mean size at time t+1, was utilised to generate the parameters for the variance around the mean size at t+1 by taking the squared residuals and fitting them against a statistical function of the same form as the mean size to estimate the variance in size at time t+1. The growth (3) and inheritance functions (5) were then constructed using the equation following Easterling et al. (2000):

$$y_i = \frac{1}{\sqrt{2\pi\sigma_i}} e^{\frac{-(z' - \mu_i)^2}{2\sigma_i^2}} \qquad \text{(Eq. S2)}$$

where y_i is either the growth or inheritance function, μ_i describes the mean effect of the significant predictors on growth or body size inheritance, and σ_i describes the squared residuals around μ_i.

For details on construction the IPMs from these parameters see main text. Note that mite identity was included in the statistical analyses (except in the inheritance function) but was not modelled within the IPMs.

Parameter values of character-demography functions

Survival rates for the dispersal IPM (fraction per day)

E: $y = 0.956$ (n = 297); L: $y = 0.999$ (n = 112); P: $y = 0.910$ (n = 166); D: $y = 0.999$ (n = 426); TP: $y = 0.999$ (n = 132); TD: $y = \frac{1}{1 + \frac{1}{e^{(-0.4175+6.9435B)}}}$ (n = 119); AP: $y = 0.999$ (n = 115); AD: $y = 0.933$ (n = 60).

Survival rates for the non-dispersal IPM (fraction per day)

E: $y = 0.956$ (n = 297); L: $y = 0.999$ (n = 112); P: $y = 0.909$ (n = 166); T: $y = 0.999$ (n = 132); A: $y = 0.999$ (n = 115)

Life stage transition rates for the dispersal IPM (fraction per day)

E→L: $y = \frac{1}{1 + \frac{1}{e^{(-1.437+8.674B)}}}$ (n = 97); L→P: $y = \frac{1}{1 + \frac{1}{e^{(-6.933 + 29.429B)}}}$ (n = 47);

P→D: $y = \frac{1}{1 + \frac{1}{e^{(-2.601 + (-5.673)B)}}}$ (n = 137); P→T: $y = \frac{1}{1 + \frac{1}{e^{(-11.220 + 26.235B)}}}$ (n = 137); D→T: $y = \frac{1}{1 + \frac{1}{e^{(55.05-385.54B+654.02B^2)}}}$ (n = 155); $T_P \rightarrow A_P$: $y = \frac{1}{1 + \frac{1}{e^{(-6.703 + 13.100B)}}}$ (n = 76); $T_D \rightarrow A_D$: $y = \frac{1}{1 + \frac{1}{e^{(-6.275 + 14.933B)}}}$ (n = 45).

Life stage transition rates for the non-dispersal IPM (fraction per day)

E→L: $y = \frac{1}{1 + \frac{1}{e^{(-1.437 + 8.674B)}}}$ (n = 97); L→P: $y = \frac{1}{1 + \frac{1}{e^{(-6.933 + 29.429B)}}}$ (n = 47); P→T: $y = \frac{1}{1 + \frac{1}{e^{(-7.170 + 21.425B)}}}$ (n = 108); T→A: $y = \frac{1}{1 + \frac{1}{e^{(-6.703 + 13.100B)}}}$ (n = 76).

Reproduction rate for the dispersal IPM (no. per day)

A_P: $y = 0.5(-18.446 + 35.209B)$ (n = 190); A_D: $y = 0.5(-13.592 + 33.892B)$ (n = 172)

Reproduction rate for the non-dispersal IPM (no. per day)

$y = 0.5(-18.446 + 35.209B)$ (n = 190)

Mean growth rates for the dispersal IPM (when staying in the same life stage) (mm)

E: $y = L$ (n = 65); L: $y = 0.11739 + 0.64316B$ (n = 29); P: $y = 0.0772 + 0.904B$ (n = 39); D: $y = L$ (n = 153); T_P: $y = 0.0776 + 0.9538B$ (n = 44); T_D: $y = -0.0772 + 1.3570B$ (n = 23); A_P: $y = 0.3977 + 0.5359B$ (n = 215); A_D: $y = 0.2816 + 0.6355B$ (n = 238)

Variance in growth rates for the dispersal IPM (when staying in the same life stage) (mm^2)

E: $y = 0.0001$ (n = 65); L: $y = -0.0008 + 0.0050B$ (n = 29); P: $y = -0.0007 + 0.0040B$ (n = 39); D: $y = 0.0001$ (n = 153); T_P: $y = 0.0039 - 0.0042B$ (n = 44); T_D: $y = -0.0044 - 0.0060B$ (n = 23); A_P: $y = 0.0009 - 0.0004B$ (n = 215); A_D: $y = 0.0014 - 0.0016B$ (n = 238)

Mean growth rates for the non-dispersal IPM (when staying in the same life stage) (mm)

E: $y = B$ (n = 65); L: $y = 0.1174 + 0.6432B$ (n = 29); P: $y = 0.0772 + 0.9040B$ (n = 39); T: $y = 0.0776 + 0.9538B$ (n = 44); A: $y = 0.3977 + 0.5359B$ (n = 215)

Variance in growth rates for the non-dispersal IPM (when staying in the same life stage) (mm^2)

E: $y = 0.0001$ (n = 65); L: $y = -0.0008 + 0.0050B$ (n = 29); P: $y = -0.0007 + 0.0004B$ (n = 39); T: $y = 0.0039 - 0.0042B$ (n = 44); A: $y = 0.0009 - 0.0004B$ (n = 215)

Inheritance function (mean offspring-mother difference) for the dispersal IPM (mm)

A_P: $y = 0.1638$ (n = 96); A_D: $y = 0.1689$ (n = 175)

Variance around inheritance function for the good environment (mm^2)

A_P: $y = 0.00008$ (n = 96); A_D: $y = 0.0001$ (n = 175)

Inheritance function (mean offspring-mother difference) for the non-dispersal IPM (mm)

$y = 0.1638$ (n = 96)

Variance around inheritance function for the non-dispersal IPM (mm^2)

$y = 0.00008$ (n = 96)

Supporting figures

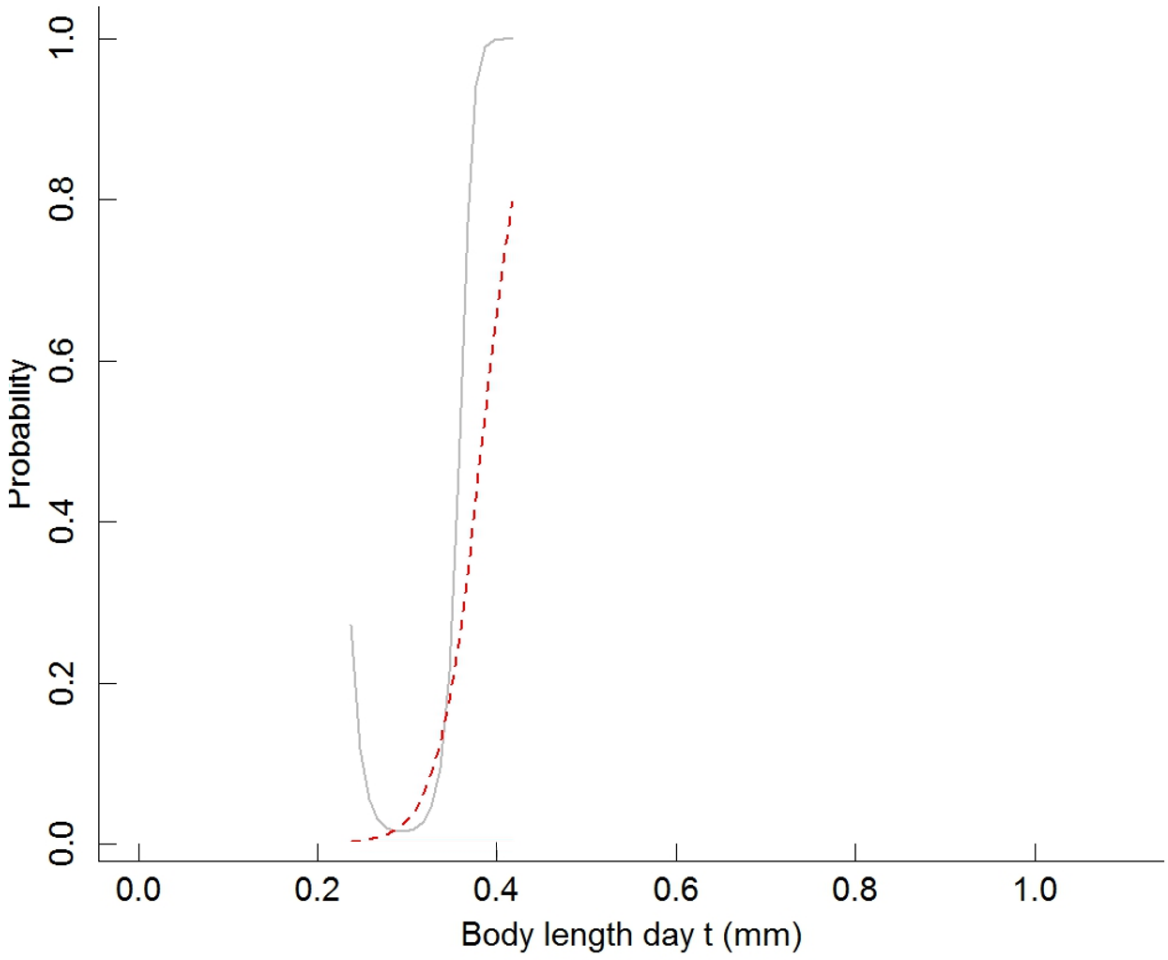
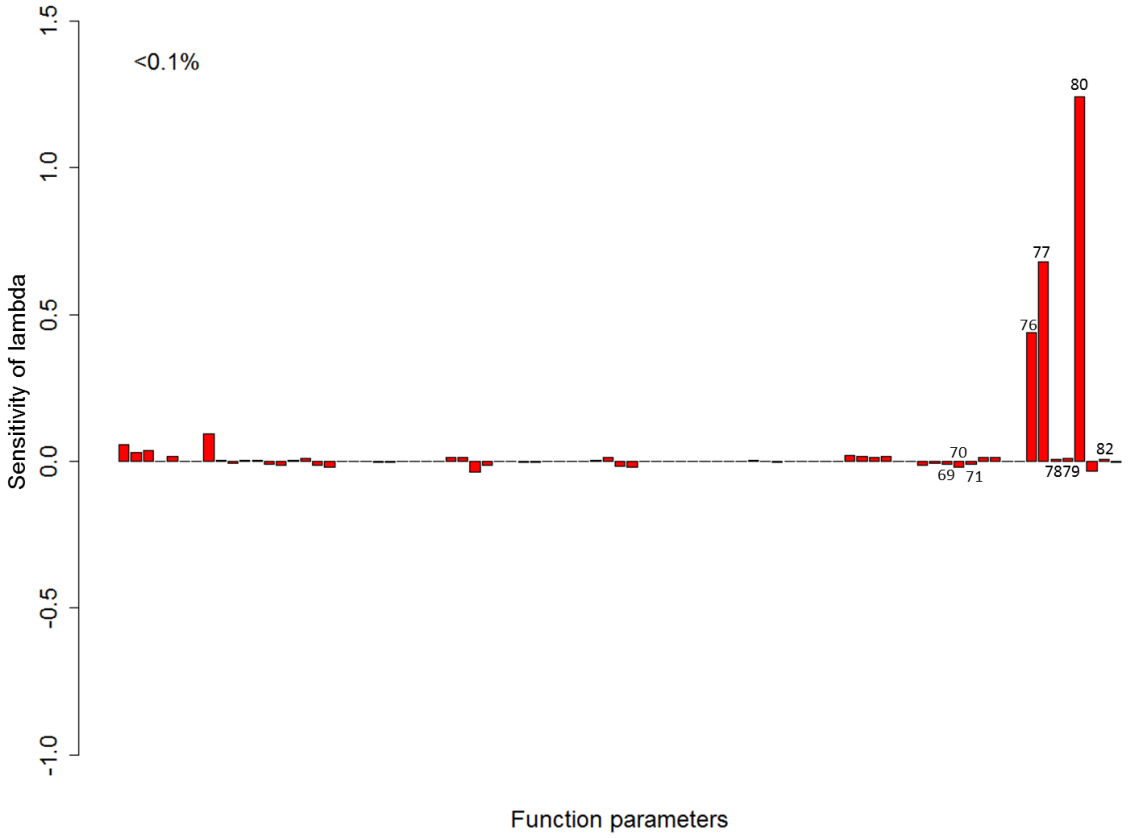


Figure S1. Deutonymph transition rate used in the dispersal IPM. Solid line indicates a logit function with a quadratic term, parameterised from dispersal life history data (quadratic logit transition function). Dashed line is a logit function without a quadratic term fitted to dispersal data which would be more representative of species with a similar life history to that of the bulb mite (linear logit transition function) (see text for details).

Figure S2. Perturbation analysis. Sensitivity of population growth rate (lambda/λ_0) to each of the parameters of each of the statistical functions used to construct the integral Projection Model (IPM) (83 parameters in total) (see text for details). In all cases the deutonymph transition function includes the quadratic term (quadratic logit). Sensitivities were calculated for increasing deutonymph expression from (A) <0.1% to (K) 100%. Numbers indicate the various function parameters (only parameters which change significantly with increasing deutonymph expression shown): 69 – 71 (deutonymph transition function); 76 – 77 (non-disperser reproduction function); 78 – 79 (disperser reproduction function); 80 (non-disperser offspring inheritance function); 82 (disperser offspring inheritance function).

(A)


Sensitivity of lambda
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1.0
0.5
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-1.0
<0.1%
80
77
76
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69
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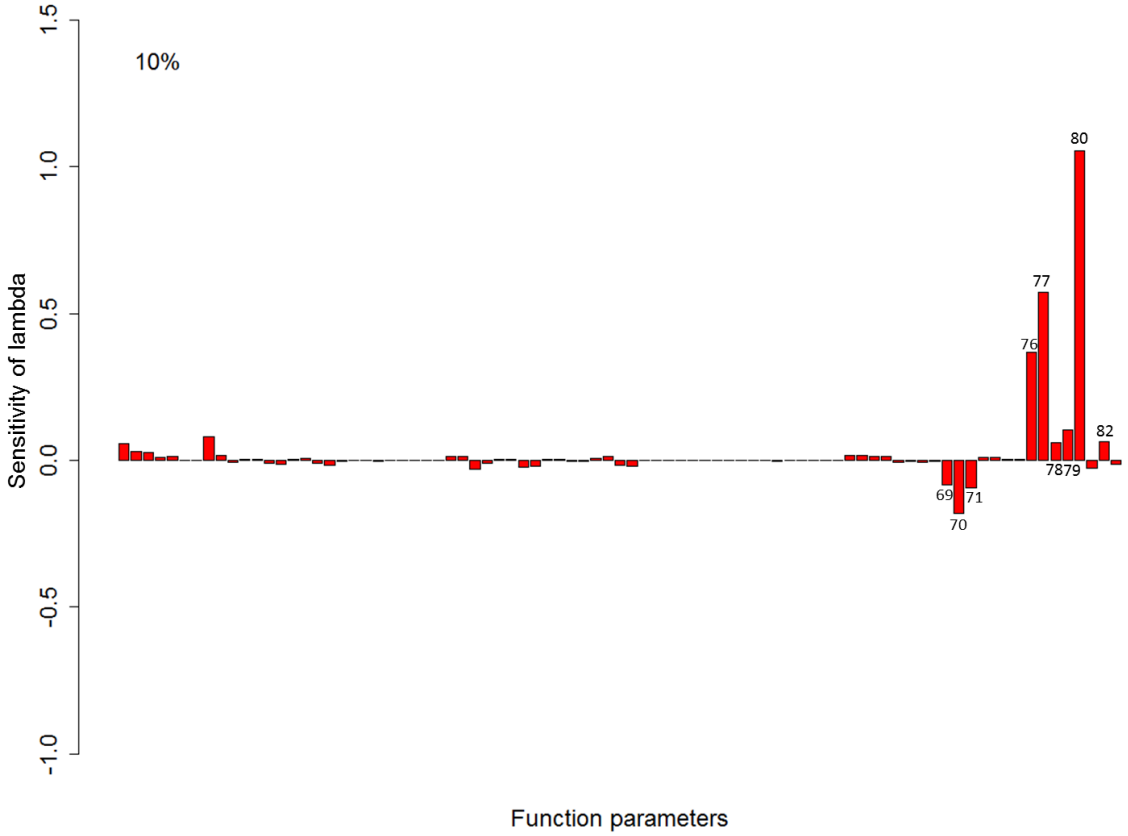
78
79
82
Function parameters

(B)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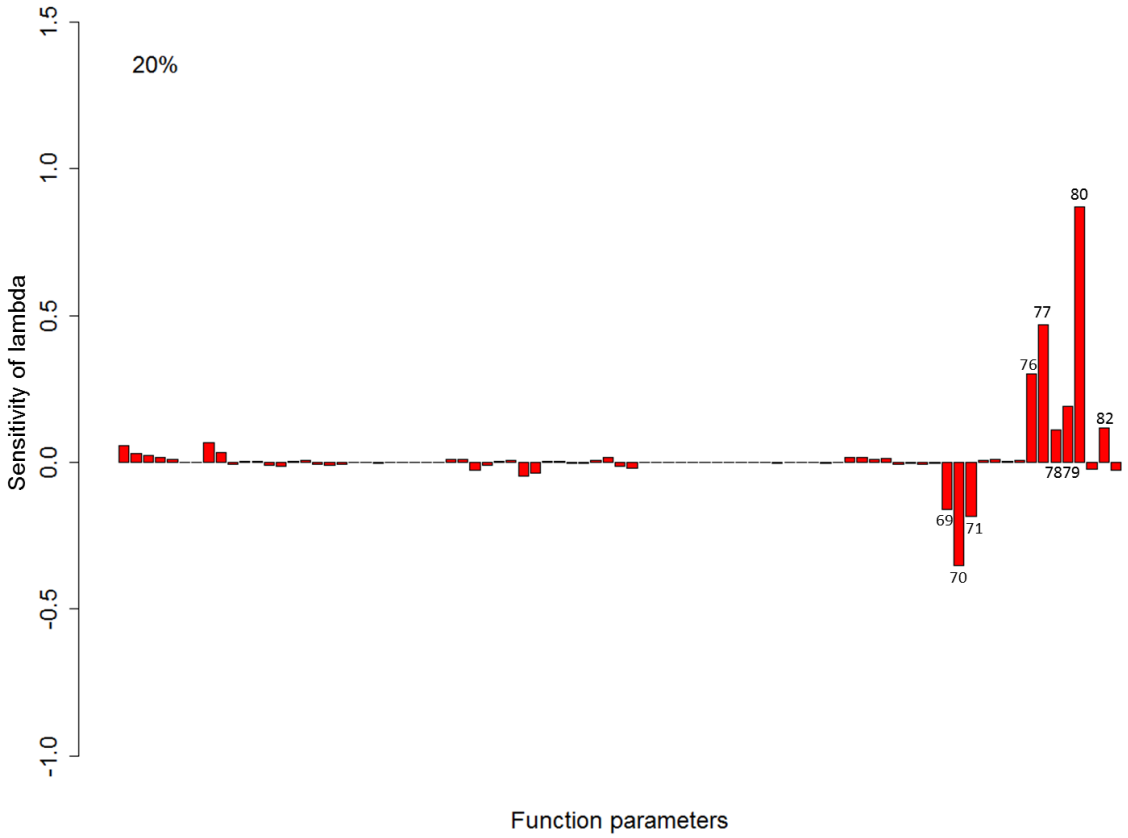
Function parameters

(C)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7879
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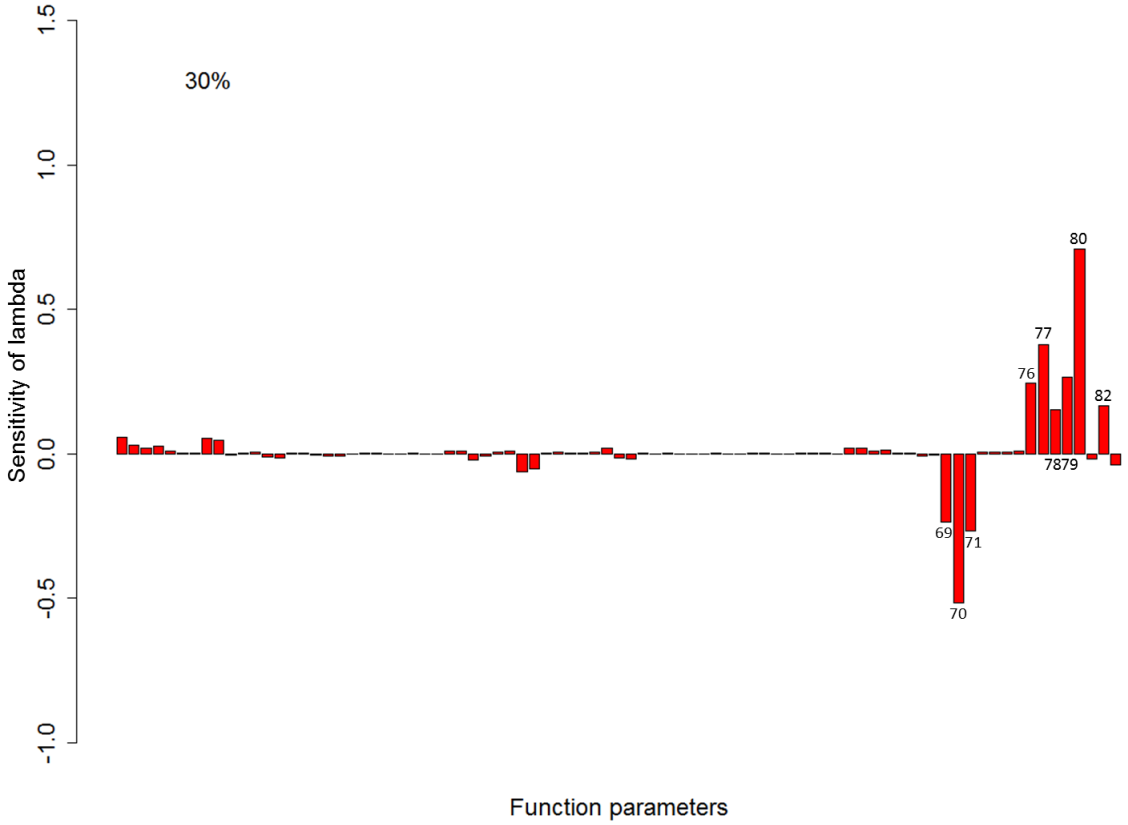
Function parameters

(D)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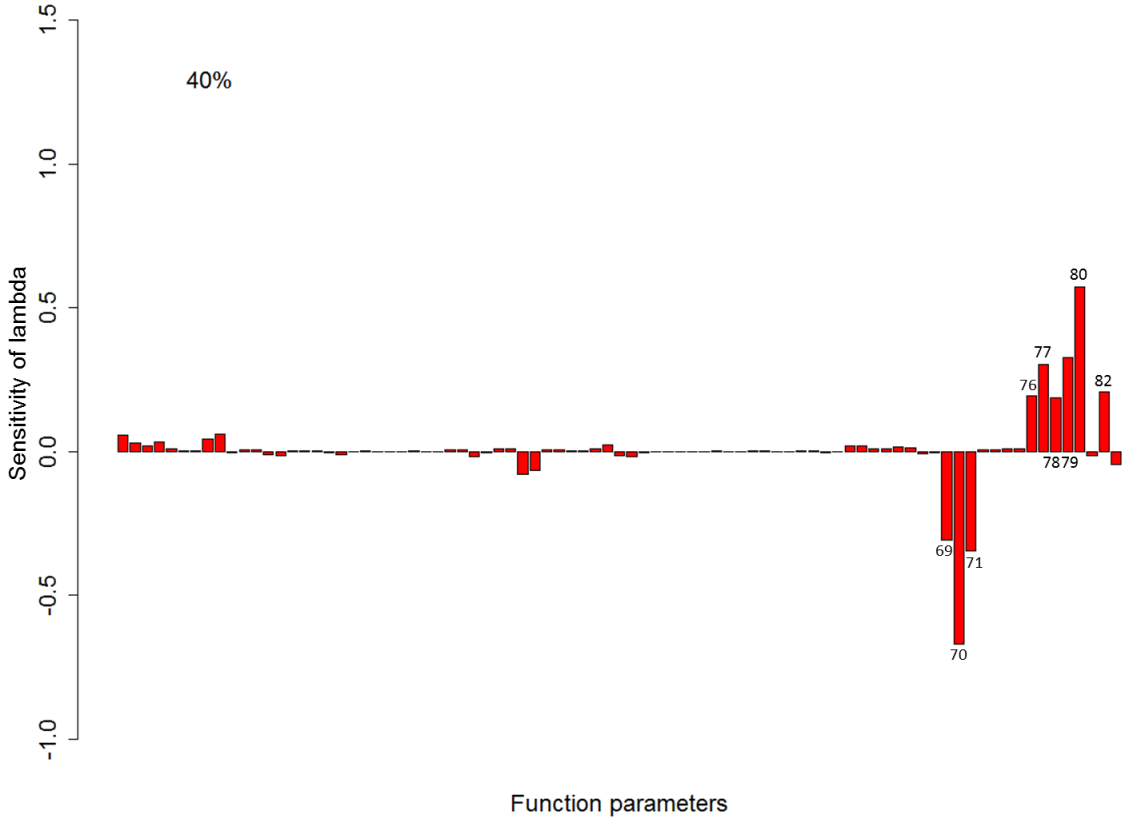
Function parameters

(E)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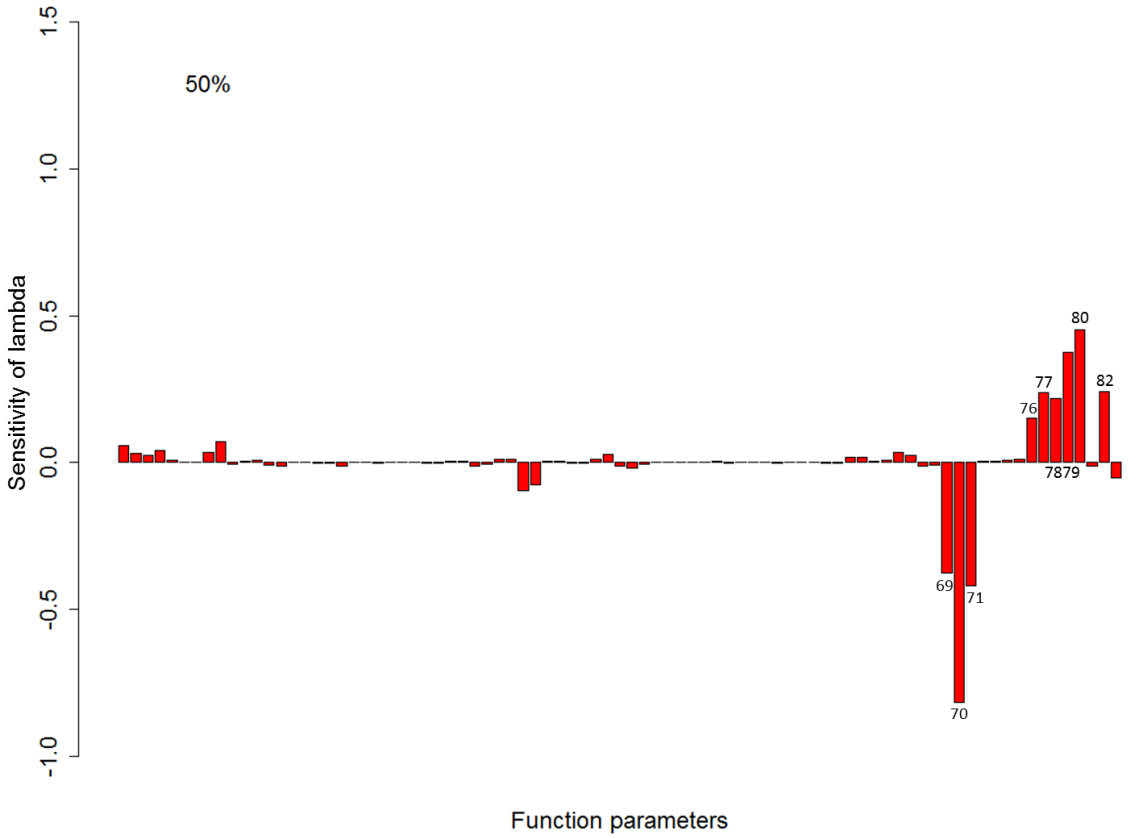
Function parameters

(F)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7879
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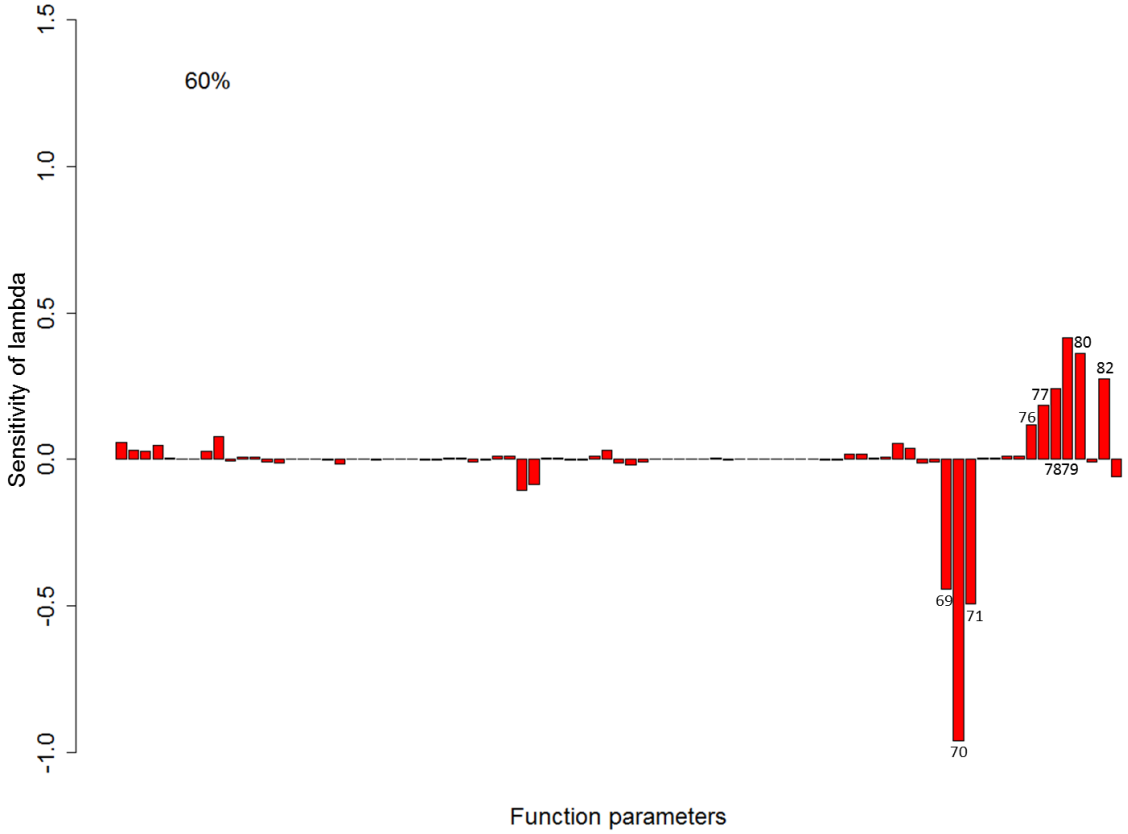
Function parameters

(G)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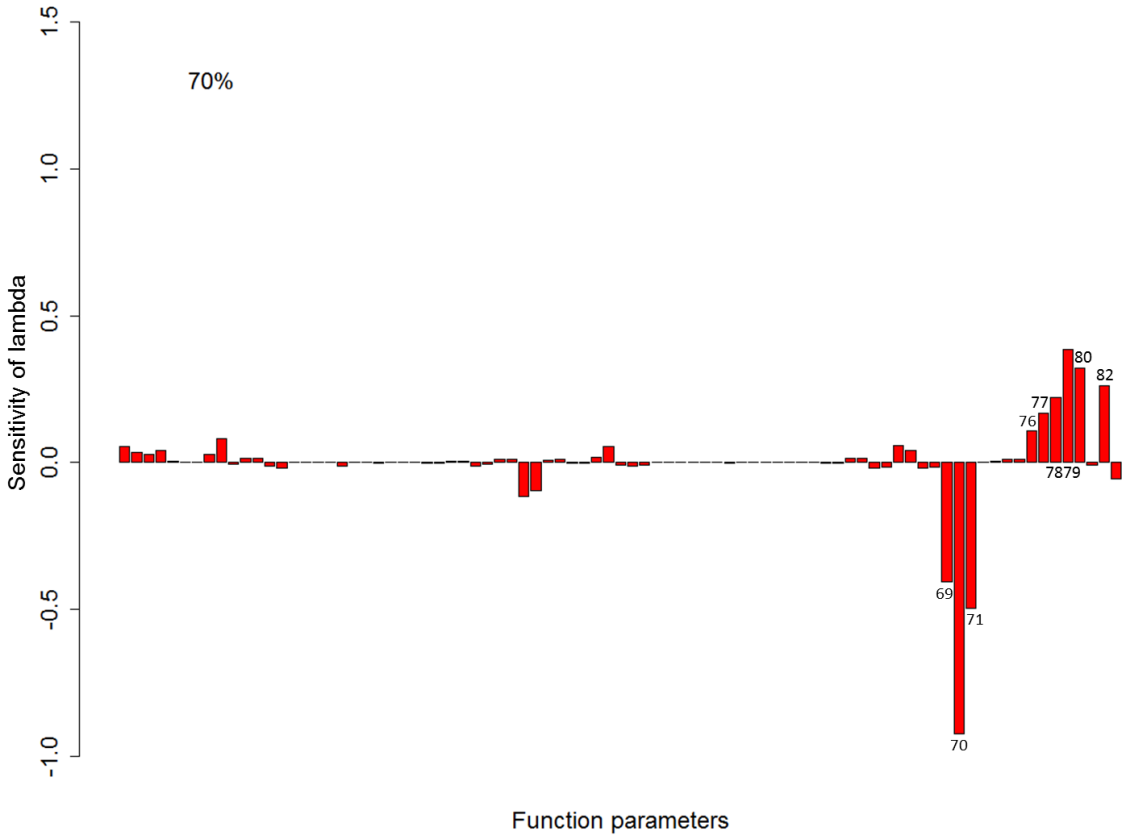
Function parameters

(H)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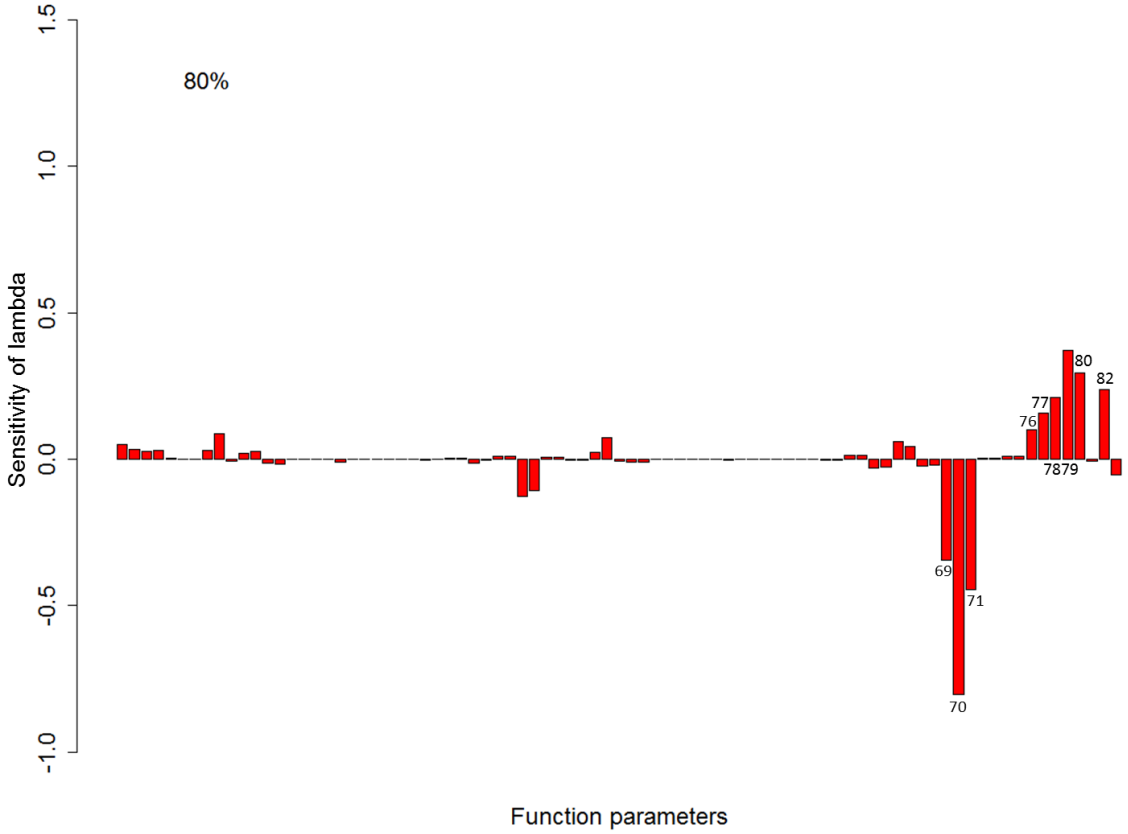
Function parameters

(I)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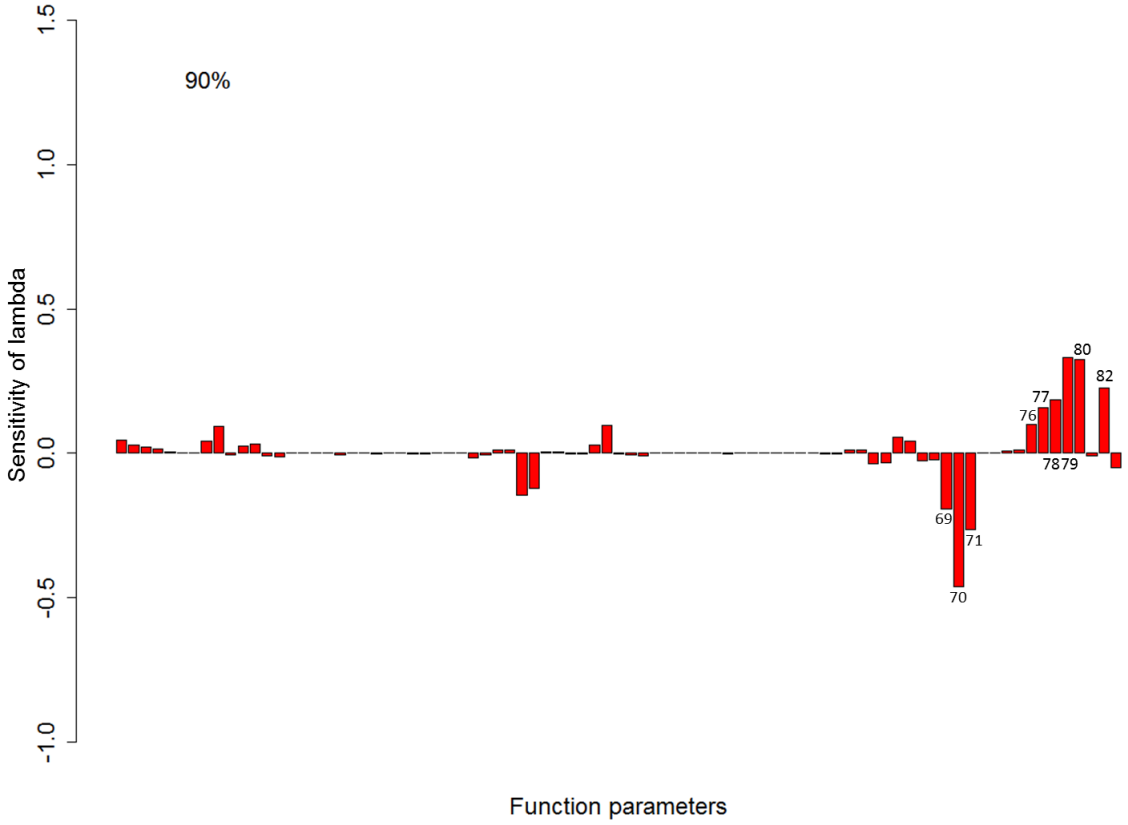
Function parameters

(J)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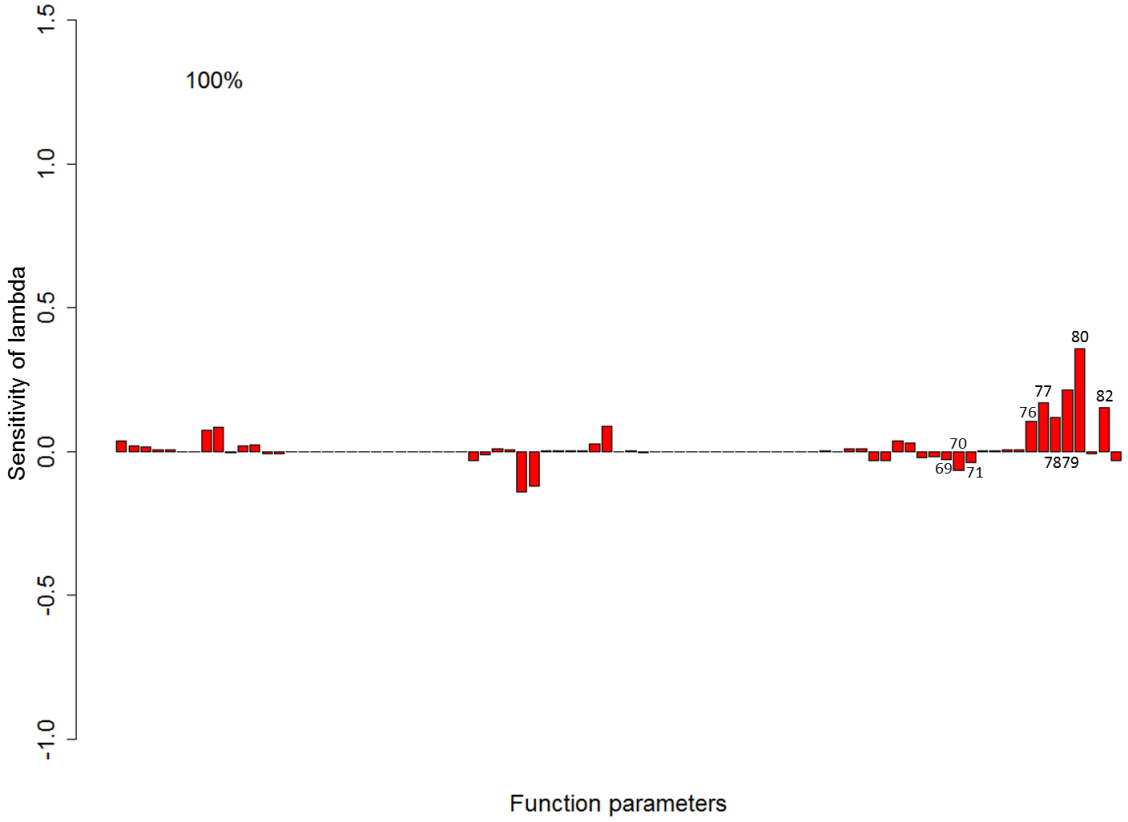
Function parameters

(K)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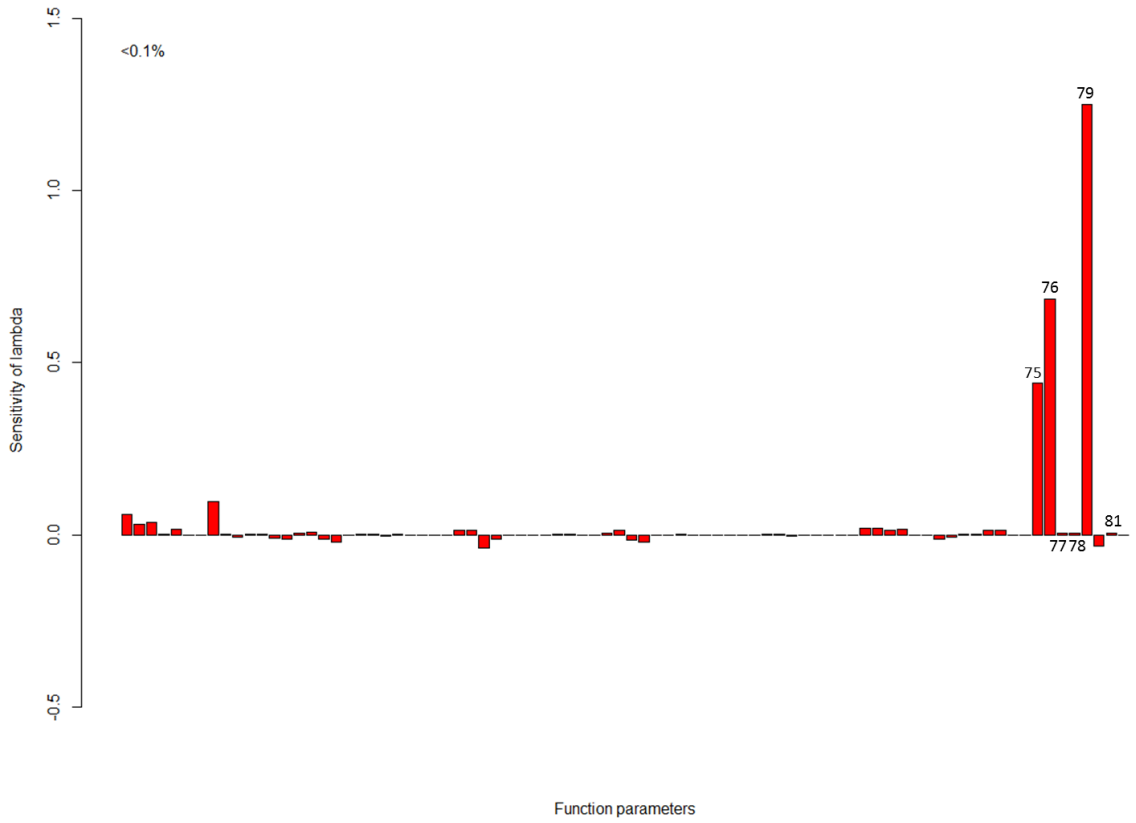
Function parameters

Figure S3. Perturbation analysis. Sensitivity of population growth rate (lambda/λ_0) to each of the parameters of each of the statistical functions used to construct the integral Projection Model (IPM) (82 parameters in total) (see text for details). In all cases the deutonymph transition function includes a linear and not the quadratic term (linear logit). Sensitivities were calculated for increasing deutonymph expression from (A) <0.1% to (K) 100%. Numbers indicate the various function parameters (only parameters which change significantly with increasing deutonymph expression shown): 75 – 76 (non-disperser reproduction function); 77 – 78 (disperser reproduction function); 79 (non-disperser offspring inheritance function); 81 (disperser offspring inheritance function).

(A)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76
75
81
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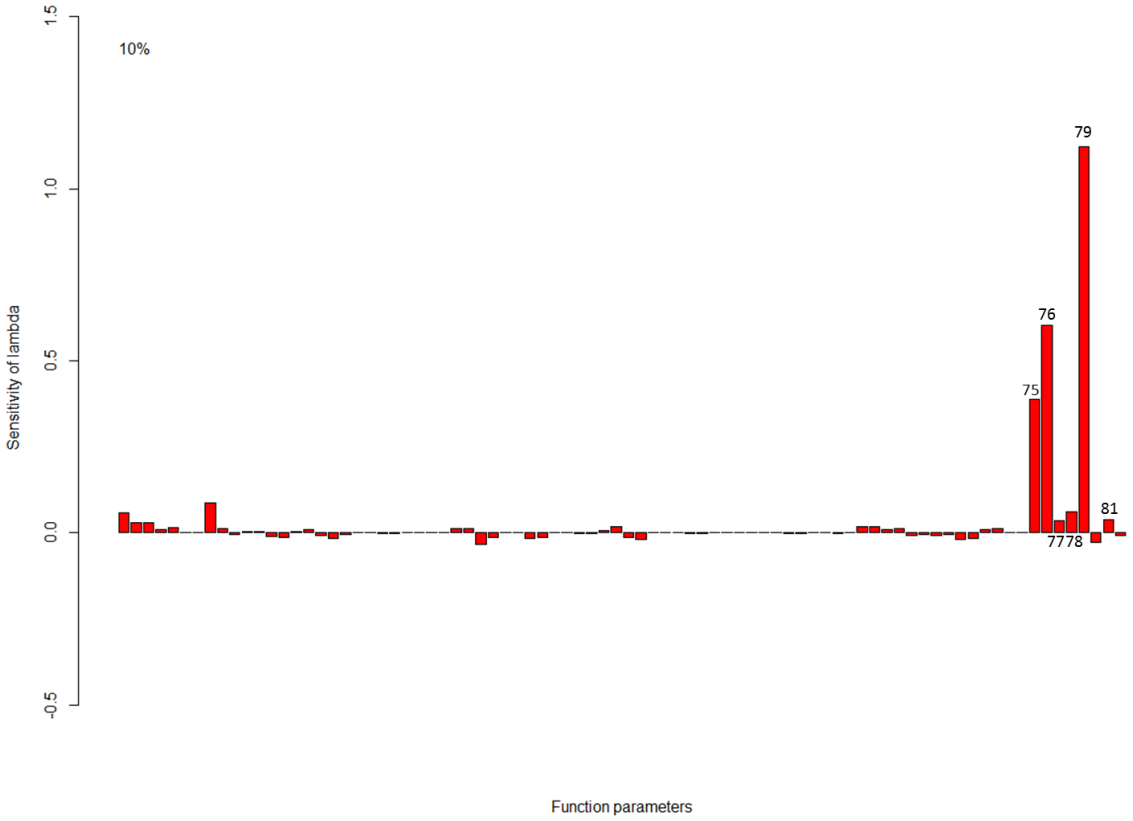
78
Function parameters

(B)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75
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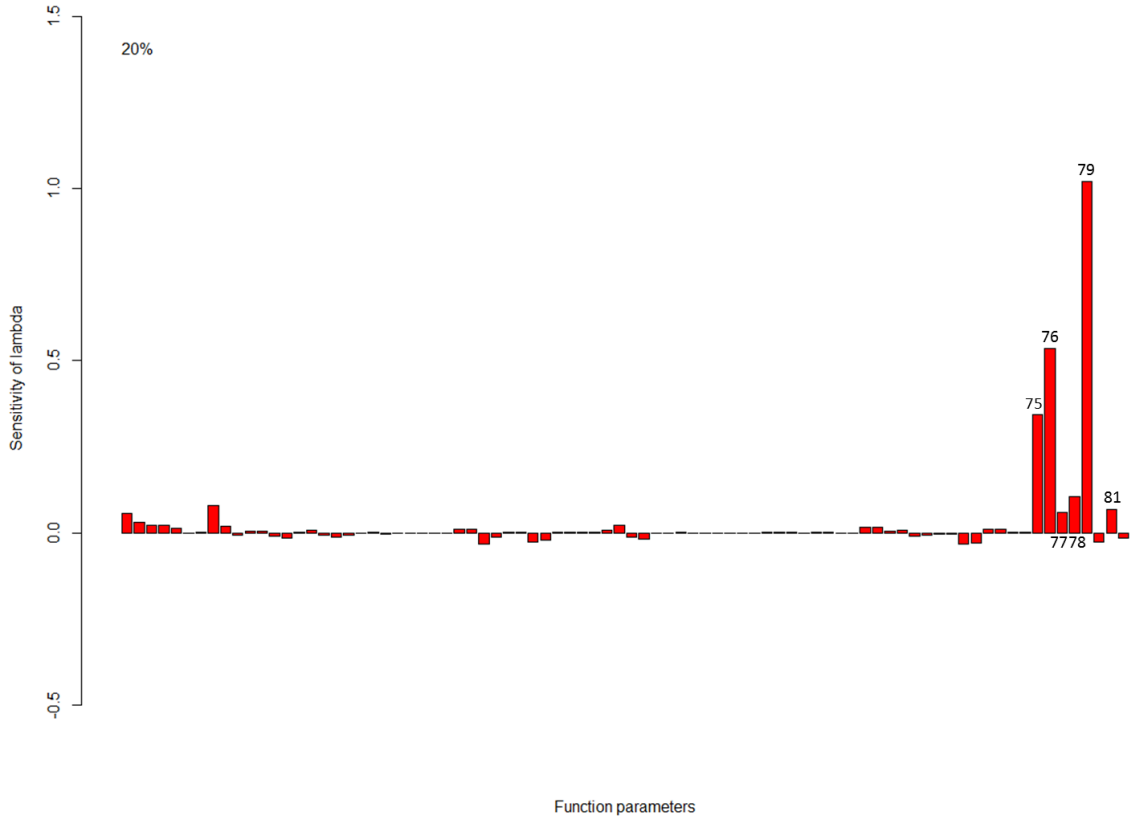
Function parameters

(C)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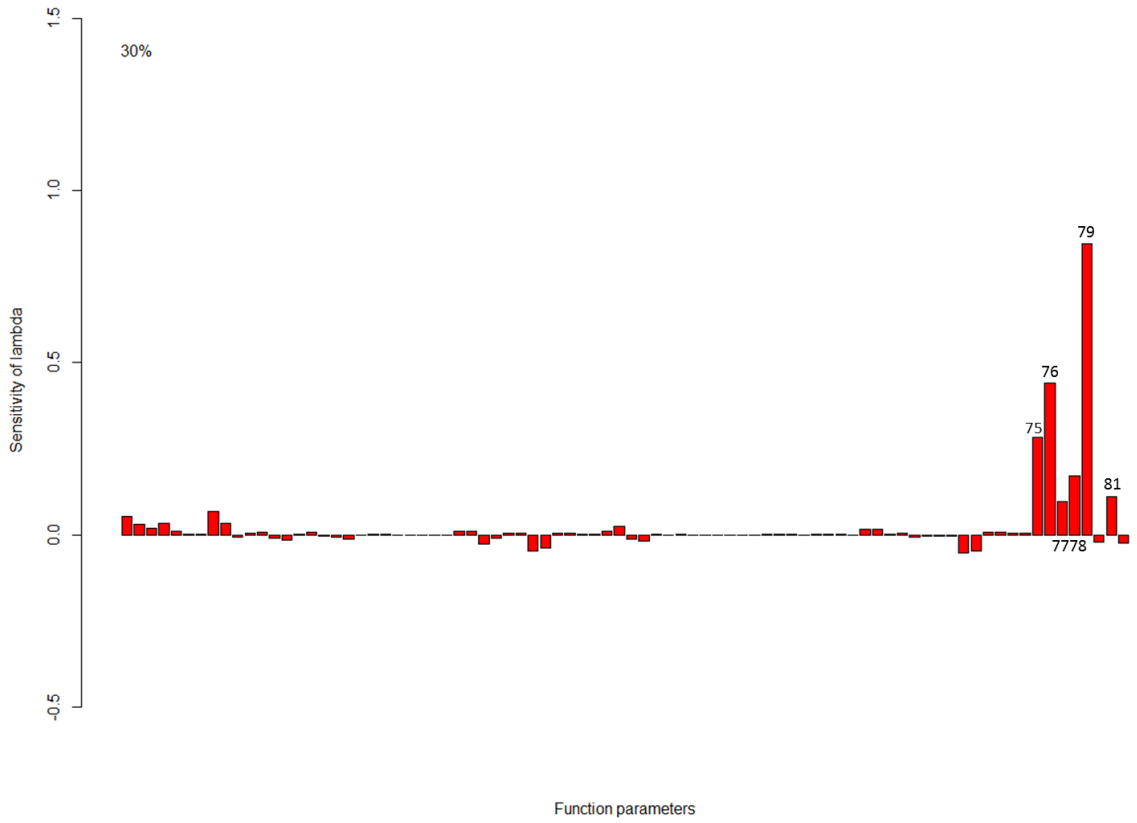
Function parameters

(D)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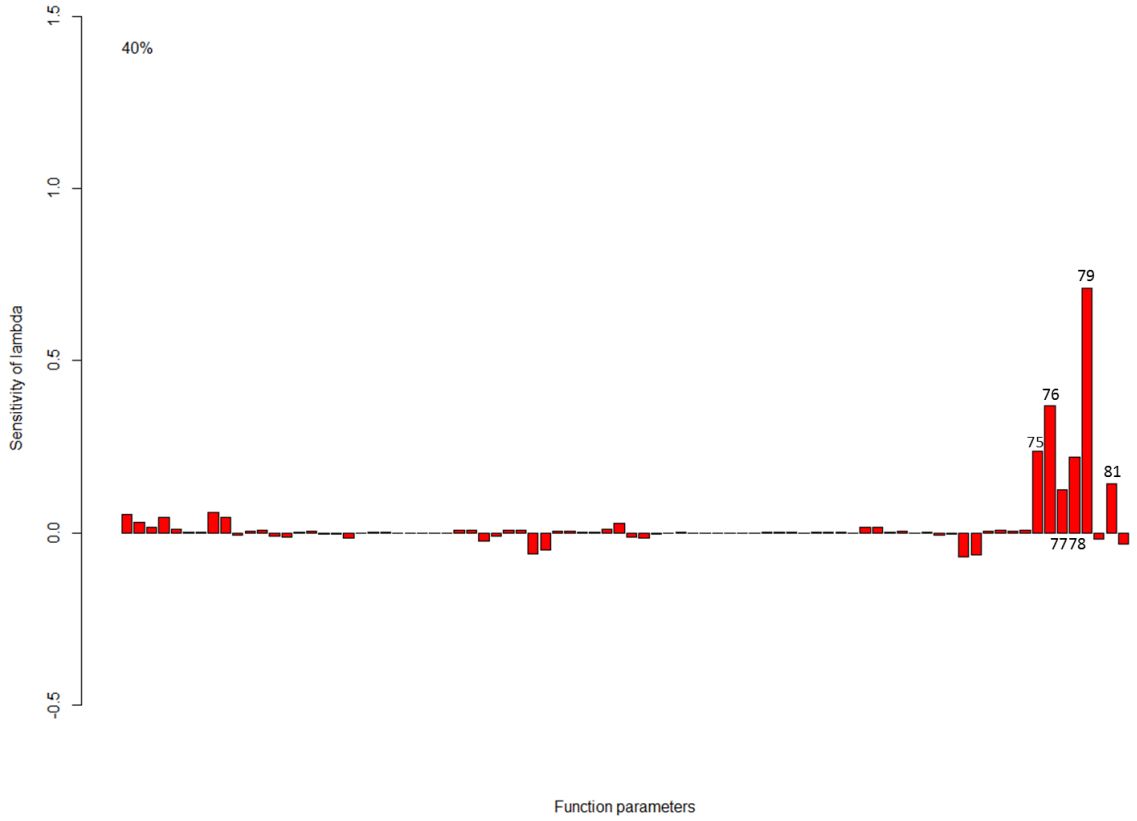
Function parameters

(E)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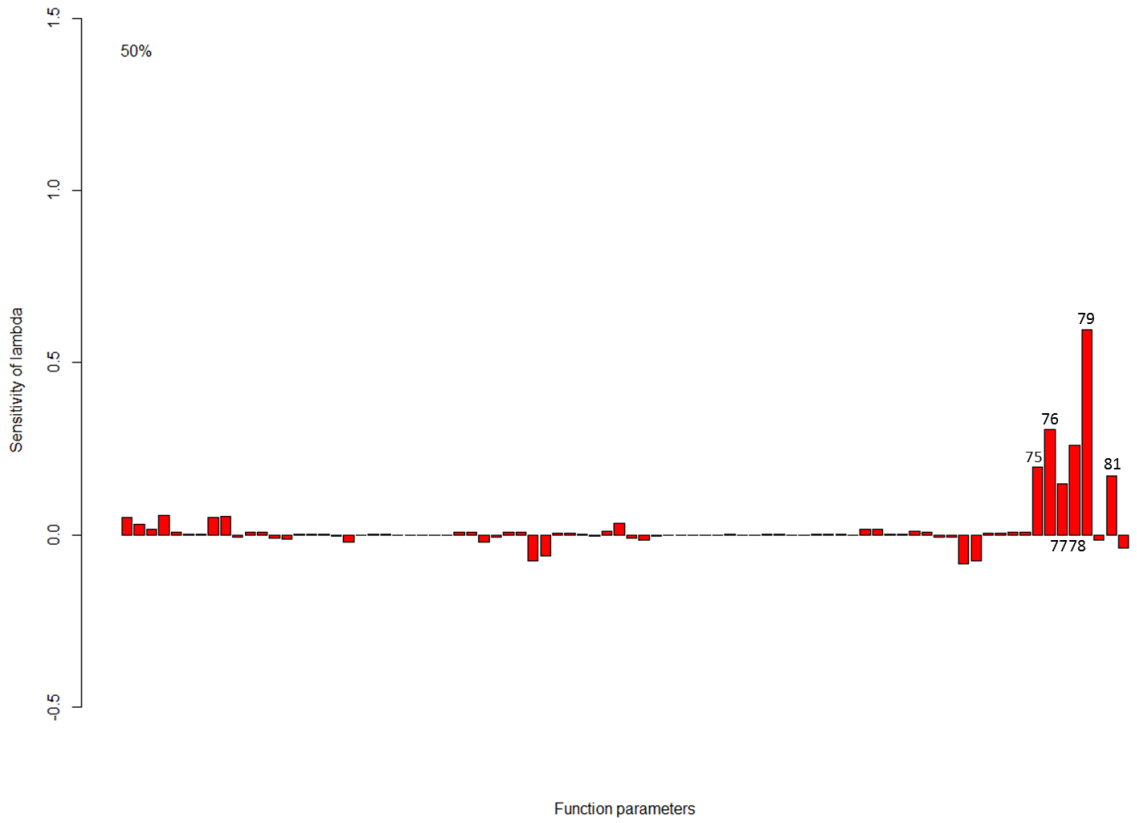
Function parameters

(F)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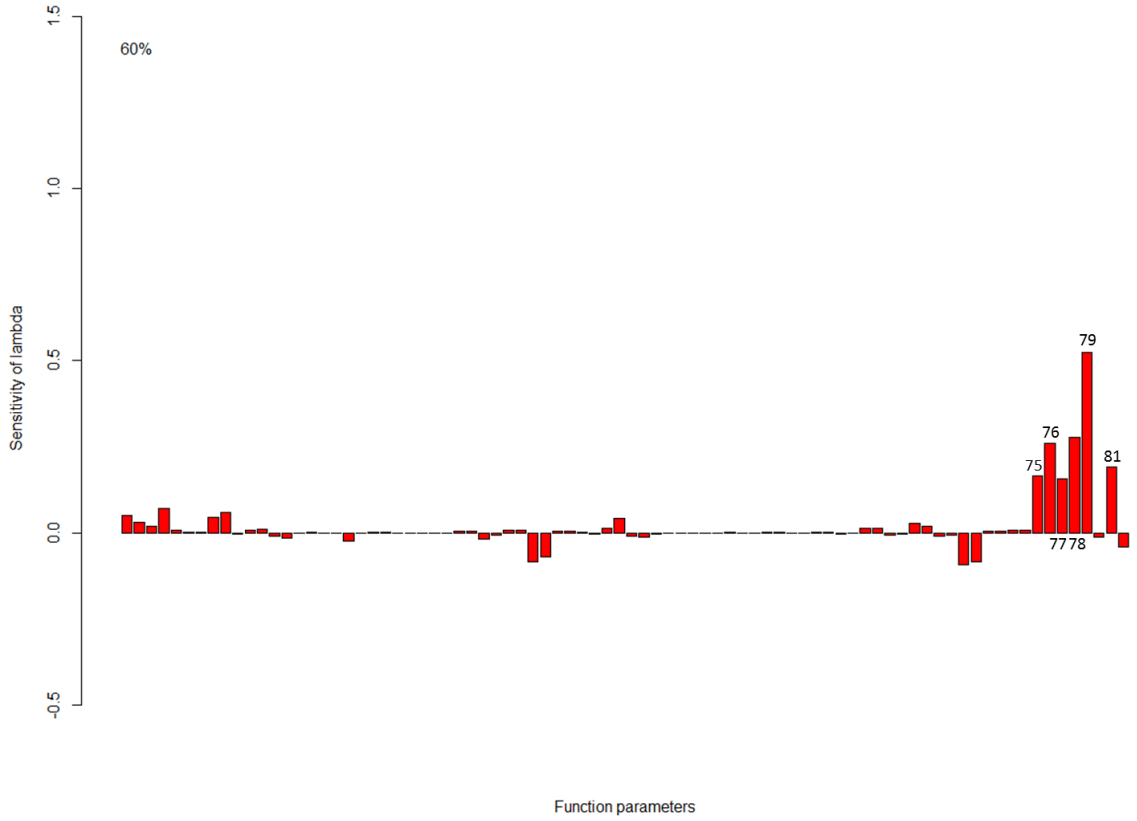
Function parameters

(G)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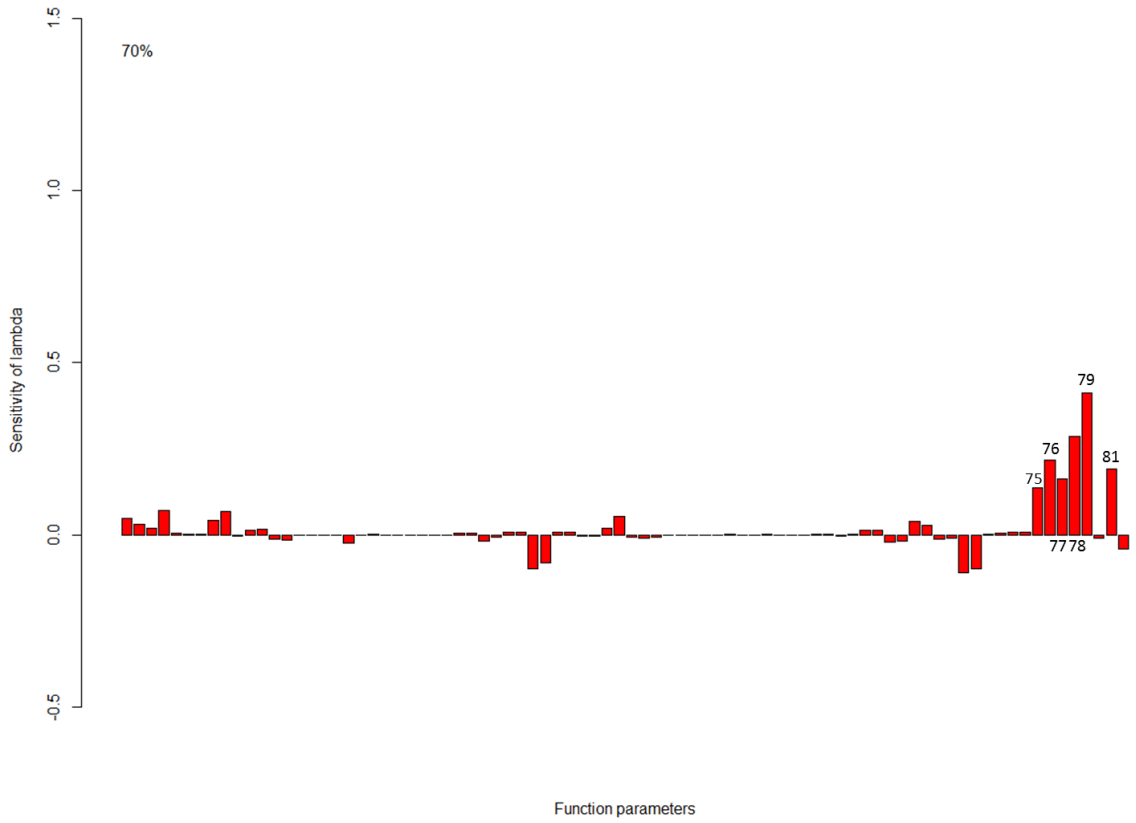
Function parameters

(H)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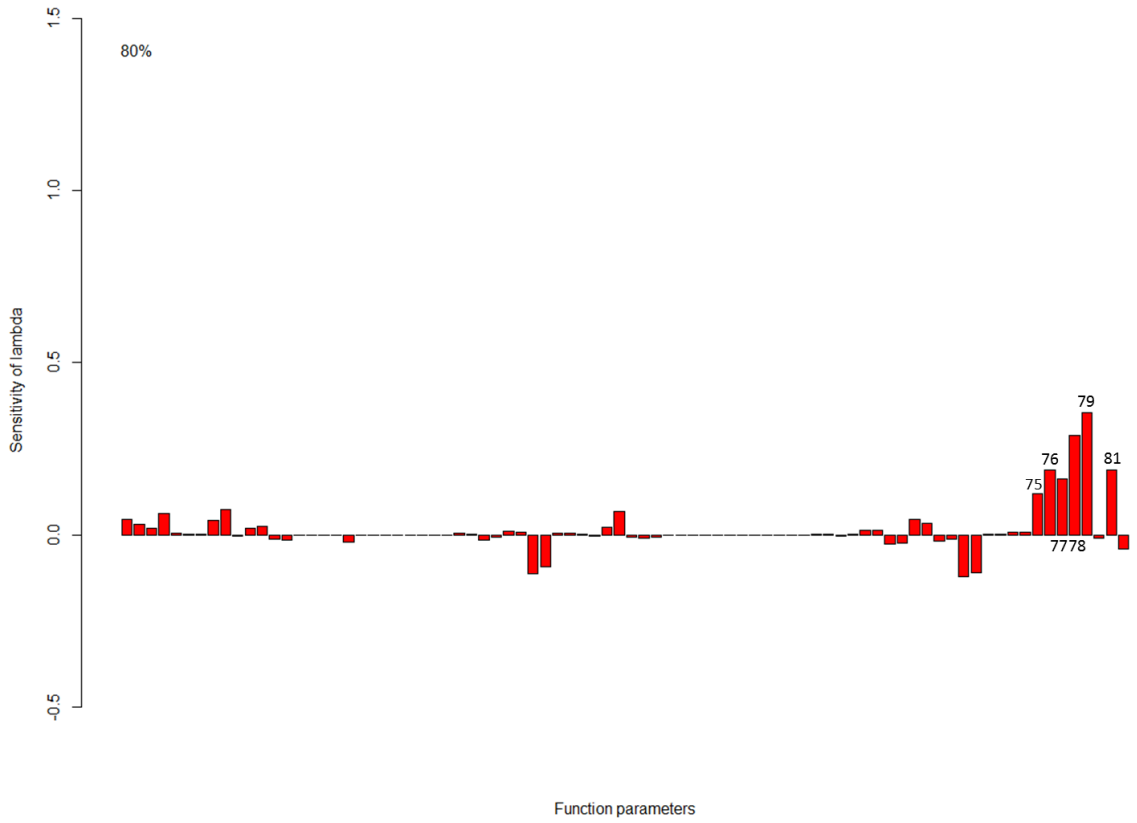
Function parameters

(I)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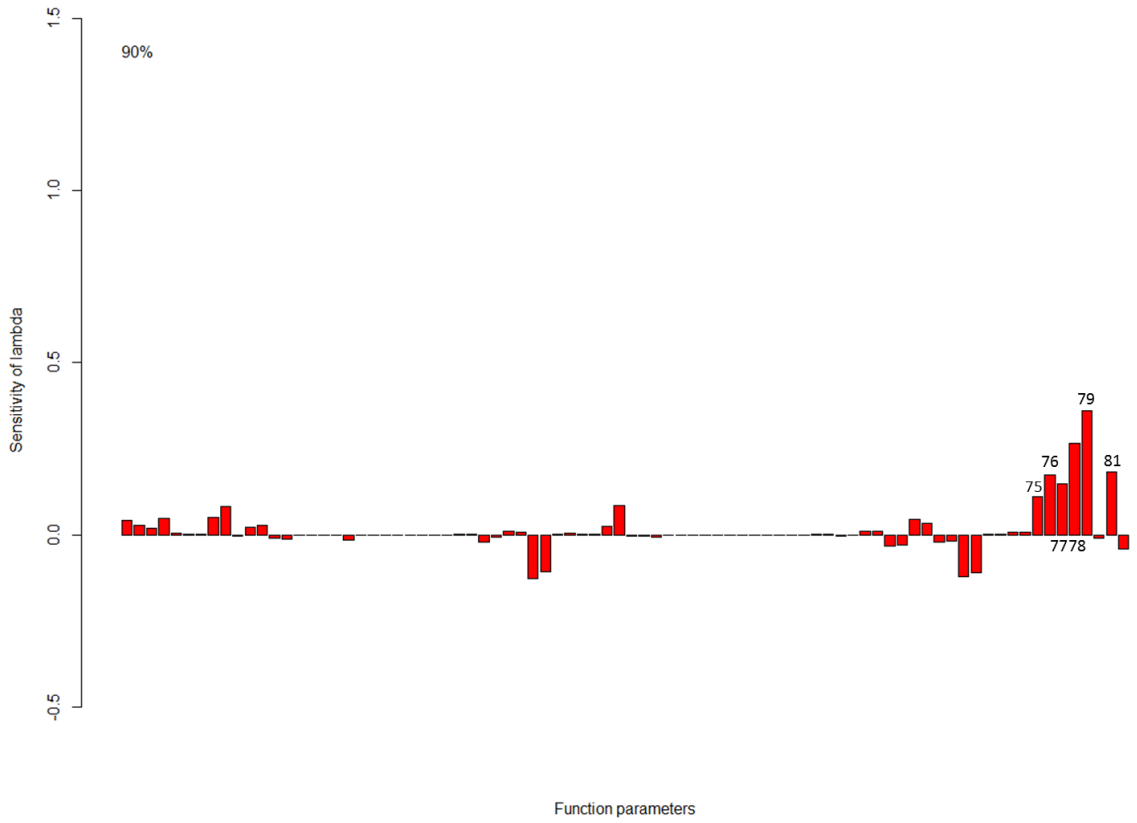
Function parameters

(J)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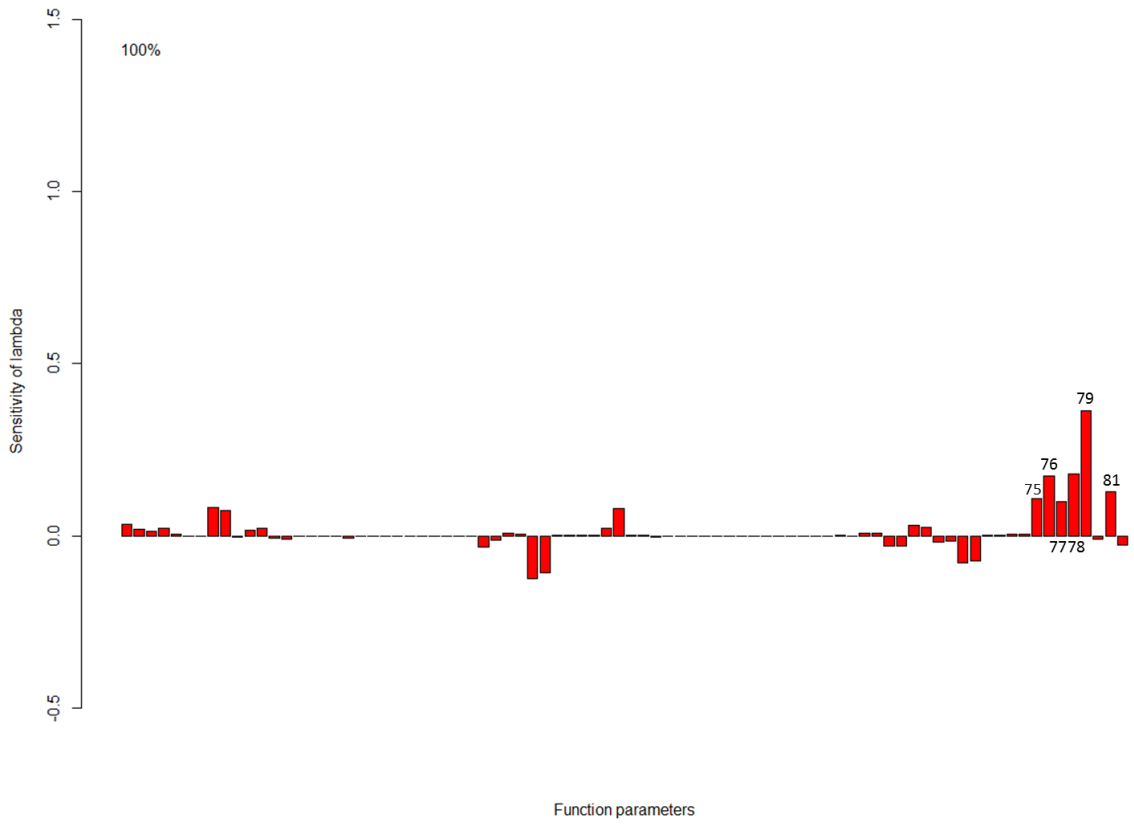
Function parameters

(K)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Sensitivity of lambda
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Function parameters

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Chapter 5

Influence of coloured environmental noise and dispersal on the stochastic growth rate

Running head

Effects of noise colour and dispersal on the stochastic growth rate

Title

Influence of coloured environmental noise and dispersal on the stochastic growth rate

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Abstract

Environmental variation due to anthropogenic change is increasing. In order to manage populations in the face of change it is necessary to understand the influence environmental variation has on population dynamics. Environmental variation, or noise, can be characterised by colour, and the type of colour can affect demographic processes including dispersal. However, it is not yet known if and how environmental variation and dispersal interactively affect characteristics of the population. Here, we use a stochastic integral projection model (IPM) to assess what the combined influence of environmental properties and the proportion of the population that disperses have on population fitness measured as the long run stochastic growth rate λ_s. We parameterise the model using life history data of the bulb mite (*Rhizoglyphus robini*), which life-cycle includes a facultative dispersal stage. We find that environmental noise colour does not directly influence λ_s, but that the frequency of good environments and the proportion of dispersers does. Unsurprisingly, as the proportion of dispersers increases, sensitivity of the stochastic growth rate to demographic rates of non-dispersers decreases while the sensitivity of demographic rates to dispersers increases. However, this is only the case when good environments occur infrequently, irrespective of noise colour. In contrast when bad environments are rare (high good environmental frequency) stochastic growth rate is most sensitive to demographic rates of non-dispersers but only in red and white environments. We surmise that the subtle, combined effects of the proportion of dispersers and environmental properties on population dynamics can influence selection for dispersal. We conclude that environmental properties interact, in different ways, with life-history to influence population biology quantities including the population growth rate.

Key words

Environmental variation, demography, environment frequency

Introduction

Variability in environmental conditions is changing and becoming increasingly higher, partly due to anthropogenic-induced climate change (Easterling et al. 2000; Emanuel 2005; IPCC 2007; Lawson et al. 2015). Populations are influenced by their environment and increased variability in climate conditions will affect their dynamics (Gaillard et al. 2000; Boyce et al. 2006; Lawson et al. 2015). Therefore understanding how environmental variation influences population dynamics is necessary to successfully manage populations in a changing climate (Lande et al. 2003, Reigada et al. 2015).

One aspect of environmental variation that is changing is the autocorrelation in environmental variables. For example, the frequency of consecutive drought years appears to be increasing in some areas. The autocorrelation structure can be characterised by noise colour. White noise characterises no autocorrelation (i.e. the environment states are independent and occur randomly), red noise characterises positive autocorrelation (long runs in one environmental state, and only occasional switches between states) while blue noise characterises negative autocorrelation (very frequent fluctuations between states) (Kaitala et al. 1997; Caswell 2001; Vasseur 2007). Although much of the past theoretical work on environmental noise assumed the noise to be white, white noise is not characteristic of nature and so the broader colour spectrum of red through to blue is considered to be more accurate when investigating the effects of environmental variation (Travis 2001; Vasseur and Yodzis 2004). A large number of studies have shown that noise colour differentially affects population-level processes (Ripa and Lundberg 1996; Kaitala et al. 1997; Petchey et al. 1997; Heino 1998; Morales 1999; Heino et al. 2000; Petchey 2000; Heino and Sabadell 2003;

Laakso et al. 2003; Fontaine and Gonzalez 2005; Schwager et al. 2006; Vasseur 2007; Smallegange et al. 2014). For example, noise colour has significant effects on population and metapopulation persistence. Some studies show that populations that are exposed to a shift in noise colour, where environments become redder, show increased extinction risk (e.g. Petchey et al. 1997; Heino 1998). However in other studies, opposite effects are found (Ripa and Lundberg 1996; Morales 1999; Heino et al. 2000; Heino and Sabadell 2003; Schwager et al. 2006). It has also been shown that different life-histories have contrasting effects on extinction risk; annual species have reduced extinction risk with redder noise as compared to biennial or perennial life-histories (Heino and Sabadell 2003). The increased extinction risk is driven by changes in the population structure at low densities resulting in lower equilibrium population sizes. In another study, the opposite effect on extinction risk was found to occur when populations are sensitive to extreme events of bad environments (Schwager et al. 2006). In this case increased extinction risk is due to more overcompensatory density regulation in the population which increases the risk of population crashes. Other population-level processes influenced by environmental noise colour include population synchrony, which increases with redder noise (Fontaine and Gonzalez 2005). Environment colour also affects life-history variables, although life-history variables differ in their response to redder noise (Laakso et al. 2003; Smallegange et al. 2014).

Autocorrelation of environmental noise is not the only environmental property that influences population-level processes. Seasonality has also been shown to be an important factor influencing life-history traits and Marshall and Burgress (2014) showed that the relationship of a number of life-history traits depends on a combination of environmental noise colour and seasonality. Another factor is the frequency of disturbances (or environmental state) which can dictate the relative importance of

specific demographic rates to the growth rate of populations (Tuljapurkar et al. 2003; Smallegange et al. 2014). Smallegange et al. (2014) found that the response of different life-history variables is determined by both environmental noise colour and the frequency of favourable environments. As such, populations are expected to respond to a combination of environmental properties in different ways. This environmental stochasticity is important in both small and large populations and is the temporal fluctuations in the probability of mortality and reproductive rate of all individuals (all individuals are affected in the same way) (May 1973; Shaffer 1981; Lande 1993; Engen et al. 1998; Lande et al. 2003). The changes brought about by environmental stochasticity is one form of stochasticity that the population will experience. Populations are also subject to demographic stochasticity, which is the chance events of individuals dying and reproducing with these events operating independently among individuals (May 1973; Shaffer 1981; Lande 1993; Engen et al. 1998; Lande et al. 2003). In contrast to environmental stochasticity, demographic stochasticity is most important in small populations as individual, independent events tend to average out in large populations (Lande 1993). Both demographic and environmental stochasticity therefore need to be considered when trying to understand population fluctuations.

Dispersal is an important demographic process in determining population trajectories (Clobert et al. 2001). Which dispersal strategy is best critically depends on environmental conditions. Theoretical models, and a few empirical studies, have shown that selection for dispersal is influenced by environmental variation (Marshall and Burgess 2014) and that selection on dispersal is weaker in temporally constant than in more variable environments (e.g. McPeek and Holt 1992; Travis and Dytham 1998; Travis 2001; Marshall and Burgess 2014). However, we have yet to understand if and how this demographic characteristic and environmental variation interactively affect the

characteristics of populations including their population growth rate, generation time and lifetime reproductive success. A change in environmental variability could favour dispersal in populations that were adapted to conditions under which selection for dispersal was weak. But would such a change be the same for a shift from red to white noise, or from white to blue noise? And how would these shifts and associated changes in dispersal expression within a population affect population biology quantities? These are still unanswered questions.

Here we use a stochastic integral projection model to assess the combined influence of (i) noise colour, (ii) frequency with which favourable environmental conditions occur and (iii) the proportion of dispersing individuals within a population on the long run stochastic growth rate (λ_S) of a single (natal) population. We use integral projection models (IPMs; Easterling et al. 2000) as they have been shown to be a useful tool when investigating the concurrent change in life-history variables with changing environmental conditions within a population (Coulson et al. 2011; Smallegange et al. 2014; Deere et al. in prep, Chapter 4). Life-history data from the bulb mite (*Rhizoglyphus robini*) are used to parameterise our model. We use this model species for two reasons. First, it has a facultative dispersal stage in its life-history (Smallegange and Coulson 2011; Deere et al. in prep, see Chapter 3); juvenile mites develop into this stage during unfavourable conditions. Second, we have a detailed understanding of how the environment affects this species' life-history (Smallegange 2011a, 2011b; Smallegange and Coulson 2011; Smallegange et al. 2014; Deere et al. in prep, Chapter 3). We only explore temporal variability/autocorrelation as we only focus on a single (natal) population exposed to either good or bad environments, where bad environments (unfavourable conditions) produce dispersing individuals that do not disperse from the natal population. For simplicity, in this study, we do not model

immigration or emigration into the population by dispersing individuals but this could be included in an extended model. For the same reason we do not include spatial autocorrelation of environments but we are aware that populations could respond differently to a combination of spatial and temporal autocorrelation. We predict that increasing the proportion of dispersers will have a greater negative effect on stochastic growth rates when: (i) the environmental noise colour is white (unpredictable environment state) or red (predictable long cycles of environment state) and (ii) the frequency of favourable environmental conditions is low. This study provides insight into the subtle effects that the combination of life-history and variation in multiple environmental properties can have on local population dynamics.

Methods

Stochastic Integral Projection Models

An integral projection model (IPM) is a structured population model that can use a continuous individual attribute (e.g. size) that affects demographic rates to define a population's structure (Ellner and Rees 2006). The IPM can also be used when multiple attributes affect demographic performance by extending the original IPM. For example, a size-structured IPM can be extended to include multiple life-history stages of which dispersal can be included as a stage.

We use a previously developed IPM that includes dispersal (Deere et al. in prep, see Chapter 4). In brief, this IPM is constructed from four fundamental functions: survival, growth, reproduction and inheritance. These functions describe how distribution of the body size z at time t is influenced by survival, growth, reproduction and inheritance, resulting in a new distribution of body size at time t+1 and can be

written as follows: (1) the survival probability at time t+1, $S(z,s,t)$; (2) the transition probability that females develop into the next stage s at time t+1, $P(z,s,t)$; (3a) the change in body size among survivors of size z that stay in stage s at time t+1, $G(z'|z,s,t)$; (3b) the change in body size among survivors of size z that have moved to stage s+1 at time t+1, $G(z'|z,s+1,t)$; (4) the number of eggs produced by individuals of size z at time t+1 (assuming a pre-breeding census), $R(z,s,t)$; and (5) the size of eggs, z', produced by individuals of size z at time t+1, $D(z'|z,s,t)$. The IPM was parameterised for two contrasting environments using life-history data of female mites (see Fig. 5.1) that were collected in a good environment (ad lib access to yeast: Smallegange 2011a, 2011b; Smallegange et al. 2014) and in a bad environment (ad lib access to oats: Deere et al. in prep, Chapter 3). However, as no deutonymphs develop in a good environment, the transition to the deutonymph stage (β in Fig. 5.1) (and all subsequent stages from the deutonymph) is set to zero when the IPM is parameterised for the good environment. When, in a stochastic time series (see below) and a good environment follows a bad environment, the transition rate from deutonymph (coming from the bad environment) to tritonymph is set equal to 1 (G_6 in Fig. 5.1). A value of 1 is used as we assume that when a deutonymph enters the good environment there is no delay in transition into the next stage in order to continue developing. A full description of how the parameter functions were calculated and the IPM constructed can be found in Smallegange et al. (2014) and Deere et al. in prep (see Chapter 4). A brief description of the construction of the IPM and the parameter estimation can be found in the supporting information and the final IPM equations can be found in Table 5.1. Equation (1) (Table 5.1) provides a description of the dynamics of body size z (which is a continuous trait) for a population in a good environment with Eq. (2) (Table 5.1) a population in a bad environment. The equations vary slightly depending on the environment concerned.

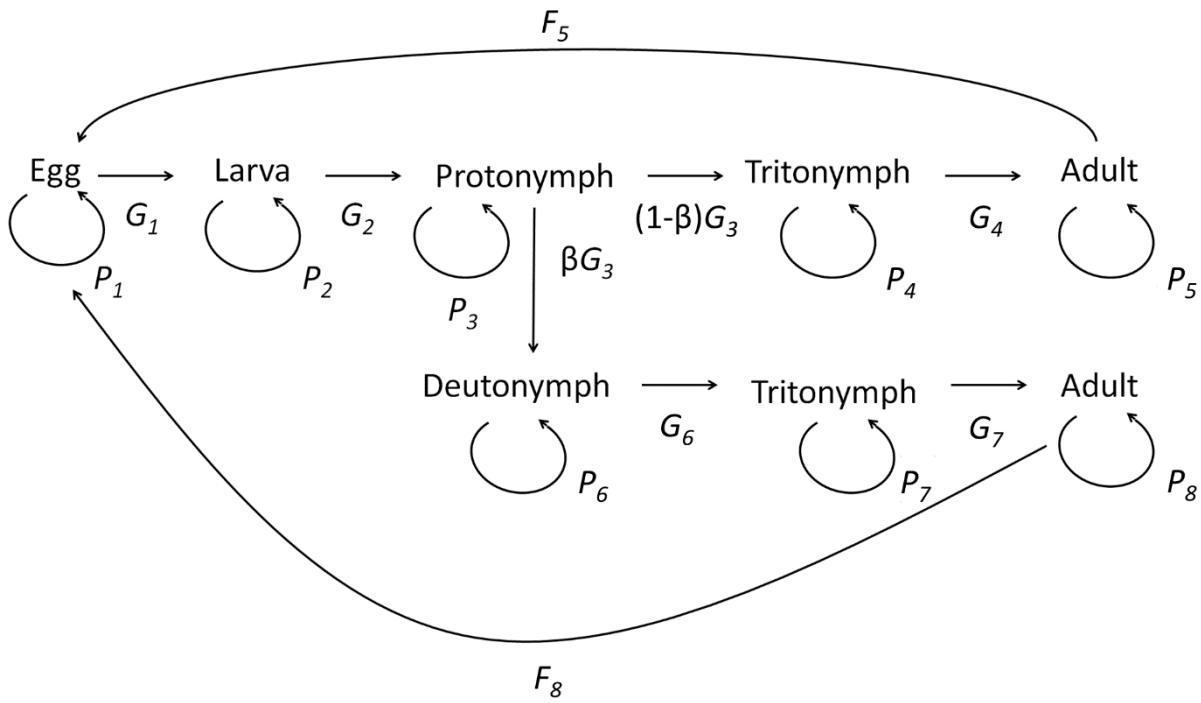

Figure 5.1. Life cycle of the bulb mite indicating the six life stages. From the life cycle we calculated survival (*P*) and fecundity (*F*) rate and the probability of surviving and growing into the next stage (*G*). The deutonymph stage is the facultative dispersal stage.

Stochastic demographic model

In order to incorporate environmental variability into the stochastic model we calculate autocorrelation values that cover the noise colour spectrum of red, white and blue. We do so using a two-state Markov chain that gives the probability distribution of environment states at time *t*. Two environmental states were identified: 1 for the good environment and 2 for the bad environment. The Markov chain transition matrix **M** (Caswell 2001, p. 379) can then be written as follows:

$$\mathbf{M} = \begin{bmatrix} 1-p & q \\ p & 1-q \end{bmatrix}, \tag{5.1}$$

Table 5.1. The Integral Projection Model (IPM) is a combination of equations. Equations are constructed from five statistical demography functions: 1) Survival *S(z,s,t)*; 2) Transition *P(s|z,s,t)*; 3) Growth *G(z'|z,s,t)*; 4) Reproduction *R(z,s,t)* and 5) Inheritance *D(z'|z,s,t)* (see Appendix 1 and Chapter 4 for details). The equations calculate the number of females in each stage *s* at time *t* which is described by *n(z,s,t)* with the *R(z,s,t)* and *D(z'|z,s,t)* functions zero for all non-adult stages as only adults reproduce. Ω_s is a closed interval indicating the size domain of stage *s*.

	Life stage Equation	Description
Bad-environment IPM		
1.1	$n(z',1,t+1) = \int D(z'\mid z,5,t)R(z,5,t)n(z,5,t)dz + \int D(z'\mid z,8,t)R(z,8,t)n(z,8,t)dz,$	Egg production by adults that have and have not passed through the dispersal stage
1.2	$n(z',s+1,t+1) = \int_{\Omega_s} G(z'\mid z,s+1,t)P(s+1\mid z,s,t)S(z,s+1,t)n(z,s,t)dz$ $n(z',s,t+1) = \int_{\Omega_s} G(z'\mid z,s,t)P(s\mid z,s,t)S(z,s,t)n(z,s,t)dz$ } $1 \le s \le 2$	Eggs and Larvae developing into the next stage and staying in the same stage
1.3	$n(z',3,t+1) = \int G(z'\mid z,3,t)P(3\mid z,3,t)S(z,3,t)n(z,3,t)dz$	Non-dispersal Protonymphs staying Protonymphs
1.4	$n(z',4,t+1) = \int G(z'\mid z,4,t)P(4\mid z,3,t)S(z,4,t)n(z,3,t)dz$	Non-dispersal Tritonymphs developing from Protonymphs

Table 5.1 (cont.)

	Life stage Equation	Description
(Bad-environment IPM cont.)		
1.5	$n(z',4,t+1)=\int G(z'\mid z,4,t)P(4\mid z,4,t)S(z,4,t)n(z,4,t)dz$	Non-dispersal Tritonymphs staying Tritonymphs
1.6	$n(z',6,t+1)=\int G(z'\mid z,6,t)P(6\mid z,3,t)S(z,6,t)n(z,3,t)dz$	Deutonymphs developing from a Protonymphs
1.7	$n(z',6,t+1)=\int G(z'\mid z,6,t)P(6\mid z,6,t)S(z,6,t)n(z,6,t)dz$	Deutonymphs staying Deutonymphs
1.8	$n(z',7,t+1)=\int G(z'\mid z,7,t)P(7\mid z,6,t)S(z,7,t)n(z,6,t)dz$	Dispersal Tritonymphs developing from Deutonymphs
1.9	$n(z',7,t+1)=\int G(z'\mid z,7,t)P(7\mid z,7,t)S(z,7,t)n(z,7,t)dz$	Dispersal Tritonymph staying Tritonymphs
1.10	$n(z',5,t+1)=\int G(z'\mid z,5,t)P(5\mid z,5-1,t)S(z,5-1,t)n(z,5-1,t)dz+\int G(z'\mid z,5,t)S(z,5,t)n(z,5,t)dz$	Non-dispersal adults developing from non-dispersal Tritonymphs and surviving non-dispersal adults
1.11	$n(z',8,t+1)=\int G(z'\mid z,8,t)P(8\mid z,8-1,t)S(z,8-1,t)n(z,8-1,t)dz+\int G(z'\mid z,8,t)S(z,8,t)n(z,8,t)dz$	Dispersal adults developing from dispersal Tritonymphs and surviving dispersal adults

Table 5.1 (cont.)

	Life stage Equation		Description
Good-environment IPM			
2.1	$n(z',1,t+1)=\int_{\Omega_s} D(z'\mid z,s,t)R(z,s,t)n(z,s,t)dz,$	$s=5$	Egg production adults
2.2	$n(z',s+1,t+1)=\int_{\Omega_s} G(z'\mid z,s+1,t)P(s+1\mid z,s,t)S(z,s+1,t)n(z,s,t)dz$ $n(z',s,t+1)=\int_{\Omega_s} G(z'\mid z,s,t)P(s\mid z,s,t)S(z,s,t)n(z,s,t)dz$	$1\le s\le 2$	Eggs and Larvae developing into the next stage and staying in the same stage
2.3	$n(z',3,t+1)=\int G(z'\mid z,3,t)P(3\mid z,3,t)S(z,3,t)n(z,3,t)dz$		Non-dispersal Protonymphs staying Protonymphs
2.4	$n(z',4,t+1)=\int G(z'\mid z,4,t)P(4\mid z,3,t)S(z,4,t)n(z,3,t)dz$		Non-dispersal Tritonymphs developing from Protonymphs
2.5	$n(z',4,t+1)=\int G(z'\mid z,4,t)P(4\mid z,4,t)S(z,4,t)n(z,4,t)dz$		Non-dispersal Tritonymphs staying Tritonymphs
2.6	$n(z',7,t+1)=\int G(z'\mid z,7,t)P(7\mid z,6,t)S(z,7,t)n(z,6,t)dz$		#Dispersal Tritonymphs developing from Deutonymphs
2.7	$n(z',7,t+1)=\int G(z'\mid z,7,t)P(7\mid z,7,t)S(z,7,t)n(z,7,t)dz$		Dispersal Tritonymph staying Tritonymphs

Table 5.1 (cont.)

	Life stage Equation	Description
(Good-environment IPM cont.)		
2.8	$n(z',5,t+1)=\int G(z'\mid z,5,t)P(5\mid z,5-1,t)S(z,5-1,t)n(z,5-1,t)dz+$ $\int G(z'\mid z,5,t)S(z,5,t)n(z,5,t)dz$	Non-dispersal adults developing from non-dispersal Tritonymphs and surviving non-dispersal adults
2.9	$n(z',8,t+1)=\int G(z'\mid z,8,t)P(8\mid z,8-1,t)S(z,8-1,t)n(z,8-1,t)dz+$ $\int G(z'\mid z,8,t)S(z,8,t)n(z,8,t)dz$	Dispersal adults developing from dispersal Tritonymphs and surviving dispersal adults

#The point where dispersers enter the population as deutonymphs and transition to the next stage with 100% probability as they are in a good environment

where p is the probability of switching from the good environment to the bad environment and q is the probability of switching from the bad environment to the good environment (both values must range between 0 and 1). The serial autocorrelation of the Markov chain equals $\rho = 1 - p - q$ (Caswell 2001, p. 379), which informs on environmental noise colour (see *Introduction*). We then iterated **M** and generated a time series of $S = 2500$ time steps; this sequence determines the environmental state that a population experiences at each time step (see Tuljapurkar et al. 2003). An initial transient length of 200 was discarded; the starting population comprised one individual in each size bin (see supporting information for calculation of size bins) with the initial environmental state chosen randomly. The state of the environment at time t then determines which IPM matrix is used. A good environment at time t results in use of the matrix approximation for a good environment IPM, a bad environment at time t results in the use of the matrix approximation for a bad environment IPM.

We calculated long run stochastic growth rate (λ_S) as an indicator of population fitness. The quantity λ_S can be defined as the geometric mean growth rate of the population and is considered a more accurate descriptor of the long term population dynamics in fluctuating environments than the arithmetic mean growth rate (λ_A) (Tuljapurkar and Orzack 1980; Tuljapurkar et al. 2003). For a stationary temporal sequence of habitats, λ_S is the long term growth rate of the population as a result of a typical realization of sequence of habitats (Tuljapurkar et al. 2003). It is calculated from the product of multiple distinct matrices. λ_A is the long term growth rate of a population experiencing a constant environment given by the mean habitat (Tuljapurkar et al. 2003). It is calculated as the dominant eigen value of the matrix of average transition rates (as a result of a sequence of habitats) (Tuljapurkar and Orzack 1980; Tuljapurkar et al. 2003). The differences in how the two values are calculated often results in λ_A

tending to overestimate growth rate compared to λ_S. Additionally, λ_A does not describe the dynamics of the average population unless there is no serial autocorrelation of habitat state (Tuljapurkar et al. 2003). Therefore λ_A does not take into account the sequences of habitat states, which λ_S is able to do, and this is key to the dynamics in temporally varying environments. It is for these reasons that λ_S is generally favoured over λ_A as an indicator of population fitness. The long run stochastic growth (λ_S) was calculated over a period of length *S* by taking the exponent of

$$\log(\lambda_s) = \frac{1}{S}\sum_{t=0}^{S-1} r_t, \qquad (5.2)$$

with $r_t = \log[\Sigma_s \mathbf{p}_s(t+1)/\ \Sigma_s \mathbf{p}_s(t)]$ and $\mathbf{p}(t)$ is the population vector at time *t*.

The long run stochastic growth rate summarises how a natal population responds to environmental stochasticity and can be used to explore how disperser abundance in the natal habitat influence population size and structure in a stochastic environment.

Proportion of dispersers

In a source-sink population context, environmental change could impact the viability of source populations (Hanski 1999) and this impact will be influenced by the frequency of the population that goes through the dispersal stage and remain in the population. Scenarios of unsuccessful dispersers in a population may be more prevalent than one might think. Any species that has probabilistic dispersal (Houck and OConnor 1991; King and Roff 2010) will, by definition, have populations that have individuals that have failed to disperse if there is dispersal expression in the population. Furthermore, species may be restricted in their ability to move by habitat fragmentation (e.g. reduced accessibility to habitat patches, Clobert et al. 2001) or environmental

conditions (e.g. reduced migration levels during cloudy and rainy summers, Johnson 1969). Therefore we manipulated the proportion of individuals that transition into the dispersal stage to identify whether this has an impact on the population dynamics.

We explored four different transition probabilities of developing from a protonymph to a deutonymph (deutonymph probability): the default value (<3%), 10%, 50% and 80%. Higher values were not explored as these were considered to be biologically unrealistic. For each of these probabilities, which we refer to as "proportion of dispersers" throughout the rest of the manuscript, we calculated the stochastic growth rate for all combinations of p and q.

Perturbation analyses

Perturbation analysis is one way in which we can understand the relative importance of the different life-stages of a species by identifying which vital rate affects fitness most (Benton and Grant 1999). In our case we can assess the relative importance of different life-stages for a life cycle that does or does not include the dispersal stage (Fig. 5.1). By applying a small change to the vital rates we can assess how the model predictions change as an individual model parameter is altered. If we find that changing the vital rates of the dispersal stage (deutonymph) results in a larger change in population growth rate relative to the other life-stages, we know that the dispersal stage has an effect on the population growth rate (fitness) of the population. The perturbation analysis we chose was elasticity analysis, which estimates the effect of a proportional change in a vital rate on the population growth rate (Benton and Grant 1999).

We examined the elasticity of the long run stochastic growth rate (λ_s) to perturbation of the parameters of the character-demography functions by performing a

stochastic elasticity analysis. Each parameter of each character-demography function was independently perturbed upwards; positive values were multiplied by 1.001 and negative values were multiplied by 0.999 (making negative values less negative). Perturbations were done simultaneously in both environments. This would identify which parameters of which functions, and under which stochastic regimes, were most influential to λ_s. As the shape of a few character-demography functions between the good and bad environment IPM differed (e.g. for dispersers a function was quadratic whereas its non-dispersers counterpart was linear) (Fig. S1), only the most significant coefficient within each function that occurred in both environment IPMs was perturbed. For example, where a function in the bad environment IPM was quadratic but the same function in the good environment IPM was linear only the intercept and slope parameters in the IPMs were perturbed to allow for a like-for-like interpretation in the stochastic model. Elasticity values for each combination of p and q (see above *Stochastic demographic model* for *pq* calculations) were generated for each parameter of each character-demography function. For each *pq* combination, and for each character-demography function the parameter value that had the maximum elasticity value (most influential to a change in λ_S) was identified. These functions included ones capturing both disperser and non-disperser demography; which were scaled to sum to one to identify the proportional contribution of disperser and non-disperser parameters to the change in λ_s. If the scaled elasticity of λ_s to parameters in the disperser function is greater than 0.5, then λ_s is most sensitive to a change to parameters in the disperser function. Alternatively, if this scaled value is less than 0.5 then λ_s is most sensitive to a change in the non-disperser functions (results discussed in terms of disperser contributions). The relative disperser contribution for each ρ - and f - value was then plotted as a function of λ_s, where λ_s is the stochastic growth rate values generated over

the length of time of the stochastic sequence. This process was repeated for each stochastic model for each disperser proportion.

Results

Perturbation analysis

The stochastic perturbation analysis indicates how much average fitness (λ_s) changed after perturbing the function parameters (i.e. the elasticity) across all environmental regimes (*pq* combinations) and for all good environment frequencies (*f* – values; 0 to 1). The results revealed that parameters of four character demography functions are most influential to λ_s (they showed the maximum change in λ_s/maximum elasticity of λ_s): one parameter from one disperser function (intercept of deutonymph transition rate) and three parameters from three non-disperser functions (slope of reproduction rate, slope of protonymph growth rate and intercept of tritonymph transition rate); although not all the parameters of the three non-disperser functions were influential at the different proportions of dispersers.

We then wanted to know, given the maximum elasticity of λ_s for a certain *pq* combination and *f*-value, what the contribution of the abovementioned disperser and non-disperser parameters was to that elasticity. For example, if a disperser parameter elicits the maximum change in λ_s with the change equal to 0.02 and a non-disperser parameter results in a value of 0.019 (for the same *pq* combination and *f* - value), the disperser value is the maximum value, yet the difference between the values is not large. If you consider values of 0.05 vs. 0.01 (for disperser and non-disperser respectively), here the disperser value is still the maximum value but the difference between the two values is significantly higher. This tells us how strong the influence of the disperser

parameter is relative to the non-disperser parameters on the elasticity of λ_s for a *pq* combination and *f*-value, and is calculated as the relative difference between the disperser value and the non-disperser value. A large disperser contribution suggests that dispersers dominate the change in λ_s to an extent that nullifies the influence of non-dispersers (positive values) (Fig. 5.2). If the disperser contribution is intermediate then the influence of dispersers and non-dispersers to the elasticity of λ_s are similar (values close to zero; $\Delta = 0$ line in Fig. 5.2). Small disperser contributions suggest that the influence of non-dispersers to the elasticity of λ_s is higher than dispersers (negative values) (Fig. 5.2).

When the proportion of individuals that go through the dispersal stage is at the lowest (<3%), only parameters from non-disperser functions (i.e. reproduction rate and tritonymph transition rate) contribute to the change in λ_s. Therefore λ_s is most elastic to the parameters of non-disperser functions (Fig. 5.2). This is true across all *f* - values (good environment frequency) and across all ρ - values (autocorrelation values indicate environmental noise colour: high, positive values of ρ denote red noise; high, negative values of ρ denote blue noise; and $\rho = 0$ denotes white noise).

At a proportion of dispersers of 10% we see a large difference in the contribution of parameters of disperser and non-disperser functions to the change in λ_s (Fig. 5.2). The area above the line where the difference in the elasticity to λ_s to perturbation of disperser versus non-disperser function is zero ($\Delta = 0$ line), indicates where λ_s was most elastic to the parameter of the disperser function (deutonymph transition rate). This occurs over all environmental noise colours but only for *f* - values less than approximately 0.6. The area below the line indicates where λ_s was most elastic to only one parameter of the non-disperser function (reproduction rate). Again this

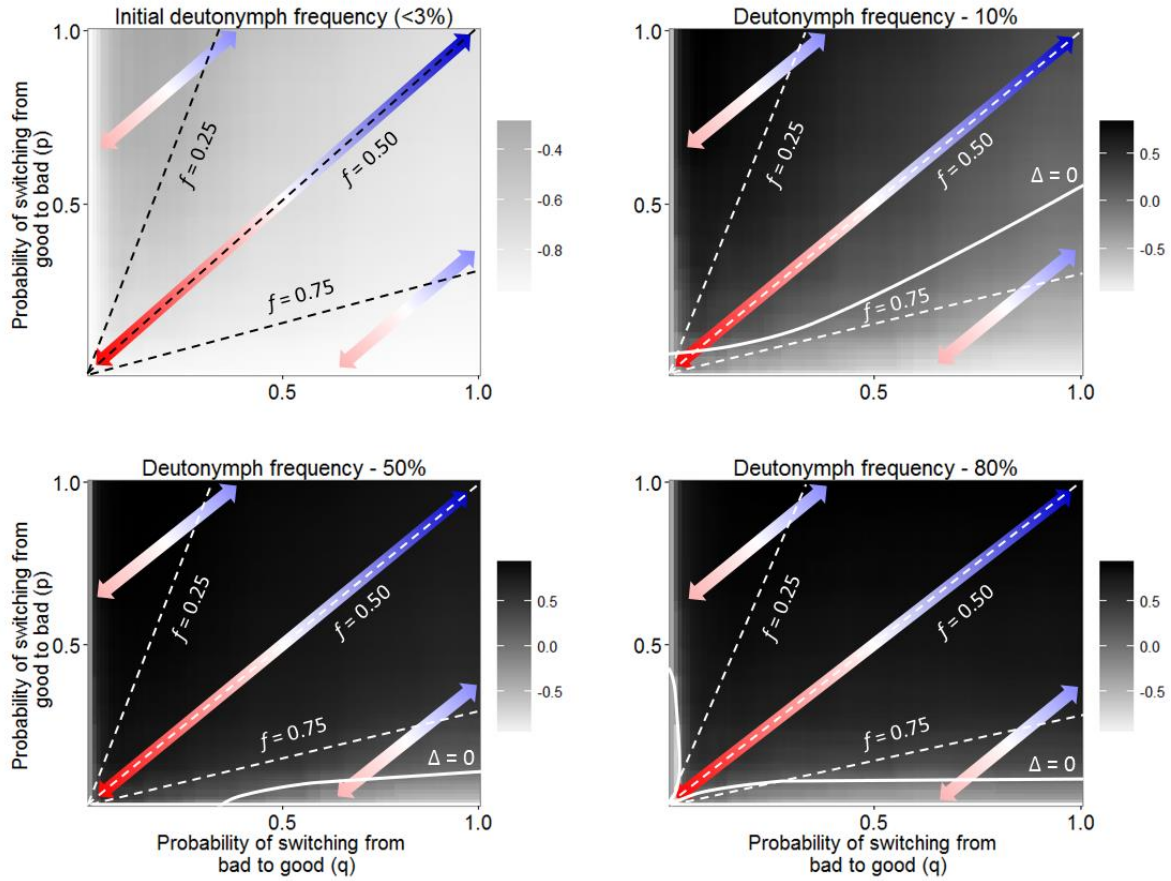

Figure 5.2. Difference (Δ) in the elasticity of λ_s to perturbation of the parameters in the disperser (deutonymph transition) versus non-disperser functions (reproduction rate, protonymph growth rate and tritonymph transition rate) in relation to the probability of switching from the bad to the good environment, *q*, and from the good to the bad environment, *p*, for all proportions of dispersers. The area below the Δ = 0 line denotes where λ_s was most elastic to parameters of the non-disperser functions in both the good and bad environment IPMs, and the area above the Δ = 0 line denotes where λ_s was most elastic to parameters of the disperser functions. The autocorrelation, *ρ*, in the environmental regimes (coloured arrows) is red in the bottom-left corner, white along the antidiagonal and blue in the top-right corner. The three dashed lines indicate values of *p* and *q* where the good environment frequency *f* equals 0.25, 0.50 and 0.75.

occurs over all environmental noise colours but only for f - values more than approximately 0.6.

When the proportion of dispersers reaches 50%, the position of the $\Delta = 0$ line changes and is below $f = 0.75$ (higher values of f); λ_s is now most elastic to the parameter of the disperser function (deutonymph transition rate) across a higher range of f - values but still encompasses all environmental noise colour (Fig. 5.2). Additionally, λ_s is now elastic to two non-disperser parameters from separate functions (reproduction rate and protonymph growth rate) and is restricted to the bottom right of the graph, the area where there are very high f - values and only in red and white environments.

Higher proportions of dispersers elicit a change in how the $\Delta = 0$ line shifts. At a proportion of dispersers of 80% the line extends to include a larger range of q values (probability of switching from the bad to the good environment) but λ_s is still most elastic to the parameter of the disperser function (deutonymph tranition rate) (Fig. 5.2). The elasticity of λ_s to parameters of the non-disperser functions (reproduction rate and protonymph growth rate) is now at f - values above 0.75 in red and white environmental noise colour, and a small range of values below 0.25 but only in red noise (bottom right and bottom left of Fig. 5.2 respectively).

Relationship between λ_s and its sensitivity to disperser life-history

We wanted to investigate the correlative response of average fitness to changes in environmental noise colour (ρ) and in good environment frequency (f), and see how this changes with the proportion of individuals that transition through the disperser stage. Overall the demographic costs of dispersal are the consequence of an interaction

between the proportion of time spent in good and bad environments and the proportion of individuals that transition through the disperser stage, regardless of environmental noise colour (Fig. 5.3). The range of stochastic growth rates increases (more values of the stochastic growth rate below 1.2) when the proportion of individuals that transition through to the disperser stage increases, which suggests that dispersers are associated with low average fitness. These results are qualitatively similar to the deterministic IPM for the good and bad environments (Table 5.2). Individuals in the good environment have a higher average fitness compared to individuals in the bad environments (Table 5.2).

At the lowest proportion of dispersers (<3%), λ_s is always most sensitive to perturbation of the parameters in the non-disperser functions, reproduction rate and tritonymph transition rate (relative elasticity values are all below 0.5), at all stochastic growth rates (Fig. 5.3). Furthermore, with increasing *f* - values (good environment frequency) there is an increase in the average fitness (λ_s), however high λ_s values only occur in red and white environments. As the proportion of dispersers increases, both dispersers and non-dispersers contribute to the change in λ_s (Fig. 5.3). Initially, there is a higher relative contribution of dispersers to the change in λ_s when λ_s is low; this is true for all environmental colours but only at low *f* - values (good environment frequency). At higher λ_s values, the contribution of non-dispersers to a change in λ_s is more than dispersers, in all environmental noise colours and at intermediate and high *f* - values. When the proportion of dispersers reach intermediate levels (approx. 50%) the range of λ_s values begins to increase as the smallest λ_s values become smaller. Here dispersers have a higher contribution to changing λ_s, only in red and white environmental noise and at high *f* - values does the contribution of non-dispersers become higher (Fig. 5.3).

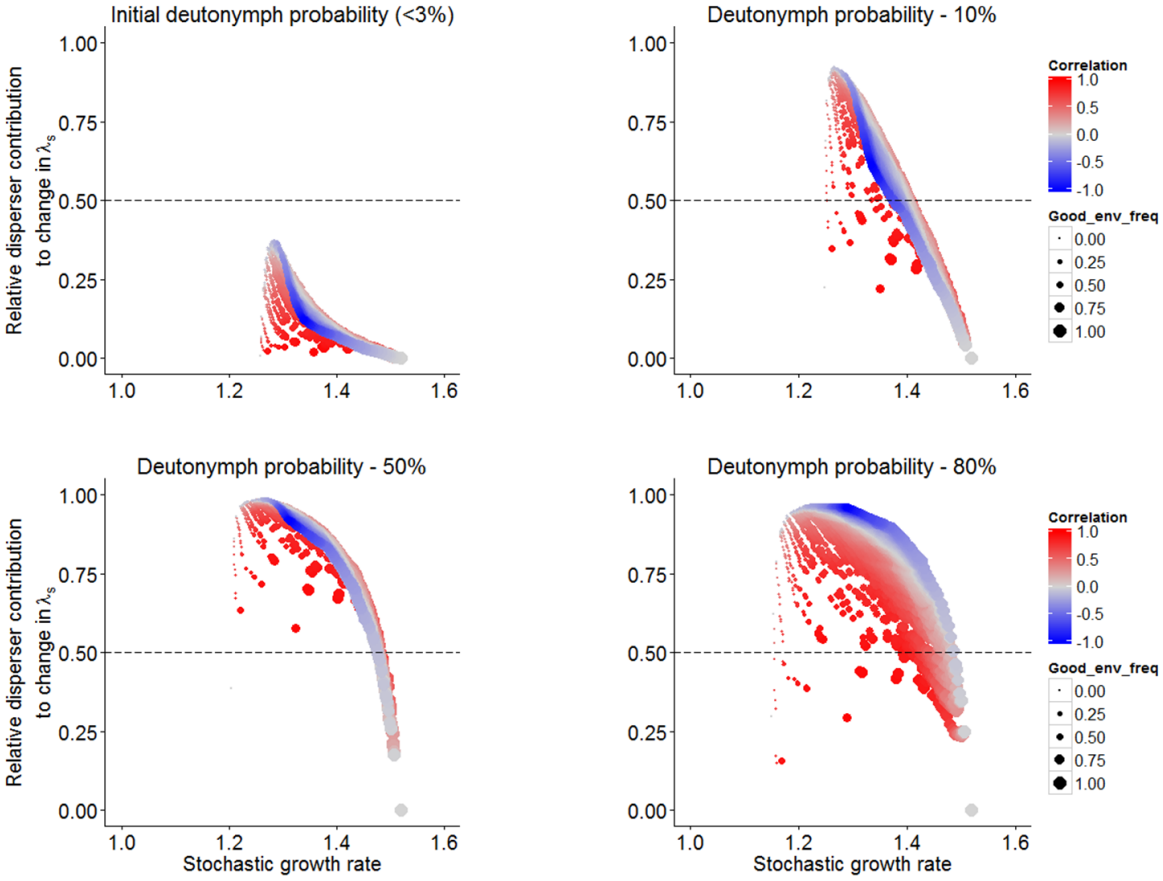

Figure 5.3. The relative disperser contribution to the scaled change in elasticity of λ_s as a function of stochastic growth rate (λ_s). The points are values that cover all the environmental regimes (all combinations of *p* and *q*; Fig. 5.2). Colour of the points indicate the autocorrelation value (ρ): ρ = 1 is red noise; ρ = 0 is white noise; ρ = -1 is blue noise, while size of the points increase with increasing good environment frequencies (*f* - values). The horizontal dashed line indicates where the scaled change in elasticity of λ_s equals 0.5; values above the line indicate that λ_s is more sensitive to dispersers and values below the line indicate λ_s is more sensitive to non-dispersers.

Additionally, when the proportion of dispersers is low and intermediate, when λ_s values are low the contribution of dispersers to the change in λ_s is higher under blue environmental noise colour than in white or red (Fig. 5.3, deutonymph probabilities of

Table 5.2. Comparison between population biology quantities predicted by the good environment IPM and at the four different proportions of dispersers in the bad environment IPM. The quantities are: population growth rate (λ_0); generation time (*GT*, days), and mean lifetime reproductive success (R_0).

	Quantity		
	λ_0	R_0	*GT*
Good environment	1.52	113.87	11.32
Bad environment			
Proportion - Initial (<3%)	1.26	50.89	17.12
Proportion - 10%	1.25	50.26	17.67
Proportion - 50%	1.21	28.15	17.88
Proportion - 80%	1.15	20.02	21.41

<3%, 10% and 50%). However at intermediate and high values of λ_s, the contribution of dispersers is higher under red environmental noise colour than in blue or white (Fig. 5.3, deutonymph probabilities of <3%, 10% and 50%). This is not true at a high proportion of dispersers where at all values of λ_s, the contribution of dispersers to the change in λ_s is higher in blue environmental noise than in white or red (Fig. 5.3, deutonymph probabilities of 80%).

Discussion

Here we determined how the proportion of dispersers in a population, the noise colour and good environment frequency that a population experiences affects fitness measured as long-term stochastic growth rate (λ_s). Overall, as disperser proportion increases, the maximum elasticity of λ_s to demographic rates shifts from those associated with non-disperser functions to those associated with disperser functions, regardless of noise colour, but only at low and intermediate frequencies of the good

environment. The fact that a relative increase in dispersers increases the contribution of dispersers to the elastic of λ_s is not surprising, but what is surprising is that this relationship does not hold when a population experiences good environmental conditions at a high temporal frequency. We found a similar phenomenon when investigating the relative disperser contribution to the elasticity of λ_s with increasing values of the stochastic growth rate. We find that there is a positive correlation between the stochastic growth rate and good environment frequency; at the same time, the contribution of dispersers to a change in λ_s declines irrespective of noise colour. At high good environment frequencies, λ_s was always most sensitive to perturbation of non-disperser demographic rates. These results suggest that only at the highest frequencies of good environments will non-disperser contribution be higher. One reason for this may be that, at low and intermediate frequencies of good environments, any contribution that non-dispersers have to a change in λ_s is countered by disperser contributions in the population when there is a switch between environment states. In contrast to the influence of good environment frequency, we found that noise colour had little influence on whether perturbation of disperser or non-disperser demographic rates has the greatest influence on λ_s. These contrasting responses to variation in two different characteristics of stochastic environments (noise colour and good environment frequency) could be of relevance in a wider context. Elasticity analyses identifies which vital rate (e.g. juvenile survival rate) has the greatest impact on population growth and is considered to be a useful way to quantify the relative importance of different life-history stages (Benton and Grant 1999). However, as we show, when incorporating environmental properties (variation and frequency) and changes in life-history of a proportion of individuals in the population, the impacts that demographic functions have on population growth rate vary depending on the individual environmental

properties. Therefore, to identify the relative importance of different life-history stages, consideration of how different environmental properties interacts with life-history (such as disperser individuals) to affect elasticity of λ_s need to be taken in to account.

We predicted that an increase in the proportion of dispersers in a population will have a greater negative effect on stochastic growth rate when there are long cycles of predictable but poor environments (red noise), and the frequency of the good environment is low. This is indeed what we observed as the values of λ_s associated with low frequency of good environments under red noise (i.e. the small red points in Fig. 5.3), decrease with increasing proportions of disperser. These conditions of long cycles of predictable but poor environments will favour dispersal expression so that individuals can find seek better quality habitats (Bower and Benton 2005). The greater the proportion of these dispersers that fail to disperse, the greater the negative impact on stochastic growth rate. We also predicted that this would be the case for unpredictable environments (white noise) with low good environment frequency. Again, our findings are in line with the predictions: values of λ_s associated with low frequency good environments and white noise (i.e. the small white points in Fig. 5.3) decrease with increasing proportion of dispersers. The dispersal response under such scenarios can be considered a bet-hedging strategy in an attempt to reduce the variation in fitness (den Boer 1968). What we did not predict was that this pattern also applies to blue noise environments (i.e. the small blue points in Fig. 5.3). It is difficult to see what the mechanism for this would be as blue noise represents short cycles of predictable environments which should not favour dispersal and should lessen the impact of dispersers on stochastic growth rate. One possibility may be that these short cycles of predictable environments when the good environment frequency is low result in, on average, increased bad environment-cycles which, along with increasing proportion of

dispersers, reduces the stochastic growth rate. The response to environmental variation we find is similar to a previous study by Smallegange et al. (2014) who investigated the response of life-history variables to the combination of environmental noise colour and frequency of environmental states. Their study showed that noise colour had little influence on the life-history variables but that there was a positive correlation with average fitness (λ_s) and good environment frequency. From this it appears that the structure of the population, specifically the presence of dispersers, and frequency of environments rather than the colour of the external environment, drive the population dynamics. The population response to the presence of dispersers, given the environmental properties, could then influence the strength of selection for dispersal.

Selection on dispersal is weaker in temporally constant environments (McPeek and Holt 1992; Travis and Dytham 1998; Travis 2001; Marshall and Burgess 2014) which would suggest that in redder environments, there is reduced selection for dispersal. However, we show that even when the proportion of dispersers is low (10%), and in increasing good environment frequencies (up to approximately 0.7) in red environments (long cycles of the environmental state), dispersers have a stronger influence to changes in λ_s than non-dispersers. This indicates that in intermediate to high periods of constant good environments (conditions under which selection for dispersal would be weak) characteristics of the population (in this case λ_s) are more elastic to dispersers. This, in turn, signifies that dispersal could be favoured in these situations. Elasticities can inform on the selection pressure on an organism's life history, as the response to selection is the product of the elasticities and evolvabilities (i.e. mean standardised genetic variances) (Benton and Grant 1999). Here we see that a shift in selection pressure on the elasticity of population growth rate from non-dispersers to dispersers requires only a small proportion of dispersers to be present in

the population. Selection for dispersal is now potentially a combination of not only environmental variation but also the combination of other environmental properties and the structure of the population, if the population includes disperser individuals. However, in order for dispersal to be selected for, the benefits of the strategy need to outweigh the costs of dispersing. We show there are demographic cost of dispersal on stochastic growth rate, with similar effects in the deterministic model and stochastic model (Table 5.2). We also show how stochastic growth rate changes with an increase in the proportion of dispersers and with the different environmental properties. Therefore this gives us an indication of what the benefits of dispersal need to be in order for the strategy to evolve as our model only includes the cost of developing into a disperser. Of course, this does not include other factors that can influence dispersal, such as inbreeding, but it does provide an indication of what the benefits of dispersing to a new habitat would need to be. The fact that we show a change in selection pressure for dispersal in environments that should not favour dispersal, suggests that the costs may not be outweighing the potential benefits of dispersal in this system. A true response to selection, however, can only be determined when considering elasticities together with evolvabilities. This outcome would also need to be tested empirically in a metapopulation context, where the response of local populations to dispersal would indicate the benefits in dispersing and if they outweigh the costs in our model.

A population response to environmental changes does not automatically suggest there will be a change in selection pressure on dispersal. How a population responds, therefore, is also influential in dictating the selection for dispersal. The response of a population to environmental variability is based on the frequency of the environmental variability (Cuddington and Yodzis 1999; Petchey 2000). For example, if environmental reddening occurs at a certain frequency but the response time of populations occurs at a

lower frequency relative to the reddening, populations will not be able to track the change (Petchey 2000). We see this in our study, under red environmental noise. In Fig. 5.3, red noise has high variance compared to the white and blue noise, which can be attributed to the high positive autocorrelation values (ρ close to one). In these instances, there are very long cycles of environmental states and the environments have not been able to change from either a good-to-bad or bad-to-good state within the time frame of the stochastic model. However, at less extreme positive ρ-values the variance disappears (Fig. S2), indicating that environments at these values are able to switch between states. This has been highlighted before, where short time intervals with high values of ρ can result in the average cycle time of the process being too large for the process to equilibrate within the given time interval (Tuljapurkar & Orzack 1980). Given a long enough timeframe, the variance we see will most likely be reduced at the high positive ρ-values. What a meaningful timeframe to assess the effects of environmental variation would be will ultimately depend on the study system. Therefore any population response due to environmental variation, and any subsequent interpretations, should be population specific.

Organisms will also respond differently to the predictability of environments, for example bet-hedging reproductive strategies employed by females may be one strategy in which an organism can respond to unpredictability of environments. If, for example, body size affects the initiation of dispersers then females could produce offspring of various sizes thereby ensuring that there are at least some dispersers, regardless of the environment. Future development of IPMs could incorporate such a process by including the bet-hedging decision in the female reproduction function. A limiting factor with the current model, however, is that given the relative proportions of dispersers and non-dispersers there is limited opportunity for more unexpected results

without a potential feedback. As mentioned earlier, it is not surprising that a relative increase in dispersers increases the contribution of dispersers to the elastic of λ_s but this will always be the dominating factor in the current model. A feedback can be incorporated in an IPM by extending it to make dispersal initiation resource dependent. Including resource dynamics would provide the opportunity to test if the outcomes found here still hold in the same way, as the proportions of dispersers may vary during the stochastic simulations given each autocorrelation value. This extended IPM could then also include varying rates of dispersal out of the natal population which would also affect any potential feedback.

We have shown how a change in a life-history characteristic (increased proportion of dispersers) within a population that is exposed to different environmental properties has subtle effects on local population dynamics. These local population effects in turn may influence the strength of selection on dispersal in environments where selection for dispersal would normally be considered weak. Whether this effect influences the metapopulation response for selection would need to be tested. Our model estimates the cost of developing in the dispersal stage. Identifying the benefits of dispersing and successful colonization of a new habitat would be the first step in addressing this question, assuming that dispersal, on average, increases fitness. Additional factors influencing the selection pressure on dispersal could then also be included, such as spatial variability in the environment (and the colour of this variation).

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Supporting Information

Supporting methods Construction and parameterisation of IPM

Figure S1 Comparison of the survival character demography function of disperser and non-disperser

Figure S2 Relative disperser contribution to change in λ_s with adjusted autocorrelation values (ρ)

Supporting methods

Construction of the integral projection model (IPM)

The IPM comprises a number of equations that are constructed from the character demography functions (*S(z,s,t)*, *P(z,s,t)*, *G(z'|z,s,t)*, *G(z'|z,s+1,t)*, *R(z,s,t)* and *D(z'|z,s,t)*. The number of stages in the IPM is eight: egg (E), larva (L), protonymph (P), deutonymph (D), tritonymph from protonymph (T_P), tritonymph from deutonymph (T_D), adult with no deutonymph in development (A_P) and adult with deutonymph in development (A_D). These equations calculate the number of females in each stage *s* at time *t* which is described by *n(z,s,t)*, with all the equations taking a similar form (see Table 5.1 in manuscript).

Predicted values from these equations are calculated to create a discrete approximation of the IPM. This is done by dividing the size domain of each stage into very small-width discrete bins ('mesh points'). The body size domain of each stage is divided into 50 size bins as a higher number of bins did not produce notably different results. Transition rates for the midpoint of two adjacent mesh points were estimated for each stage class. The final matrix size for the IPM was 400X400 (50 bins x 8 stages = 400 mesh points). The population growth rate was calculated as the dominant eigenvalue from the matrix approximation of the IPM.

Parameter estimation

Function parameters were estimated using the following statistical models: (1) Survival - generalised linear mixed model (GLMMs) with binomial error structure; (2) Transition rates – GLMM with binomial error structure; (3) Growth – GLMM with a

Gaussian error structure; (4) Fertility rate (Reproduction) – GLMM with a Gaussian error structure; (5) Inheritance kernel – generalised linear model (GLM) with a Gaussian error structure. In all cases body length and body length squared were linear predictors and, with the exception of the inheritance function where we fitted a GLM, mite identity was included as a random factor. The response variables for the five functions were: (1) Survival – from time t to time t+1 (this is binary and set as 0 or 1), (2) Transition - probability of growing to the next stage at time t+1 (see below), (3) Growth - mean and variance in body size at time t+1, (4) Fertility - the number of eggs produced at time t+1, and (5) Inheritance - mean and variance in size of eggs produced at time t+1 by each individual at time t. In the case of eggs their size at time t+1 equalled their size at time t as eggs do not increase in size. In the case of the bad-environment IPM, the linear deutonymph transition function from Deere et al. (in prep, Chapter 4) was used.

In the event dispersers (deutonymph) enter the good environment IPM, stages subsequent to the deutonymph stage take on the parameter values of the good environment IPM. For example, an adult that developed from a deutonymph (A_D) that entered the good-environment IPM would take on the growth and survival parameters of the adult that did not develop from a deutonymph in the good environment IPM (A_P) (see below).

Parameter values of character-demography functions

Survival rates for the bad-environment IPM (fraction per day)

E: $y = 0.956$ (n = 297); L: $y = 0.999$ (n = 112); P: $y = 0.910$ (n = 166); D: $y = 0.999$ (n = 426); T_P: $y = 0.999$ (n = 132); T_D: $y = \frac{1}{1 + \frac{1}{e^{(-0.4175+6.9435z)}}}$ (n = 119); A_P: $y = 0.999$ (n = 115); A_D: $y = 0.933$ (n = 60).

Survival rates for the good-environment IPM (fraction per day)

E: $y = 0.999$ (n = 110); L: $y = 0.948$ (n = 58); P: $y = 0.999$ (n = 38); D: $y = 0.999$; T_P: $y = 0.999$ (n = 152); T_D:= T_P; A_P: $y = \frac{1}{1 + \frac{1}{e^{(26.05-54.20B+31.47z^2)}}}$ (n = 1103); A_D = A_P.

Life stage transition rates for the bad-environment IPM (fraction per day)

E→L: $y = \frac{1}{1 + \frac{1}{e^{(-1.437+8.674z)}}}$ (n = 97); L→P: $y = \frac{1}{1 + \frac{1}{e^{(-6.933 + 29.429z)}}}$ (n = 47);

P→D: $y = \frac{1}{1 + \frac{1}{e^{(-2.601 + (-5.673)z)}}}$ (n = 137); P→T: $y = \frac{1}{1 + \frac{1}{e^{(-11.220 + 26.235z)}}}$ (n = 137); D→T: $y = \frac{1}{1 + \frac{1}{e^{(-15.864+41.321z)}}}$ (n = 155); T_P→A_P: $y = \frac{1}{1 + \frac{1}{e^{(-6.703 + 13.100z)}}}$ (n = 76); T_D→A_D: $y = \frac{1}{1 + \frac{1}{e^{(-6.275 + 14.933z)}}}$ (n = 45).

Life stage transition rates for the good-environment IPM (fraction per day)

E→L: $y = \frac{1}{1 + \frac{1}{e^{(-0.909+4.149z)}}}$ (n = 80); L→P: $y = \frac{1}{1 + \frac{1}{e^{(-3.107 + 16.973z)}}}$ (n = 48); P→T: $y = \frac{1}{1 + \frac{1}{e^{(-4.100 + 12.591z)}}}$ (n = 37); D→T: $y = 0.999$; $T_P \rightarrow A_P$: $y = \frac{1}{1 + \frac{1}{e^{(-8.006 + 15.001z)}}}$ (n = 150); $T_D \rightarrow A_D = T_P \rightarrow A_P$.

Reproduction rate for the bad-environment IPM (no. per day)

A_P: $y = 0.5(-18.446 + 35.209z)$ (n = 190); A_D: $y = 0.5(-13.592 + 33.892z)$ (n = 172)

Reproduction rate for the good-environment IPM (no. per day)

A_P: $y = 0.5(-213.3 + 510.40B - 273.49z^2)$ (n = 701); $A_D = A_P$

Mean growth rates for the bad-environment IPM (when staying in the same life stage) (mm)

E: $y = L$ (n = 65); L: $y = 0.11739 + 0.64316z$ (n = 29); P: $y = 0.0772 + 0.904z$ (n = 39); D: $y = L$ (n = 153); T_P: $y = 0.0776 + 0.9538z$ (n = 44); T_D: $y = -0.0772 + 1.3570z$ (n = 23); A_P: $y = 0.3977 + 0.5359z$ (n = 215); A_D: $y = 0.2816 + 0.6355z$ (n = 238)

Variance in growth rates for the bad-environment IPM (when staying in the same life stage) (mm^2)

E: $y = 0.0001$ (n = 65); L: $y = -0.0008 + 0.0050z$ (n = 29); P: $y = -0.0007 + 0.0040z$ (n = 39); D: $y = 0.0001$ (n = 153); T_P: $y = 0.0039 - 0.0042z$ (n = 44);

T_D: $y = -0.0044 - 0.0060z$ (n = 23); A_P: $y = 0.0009 - 0.0004z$ (n = 215); A_D: $y = 0.0014 - 0.0016z$ (n = 238)

Mean growth rates for the good-environment IPM (when staying in the same life stage) (mm)

E: $y = B$ (n = 59); L: $y = 0.0977 + 0.8171z$ (n = 24); P: $y = 0.1973+0.6840z$ (n = 29); T_P: $y = 0.0900 - 1.062z$ (n = 118); $T_D = T_P$; A_P: $y = 0.5896 - 0.3417z$ (n = 422); $A_D = A_P$

Variance in growth rates for the good-environment IPM (when staying in the same life stage) (mm^2)

E: $y = 0.0001$ (n = 59); L: $y = -0.0003 + 0.0150z$ (n = 24); P: $y = -0.0037 + 0.0155z$ (n = 29); T_P: $y = 0.0095 - 0.0109z$ (n = 118); $T_D = T_P$; A_P: $y = 0.5896 - 0.3417z$ (n = 422); $A_D = A_P$

Inheritance function (mean offspring-mother difference) for the bad-environment IPM (mm)

A_P: $y = 0.1638$ (n = 96); A_D: $y = 0.1689$ (n = 175)

Variance around inheritance function for the bad-environment IPM (mm^2)

A_P: $y = 0.00008$ (n = 96); A_D: $y = 0.0001$ (n = 175)

Inheritance function (mean offspring-mother difference) for the good-environment IPM (mm)

A_P: $y = 0.1663$ (n = 208); $A_D = A_P$

Variance around inheritance function for the good-environment IPM (mm^2)

$A_{P:}\ y = 0.00019$ (n = 208); $A_D = A_P$

Supporting figures

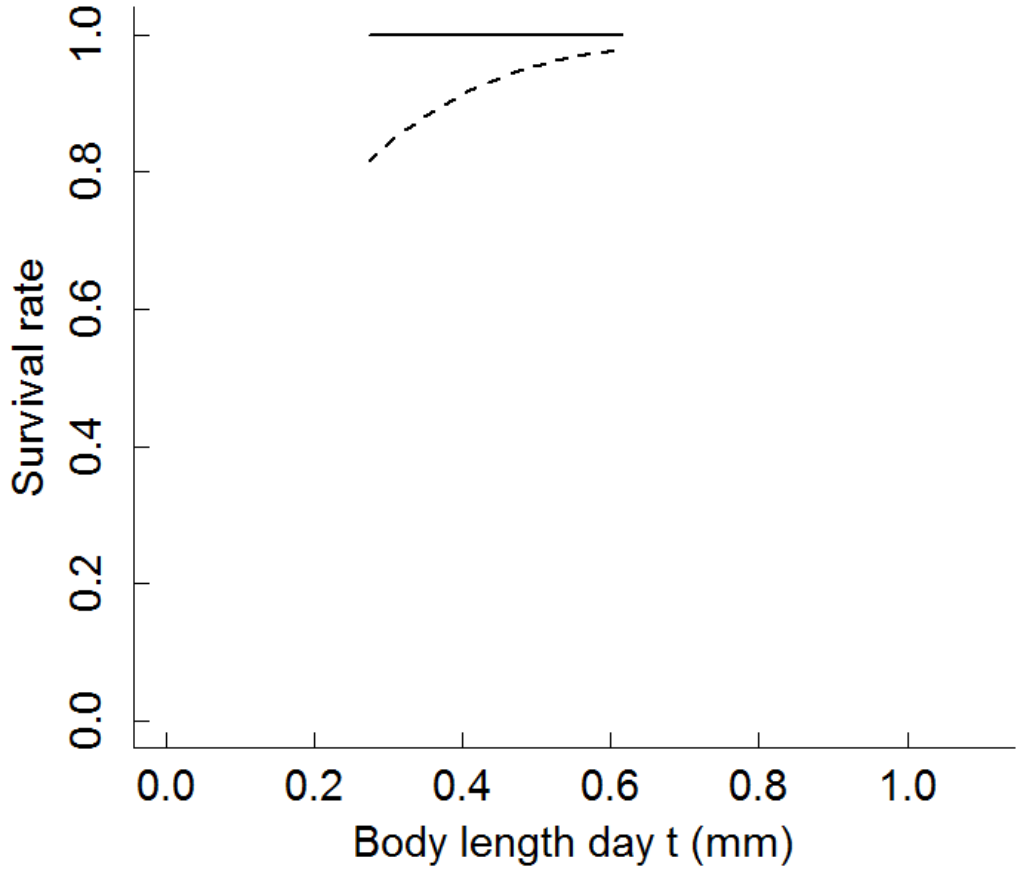

Figure S1. Survival character demography functions of adult females in a good- and bad-environment (solid and dashed lines respectively) (see Smallegange et al. 2014 and Deere et al. in prep, Chapter 4).

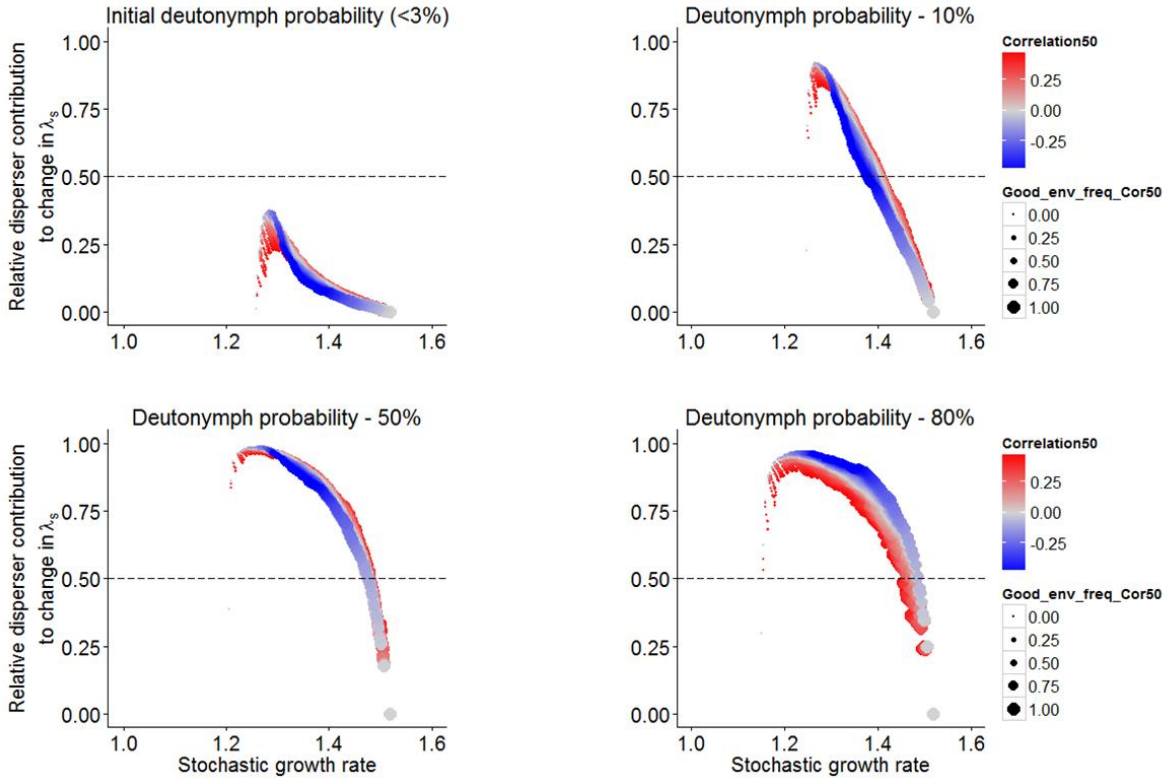

Figure S2. Scaled relative disperser contribution to change in λ_S as a function of stochastic growth rate for low, intermediate and high deutonymph probability with adjusted autocorrelation values (ρ). Extreme autocorrelation values are removed, with adjusted values spanning ρ = -0.5 to ρ = -0.5.

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Chapter 6

Discussion

In this discussion I aim to provide the context in which this work contributes to the field as well as addressing limitations and providing some thoughts on possible future work.

Complexities of interacting life-history processes

I have focused on two life-history strategies, alternative reproductive phenotypes (ARPs) and dispersal, both of which are found in most taxa and have received much attention (Gross 1996; Clobert et al. 2001; Oliveira et al. 2008). These two processes can be classed as conditional strategies, where phenotype expression (i.e. alternate morphs; dispersal morphs) depends on a threshold response to an environmental cue (Gross 1996; Clobert et al. 2001). Currently, the environmental threshold (ET) model best explains the evolution of conditional strategies (Hazel et al. 2004; Tomkins and Hazel 2007; Buoro et al. 2012). However, I show that these two processes interact, as juvenile males that disperse (by developing into the facultative dispersal stage) always develop into one of the two male morphs (fighters). Therefore, incorporating this interaction into the ET model may be problematic as identifying the threshold for expression would be difficult to resolve because of how male morph and dispersal interact. I discuss two possible scenarios. In the first, there is one alternating threshold during ontogeny; in this scenario if a threshold for the expression of dispersal is reached then there is no subsequent threshold for ARP expression (i.e. only fighters are expressed); alternatively if no threshold for dispersal expression is reached then there is a threshold for ARP expression. In the second scenario there are always two distinct thresholds during ontogeny (e.g. Rowland and Emlen 2009); one involving the initiation of dispersal and one the initiation of male morphs (here ARP is not dependent on dispersal expression). In the scenario for one threshold, the ET model could explain the

expression of ARPs given there is no dispersal expression. However, to identify if there is only one threshold would first require knowing when during ontogeny, the decision to express male morph and dispersal are made. In this scenario I assume that dispersal is expressed first during ontogeny but it may be just as likely that ARP expression occurs earlier during ontogeny and in the above scenario the two strategies would be reversed. If there are indeed two distinct thresholds in ontogeny then this may also be explained within the ET model. The ET model is a quantitative genetic model and selection is on the distribution of thresholds within the population with the effect of selection acting on the mean threshold that evolves (Tomkins and Hazel 2007). If there are two strategies that interact, and the result alters the mean threshold in one or both strategies, this would suggest that the threshold distributions of both strategies need to be accounted for in the ET model. The current ET model would then need to be extended to include multiple conditional strategies, with multiple thresholds, that interact. If the ET model cannot be extended, then this may not be the best model to explain interacting conditional strategies and how they evolve.

The life-history outcome of the interaction of dispersal and ARPs, where only fighter morphs develop from juvenile male deutonymphs, is puzzling. One possible explanation for this observation is that deutonmyphs always have sufficient resources to grow past the size threshold for male morph expression, which would explain why only fighter males develop from deutonymphs. If this was the case, the resources that need to be stored would be significant as this would need to allow for surviving potentially long distant dispersal (Houck and Oconnor 1991) as well as developing costly fighter morphology (Radwan et al. 2002). Many species show dispersing individuals that store fat reserves, however dispersers are generally larger than non-dispersers (Clobert et al. 2001). In this study, deutonymphs do not have a larger body size than individuals of the

protonymph stage, the stage from which deutonymphs develop, or non-dispersing individuals of the tritonymph, the stage following the deutonymph stage in disperser development. Therefore, it is unlikely that they can store enough resources for fighter development, and maintenance during dispersal. Additionally, the deutonymph stage is non-feeding, has no hollow gut and has extensive sclerotization (Houck and Oconnor 1991); features which aid in desiccation resistance more than fat storage. One way to determine whether, and how many, resources are stored would be to identify energetic expenditure and fat reserves of deutonymphs. Determining metabolic rate (resting and active) and lipid content (through lipid extraction) has been done experimentally in a number of species (Chown and Nicholson 2004; Hahn and Denlinger 2007) and these approaches can be applied to deutonymphs. Alternatively, dispersal could be an environmental cue for male morph expression. Dispersal is expressed when the benefits of successfully colonizing a new habitat will potentially outweigh the costs of dispersing or staying in the natal habitat (Bowler and Benton 2005). If only male fighter morphs develop from deutonymphs this could ensure the best chance of establishment after dispersing into a new habitat, as fighter morphs are traditionally considered to have higher fitness than scramblers (Maynard Smith 1982). Fighter morphs also have modified legs which could be useful tools in a new habitat as they can be used to fend off competing males, kill and consume other con- or heterospecific mites (Lukasik 2010), or to defend themselves from predatory mites (Iza Lesna, personal communication). If dispersal was an environmental cue for male morph expression, this would have consequences for the environmental threshold (ET) model. The ET model acknowledges the possibility of multiple cues in condition dependent traits, but this would again require knowing when during ontogeny the decision to express male morph and dispersal are made. There could also be the possibility that levels of fighter

expression from deutonymphs could be dependent on deutonymph frequency in the population. Deutonymph frequency is dependent on environment quality (e.g. diet) with higher deutonymph expression in environments of poorer quality. Our culture on an oats diet did produce deutonymphs but only at levels of deutonymph expression of < 3%. However, identifying diets that could produce various proportions of deutonymph expression within the population would provide an opportunity to test whether fighter expression is influenced by the proportion of deutonmyphs. The question can then be asked, if in an environment where the population is producing a larger proportion of deutonymphs, would male deutonymphs still only develop into fighters. If not, is there a male morph trade-off with deutonymph (dispersal) frequency? This would then assume that after a certain proportion of deutonymphs in the population is reached, having the highest possible fitness in a new habitat is less essential.

Finally, the interaction between ARPs and dispersal suggests that the role that frequency-dependence plays in maintaining ARPs may need to be teased apart even further. This could also explain the weak frequency-dependence found in Chapter 2 (dispersal morphs were not taken into account in the study) as opposed to the suggested weak stabilizing mechanisms. Future work to identify frequency-dependence should account for deutonymphs during development. I would redo the frequency-dependence experiment initially using survival again but then assessing other traits (e.g. body size or survival in juveniles) as well as using two environmental states. The first would be one where I would alter the fighter:scrambler ratio where the fighters developed from deutonymphs. I would then compare male morph survival in that state to one where the fighter:scrambler ratios were composed of fighters that did not go through the deutonymph morph. This would show if there was frequency-dependence in either state and if so, would going through the deutonymph stage affect the outcome. This could

then be done over a range of conditions which produce various proportions of deutonymphs in the population to test if proportions of dispersers have an effect on frequency-dependence. Identifying a genetic component to deutonymph development would also be needed to determine if the trait is heritable in both male morphs, if this is the case then the notion that deutonypmhs only produce fighter morphs is false and the challenge would be to identify the conditions under which scrambler morphs develop. However, an alternate explanation to frequency dependence maintaining the male polymorphism may be male senescence. In chapter 2, Fig. 4, I show that in a good quality environment after 10 days fighter male survival is significantly lower than scrambler male survival. This difference in survival could well ensure that scrambler males can have an equivalent fitness to fighter males over their lifetime which may help maintain the male polymorphism. This, though, would need further investigation to identify whether senescence could be a potential mechanism to maintain ARPs in this species.

Disperser costs and natal populations

The proportion of dispersers in a population that ultimately disperse affect the dynamics and persistence of spatially structured populations and communities differently. The changes in dispersal rate between environments can cause asymmetry between populations and can ultimately affect population persistence in a metapopulation (Vuilleumier and Possingham 2006). However, conditions experienced in the natal habitat are shown to affect dispersal probability and, as such, asymmetry between populations (Benard and McCauley 2008). This suggests that changes in natal populations play an integral role in metapopulations. I have shown that in natal populations, where individuals have developed into disperser individuals and have

failed to disperse, there is a negative effect on the population. What we show is no different to what is found in populations with individual specialization, where the population's phenotype distribution can affect population-level ecological traits (Roughgarden 1972; Collins et al. 1993; Bolnick et al. 2003, Smallegange et al. 2014). The idea that natal habitats can affect the probability of dispersal has been highlighted (see review by Benard and McCauley 2008), however this does not consider the fact that unsuccessful dispersers in turn can affect the natal habitats. This has meant that costs to the natal population of unsuccessful dispersers have so far not been considered in a metapopulation context. Including these costs in a metapopulation would be beneficial in a number of ways. First, the effect of unsuccessful dispersers on natal populations can be included with other environmental factors that may affect dispersal rates (e.g. low resources). Changes in dispersal rates between populations can generate asymmetry that can effect ecological and evolutionary processes. For example, asymmetry in the dispersal rates in a metapopulation affects population persistence. Asymmetry in dispersal rates affect evolutionary processes by altering the magnitude and symmetry of gene flow between populations (Benard and McCauley 2008). How relevant the costs of unsuccessful dispersers would be in influencing asymmetry in dispersal, and the subsequent ecological and evolutionary effects on populations, can only be investigated by comparing the outcome when these costs are included and when they are not. Second, including them in a metapopulation context would allow for the opportunity to characterise other dispersal costs and benefits of successful dispersal into a new habitat, which would identify the risk-benefits of individuals that have successfully dispersed. The demographic costs give us a potential estimate of what the benefits would need to be in order for dispersal to evolve. We did not include other costs such as inbreeding, but the estimate would still be a good indicator of what the

benefits should be. Finally, the effect on natal populations (e.g. population growth rate) when individuals that develop into dispersers actually disperse as opposed to not dispersing (i.e. allow emigration) can be detected. Here, the proportion of dispersers that actually disperse can also be manipulated together with the proportion of individuals that invest in dispersal. This would provide a more robust alternative to the standard calculation of dispersal rate from a population which is calculated as the proportion of the population that disperses each generation (Hanski 1999). Assessing the results of this study in a metapopulation context would provide the opportunity to test if the costs to natal populations are mitigated in a metapopulation and how the strength of selection we see in the natal population changes at the metapopulation level.

The importance of natal population conditions and how life-history processes influence them stress that the role of eco-evolutionary dynamics need to be considered in line with this work. The model approach I have used does not include any population feedbacks through, for example density dependence, but accounting for feedback is important as previous work has shown how eco-evolutionary feedback can play a role in life-history evolution (Cameron et al. 2013), including this system (Smallegange and Deere 2014). Smallegange and Deere (2014) show that eco-evolutionary feedback influences male morph expression. When scrambler males were removed from the populations, scrambler frequency instead of fighter frequency increased in contrast to evolutionary theory. This counter-intuitive response was attributed to reduced cannibalism opportunities for fighters which was hypothesised to be a stronger selection force on fighter expression than the harvesting of scramblers. However, in the study by Smallegange and Deere (2014) populations were kept on a restricted, high quality diet with populations, on average, producing less than 1% deutonymphs; therefore the role of the deutonymph stage in this feedback process could not be assessed. Given my

finding that only fighter males develop from deutonymphs it is likely that feedbacks from deutonymph expression will play a role in how population size and structure fluctuate. For example, if there is selection against dispersal there will be fewer fighters resulting in less cannibalism, this in turn will have a knock on effect on population size and structure. The question would then be if including dispersal would alter the evolutionary feedbacks that was found by Smallegange and Deere (2014). It would be interesting to see if including dispersal reduces or enhances existing eco-evolutionary feedbacks or whether dispersal would elicit an eco-evolutionary feedback of its own, resulting in multiple feedbacks that vary in their strength depending on current population conditions. These questions could not be answered using standard experimental evolution studies as these studies are not adequate at disentangling ecological and evolutionary change. The reason for this is that in such studies, discrete generation methods are often used (e.g. Tomkins et al. 2011) which changes the structure of the populations at the start of each new generation and, in so doing, interrupts ongoing ecological change and any population feedback effects (Smallegange and Deere 2014). What this highlights, and what has been echoed before (Smallegange and Deere 2014), is that population feedbacks should not be ignored in experimental evolution studies.

Study limitations and recommendations

This study shows how a natal population can be influenced by individuals that have invested into dispersal morphology but are unsuccessful in dispersing from the natal population. These findings are novel but follow-up work should take into account some of the limitations of this study. Firstly, this study focuses on one population, the natal population, but has not quantified consequences for recipient populations. Also,

because no density-dependence has been included in the model, predictions are limited to density-independent scenarios. It should be said, though, that the appropriate growth rate to use to describe outcomes in density-dependent environments is context dependent (Metz et al. 1992), greatly complicating model analyses. Furthermore, density-independent demographic models may under certain circumstances provide adequate approximations when density or frequency dependence operates (Caswell 2001). Nevertheless, context-dependent models that do allow for population feedbacks could shed light on how dispersal propensity may feedback to influence population fluctuations, especially in response to environmental stochasticity.

In this study, I have also not addressed the evolution of dispersal. I highlight to what extent population growth rate is sensitive to the dispersal stage which could inform on the selection pressure on an organism's life history. Previous studies on evolutionary stable dispersal strategies and evolution of dispersal polymorphisms have included spatially and temporally varying environments, density-dependence, distance-dependent factors (e.g. competition, costs) as well as chaotic population dynamics (Comins et al. 1980; Comins 1982; Frank 1986; Cohen and Levin 1991; McPeek and Holt 1992; Olivieri et al. 1995; Holt and McPeek 1996, King and Roff 2010; Fronhofer et al. 2015; Massol and Débarre 2015). I also do not address temporal changes in populations and the stochastic properties of population fluctuations. Often the role of dispersal in population synchrony is investigated; this can be done in combination with demographic and environmental stochasticity, and population density regulation (Hanski 1999; Lande et al. 1999; Kendall et al. 2000; Engen et al. 2002; Lande et al. 2003). How dispersal influences other properties of fluctuating populations such as extinction probability and minimum viable population size are also investigated (Lande et al. 1998; Hanski 1999; Engen et al. 2002; Lande et al. 2003). These dispersal models,

also include, in some combination, stochasticity (demographic and environmental) and population density regulation. However, the model framework used here, IPMs, do allow for the inclusion of feedbacks (e.g. Coulson et al. 2011) such as dispersal rates that are influenced by the proportion of individuals that remain in the natal population. The extended model will allow one to investigate the evolution of dispersal and how dispersal influences properties of fluctuating populations, such as population synchrony and persistence.

Given the opportunity I would improve on a number of aspects of the work done in this thesis. Firstly I would re-parameterise the dispersal IPM with data from a gradient of habitat qualities that produced various proportions of dispersers, as currently we manipulate the proportions that become deutonmyphs within the IPM (Chapter 4). Secondly I would also, during the life-history, place deutonymphs on a good quality habitat which would provide more robust data to parameterise deutonymphs that enter a good environment in a stochastic model (as in Chapter 5). I would then like to expand the IPM to a two-sex IPM which includes both male morphs, as currently the model only includes females. The inclusion of both sexes in an IPM has recently been developed (Schindler et al. 2013) and this would provide a more robust indication of the interaction of dispersal and ARPs within a population and the potential cost to the natal population. Finally, I would extend the IPM to include population feedback. Initially, I would focus on a mechanistic representation of individual life history trajectories (e.g. dynamic energy budget theory, Kooijman 2000). The IPM can then be extended to include population feedback on resources. In so doing feedback to the body size distributions and the condition of individuals, would affect dispersal development. With these, more robust, models I would then include them in a metapopulation model to try and identify how the costs and benefits of dispersal may change in a metapopulation

model that incorporates asymmetry in the dispersal of individuals. I would also like to take an experimental approach so a comparison can be made with the results from the model. A long term metapopulation experiment where natal populations and dispersal rates are manipulated will also provide an opportunity to detect any eco-evolutionary feedback on ARPs and dispersal in the populations. Only by accounting for eco-evolutionary feedbacks can we get a grasp of the effect of interacting life-history strategies and how they may evolve.

Benefits to other areas/systems

This work could benefit other areas of movement ecology, where movement involves only a proportion of individuals in a population (e.g. within-population migratory dimorphism or variation in individual movement ability). One such area is partial migration. Partial migration occurs when a population has both sedentary and migratory individuals, which occurs in many species of birds, mammals, fish and insects (Adriaensen and Dhondt 1990; Kaitala et al. 1993; Ball et al. 2001; Brodersen et al. 2008; Chapman et al. 2011). These discrete individuals differ in their vital rates depending on whether they migrate or remain in the natal population. If not all migratory individuals are able to migrate then the natal population structure and dynamics will differ to a population where all migrating individuals do migrate. Another area of movement is heterogeneous movement. In this case individuals in populations can vary in their dispersal distance or mobility. This is found in some fish species where a positive correlation with distance and individual length is found, in these cases stream size can also play a role (Rodrígues 2002; Radinger and Wolter 2014). Therefore populations where larger individuals disperse will differ in their structure and dynamics compared to a population where larger individuals may be

prevented from dispersing (e.g. by changes in lake sizes). Heterogeneous movement is also seen in the Glanville butterfly, however here it is the combination of physiological differences in flight metabolic performance and habitat connectivity that contribute to the observed variation in mobility (Hanski et al. 2004). However, it may be difficult to determine how the presence of mobile individuals that cover a gradient of dispersal abilities proportionally affect a population. The outcome of this thesis would benefit systems with either movement (migratory) dimorphisms or more distinct movement polymorphisms (in the case of heterogeneous movement), as, in such cases fitness trade-offs can be measured more easily.

In both movement areas, partial migration and heterogeneous movement, populations are prone to habitat fragmentation, which could influence the movement of individuals. Increased fragmentation may prevent mobile individuals from leaving populations which in turn will affect the population structure and dynamics. Therefore this work could also be beneficial for areas such as conservation or population management where habitat fragmentation can play a large role in how populations persist.

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Appendix – Other published work

Smallegange, I. M., J. A. Deere, and T. Coulson. 2014. Correlative Changes in Life-History Variables in Response to Environmental Change in a Model Organism. The American Naturalist 183:784–797

Correlative Changes in Life-History Variables in Response to Environmental Change in a Model Organism

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abstract: Global change alters the environment, including increases in the frequency of (un)favorable events and shifts in environmental noise color. However, how these changes impact the dynamics of populations, and whether these can be predicted accurately has been largely unexamined. Here we combine recently developed population modeling approaches and theory in stochastic demography to explore how life history, morphology, and average fitness respond to changes in the frequency of favorable environmental conditions and in the color of environmental noise in a model organism (an acarid mite). We predict that different life-history variables respond correlatively to changes in the environment, and we identify different life-history variables, including lifetime reproductive success, as indicators of average fitness and life-history speed across stochastic environments. Depending on the shape of adult survival rate, generation time can be used as an indicator of the response of populations to stochastic change, as in the deterministic case. This work is a useful step toward understanding population dynamics in stochastic environments, including how stochastic change may shape the evolution of life histories.

Keywords: bulb mite *Rhizoglyphus robini*, integral projection models, perturbation analysis, slow-fast life-history continuum, stochastic population growth rate.

Introduction

Environmental change greatly influences the dynamics of populations (Andrewartha and Birch 1954; Boyce et al. 2006) and can generate simultaneous responses in population fluctuations, life history, and phenotype distributions (Hairston et al. 2005; Coulson et al. 2011). However, the wide range of observed and expected ecological and evolutionary responses to changes in environmental conditions (Reznick et al. 2001; Post et al. 2009; Coulson et al. 2011) means that understanding the population consequences of environmental change has become one of the greatest challenges in biology (Schoener 2011). This challenge is even more current today as several aspects of the environment are changing simultaneously: the frequency of ecological and climate events (e.g., algal blooms or El Niño years) is increasing and there have also been temporal changes in the amplitude and probability distribution of climate variables (Emanuel 2005; Katz et al. 2005; García-Carreras and Reuman 2011). Detailed investigations into the consequences of such changes largely fall into two groups. The first group uses data from field populations to decompose variation in demographic rates into different ecological and/or evolutionary contributions (Coulson et al. 2001; van de Pol et al. 2010), and it also uses data from field populations to assess how populations respond to perturbation of demographic rates (e.g., Forcada et al. 2008; Morris et al. 2008; Dalgleish et al. 2010; Coulson et al. 2011) and to recurrent disturbances (e.g., Tuljapurkar et al. 2003; Barrows et al. 2010; Vincenzi et al. 2012). The second group consists of theoretical studies that investigate the demography of populations and stylized life histories in stochastic environments (e.g., Wilmers et al. 2007; Lande et al. 2009; Tuljapurkar et al. 2009). Results from the first group of studies indicate that the relative importance of specific demographic rates to the growth rate of populations can depend on the frequency of disturbances (Tuljapurkar et al. 2003). They also forecast that the population growth rate of longer-lived species will be less sensitive to increases in climate variability (Morris et al. 2008; Dalgleish et al. 2010) but that the population consequences of a change in climate variability can be outweighed by the consequences of changes in the mean environment (van de Pol et al. 2010; Coulson et al. 2011). The theoretical studies, in turn, indicate that a change in the serial correlation of demographic rates may increase or decrease population growth rate depending on the structure of the life history (Tuljapurkar et al. 2009) and that a change in the magnitude or type of environmental stochasticity can induce a shift from fast to slow life histories (Lande et al.

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2009) or even lead to extinction due to increased population fluctuations (Wilmers et al. 2007). These are notable insights but the question of how common concurrent change in life-history variables such as the population growth rate, generation time, and lifetime reproductive success is in response to changing environmental change, is still open. In a recent study, Coulson et al. (2011) concluded from their analysis on the demography of Yellowstone gray wolves that different types of environmental change can generate a wide range of responses in population biology quantities such as life-history variables. However, only a consistent population response, that is, one where life-history variables show a correlative response to environmental change so that testable predictions on the consequences of environmental change can be derived, improves our understanding of population dynamics in variable environments. Accurate predictions of the life-history response can be achieved when the relationship between a species' life history and its environment is known in great detail (Coulson et al. 2011). We therefore set out to explore the impact of environmental change on the life-history response of a model organism, the bulb mite (*Rhizoglyphus robini*), of which we have detailed knowledge of how environmental quality affects its life history (Smallegange 2011*a*, 2011*b*; Smallegange and Coulson 2011). To this end, we combine recently developed population modeling approaches and theory in stochastic demography to predict how different life-history variables vary in response to environmental change across a range of stochastic environments.

Changes in environmental conditions translate into changes in demographic rates (Boyce et al. 2006), which can in turn affect the dynamics of a population (Coulson et al. 2011). Integral projection models (IPMs; Easterling et al. 2000) provide a powerful approach to investigate concurrent change in life-history variables within populations (Coulson et al. 2011). Here we develop IPMs for the bulb mite to examine the likely life-history response of populations to changes in important properties of the environment: (i) the temporal frequency of favorable environment states and (ii) the serial correlation of environment states. Investigating population responses to these two types of changes is paramount to understanding the effects of climate change on the dynamics of structured populations (Boyce et al. 2006; Wilmers et al. 2007) and also informs on how stochastic change shapes the evolution of life histories (Tuljapurkar et al. 2009). We use a scenario in which the environment is in one of two states, good or bad, and investigate the life-history response to changes in the environment in four steps. First, we construct the character-demography functions that comprise the good- and bad-environment IPMs and that describe the associations between body size and survival, growth, and fertility for a population in the good and in the bad environment. Life-history data on bulb mites that were reared on yeast (good environment) and on filter paper (bad environment) are used to parameterize the functions. Yeast is rich in protein, whereas filter paper contains only cellulose on which the mites feed. These two diets represent extremes in terms of food quality and for this reason are commonly used in life-history studies to assess effects of food quality on growth and development of acarid mites (Gerson et al. 1983; Smallegange 2011*a*, 2011*b*). This first step will show how character-demography associations and IPM outputs vary between different environment states, which will aid in identifying the drivers that influence these associations. Second, using a stochastic demographic model in which the temporal sequence of good and bad environments is driven by a Markovian process that governs the serial correlation of environment states, a perturbation analysis is conducted to investigate how the two types of environmental change affect the relative importance of demographic rates to the long-run stochastic population growth rate, λ_s. This will reveal if different stochastic environments select for fast life histories, which are characterized by early maturation, short life span, and high fecundity, or for slow life histories that have the opposite characteristics (Stearns 1983; Gaillard et al. 1989; Heppell et al. 2000; for examples of life-history speed in stochastic environments, see Lande et al. 2009; Miller et al. 2011). Note that the latter covariation between age at maturity, fecundity, and survival is captured in the life-history variable generation time (Stearns 1992). Because generation time is directly involved in the calculation of elasticities of λ to vital rates (Hamilton 1966), life-history speed can be linked to demographic sensitivity: the shorter the generation time and the faster the speed of life history, the more sensitive populations are to perturbation of fertility rate, whereas a long generation time is indicative of slower populations that are more sensitive to perturbation of survival rate (Lebreton and Clobert 1991). To cover a wide range of stochastic environments in this second step, the serial correlation varies from blue to red noise color (corresponding to negative and positive first-order autocorrelation of the temporal sequence, respectively), and the frequency at which the good environment occurs varies from zero to one. Third, we examine the response of the stochastic population growth rate (λ_s), generation time, lifetime reproductive success, and body size to assess whether changes in these quantities correlate with changes in the environment so that testable predictions on population responses can be derived. Fourth, and finally, we explore which quantities can be used as indicators of the effects of future environmental change on the growth rate (fitness) of populations and their life-history speed.

Methods

Size- and Stage-Structured Integral Projection Model

The life cycle of the bulb mite comprises five stages: egg ($s = 1$), larva ($s = 2$), protonymph ($s = 3$), tritonymph ($s = 4$), and the adult stage ($s = 5$). We construct a size- and stage-structured integral projection model that tracks the body size distribution of females within each of these stages. Juvenile bulb mites also have a facultative dispersal stage (deutonymph), but few individuals develop into this stage when raised on ad lib. yeast or ad lib. filter paper (Smallegange and Coulson 2011). For simplicity the facultative dispersal stage is not included in the model. The IPM will be parameterized for mites living in a good and in a bad environment (see below).

The rationale of the IPM is as follows. If a female survives from day t to $t + 1$, she stays in the same life stage or moves to the next life stage and then grows from size z' to size z. If a female is an adult, she also produces eggs. In the IPM, these events are captured by statistical, character-demography functions that relate body size z at each time t to: (1) the survival probability at time $t + 1$, $S(s, t, z')$; (2) the transition probability that females stay in stage s at time $t + 1$, $P(s|s, t, z')$; (3a) the increase in body size among survivors that stay in stage s at time $t + 1$, $G(z|s, t, z')$; (3b) the increase in body size among survivors that have moved to stage $s + 1$ at time $t + 1$, $G(z|s + 1, t, z')$; (4) the number of eggs produced at time $t + 1$ (assuming a prebreeding census), $R(s, t, z')$; and (5) the size of eggs produced at time $t + 1$, $D(z|s, t, z')$. Functions (3) and (5) are probability density functions that not only describe how body size at time $t + 1$ is related to body size at time t among survivors but also describe how the variance in size at time $t + 1$ is affected by size at time t among survivors. These functions therefore capture how individuals of identical size at time t can develop to different sizes and produce eggs of different sizes at time $t + 1$. Denoting the number of females of stage s at time t by $n(s, t, z')$ means that the dynamics of this number distribution from t to $t + 1$ can be written as

$$n(1, t + 1, z) = \int_{\Omega_s} D(z|s, t, z') \times R(s, t, z')n(s, t, z')dz', \quad (1a)$$

where $s = 5$,

$$n(s + 1, t + 1, z) = \int_{\Omega_s} G(z|s + 1, t, z') \times P(s + 1|s, t, z')S(s, t, z') \times n(s, t, z')dz', \quad (1b)$$

$$n(s, t + 1, z) = \int_{\Omega_s} G(z|s, t, z')P(s|s, t, z') \times S(s, t, z')n(s, t, z')dz',$$

where $1 \le s \le 4$, and

$$n(5, t + 1, z) = \int_{\Omega_s} G(z|s, t, z')P(s|s - 1, t, z') \times S(s - 1, t, z')n(s - 1, t, z')dz' + \int_{\Omega_s} G(z|s, t, z')S(s, t, z') \times n(s, t, z')dz', \quad (1c)$$

where $s = 5$. The closed interval Ω_s denotes the size domain of stage s. Since only adults reproduce, the $R(s, t, z')$ and $D(z|s, t, z')$ functions are zero for $1 \le s \le 4$ (eq. [1b]). The number distribution of adult females at time $t + 1$ (eq. [1c]) is determined by the number of tritonymphs that have moved to the adult stage at time t (first part of eq. [1c]) and by the number of surviving adult females at time $t + 1$ (second part of eq. [1c]).

Equations (1) describe the dynamics of the continuous trait body size z. Predicted values from these equations are calculated by dividing the size domain of each stage (Ω_s) into very small-width discrete bins, defined as "mesh points," to create a discrete approximation of the IPM. For each stage, each transition rate was estimated for the midpoint of two adjacent mesh points. The numerical accuracy of the approximation increases with the number of mesh points (Ellner and Rees 2006), and here, the body size domain of each stage was divided into 50 size bins (because body size domains differed between life stages, bin widths differed between life stages; fig. 1). A higher number of bins did not produce different results. The asymptotic population growth rate λ_0 was estimated as the dominant eigenvalue of the resulting 250 × 250 matrix (there are 50 size bins × 5 stages = 250 mesh points). We furthermore calculated the key life-history variables generation time (T) and lifetime reproductive success (R_0). Lifetime reproductive success was calculated as the dominant eigenvalue of the matrix $\mathbf{F}(\mathbf{I} - \mathbf{T})^{-1}$, where $\mathbf{I}$ is the identity matrix and $\mathbf{F} = \mathbf{DR}$, where $\mathbf{D}$ is a matrix that

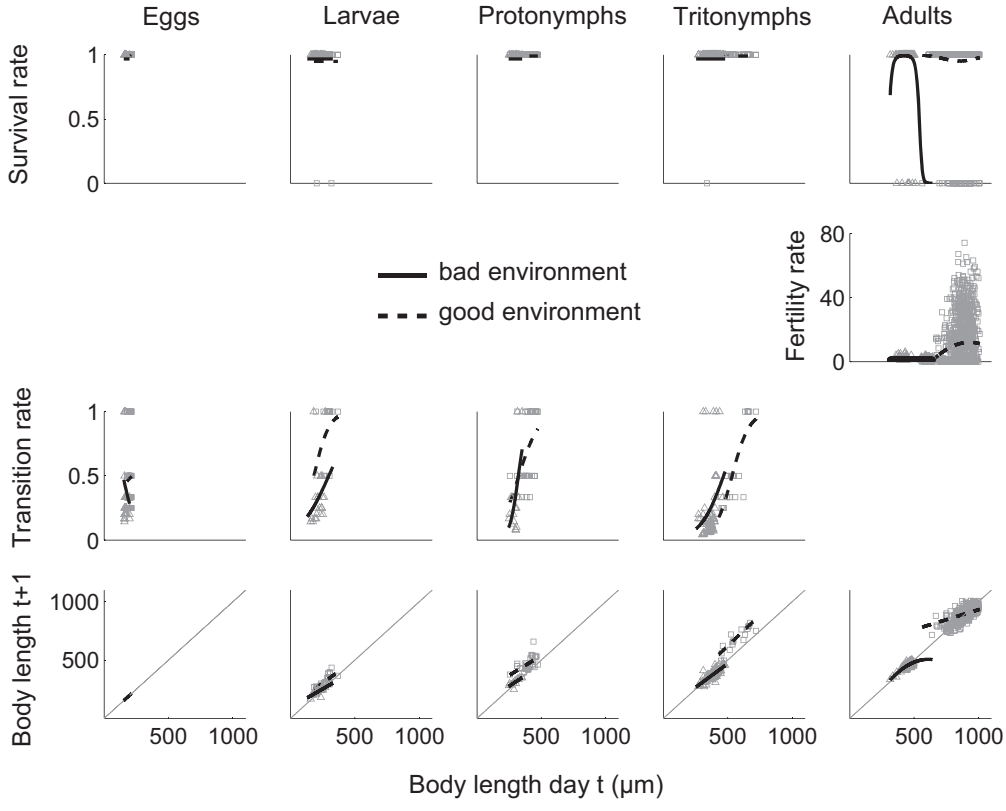

Figure 1: Character-demography functions for each of the five life stages showing the relationships between body length and survival, fertility, transition rate of moving to the next life stage, and mean growth rate when staying in the same life stage, which are used to parameterize the good-environment integral projection model (IPM; dashed lines and squares) and the bad-environment IPM (solid lines and triangles). Statistical functions that are not displayed are those that describe inheritance; in this specific case, egg size was independent of maternal size so that the expected mean egg size and variance at time $t + 1$ are constant (see also appendix) and growth when growing into the next life stage (in which case growth between time t and $t + 1$ is described by the growth rate of stage $s + 1$, as shown in this figure). Lines represent predictions from regressions, and points represent raw data. In the bottom panels the zero-growth line is also plotted. Growth of the largest adults in the bad environment was not measured, which is why the fitted line—which ranges from the minimum to the maximum observed adult body length—in the bottom right corner panel extends beyond the data points. The size of the smallest and largest individual observed within each life stage determined the minimum and maximum size of the size domain of each life stage.

approximates the inheritance kernel and **R** is a matrix that approximates the reproduction kernel. The matrix **T** is given by $\mathbf{T} = \mathbf{GS}$, where **G** is a matrix that approximates the growth kernel and **S** is a matrix that approximates the survival kernel (Caswell 2001, 2009). Generation time was approximated as $T = \log(R_0)/\log(\lambda)$ (Coale 1972; Caswell 2001).

Data Collection

We parameterized the good and bad environment IPMs using life-history data on mites that were reared individually on ad lib. access to yeast (good environment) and ad lib. access to filter paper (bad environment), respectively. Data on the growth and survival of adult females, egg production, and the relationship between size of the mother and her offspring were taken from Smallegange (2011*a*, 2011*b*). Data on juvenile growth and survival were collected here following the methods of Smallegange (2011*a*, 2011*b*) and which is described in the appendix, available online. All data are deposited in the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.df98r (Smallegange et al. 2014).

Parameterizing the Character-Demography Functions

For each IPM, parameters of five character-demography functions were estimated: (1) the survival function; (2) the transition function, which gives the probability of moving from the current life stage to the next; (3) the

growth function; (4) the reproduction function, which gives the number of female offspring produced; and (5) the inheritance function, which gives the relationship between size of the mother and size of her eggs. To construct functions (1)–(5), generalized linear mixed models (GLMMs) were used with body length and body length squared as linear predictors and, except for function (5) where a GLM was used, mite identity as a random factor. The response variables respectively were (1) survival at time $t + 1$ (0 or 1), (2) probability of growing to the next stage at time $t + 1$, $\gamma_{s,t+1}$, (3) mean and variance in body size at time $t + 1$, (4) the number of eggs produced at time $t + 1$, and (5) mean and variance in size of eggs produced at time $t + 1$ by each individual at time t. Eggs do not increase in size so that their size at time $t + 1$ equaled their size at time t.

During data collection it was not always possible to locate each individual every day. For those individuals that were not seen but were alive (e.g., that were seen alive the next day), omitting these observations would result in an underestimation of the survival function. Therefore, we filled in these missing values by estimating the body length of a female of age a using the Gompertz function (fig. A1; figs. A1–A8 available online):

$$z_a = z_\infty e^{-e^{-k(a-a_0)}}, \tag{2}$$

where z_a is body length (μm) at age a (days), z_∞ is the mean maximum length (μm, at $a = \infty$), k is the instantaneous growth rate at age a_0, and a_0 is the inflection point of the curve and the age at which absolute growth rate begins to decline. Mean maximum length of females was calculated using data from Smallegange (2011*a*, 2011*b*). We did not use alternative growth models such as the logistic or von Bertalanffy growth model. The reasons for this is that the von Bertalanffy growth model is not applicable to sigmoidal growth data (unless the cubic version is used, which requires estimating an additional parameter). In the logistic model, in turn, the regions above and below the inflection point are symmetrical, whereas those of the Gompertz function are not. Since our data are not symmetrical around the inflection point, we chose the Gompertz function. It turned out that on the filter paper diet, the survival probability of juveniles of all stages was unaffected by body length (or body length squared) and did not differ between juvenile life stages. Therefore, we lumped survival data of all juveniles and estimated daily survival probability from a survivorship function by estimating the slope of the regression of log-transformed proportion of surviving mites against age (Caswell 2001; fig. A2). For function (2), we used the notion that the probability of growing to the next stage depends on the time spent in the current stage (Caswell 2001) so that $\gamma_{s,t+1}$ is given by $\gamma_{s,t+1} = 1/d_{s,t}$, where $d_{s,t}$ is the number of days left in stage s, or in other words, the number of days left prior to molting into the next stage $s + 1$. This means that d_s equals the total duration of stage s on the first day that a female is in stage s, and that when an individual develops from stage s into stage $s + 1$ at time $t + 1$, $d_s = 1$ so that $\gamma_{s,t+1} = 1$.

For functions (1), (2), and (4), a model simplification procedure was used whereby the full model was fitted, after which the least statistically significant term was removed (starting with body length squared) if the deletion caused an insignificant increase in deviance (significance was assessed by performing a likelihood ratio test). This procedure was repeated until the model only contained significant terms ($P < .05$). The random factor was never removed during model simplification. Parameterizing functions (3) and (5) required two steps: deriving a function describing the expected mean size at time $t + 1$ and a function describing the variance around mean size at time $t + 1$. For step 1, the predictors of mean size at time $t + 1$ were estimated using the model simplification procedure described before. Then, for step 2, the squared residuals of the minimal model of step 1 were fitted against a statistical function of the same form as in step 1 to estimate the variance in size at time $t + 1$. Following Easterling et al. (2000), the growth and inheritance functions (functions [3] and [5]) were then constructed using the following equation:

$$y_s = \frac{1}{\sqrt{2\pi\sigma_s}} e^{-(z'-\mu_s)^2/2\sigma_s^2}, \tag{3}$$

where y_s is either the growth or inheritance function of stage s, μ_s describes the mean effect of the predictors on growth or body size inheritance, and σ_s describes the squared residuals around μ_s. Binomial errors were used to estimate survival and transition probability, and Gaussian errors were used to estimate the other functions. Model assumptions on Gaussian errors and homoscedacity were confirmed by inspection of probability plots and error structures.

Stochastic Demographic Model

The stochastic demographic model was built using a two-state Markov chain that gives the probability distribution of environment states at time t. In this chain, state 1 is the good environment and state 2 is the bad environment, which results in the following Markov chain transition matrix **M** (Caswell 2001, p. 379):

$$\mathbf{M} = \begin{bmatrix} 1-p & q \\ p & 1-q \end{bmatrix}, \tag{4}$$

where p is the probability of switching from the good to

the bad environment and q is the probability of switching from the bad to the good environment. The serial or autocorrelation of the Markov chain equals $\rho = 1 - p - q$ (Caswell 2001, p. 379). High, positive values of ρ denote red noise; high, negative values of ρ denote blue noise; and $\rho = 0$ denotes white noise where the probability of switching states is independent of the current state. The temporal frequency at which the good environment occurs is given by $f = q/(p + q)$ (Caswell 2001, p. 379). In general, the stationary (long-term) mean of the environment is given by $fx_g + (1 - f)x_b$, where x_g and x_b respectively denote the values of the good and bad environments. The stationary variance of the environment is in turn given by $f(1 - f)(x_g - x_b)^2$, which shows that noise color does not affect the long-term variance of the environment. Here, the two environment states are categorical variables that affect the interpretation of the mean and variance of the environment. One could, for example, assign the arbitrary values of 1 and 0 to the good and bad environments, respectively, in which case the mean environment equals f and the variance $f(1 - f)$. By iterating **M**, a time series of length $S = 3{,}000$ (with an initial transient length of 500 discarded, a starting population of one individual in each size bin, and the initial environment state chosen randomly; see also Tuljapurkar et al. 2003) was generated (an example of a stochastic run showing the stationary time series of the stage distribution is shown in the appendix: fig. A3). This sequence determines the environment state that a population experiences at each time step. If the state at time t is the good environment, the matrix approximation of the good-environment IPM is used, and conversely, the matrix approximation of the bad-environment IPM is used if the state at time t is the bad environment. In that way an IPM is generated at each time, which is stored with associated vectors of population structure for further analysis. Note that this procedure assumes that vital rates of one environment apply to mites that developed in the other environment. Note also that although we did not explicitly include population density as an explanatory variable in the character-demography functions, the good environment IPM can be considered density-independent as mites had ad lib. access to high-quality food. The bad-environment IPM on the other hand can be considered density dependent but only to some extent as, in reality, environment and population density do not necessarily fluctuate completely synchronously over time, in which case the stochastic sequences do not completely capture the density effect. This means that different stochastic sequences to a certain extent encompass situations in which the action of density dependence is captured in terms of temporal changes in environment states between time steps.

The long-run stochastic growth rate, λ_s, is calculated over a period of length S by taking the exponent of

$$\log(\lambda_s) = \frac{1}{S}\sum_{S=0}^{S-1} r_t, \tag{5}$$

with $r_t = \log[\sum_s \mathbf{p}_s(t + 1)/\sum_s \mathbf{p}_s(t)]$ and $\mathbf{p}(t)$ is the population vector at time t. Lifetime reproductive success was calculated as the lifetime reproductive success averaged over $n = 50$ cohorts for each stochastic run:

$$R_0 = \frac{1}{n}\sum_{k=1}^{k=n}\sum_x l_k(x)m_k(x), \tag{6}$$

where $l_k(x)$ is the probability of surviving at least to age x in cohort k, and $m_k(x)$ is the average fertility of age class x in cohort k (cf. Tuljapurkar et al. 2009). Each stochastic run was divided into n/S intervals where the first cohort started life at the start of the first interval, the second at the start of the second interval, and so on. Average cohort generation time was calculated as before.

Perturbation Analysis

We first performed a deterministic perturbation analysis to each IPM to examine the elasticity of the population growth rate λ_0 when multiplying each parameter of each character-demography function by 1.001 (this increased positive parameters and decreased negative ones, and it is therefore important to note the sign of each parameter when interpreting the results). We then performed a stochastic elasticity analysis whereby we perturbed each character-demography function in both IPMs simultaneously to identify which functions under which stochastic regimes are most influential to the long-run stochastic population growth rate λ_s. Specifically, of each function of each IPM we consecutively perturbed the intercept, the linear coefficient (if it differed significantly from zero) and the quadratic coefficient (if it differed significantly from zero) by 0.1% and calculated the elasticity of λ_s to each function. The shape of a few character-demography functions differed between the two IPMs (appendix); in those cases only the most significant coefficient of each function was perturbed simultaneously in each IPM. The stochastic elasticity analysis was done such that each perturbation resulted in an increase in λ_s.

Results

Step 1: Model Performance

The first step in this investigation was to compare the fitted character-demography functions of the good- and bad-environment IPMs, which describe the associations between body size and survival, growth, and fertility for the

good and bad environments, respectively. This reveals that the shape and location of each function differed greatest between the two environment states for the adult life stage (fig. 1). Parameter values of each character-demography function are given in the appendix. We then verified that the IPMs were able to reproduce key population-level characteristics of the data. In general, the matrix approximations of the good- and bad-environment IPMs performed well in predicting life-history descriptors of mite populations at equilibrium (table 1). Individuals in the good environment have a high average fitness (λ_0), short generation time, high lifetime reproductive success and large body size, whereas individuals in the bad environment show the opposite characteristics (table 1).

Step 2: Perturbation Analysis

The deterministic perturbation analysis revealed that four character-demography functions are most influential to λ_0: adult survival rate, adult fertility rate, tritonymph growth rate, and adult growth rate (figs. A4, A5). Of each of these functions, perturbation of the slope elicited the greatest change in λ_0 (except in case of fertility rate in the bad environment where the slope did not differ significantly from zero and so an intercept-only model was used of which the intercept was perturbed; fig. A4). We then performed the stochastic elasticity analysis by consecutively perturbing each character-demography function in both IPMs. This revealed that λ_s is always most elastic to either adult survival rate or fertility rate (fig. A6). Since slower life histories are characterized by increased sensitivity to changes in survival and faster life histories by increased sensitivity to changes in fertility ("Introduction"), we can equate the elasticity results of λ_s to the speed of life history and in that way match the different stochastic regimes we investigated to life-history speed (fig. 2). This reveals that slow life histories (where λ_s is most elastic to adult survival rate) are always favored when the autocorrelation ρ is high and negative (blue noise: top right corner of fig. 2). When $\rho = 0$ (white noise: along the antidiagonal in fig. 2), a slow life history is favored if $f < 0.50$, whereas a fast life history (where λ_s is most elastic to adult fertility rate) is favored if $f > 0.50$. When ρ is high and positive, a fast life history is favored when $f > 0.25$ (red noise: bottom left corner in fig. 2). Figure 2 also shows how λ_s and the distribution of adult body length vary across all stochastic environments (inset graphs in fig. 2).

Step 3: Do Different Life-History Variables Correlate with Environmental Change?

The third step of this investigation was to assess which life-history variables show a correlative response to changes in noise color ρ and in good-environment-frequency f. For constant values of f, average fitness, that is, λ_s, increases with increasing ρ (going from blue to red noise), and for constant values of ρ, average fitness increases with increasing f (fig. 3*A*). Both generation time and lifetime reproductive success also increase with increasing ρ for constant values of f, but their increase is negligible (generation time) or minimal (lifetime reproductive success) for negative values of ρ (blue noise) and strong for high, positive values of ρ (red noise; fig. 3*B*, 3*C*). For constant values of ρ, generation time decreases and lifetime reproductive success increases with increasing f (fig. 3*B*, 3*C*). Mean adult body size decreases with increasing ρ for constant values of f, but this decrease is

Table 1: Comparison between population biology quantities predicted by the good and bad environment integral projection models and those estimated directly from data

	Good environment		Bad environment	
Quantity	Predicted	Observed	Predicted	Observed
λ_0	1.41	1.25[a]	1.08	NA
		1.13[b]		
R_0	129.84	146.5[c]	16.47	.00[c]
		187.5[d]		8.95[e]
σ_E	8.30	11.33[f]	16.27	13.76[f]
σ_L	30.69	37.46[f]	29.91	29.40[f]
σ_P	41.37	45.29[f]	39.79	38.62[f]
σ_T	92.70	48.19[f]	35.75	34.43[f]
σ_A	81.87	89.72[f]	49.24	45.12[f]
T	14.18	12.00[g]	36.43	38.28[g]
		15[c]		42[h]
V	−3.50	−3.74[f]	1.17	.13[f]
$\bar{z}_E$	194.52	194.14[f]	179.32	177.21[f]
$\bar{z}_L$	219.29	231.82[f]	212.02	210.37[f]
$\bar{z}_P$	365.30	367.10[f]	275.25	275.46[f]
$\bar{z}_T$	465.63	508.47[f]	317.93	318.34[f]
$\bar{z}_A$	818.42	841.08[d]	427.13	423.42[d]

Note: The quantities are population growth rate (λ_0); mean body length ($\bar{z}$, μm) and standard deviation of body length (σ, μm) of eggs (E), larvae (L), protonymphs (P), tritonymphs (T), and adults (A); and generation time (T, days), strength of viability selection (V, μm), and mean lifetime reproductive success (R_0). The strength of viability selection was calculated as the difference in population-level mean body length after and before survival. Observed values of R_0 are calculated over a length of time that is equal to 1 generation time after maturation. Note that for convenience we refer to the filter paper diet as the bad environment even though on filter paper $\lambda_0 > 1$ and mite populations persist and do not decline. NA = not available.

[a] Unpublished data.
[b] Bulb mites on lily bulbs: Lesna et al. (1996).
[c] Bulb mites on peanuts: Gerson et al. (1983).
[d] Smallegange (2011*a*).
[e] Smallegange and Coulson (2011).
[f] This study.
[g] Smallegange (2011*b*).
[h] Gerson et al. (1983).

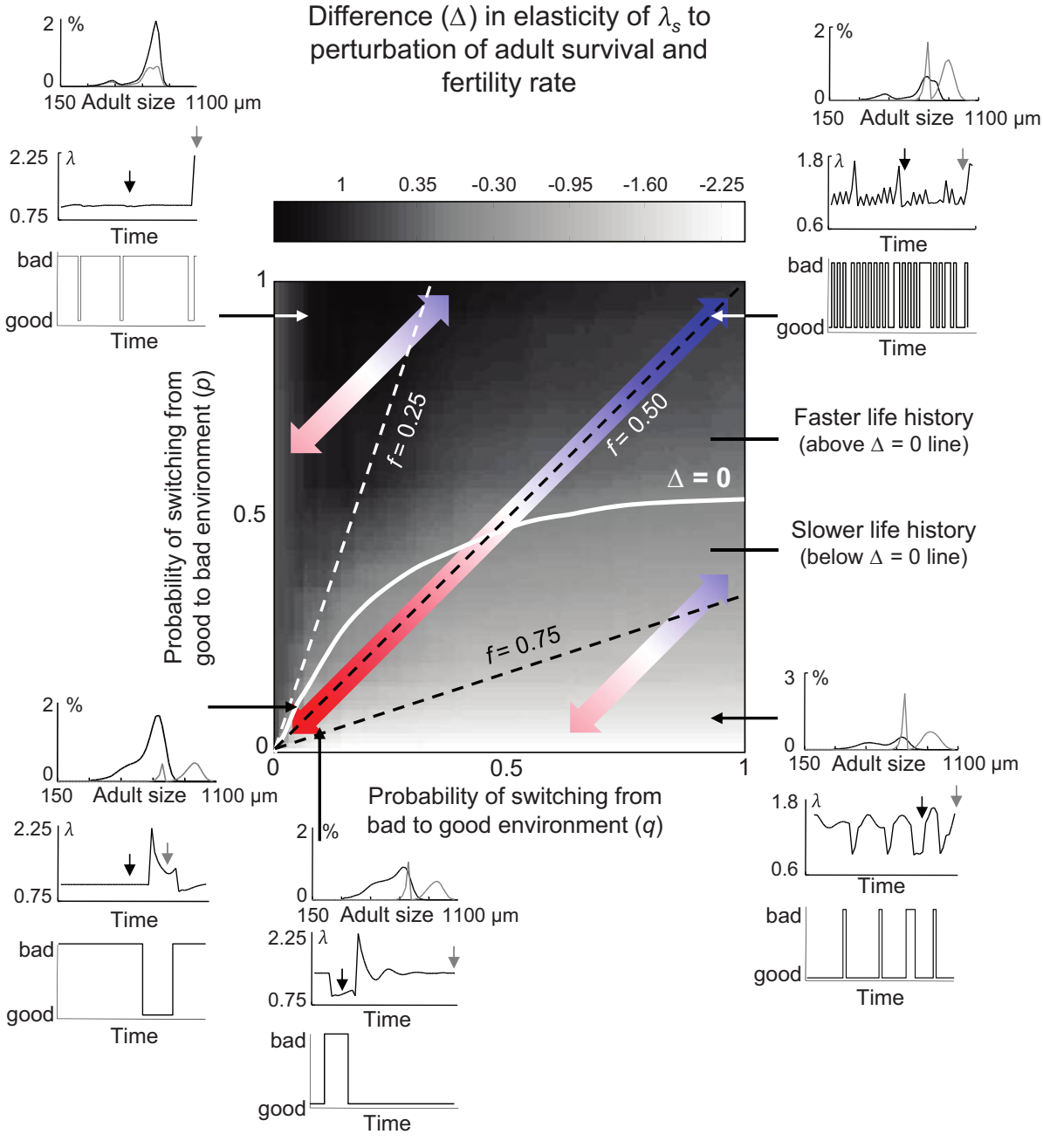

Figure 2: Difference (Δ) in the elasticity of λ_s when perturbing adult survival rate and when perturbing adult fertility rate in relation to the probability of switching from the bad to good environment, q, and the probability of switching from the good to bad environment, p. The area below the $\Delta = 0$ line denotes where λ_s was most sensitive to perturbation of the fertility rate in both the good- and bad-environment IPMs, and the area above the $\Delta = 0$ line denotes where λ_s was most elastic to adult survival rate. The autocorrelation, ρ, in environmental regimes (denoted by the colored arrows) is red in the bottom-left corner, white along the antidiagonal, and blue in the top-right corner. The three dashed lines show values of p and q where the good-environment frequency f equals 0.25, 0.50, and 0.75. Along the latter three dashed lines, the variance of the environment equals 0.22, 0.50, and 0.22, respectively (if good and environment are valued at 1 and 0, respectively). For each of five combinations of noise color and f, a set of three inset graphs give an example stochastic run (bottom graph of each set), the associated change in population growth rate (middle graph of each set), and the size distribution of adults (top graph of each set) at two points along the stochastic run (denoted by the black and gray arrows in the middle graph of each set, which correspond to the black and gray size distribution in the top graph of each set). One stochastic run was performed for each point in the figure.

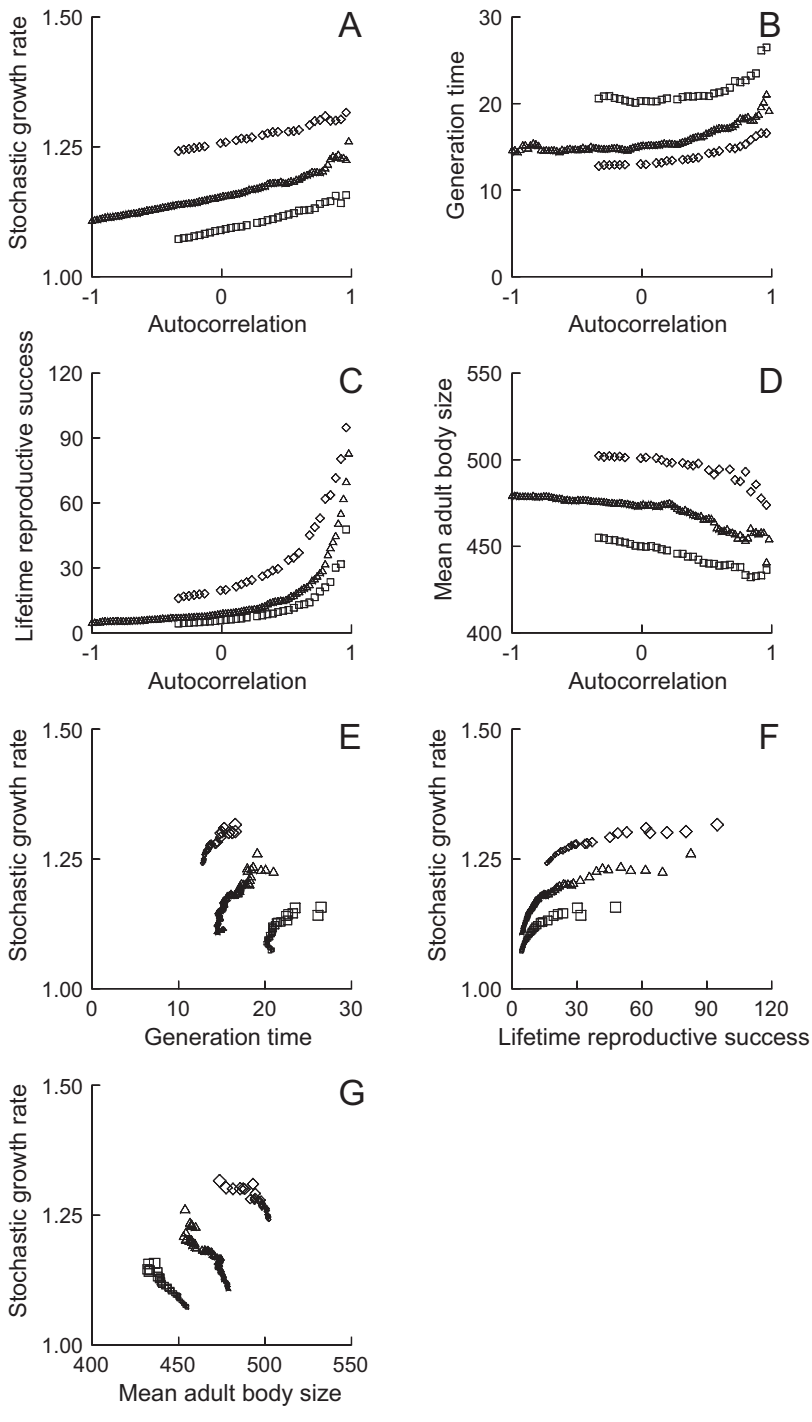

Figure 3: The stochastic growth rate λ_s (*A*), generation time (days; *B*), lifetime reproductive success (number of eggs produced; *C*), and mean adult body size (μm; *D*) as a function of the autocorrelation ρ of environmental regimes. Also shown is the fitness landscape: the stochastic growth rate in relation to generation time (*E*), lifetime reproductive success (*F*), and mean adult body size (*G*). Symbols indicate three values of *f*, the good-environment frequency: 0.25 (squares), 0.50 (triangles), and 0.75 (diamonds). In *E*–*G*, the size of these symbols increases with increasing autocorrelation (ρ) in the environmental regimes: small symbols denote blue noise (negative ρ), intermediately sized symbols denote white noise ($\rho = 0$), and large symbols denote red noise (positive ρ). The range of noise colors is larger for $f = 0.50$ than for other values of *f* (see fig. 2).

minimal when ρ is negative (blue noise) and maximal when ρ is high and positive (red noise; fig. 3*D*). For constant values of ρ, mean adult body size increases with increasing f (fig. 3*D*). These results show that average fitness, generation time, lifetime reproductive success, and body size all show a correlative response to changes in good-environment frequency (fig. 3*A*–3*D*). In response to a change in noise color, however, only average fitness shows a correlative response across the whole color spectrum, whereas generation time, lifetime reproductive success, and body size only show a correlative response if noise shifts occur in red environments (fig. 3*A*–3*D*).

Step 4: Indicators of Fitness and Life-History Speed

For step 4 we explored which quantities correlate with average fitness with changing noise color and good-environment frequency. This revealed that generation time and average fitness are negatively correlated as f increases (keeping ρ constant; fig. 3*E*). As ρ increases (keeping f constant), generation time and average fitness are positively correlated under red noise but uncorrelated under blue noise (fig. 3*E*). Lifetime reproductive success and average fitness correlate positively as f increases (keeping ρ constant; fig. 3*F*). They also correlate positively as ρ increases (keeping f constant), but average fitness values level off at high levels of lifetime reproductive success under red noise, especially at high levels of f (fig. 3*F*). This means that under red noise, for each f, there is a range of values of lifetime reproductive success at which average fitness is at a maximum. Mean adult body size and average fitness correlate positively as f increases (keeping ρ constant) but negatively as ρ increases (keeping f constant), except in the bluest environments when $f = 0.75$ and where body size and average fitness are uncorrelated (fig. 3*G*). To summarize, generation time, lifetime reproductive success, and body size inform on how average fitness changes with changing good-environment frequency, but no single quantity informs on how average fitness responds to changes in the environmental autocorrelation across the whole spectral gradient.

Finally, we explored if average fitness, generation time, or lifetime reproductive success correlate with life-history speed, that is, the slow and fast life-history patterns identified in figure 2 (body size is excluded as the covariation between maturation, longevity, and fecundity along the slow-fast life-history speed continuum is defined for a given body mass; Stearns 1983; Gaillard et al. 1989). To this end, we scaled the stochastic elasticities of λ_s to adult survival rate and fertility rate so that they sum to one. This means that if the scaled elasticity of λ_s to adult survival rate is greater than 0.5, λ_s is most sensitive to changes in adult survival rate and a slow life history exists. Conversely, a fast life history exists if the scaled elasticity of λ_s to adult survival rate is smaller than 0.5 (in which case λ_s is most sensitive to changes in fertility rate). Across all stochastic regimes, i.e. across all values of f and ρ, faster life histories are associated with higher average fitness, shorter generation time, and higher lifetime reproductive success (gray symbols in fig. 4). These results are qualitatively similar to the deterministic outcome (table 1) and to established classifications of fast and slow life histories (Stearns 1983; Gaillard et al. 1989). Of the three quantities, average fitness and lifetime reproductive success correlate best with the speed of life history (fig. 4*A*, 4*C*). Both these quantities show much less variation in response to environmental change than generation time (fig. 4*B*). Because variation in generation time is so high, the range of values that are associated with both fast and slow life histories is large compared to the total range of their observed values (fig. 4*B*). Therefore, generation time can be considered a poor predictor of the speed of life history in stochastic environments. The likely reason that generation time shows such large variation across environments is that this quantity displays opposite correlative responses to increasing ρ and f: generation time increases with increasing ρ but decreases with increasing f (fig. 4*B*; see also fig. 3*E*). Average fitness and lifetime reproductive success, on the other hand, show the same correlated response to increasing ρ and f: both quantities increase with increasing ρ and f (fig. 4*A*, 4*C*; see also fig. 3*F*) and, as a result, vary less across stochastic regimes.

Discussion

Using recently developed structured models and tools from stochastic demography, we explored how different life-history variables vary in response to environmental change. Several insights were gained. First, our graphical analyses revealed that life-history speed can be shaped by both the temporal frequency of favorable environment states and the patterning of environment states through time. In constant environments, life-history variables such as age at maturity and generation time provide a measure of the position of a given population along the slow-to-fast life-history continuum (Charlesworth 1994; Heppell et al. 2000; Oli and Dobson 2003; Gaillard et al. 2005; Stahl and Oli 2006). Our analysis revealed that in stochastic environments, lifetime reproductive success and population growth rate are reliable proxies for life-history speed. Generation time, on the other hand, seems a less reliable proxy as its direction of change in response to environmental change depends on whether the frequency of good environments or the serial correlation in environment states is changing.

Second, we showed that, as the frequency of favorable

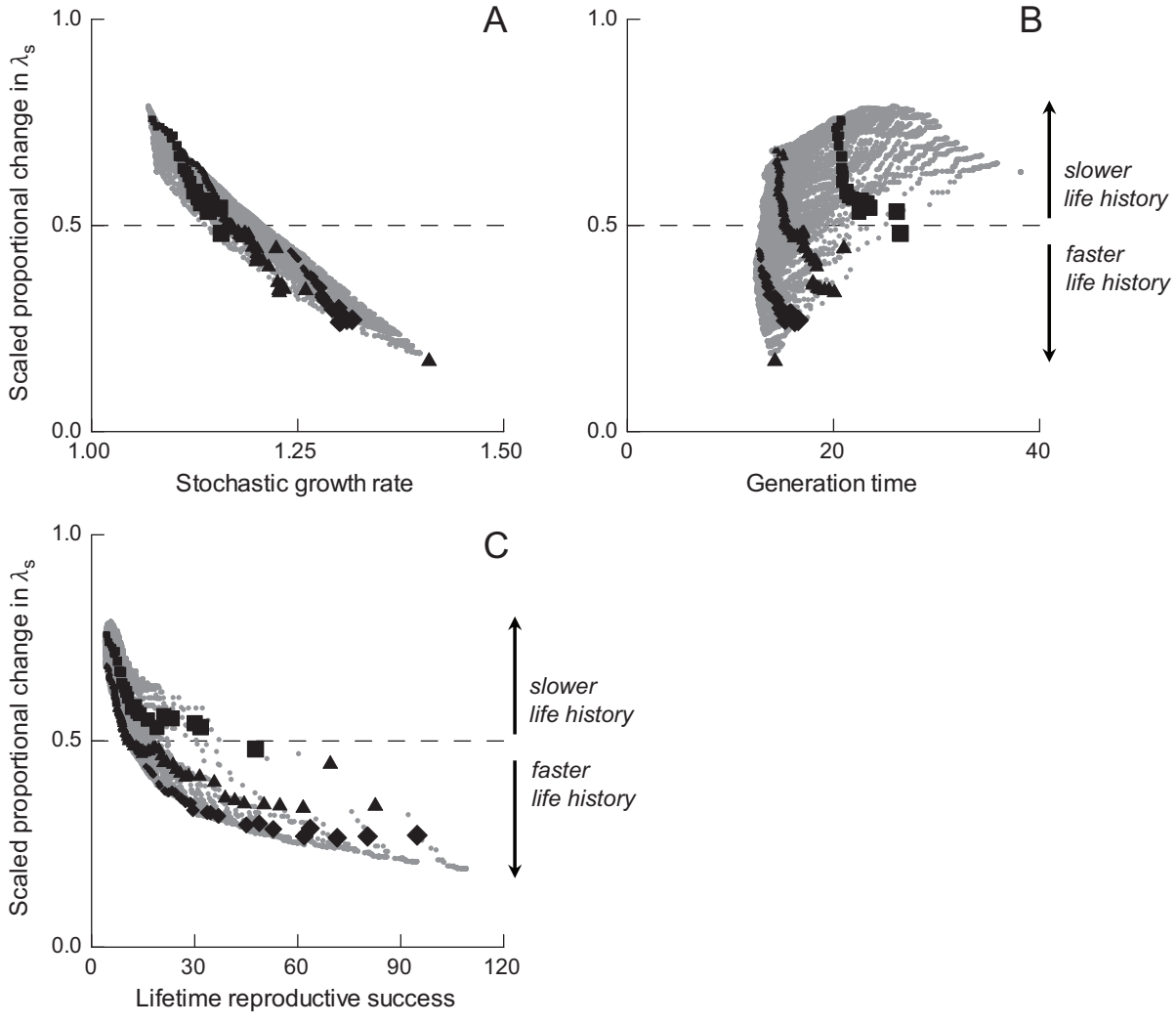

Figure 4: The scaled elasticity λ_s to perturbation of adult survival rate as a function of stochastic growth rate (*A*), generation time (*B*), and lifetime reproductive success (*C*). The black symbols represent three values of *f*, the good-environment frequency: 0.25 (squares), 0.50 (triangles), and 0.75 (diamonds). The size of these symbols increases with increasing autocorrelation (ρ) in the environmental regimes: small symbols denote blue noise, intermediately sized symbols denote white noise, and large symbols denote red noise. In gray are values associated with all environmental regimes explored in this study, that is, all combinations of the probability of switching from the bad to good environment, *q*, and the probability of switching from the good to bad environment, *p* (fig. 2). The horizontal dashed lines denote where the scaled elasticity of λ_s equals 0.5: values above this line correspond to a slow life history, and values below this line correspond to a fast life history. Calculating generation time as $(1/n)\sum_{k=1}^{k=n}\sum xl_k(x)m_k(x)/R_0$ (cf. Tuljapurkar et al. 2009) reveals the same pattern as in *B*.

environment states changes and as noise color shifts between white and red environments, different quantities can change together in a consistent and predictable way: regardless of the type of environmental change, average fitness and lifetime reproductive success always correlate positively, whereas generation time and body size always correlate negatively. However, the relationship between average fitness/lifetime reproductive success and generation time/body size depends on the type of environmental change. Furthermore, with a decrease in environmental quality due to a decrease in good-environment frequency or a shift from red to white environments, average fitness and lifetime reproductive success always decrease, but the response of generation time and body size depends on which type of stochastic change drives the environmental deterioration. We also showed that different quantities—generation time, lifetime reproductive success, and body size—can inform on how average fitness responds to a

change in the temporal frequency of favorable environment states. However, no predictors on how average fitness changes along the full spectral gradient were identified. Recent work indicates that the color of temperature-climate variables has become less red-shifted, at least on a continental scale (García-Carreras and Reuman 2011), and it is therefore of interest to investigate correlates of the change in average fitness for this shift along the color gradient. Climate variability increases as climate variables become less red (this is because the temporal frequency with which environmental conditions change increases), and theory states that increased variability of demographic rates decreases the stochastic population growth rate (Lewontin and Cohen 1969; but see Drake 2005). This is indeed what we found with our graphical analysis.

To what extent the above results can be generalized to other species depends foremost on whether the character-demography functions that form the basis of our analyses are similar to those of other species. A (nonexhaustive) review of published IPMs reveals that the shape of the bulb mite character-demography functions are comparable to those of other species (e.g., Soay sheep *Ovis aries* (Coulson et al. 2010); soil mite *Sancassania berlesei* (Ozgul et al. 2012); Nile crocodile *Crocodylus niloticus* (Wallace et al. 2012), except for the survival rate of adults in the bad environment as this is of an atypical hump shape (fig. 1). We therefore reran our analyses, setting the adult survival rate in the bad environment equal to the juvenile survival rate in the bad environment, which we considered a suitable, biologically realistic alternative. This revealed that noise color had little influence on any of the life-history variables but the life-history response to changes in good-environment frequency was the same as before (fig. A7). Importantly, lifetime reproductive success and population growth rate remain reliable proxies for life-history speed, and because variation in life-history variables is mainly determined by variation in good-environment frequency, generation time now also reliably informs on life-history speed (fig. A8). Thus, altering the shape of adult survival rate in the bad environment has no effect on the life-history response to changes in good-environment frequency or on the reliability of population growth rate and lifetime reproductive success as indicators of life-history speed. These are therefore results that may be generalized to other species with similar vital rates.

There are several ways to characterize environmental change. This can be a change in mean environment, in environmental variance, in the serial correlation of environment states (noise color), or in the frequency of favorable environment states. Here we focused on the latter two characteristics by changing the parameters p and q, which determine the probabilities of switching between environment states. However, changing the frequency of favorable environment states changes both the mean environment and environmental variance at the same time, which means that their effects cannot be separated. An alternative way that could be adopted in future studies to investigate the population response to changes in the mean environment and environmental variance using IPMs, is by altering the parameter estimates of the character-demography functions and their variance. Coulson et al. (2011) applied this approach to the character-demography functions of Yellowstone gray wolves and concluded from the wide range of expected population change that detailed knowledge on how the environment affects growth, survival, and reproduction of individuals of different life stages, genotypes, and so forth is required for an accurate prediction of the response of populations to environmental change. Here, we made a first step toward identifying reliable predictors of life-history change by analyzing demographic data that were obtained under controlled laboratory circumstances. Several predictors of the life-history response of populations to changes in the environment were identified. However, unlike Coulson et al. (2011), we did not explicitly include population density as an explanatory variable in the character-demography functions, and it would therefore be interesting to investigate whether the patterns found in this study hold when density dependence is explicitly included in the population models.

Finally, confirming the reliability of our results requires that our predictors of the life-history response are tested in a population experiment. This experiment can then also be used to test a crucial assumption of this study that environmental noise color tinges the dynamics of populations. Population studies on ciliates, for example, suggest that internal mechanisms redden population dynamics rather than the noise color of external environmental variables (Petchey 2000; Laakso et al. 2003). Yet, a study on flour beetles found that under some dynamical regimes, population power spectra can be tinged blue by external environmental variables that show blue noise (Reuman et al. 2008). This experimental test can also shed light on the links between environmental change and variability in demographic rates (e.g., Coulson et al. 2001; Drake 2005) as environmental variability sometimes does not translate into variability in demographic rates due to buffering (Pfister 1998; Morris and Doak 2004) or environmental canalization (Gaillard and Yoccoz 2003). It should also be pointed out that our approach does not accommodate potentially important effects of environmental change at the individual level, such as bet-hedging strategies (Seger and Brockmann 1987) and any delayed effects of previously experienced environmental conditions on future development. Experiments should also be conducted to test the assumption that vital rates of one environment apply to mites that developed in the other environment holds.

This assumption likely holds if functions are similar and body size distributions of individuals in different environments overlap, which was the case here for most juvenile vital rates. Adult survival and fertility rate, however, differed greatly between environments, and adult body size distributions showed little overlap. In case of adult survival, extrapolating survival rates likely poses few problems. Large adult females from the good environment that suddenly experience bad environmental conditions would be unable to obtain enough resources to meet their maintenance and reproduction costs and therefore would have very low survival rates in the bad environment, which matches the extrapolated survival rate of the bad environment to large body sizes. Vice versa, small adults from the bad environment that move to the good environment would experience an increase in survival rate as now-ample food is available. In case of fertility rate, there likely is a discrepancy between extrapolated functions and actual fertility patterns. In the bad environment, large adults from the good environment will for a short while still have a high production rate of eggs, as these were already produced in the good environment and this would introduce a reproduction lag. Small adults in the good environment, on the other hand, would never be able to reach the high levels of egg production achieved by much larger females. To what extent and under which environmental regimes these extrapolation errors would create a substantial mismatch between our predictors of the life-history response and actual life-history patterns remains to be tested.

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CHAPTER FOUR

Eco-Evolutionary Interactions as a Consequence of Selection on a Secondary Sexual Trait

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Contents

Abstract

Ecological and evolutionary population changes are often interlinked, complicating the understanding of how each is affected by environmental change. Using a male dimorphic mite as a model system, we studied concurrent changes in the expression of a conditional strategy and in the population in response to harvesting over 15 generations. We found evolutionary divergence in the expression of alternative male reproductive morphs—fighters and defenceless scramblers (sneakers)—caused by the selective harvesting of each male morph. Regardless of which morph was targeted, the direction of evolution of male morph expression in response to harvesting was always towards scramblers, which, in case of the harvesting of scramblers, we attributed to strong ecological feedback (reduced cannibalism opportunities for fighters) within the closed populations. Current evolutionary theory, however, predicts that the frequency of a morph always decreases when selected against: to understand phenotypic trait

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evolution fully, evolutionary theory would benefit from including ecological interactions, especially if traits have ecological consequences that in turn feedback to their evolutionary trajectory.

1. INTRODUCTION

Assessing the likely population dynamic consequences of environmental change and their consequent effects on community structure and ecosystem functioning requires in-depth knowledge of the complex interactions between ecological and evolutionary population dynamics (Schoener, 2011). Conceptually, we already have a solid understanding of the structure of this interaction (Carroll et al., 2007; Chevin et al., 2010; Kokko and López-Sepulcre, 2007; Post and Palkovacs, 2009; Smallegange and Coulson, 2013). For example, any process that changes ecological characteristics, such as population density or structure, can also elicit concurrent phenotypic responses, including change in the conditional expression of alternative phenotypes (e.g. Buzatto et al., 2012; Tomkins et al., 2011), which underpins life-history decisions and many dichotomous traits (Gross, 1996; Hazel et al., 2004; Roff, 1996; Smallegange, 2011a,b; West-Eberhard, 1989, 2003). Evolved change in the conditional expression of alternative phenotypes in turn affects population structure and dynamics (Myers, 1984; Piou and Prévost, 2013), creating an eco-evolutionary feedback with population and community-level consequences. However, few studies have disentangled ecological from evolutionary change in either natural or experimental settings. Studies that have separated observed life-history change (mainly change in population growth rate) into both compartments have rarely tested whether phenotypic change came about through environmental or through genetic, and therefore, evolutionary processes (Coulson et al., 2011; Ezard et al., 2009; Hairston et al., 2005; Losos et al., 1997; but see Ozgul et al., 2009). In contrast, experimental studies that explicitly focused on disentangling environmental from genetic drivers of phenotypic change rarely recorded concurrent ecological responses to treatments (Dodd, 1989; Garland and Rose, 2009; Kawecki et al., 2012; Moya et al., 1995; but see Cameron et al., 2013; Reznick et al., 2001; van Doorslaer et al., 2009). This is largely because such experimental evolution studies employ discrete generation methods, in which a sample of individuals of one generation is used to start the next (e.g. Kolss et al., 2009; Santos et al., 1997; Tomkins et al., 2011). This alters the structure of populations at the start of each new generation, interrupting ongoing ecological change, so

the population dynamical response to experimental treatments cannot be tracked. Thus, there is an important caveat in our understanding of the mechanisms and the population consequences of concurrent ecological and evolutionary change and their interaction.

Conditional strategies, whereby phenotype expression depends on a threshold response to an environmental cue (Roff, 1996), allow individuals to respond rapidly to environmental change (Gross, 1991). Because this response depends on system productivity, understanding it is also of economic significance to the commercial exploitation of populations (Myers, 1984). For example, the conditional expression of alternative reproductive phenotypes (ARPs) in males of many salmon species can vary in response to selective harvesting (Gross, 1991), as well as climate change (Piou and Prévost, 2013). ARPs in males are commonplace and usually comprise fighters, which compete over access to females, and 'sneakers' that avoid direct competition (Oliveira et al., 2008). Although we have a thorough understanding of how drivers such as food quality and habitat complexity influence the evolution and expression of ARPs (e.g. Łukasik et al., 2006; Smallegange, 2011a,b; Tomkins et al., 2011), how a change in the environment (e.g. by selective harvesting) affects the interplay between ecological and evolutionary dynamics within ARP populations is still unclear (Johnstone et al., 2013; Piou and Prévost, 2013; Smallegange and Johansson, 2014).

The environmental threshold (ET) model is currently at the forefront to explain the evolution of conditional strategies (Buoro et al., 2012; Tomkins and Hazel, 2007): this model was first conceptualized over 30 years ago (Hazel, 1977; Hazel and West, 1982) and subsequently formalized mathematically over the last couple of decades (Hazel et al., 1990, 2004). It can take into account frequency-dependent selection (but this is not a requirement) and can model both the trajectory and the outcome of selection on any conditional strategy (Hazel et al., 2004). Its results are almost identical to those of an earlier model on the conditional strategy (Lively, 1986), but unlike the latter, the ET model uses quantitative genetic theory (Hazel et al., 1990, 2004). The ET model assumes that the two male morphs have different, crossing fitness functions (i.e. the slopes of fitness plotted against status (e.g. size) for each morph differ) and that male morph expression depends on whether or not an individual reaches a critical threshold during ontogeny. This threshold, in turn, is assumed to be under polygenic control and influenced by a cue such as juvenile body size. If a juvenile reaches the size threshold, it develops into a fighter; otherwise, it develops into a sneaker.

In this study, we use the ET model to derive predictions on the evolutionary response to selection against male morph in the bulb mite *Rhizoglyphus robini*. Adult fighter bulb mites have a greatly thickened third pair of legs with sharp ends that can be used to kill rival males by grabbing them and puncturing their skin. Fighters also use their fighter legs to kill other adults as well as larger juveniles to cannibalize them if other food is scarce (Łukasik, 2010). This third pair of legs is unmodified in defenceless sneakers, which are called scramblers (Radwan et al., 2000). Whether or not a male bulb mite develops into a fighter predominantly depends on whether a male reaches a critical size threshold during its final instar stage (quiescent tritonymph stage): males larger than this quiescent tritonymph size threshold (henceforth, referred to as tritonymph size threshold) are most likely to develop into a fighter and males smaller than this size into a scrambler (Smallegange, 2011a). The development of most male bulb mites follows this conditional expression of male morphology (Buzatto et al., 2012; Smallegange, 2011a,b), but, sometimes, small individuals develop into a fighter and large individuals into a scrambler (Leigh and Smallegange, 2014; Smallegange, 2011a). This can occur because there is large genetic variation in the size threshold (Buzatto et al., 2011), or because some males have lost the ability to express the fighter morph (Buzatto et al., 2012). Male morph expression in *R. robini* is also heritable. Smallegange and Coulson (2011) assumed, based on Radwan (1995), that the male dimorphism in the bulb mite is a genetic polymorphism, but we now know that environmental conditions play a large role in male morph expression in the bulb mite (Smallegange, 2011a). The estimated heritability for scrambler morph is 0.41 and for the fighter morph 0.30 (Smallegange and Coulson, 2011); hence, a conditional strategy, which assumes male morph expression to be a polygenic trait (Tomkins and Hazel, 2007), best matches the underlying genetics of the bulb mite male dimorphism. Unlike in other soil and bulb mites (e.g. *R. echinopus* and *Sancassania berlesei*), male morph expression in *R. robini* is unaffected by signals of population density such as airborne substances (Radwan, 1995) or by male morph frequency (Deere and Smallegange, 2014). Further differences between the two morphs are that scramblers live longer than fighters (Radwan and Bogacz, 2000). The reproductive success of each morph, in turn, depends on a number of variables. In the absence of male–male competition, the reproductive success of fighters is independent of their adult size but the reproductive success of scramblers increases with adult size (Smallegange et al., 2012). When in competition with other males, scrambler mating duration, and thereby the number of offspring produced by its mate, was unaffected by the size of its mate, whereas

fighter mating duration decreased with increasing size of its mate (Smallegange et al., 2012). Which morph has the highest fitness as determined by its survival rate and reproductive success, is, therefore, context dependent.

Predictions from the ET model are derived from functions that relate male morph fitness to a cue such as body size (Tomkins and Hazel, 2007). Selection against a morph lowers its fitness, shifting the intersection point of the fitness functions and hence the mean size threshold from its position prior to selection to one associated with a reduced expression of this morph. According to the ET model, we therefore expect that if fighter fitness changes due to, e.g., selective harvesting, then this should result in evolutionary divergence of the mean final instar size threshold: if fighter (scrambler) expression is suppressed, the mean size threshold should increase (decrease) and fighter frequency should decrease (increase) as fewer (more) males will reach the higher (lower) size threshold and develop into fighters (scramblers) (cf. Tomkins et al., 2011). Results from a previous experiment, where we created male morph selection lines using the aforementioned discrete generation method, are in line with this expectation as we found that selection against fighters reduced fighter expression (i.e. the proportion of males that are fighters) and selection against scramblers reduced scrambler expression (Smallegange and Coulson, 2011). To test our hypothesis within a population setting with overlapping generations and uninterrupted ecological change, we established replicate experimental populations and applied proportional harvesting regimes to fighters and scramblers. The experimental populations were created by collecting mites from source populations and culturing them in enclosed populations with a constant food supply. To record the ecological response of experimental populations, we regularly performed a census of the total size of each population and its structure, i.e., the number of individuals of each life stage, sex and male morph. To score the evolutionary response of populations, we measured the size of final instars (as final instar size is the cue for fighter expression) and the size of adult fighters, scramblers and females (females were included to compare the response in male body size with that of females) throughout the experiment. After approximately nine generations (at the end of the experiment), we performed a common garden environment life-history assay to assess whether phenotypic change in body size and morph expression observed during the experiment was plastic or genetic. Using these data, we reveal complex links between ecological and evolutionary change and an unexpected response to selection in populations of a sexually reproducing organism.

2. METHODS

2.1. Predictions on evolution of fighter expression in response to harvesting

The ET model uses fitness functions of the two male morphs to predict the direction of evolution (Hazel et al., 1990, 2004; Tomkins and Hazel, 2007; Tomkins et al., 2011). Fitness functions of fighters and scramblers in relation to adult body size intersect (fitness was measured as reproductive success) (Smallegange et al., 2012). From this, we predict that selection against fighters (scramblers) through selective harvesting shifts the intersection point and mean tritonymph size threshold below and above which males most likely develop into scramblers and fighters, respectively, reducing the frequency of fighters (scramblers) in the male population (Appendix). We did not incorporate frequency-dependence into the ET model as this is non-significant in bulb mites (Deere and Smallegange, 2014; Radwan and Klimas, 2001).

2.2. Source population and bulb mite life cycle

Mites were collected from storage rooms of flower bulbs in North Holland (the Netherlands) in December 2010 and kept on yeast as described in Smallegange (2011a). The life cycle of the bulb mite consists of six stages: egg, larva, protonymph, deutonymph, tritonymph and adult. Mites moult to grow from one life stage to the next, and during this quiescent stage, they are immobile and do not feed. The deutonymph is a facultative dispersal stage to escape unfavourable environmental conditions and its development is induced by low food quality and quantity (Díaz et al., 2000). Few were observed in this study (on average three on each census day, which is less than 1% of the total population) and were therefore not included in the analyses.

2.3. Experimental procedure

The experiment comprised three treatments: (i) harvesting of fighters (FH), (ii) harvesting of scramblers (SH) and (iii) no harvesting (control treatment) (C) and was conducted from July 2011 to July 2012 (365 days). Each treatment was replicated three times, resulting in nine populations. Each population was initiated with 50 randomly selected adult mites from the source population. To reduce founder effects, we removed these founder mites after 15 days, by which time the next generation had matured (founder mites

were much larger than mites from subsequent generations and could therefore easily be distinguished). Each population was fed eight rods of yeast per day (rods range in size from 750 to 1000 μm and eight rods per day amounts to ~0.50 mg yeast per day). After initialization, for the first 230 days, on each Tuesday and Friday, mites of all life stages in each population were counted using a hand counter at 15 × magnification, and photos were taken at 15 × magnification of up to 10 individual adult females, fighters, scramblers and preimaginal (final instar) quiescent tritonymphs using a Lumenera Infinity 3.1 camera connected to a Meiji EMZ-8TRD (10–45 ×) stereomicroscope. The body length of mites (with mouthparts) on each photo was measured to the nearest 0.1 μm using Infinity Analyze Imaging Software (Lumenera Corporation, Ottawa, Ontario, Canada). For the next 135 days of the experiment, mites were counted every week on Tuesday and in addition their sizes were measured every other Tuesday. Under a constant feeding regime, lab-conditioned acarid mite populations' stabilize at about 40 days after initialization (Cameron and Benton, 2004). We started our harvesting treatment 60 days after populations were initialized. Harvesting was carried out once per week (on Tuesday), after counting and before feeding, by removing 50% of fighters (FH treatment) or 50% of scramblers (SH treatment). Generation time of mites varies with food supply, but under good conditions the minimum egg-to-egg time is 11 days (Smallegange, 2011b). Assuming a generation time of 35 days under strong, density-dependent conditions (Cameron et al., 2013), this means that harvesting lasted for $305/35 \approx 9$ generations, which is sufficiently long for evolutionary shifts in fighter expression to occur (Smallegange and Coulson, 2011). Populations were kept in 20 mm diameter, flat-bottomed glass tubes with a plaster of Paris and powdered charcoal base, which was kept moist to avoid desiccation of the mites. Tubes were sealed by a circle of very fine mesh (allowing gaseous diffusion), which was held in place by the tubes' standard plastic caps with ventilation holes cut into them. Populations were kept in an unlit incubator at 25 °C and >70% relative humidity.

After completion of the experiment, we performed a common garden environment life-history assay to assess whether differences in male morph expression between treatments were due to evolutionary shifts in the mean tritonymph size threshold, and whether differences in body size between treatments were plastic or genetic. Ten adult females were taken from each population, individually isolated, given *ad lib* access to yeast, and allowed to lay eggs. From these ten females, three were selected (several females died or did not lay eggs) and their offspring followed until they reached the

quiescent tritonymph stage. No adults, except the mother, were ever present among the offspring. Once quiescent tritonymphs were present, they were collected, their size measured as before, after which we individually isolated each quiescent tritonymph and scored its sex, morph and adult size 2–5 days after maturation. Up to 20 individuals from each female were measured this way, resulting in a total of 418 observations. Nutrition during ontogeny is the strongest environmental determinant of male morph development. Paternal morph only has an effect on offspring male morph expression under poor food quality conditions (here, mites were kept under rich food conditions) and effects of maternal nutritional conditions are negligible in this species (the effect size of offspring environment is 15 times larger than that of maternal environment; Smallegange, 2011a). Therefore, rearing mites in a common garden environment for one generation is sufficient to eliminate any maternal effects. We also isolated 100 quiescent tritonymphs from the source population to assess their mean tritonymph size threshold above and below which males are most likely to develop into scramblers and fighter, respectively. Females and their offspring, and all isolated quiescent tritonymphs were given *ad lib* access to yeast and kept in 25 mm diameter (females and offspring) and 10 mm diameter tubes (quiescent tritonymphs) with a plaster of Paris and powdered charcoal base, which were kept in an unlit incubator at 25 °C and >70% relative humidity.

2.4. Statistical analyses

To analyse the effects of harvesting on the number and size of individuals, we used data from day 100 onwards, i.e., 40 days after the start of the harvesting regimes, to reduce the impact of transients (cf. Cameron and Benton, 2004) (see Appendix for an overview of the observed population dynamics). This resulted in 783 observation days on life stage numbers and 466 observation days on life stage body sizes. We used bootstrap resampling to estimate 95% confidence intervals (CIs) for the mean number and the mean size of individuals of relevant life stages per treatment group (cf. Benton et al., 2004). We furthermore divided the experimental period into three periods, day 100–190, day 191–280 and day 281–365, and calculated 95% CIs per period to assess long-term (instead of transient) temporal changes in life stage number and mean body size over time within each treatment group. Bootstrap resampling was done by taking 1000 resamples, which were stratified by population tube within each treatment group to ensure that there were no biases due to tube effects. These samples were used to estimate the

bias-corrected and adjusted (BCa) 95% CI. If a statistic's 95% CI does not overlap between treatment groups or between time periods within a treatment group then, by definition, the statistic differs across groups at $\alpha = 0.05$.

To analyse the results from the life-history assay, we used a generalized linear mixed model (GLMM) with Gaussian errors to analyse the effects of harvesting treatment, morph (including fighters, scramblers and females) and their interaction on quiescent tritonymph size and adult size (μm). We also used a GLMM with binomial errors to analyse the effects of harvesting treatment and quiescent tritonymph size on male morph expression (0 if scrambler; 1 if fighter) (cf. Tomkins et al., 2011) and used a GLMM with binomial errors to analyse the effects of harvesting treatment on the probability of female expression (0 if male; 1 if female). In each GLMM, maternal identity and population tube were included as random terms. To assess significance of treatment effects on quiescent tritonymph size and adult size, a model simplification procedure was used whereby the full model was fitted after which the least significant term was removed (starting with the highest order interaction) if the deletion caused an insignificant increase in deviance. Model simplification using likelihood ratio tests is not recommended for GLMMs (Bolker et al., 2009) and instead we used a Markov Chain Monte Carlo (MCMC) approach and calculated 95% CIs of the model parameters using the underlying Gaussian distribution of the model residuals. First, an MCMC sample was generated from the posterior distribution of each parameter estimate using the function *mcmcsamp* in the R package *lme4* (Bates and Sarkar, 2007). The Bayesian highest posterior density (HPD) 95% CIs of the MCMC sample for each parameter estimate was then computed using the function *HPD interval* in the R package *coda* (Plummer et al., 2006). If the HPD interval of a parameter estimate overlaps with zero, then the associated factor has no significant effect on the response variable. If the full model contained non-significant parameter estimates, the parameter that was the least significant was removed and new HPD intervals calculated. This process was repeated until the model only contained significant terms. In Section 3, we report the parameter estimates ($\hat{e}$) and associated statistics of (non)significant terms. Model assumptions on Gaussian errors and homoscedacity were confirmed by inspection of probability plots and error structures. Models were fitted by maximum likelihood in R.

3. RESULTS

We first present the results of the life-history assay conducted at the end of the experiment to show that evolutionary change in fighter

expression had occurred, followed by the ecological changes in population number and structure associated with changes in fighter expression.

3.1. Evolution of fighter expression

The life-history assay conducted at the end of the experiment revealed that the FH resulted in evolutionary change in fighter expression. Fighter expression in the assay differed significantly between treatments ($\hat{e} = -2.34 \pm 1.13$SE, $z = -2.081$, $p = 0.037$) and the probability of fighter expression was significantly lower in the FH lines than in the control lines (Fig. 4.1A). The probability of fighter expression in the FH and control lines of the life-history assay was not significantly different from the proportion of fighters observed during the final period of the population experiment (day 280–365) in the FH and C treatments (Fig. 4.2A): this result is inferred from the fact that, for the FH and C treatments, the standard error bars of mean probability of fighter expression observed in the life-history assay (diamonds in Fig. 4.2A [which are the same as in Fig. 4.1A]) overlap with the CIs of mean probability of fighter expression observed for the final period in the population experiment (third triangle of each treatment in Fig. 4.2A). Quiescent tritonymph size significantly affected the probability of fighter

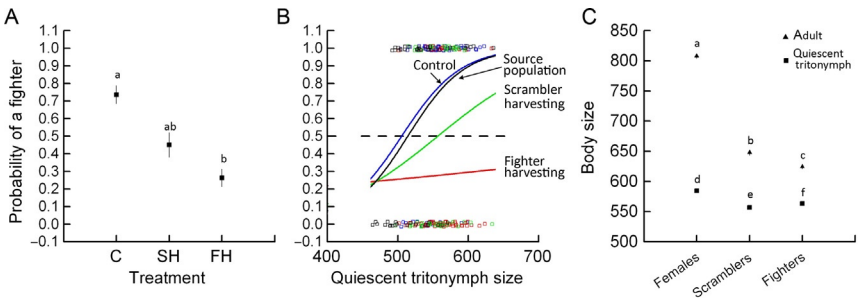

Figure 4.1 Life-history assay. (A) The probability of fighter expression differed between different treatment groups (C, control; SH, scrambler harvesting; FH, fighter harvesting). (B) The mean tritonymph size threshold (the quiescent tritonymph size at which the expected probability of fighter expression is 0.5: denoted by the horizontal dashed line) was shifted to the right for the SH treatment compared to the control treatment and for the FH treatment the predicted mean size threshold is outside of the observed range of quiescent tritonymph sizes. For comparison, the fighter expression-body size response curve of the source population is also included. (C) The body size (μm) of quiescent tritonymphs and adults differed significantly between females, scramblers and fighters. Vertical lines in (A) and (C) are standard error bars that in (C) are covered by the symbols. Letters in panels (A) and (C) denote significant differences between treatments at $\alpha < 0.05$.

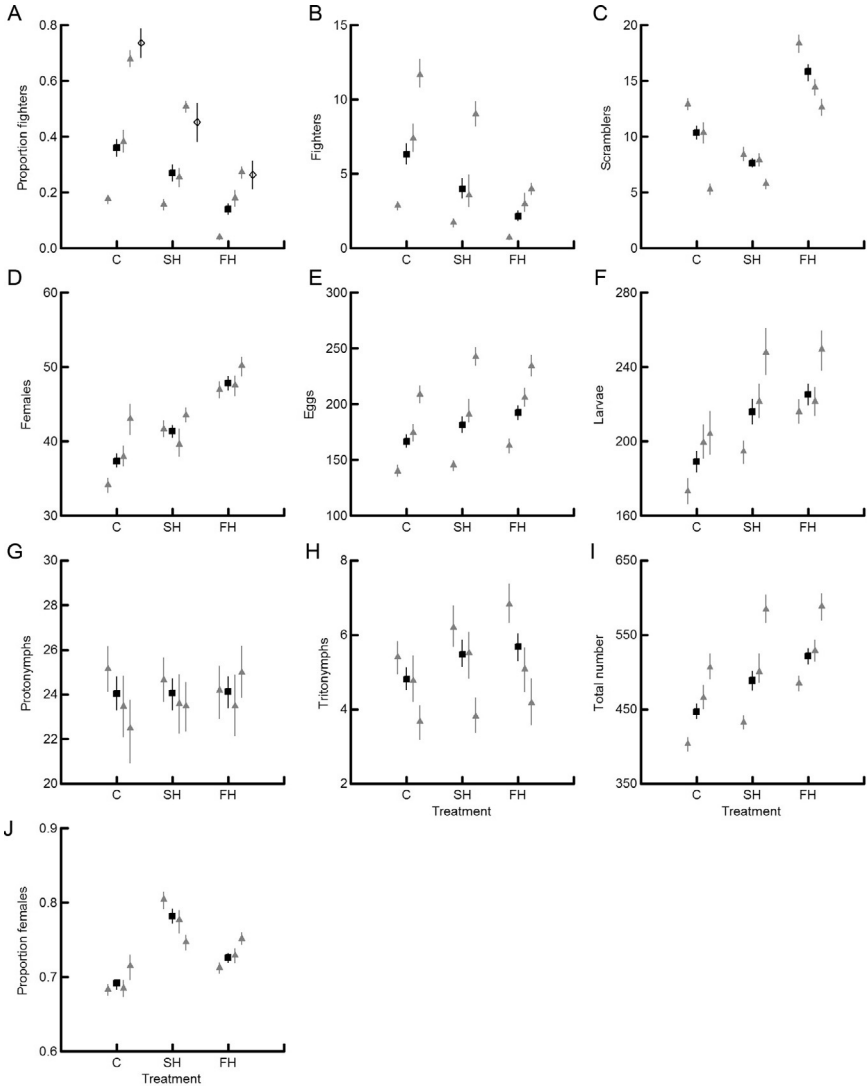

Figure 4.2 The proportion of males that are fighters (A), number of fighters (B), number of scramblers (C), number of adult females (D), number of eggs (E), number of larvae (F), number of protonymphs (G), number of tritonymphs (H), the total number of individuals in the population (I) and the proportion of the adult population that are females (J) shown per treatment group: C, control; SH, scrambler harvesting; FH, fighter harvesting. The black squares are the observed means across replicate populations from day 100 till day 365. Per treatment, the three-grey triangles denote from left to right the mean across replicate populations for day 100–190, day 190–280 and day 280–365, respectively. The probability of fighter expression values observed in the life-history assay (Fig. 4.1A) are added to panel (A) (open diamonds) to aid the visual comparison between these values and those observed at the end of the population experiment (grey triangle directly left of each diamond). Vertical lines are 95% CIs estimated by bootstrap sampling, stratified by tube, except in (A) where vertical lines associated with the open diamonds are standard error bars.

expression in the life-history assay (HPD interval: −36.61 to −23.73 μm). As expected from the selection against fighters through harvesting fighters in the FH treatment, the mean tritonymph size threshold at which the probability of fighter expression equals 0.5 was much higher for mites from the FH lines than for mites from the control lines (Fig. 4.1B: note that the predicted mean tritonymph size threshold for the FH treatment is outside of the observed quiescent tritonymph size range). An important assumption in the conditional expression of size-dependent ARPs is that, if the body size distribution changes location (e.g. mean body size becomes smaller), then the mean tritonymph size threshold for morph expression evolves such that its new location is at the same (relative) position within the body size distribution as before the change in mean body size (Tomkins et al., 2011). This means that, if there is no effect of harvesting but body size distributions differ between harvesting treatments in the life-history assay for some other, unknown reason, then the mean tritonymph size threshold will also differ between treatments as it will have tracked the change in body size distribution (all else being equal). This would then lead one to conclude incorrectly that harvesting has led to evolutionary shifts in mean tritonymph size threshold. Here, however, body size distributions (i.e. mean adult body size and mean quiescent tritonymph size) did not differ between the FH, SH and control lines in the life-history assay (HPD interval: −14.38 to 17.64 μm), so we can conclude that the differences in fighter expression and mean tritonymph size threshold observed in the life-history assay were indeed due to the different harvesting regimes. Quiescent tritonymph size furthermore differed between females and the male morphs (HPD interval: −36.61 to −23.73 μm): female quiescent tritonymphs were significantly larger than fighter quiescent tritonymphs, which were in turn significantly larger than scrambler quiescent tritonymphs (Fig. 4.1C).

Fighter expression in response to harvesting scramblers was not as we expected. The life-history assay revealed that there was evolutionary change in fighter expression in the SH treatment, but the probability of fighter expression in the SH lines was lower, instead of higher, than in the control lines (Figs. 4.1A and 4.2A). This evolutionary change was reflected in the mean tritonymph size threshold of the SH lines, which was at a larger quiescent tritonymph size than in the control lines (Fig. 4.1B). Like in the FH and control lines, fighter expression in the SH lines of the life-history assay matched the observed proportion of fighters in the SH treatment during the final period of the population experiment (standard error bars in Fig. 4.1A and CIs for the final period in Fig. 4.2A (third triangle) overlap for the SH treatment).

3.2. Effects of fighter expression on population size and structure

Overall, as a result of harvesting fighters, their mean number was significantly lower in the FH treatment than in the control treatment (squares in Fig. 4.2B: significance was inferred from the fact that the 95% CIs (vertical lines around each mean) are non-overlapping), whereas the mean number of scramblers showed the opposite relationship (squares in Fig. 4.2C). Within each treatment, the number of fighters increased significantly over the course of the population experiment and, at the same time, the number of scramblers decreased significantly over time within each treatment (triangles in Fig. 4.2B and C). Similarly, when the mean number of fighters was high for a particular treatment or time period (e.g. control treatment), the mean number of tritonymphs in that same treatment or time period was always low (e.g. in the control treatment) and vice versa. From this, we infer that there is a negative association between mean number of fighters and tritonymphs (squares and triangles in Fig. 4.2B and H).

Across treatments, the mean number of fighters was also negatively associated with the mean number of females, as, when the mean number of fighters was high for a particular treatment or time period, the mean number of females in that same treatment or time period was always low and vice versa (Fig. 4.2B and D). When more females were present, significantly more eggs were laid (Fig. 4.2E) and more larvae emerged (Fig. 4.2F). These knock-on effects of female numbers on eggs and larvae were no longer evident in the protonymph stage (Fig. 4.2G). In fact, protonymph numbers did not differ significantly across treatments and only showed a slight significant decrease over time in the control treatment (Fig. 4.2G). Within treatments, there was a positive association between the mean number of fighters and females as both increased in number over time (Fig. 4.2B and D). As the number of females increased significantly over time within treatments, so did the number of eggs and larvae (Fig. 4.2D–F). Finally, across treatments, the mean number of fighters showed a negative associated with the mean total number of individuals in the population (Fig. 4.2B and I). Within treatments, however, the association between mean fighter number and mean total number of individuals was positive and all populations increased in size over time (Fig. 4.2I).

3.3. Realized sex ratio and plasticity in body size

Harvesting had a significant effect on the sex ratio within populations: across treatments, the proportion of females was highest in the SH treatment,

followed by the FH and control treatment (squares in Fig. 4.2J). Within treatments, the proportion of females varied slightly: towards the end of the experiment the proportion of females increased significantly in the control treatment (triangles in Fig. 4.2J). In the SH treatment, the proportion of females decreased significantly over time, whereas in the FH treatment the proportion of females increased significantly over time (triangles in Fig. 4.2J). The life-history assay revealed that these differences in proportion of females were not due to evolutionary change as in the life-history assay there was no significant difference in the probability of female expression between the treatments ($\hat{e} = -0.088 \pm 0.264$SE, $z = -0.310$, $p = 0.757$). The probability of female expression was on average 0.52 ± 0.24 SE: lower than in the population experiment (Fig. 4.2J) and not significantly different from 0.50, as expected in a diploid species (Oliver, 1971, 1977).

Finally, harvesting significantly affected the mean body size of adults. Fighters, scramblers and adult females were on average significantly larger in the control treatment than in both harvesting treatments (squares in Fig. 4.3A–C). The life-history assay revealed that this variation was completely due to phenotypic plasticity as there was no significant difference in mean adult body size across the SH, FH and control lines (HPD interval: −36.57 to 15.46 μm). The life-history assay also showed that adult females, scramblers and fighters differed significantly in body size (HPD interval: −196.12 to 171.94 μm): females were larger than scramblers, which were larger than fighters (Fig. 4.1C). In the population experiment, females were on average also larger than the males (Fig. 4.3A–C), but scramblers were not significantly larger than fighters either across or within treatments (Fig. 4.3A and B). Within treatments, there was some variation in body size for each of the male morphs: fighter mean body size significantly increased towards the end of the experiment in the SH treatment (triangles in Fig. 4.3A), and scramblers significantly increased in size towards the end of the experiment in the FH treatment (triangles in Fig. 4.3B). Females, however, significantly increased in average size over time within each treatment (triangles in Fig. 4.3C). Quiescent tritonymphs, like the adults, were on average significantly larger in the control than in the harvesting treatments (squares in Fig. 4.3D). Within treatments, the average size of quiescent tritonymphs increased significantly over time in the harvesting treatments but not in the control (Fig. 4.3D). It is interesting to note here that, despite the fact that there was a difference in quiescent tritonymph size between mites in the life-history assay and mites in the experimental populations (all means in Fig. 4.3D are lower than means shown in Fig. 4.1C), the mean tritonymph size threshold (above and below which males are most likely to,

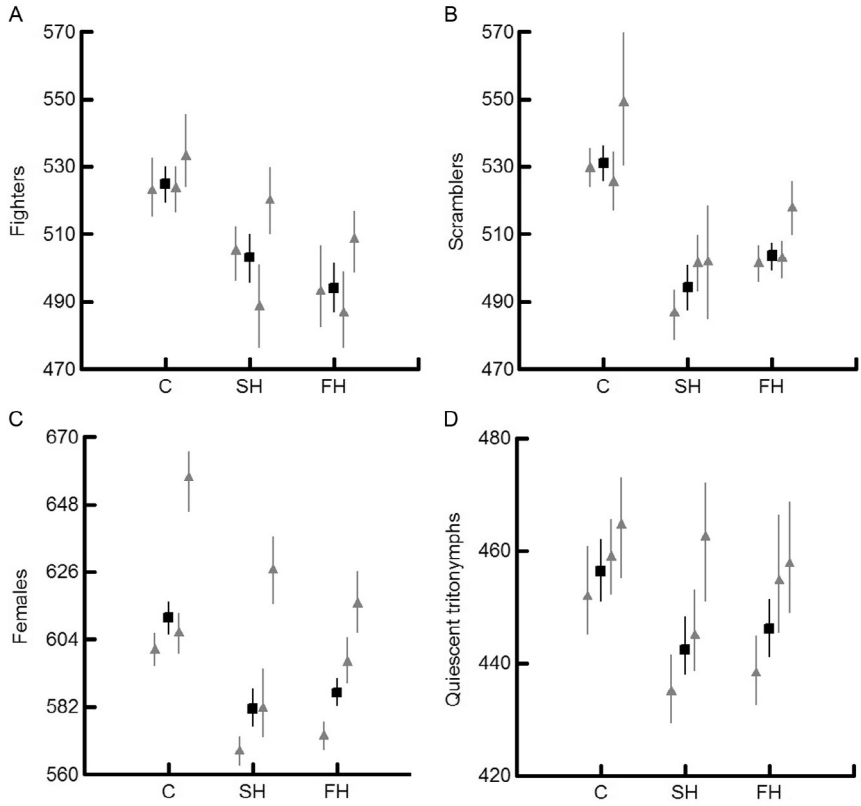

Figure 4.3 The mean size (μm) of individuals in each life stage shown per treatment group: C, control; SH, scrambler harvesting; FH, fighter harvesting. The black squares are the observed mean size across replicate populations from day 100 till day 365. Per treatment, the three-grey triangles denote from left to right the mean size across replicate populations for day 100–190, day 190–280 and day 280–365, respectively. Vertical lines are 95% CIs estimated by bootstrap sampling, stratified by tube.

respectively, develop into scramblers and fighter) of the control populations was nearly identical to that of mites in the source population (Fig. 4.1B). This suggests that mean size thresholds track plastic changes in body size.

4. DISCUSSION

To assume that selection against the expression of a particular phenotype reduces the prevalence of that phenotype in a population seems intuitive and in line with standard evolutionary theory (Falconer and MacKay, 1996). However, this effect may be very different in a setting where selection can be modified by population dynamical properties including density and

structure (Kokko and López-Sepulcre, 2007). Here, we used state-of-the-art theory on the evolution of conditional strategies, the ET model, to predict the evolutionary response of male morph expression to selective harvesting. Regardless of which male morph was targeted, the mean tritonymph size threshold above and below which they are most likely to develop into scramblers and fighters, respectively, increased and fighter frequency decreased in response to the FH and scramblers. Theory predicts this will occur if fighter fitness is reduced, but not when scrambler fitness is reduced. In a previous selection experiment, where we created male morph selection lines using the discrete generation method, we *were* able to increase fighter frequency by selecting against scramblers by killing them (Smallegange and Coulson, 2011). Because, here, unlike in the previous study, we selected against male morph in closed populations, we postulate that ecological feedback from within the population plays a role in this seemingly counterintuitive response in fighter expression to scrambler harvesting. We surmise that this feedback comprises cannibalism by fighters when food is limited (Łukasik, 2010) (but has not been observed in other contexts, e.g., when competing for mates (Deere and Smallegange, 2014; Radwan and Klimas, 2001; Smallegange et al., 2012)). Although frequency-dependence could also explain this response, its strength is non-significant in this species (Deere and Smallegange, 2014; Radwan and Klimas, 2001) and a density-dependent response is more likely. We often observed fighters killing and eating scramblers (and also tritonymphs but not smaller juveniles or adult females) and fighter and scrambler numbers always showed a negative association. We can indirectly test for the occurrence of cannibalism by inspecting the sex ratio in the control treatment where no selection through harvesting occurred: if there is no cannibalism then we expect the sex ratio to be unity, but if cannibalism occurs then we expect it to be female biased, because the consumption of scramblers by fighters will reduce the frequency of males. The latter is indeed what we found, from which we infer that the reduction in scrambler numbers (i.e. density) due to harvesting reduces food availability for fighters. We therefore propose that (at least) two selective forces determine the evolution of fighter expression in these populations: harvesting scramblers selects for fighter expression (cf. Smallegange and Coulson, 2011), but reduced food availability selects against fighter expression, resulting in an eco-evolutionary response that is intermediate between that observed in the fighter-harvested and in the control populations. Fighter fitness is determined by the ability not only to survive but also to reproduce. Given that the sex ratio in all experimental populations was female biased, competition for access to females may have been low so that

fighter fitness was more affected by perturbations to their survival than to their ability to gain access to females. One could furthermore argue that the removal of fighters increases opportunities for cannibalism as there are fewer competitor cannibalists. However, any positive selection for fighter expression through the removal of competitor cannibals is likely to be small as the number of fighters in the populations was always lower than the number of scramblers. The negative effect of the direct selection against fighters through harvesting, as well as the sustained removal of their food (scramblers); therefore, likely outweighs any positive effects of the indirect reduction in the strength of competition over food by the removal of a few fighters. Overall, we therefore conclude that our results confirm Kokko and López-Sepulcre's (2007) suspicion that the effect of a specific factor, such as high extrinsic mortality, may be very different in isolation than in a real-world setting where feedback loops between ecology and evolution are at play. Standard theories on the evolution of phenotypes, including the conditional strategy, can therefore not ignore eco-evolutionary interactions whenever phenotypic traits have ecological consequences and ecology affects the evolutionary trajectory of these traits.

We also observed that, across all treatments, fighter frequency gradually increased over time as the experiment progressed. Initially, fighter frequency was much lower than in the source population. This is likely a plastic response to the reduction in food availability (cf. Smallegange, 2011a) but could also have been partly genetic as a sufficiently high number of generations (approximately five since the start of the experiment) had passed for an evolutionary shift in fighter expression to occur in response to environmental change (Smallegange and Coulson, 2011: five generations; Tomkins et al., 2011: 10 generations). We suspect that the slow, temporal increase in fighter frequency occurred in response to natural selection to the experimental conditions (e.g. Cameron et al., 2013). This response was suppressed in the harvesting treatments compared to the control. Even though density dependence was high in our experiment (larval survival particularly was very low), which can have a suppressive effect on fighter expression because the development of fighter legs is costly (Radwan et al., 2002), the gradual increase in fighter frequency over time suggests that our laboratory circumstances favour a high proportion of fighter males. It is interesting to note, though, that fighter frequencies did not reach the frequency observed in the source population until many generations had passed. Had we terminated the experiment earlier, for example after a few generations, our interpretation would have been that the new, density-dependent conditions favour a reduction rather than an increase in fighter frequency.

By selectively harvesting fighters and scramblers, we removed some of the largest individuals from the populations. Such size-selective harvesting is common in both marine and terrestrial habitats and can lead to plastic as well as genetically based declines in age and size at maturity (e.g. Barot et al., 2004; Grift et al., 2003; Morita and Fukuwaka, 2006; Olsen et al., 2004; Sharpe and Hendry, 2009). If, in our study, harvesting resulted in a decrease in size at maturity then this could explain why males were on average smaller in the harvested than in the control populations. Interestingly, female body size varied in the same manner. Because there is a positive correlation between female body size and fecundity, population biomass and yield from harvested populations decrease if reduced average body size is not compensated for by an increase in population abundance (Ratner and Lande, 2001). In our study, the population response appears overcompensatory, as harvested populations contained in total more individuals than the control populations (e.g. De Roos and Persson, 2013). However, total population size was directly related to the number of females: the more females, the more eggs and larvae, and, since populations largely consist of eggs and larvae (Fig. 4.2), the larger the total size of the population. We therefore suspect that any (over)compensatory response in population size to mean body size is overshadowed by the population consequences of variation in female numbers. What drives this variation is, however, unclear. Mean female numbers showed a negative association with mean fighter numbers across treatments. However, because fighters are not very successful in killing adult females (Łukasik, 2010) (probably because adult females are of a much larger size: Fig. 4.1C) and because mean female and fighter numbers were positively and not negatively associated within treatments, it is likely that cannibalism is not at play here. But, if there is a causal link between fighter and female numbers, then the knock-on consequences of variation in fighter density for population structure and size are substantial.

Our results illustrate that it is imperative to consider the concurrent ecological and evolutionary consequences of selectively harvesting individuals of only one type for the sustainable management of harvested populations, which is not always strongly guided by an evolutionary framework (Young, 2004). For example, human activities in salmon breeding environments and in the oceans are altering the proportion of male salmon maturing as sneakers (Gross, 1991; Piou and Prévost, 2013), which can reduce population growth (Myers, 1984). Unravelling the ecological and evolutionary mechanisms at play here is essential to maintain viable salmon populations that can be harvested sustainably. Furthermore, sport hunting of large trophy males in wild game populations can result in evolutionary shifts in trophy size and

body size of males (Coltman et al., 2003), which, in turn, can bias the population sex ratio towards females and lower reproductive success through the (delayed) production of fewer offspring of lower quality (Milner et al., 2007). The management of such wild game populations would therefore profit from a detailed understanding of how evolutionary processes driven by hunting feedback to affect ecological change. Rigorous monitoring and carefully conducted observations, however, are required to unravel the fine details of how interactions between evolutionary and ecological processes affect wild populations, their structure, stability and growth rates (Milner et al., 2007).

In conclusion, we have identified an eco-evolutionary interaction where phenotypic trait evolution has consequences for population size and structure, and ecology imposes selection on phenotypic traits. The importance of such interactions cannot be overstated. For example, if Costantino et al. (1995) had not replaced all individuals in their experimental populations with fresh ones from separate stock cultures during each census (to counteract possible genetic changes in life-history traits), would they still have been able to experimentally induce transitions in the dynamic behaviour of *Tribolium* populations? The existence of eco-evolutionary interactions also implies that experimental evolution studies would benefit from considering ecological responses in understanding trait evolution, whereas eco-evolutionary studies would gain from identifying the drivers of phenotypic change. Merging these fields to conduct carefully designed lab and field experiments (e.g. Cameron et al., 2013; Reznick et al., 2001; Chapter 5; this study) paves the way to a deeper understanding of the components and causal routes of the eco-evolutionary response of populations to environmental change.

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APPENDIX. PREDICTING THE EVOLUTION OF FIGHTER EXPRESSION

Predictions from the environmental threshold (ET) model rely on fitness functions from which we predict that a reduction in fighter (scrambler) fitness through selective harvesting results in a lower frequency of fighters (scramblers) in the population (Fig. 4.A1).

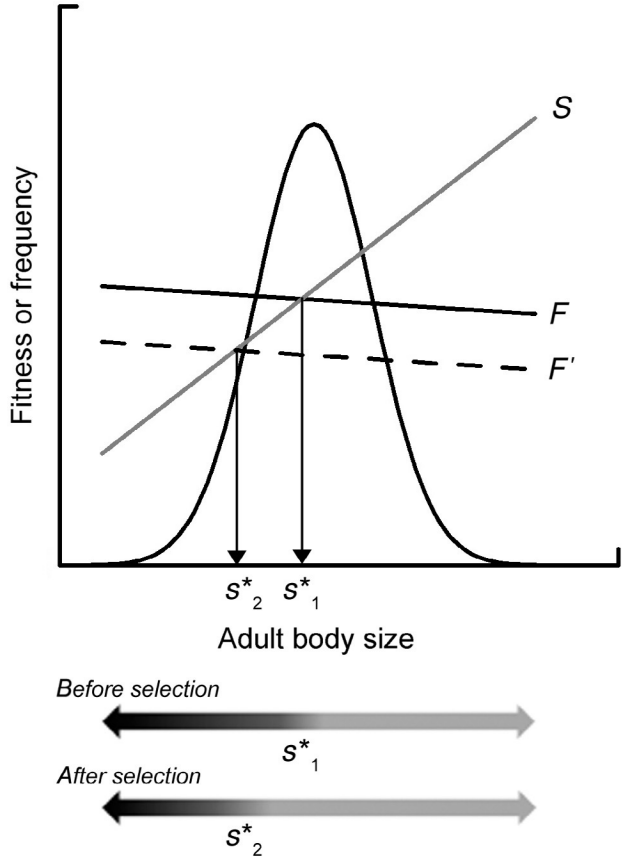

Figure 4.A1 Evolutionary predictions for selection on male morph expression using the ET model (Hazel et al., 1990, 2004). The solid straight lines are a schematic representation of the fitness functions found for scramblers (*S*; in grey) and fighters (*F*; in black) (Smallegange et al., 2012). Selection against fighters (in this case through harvesting) results in a decrease in the intercept of the fitness function of fighters (dashed line: *F′*), as a result of which the intersection point moves from its position before selection, s_1^*, to a smaller adult body size at s_2^* after selection (horizontal arrows: scramblers in grey and fighters in black), increasing scrambler expression. Note that fighter expression is negatively correlated with adult body size but positively correlated with quiescent tritonymph size (see Fig. 4.3 in Smallegange et al., 2012). This means that a reduction in fighter frequency (through reduced fighter fitness) comes about through an increase in mean tritonymph size threshold to a larger quiescent tritonymph size. Similarly, selection against scramblers increases the frequency of fighters in the population. The frequency distribution of body size (bell-shaped curve) is assumed not to be affected by selection, which was indeed the case in this study (see Section 3).

A1. Population dynamics

Time series of the number of individuals in each life stage and their mean size in each treatment is given in Fig. 4.A2.

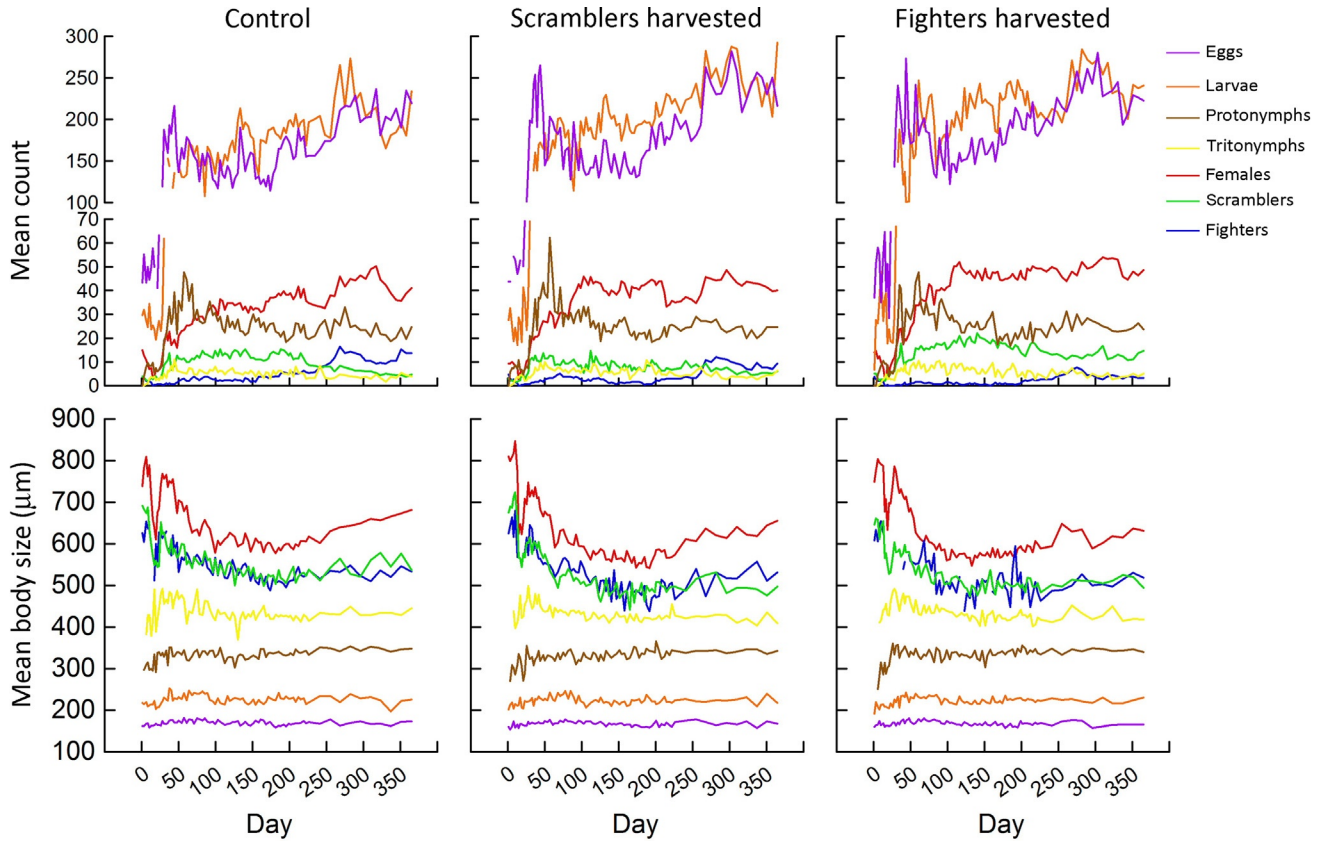

Figure 4.A2 Time series averaged per treatment. The top panels show the mean number of individuals in each life stage and the bottom panels show the mean body size of each life stage. Note the break and change of scale in the *y*-axis in the top panels. The harvesting treatment began on day 60. The dip in mean adult size of females, scramblers and fighters at the start of the experiment is caused by the removal of the large adults from the source populations at day 15.

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