DYNAMICS OF SPECIES EXTINCTION AND RECOVERY IN MULTI-TROPHIC AQUATIC SYSTEMS

by

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ABSTRACT

The current rate of species extinction is higher than at any other time in Earth's history. Despite our understanding of the causes and consequences of extinction and the development of numerous species conservation plans, it is surprising how little we know about the dynamics of extinction and recovery. Here, I explore the dynamics of population extinction and recovery across a range of meio-invertebrate species embedded in aquatic multi-trophic communities under external pressure. My results indicate that external mortality frequency has a negative impact on the dynamics of population extinction and recovery and suggest that it may be possible to predict patterns of population extinction from patterns of population growth as well as patterns of recovery from patterns of population collapse. My findings provide a valuable empirical basis from which we may increase our understanding of the factors influencing extinction risk and recovery potential to develop sustainable management strategies.

LIST OF ABBREVIATIONS AND SYMBOLS USED

Δ	Time segment
λ	Mean growth rate
$_{CV}$	Temporal variability in abundance
ANOVA	Analysis of Variance
GLM	General Linear Model
IUCN	International Union for Conservation of Nature
K	Carrying capacity
k_1	Last time population exceeds carrying capacity
k_2	First time population exceeds carrying capacity during recovery phase
NMDS	Non-metric Multi-Dimensional Scaling
NRSMs	Natural Rocky Shore Microcosms
q	Time population first declines to quasi-extinction
Q	Quasi-extinction
t_0	Starting time of the experiment
$t_{decline}$	Time when no future positive growth is observed
$t_{extinction}$	Time when population size reaches zero
t_{max}	Time of maximum value

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CHAPTER 1

INTRODUCTION

It is now common knowledge that species are currently experiencing a rate of extinction that is higher than at any background rate in the Earth's history (Pimm *et al.* 1995). It is also recognized that in order to maintain healthy ecosystems, active management of species that have been subjected to overharvesting and other disturbances is needed to facilitate species recovery (Myers *et al.* 2000). However, there exists an important gap in our understanding of extinction (Benton 2003) and recovery processes. While the causes and consequences of extinction and recovery have received significant attention, little is known about the actual dynamics of the extinction and recovery processes themselves. The goal of this present chapter is to describe the rationale for studying the dynamics of population extinction and recovery, to provide the necessary background information to do so, and to illustrate the promising implications of gaining a deeper understanding of the ecology of population extinction and recovery.

1.1. Species Extinction

The current rate of species extinction has been approximated to be up to a thousand times the background extinction rate evaluated from the fossil record (Pimm *et al.* 1995) and the latest International Union for Conservation of Nature (IUCN) Red List states that there are more than 16,000 species known to be currently threatened with extinction (Vié et al. 2009). In the last few decades, ecologists have not only learned why species go extinct but have also gained a considerable understanding of the consequences of such extinctions. For example, the effects of habitat degradation (Mora *et al.* 2007),

species invasions (Lodge 1993), and overexploitation (Pauly et al. 1998, Jackson et al. 2001) have been observed in a wide range of taxa (Stuart *et al.* 2004) and the resulting declines in biodiversity have been shown to lead to trophic cascades (Frank et al. 2005, Halpern et al. 2006), dangerous fluxes in ecosystem productivity rates (Loreau et al. 2001, Hooper et al. 2005, Isbell et al. 2008), secondary extinctions (Borrvall et al. 2000, Eklof and Ebenman 2006), and community instability (McCann 2000). However, we still do not know *how* populations go extinct, if, in other words, there exist general patterns in how population decline to extinction (Benton 2003).

The study of population dynamics is one the most established fields in ecology. By exploring the oscillations of population size over time, ecologists have gained insight into the external and internal forces that regulate a population's rate of increase (see reviews by Turchin 2001, Berryman 2003). However, there remain many areas of considerable uncertainty. Much research is still needed to understand the dynamics of population extinction and recovery as ecologists are only starting to determine the relative importance of external versus internal regulation of population abundances under deleterious environmental circumstances (e.g. Seivwright *et al.* 2005) and to understand the dynamics of populations at low abundances (Lande *et al.* 2003).

Species that are at high risk of extinction generally share similar life history attributes and are therefore regulated by similar internal factors. A small geographical range, a high trophic position within the food web, and a long generation time are all examples of life history traits that have been shown to predispose a species to a greater risk of extinction (Purvis et al. 2000, Cowlishaw et al. 2009). However, all species in the process of decline to extinction are also under one main external pressure: mortality. Deleterious events, such as habitat loss or overharvesting, cause higher mortality rates, relative to natural death rates, and therefore act as strong external regulating factors (Brooks *et al.* 2002). Additionally, populations driven to low numbers are under a greater threat of demographic and environmental stochasticity (Lande *et al.* 2003). Recently, it has been proposed that a species' biological traits, such as trophic position and geographic range size, may explain for 50% of the inter-specific variation in extinction

risk (Purvis *et al.* 2000). The remaining variance, on the other hand, may be explained by the effect of external mortality on extinction patterns (Purvis *et al.* 2000). However, such hypothesis of external control over extinction patterns remains unexplored.

Examining the dynamics of populations driven to extinction by external pressure can therefore increase our understanding of the factors regulating populations on the brink of extinction. Not only does this research question represent a promising avenue for the advancement of population ecology in general, but it also holds considerable implications for conservation. Given the wide variety of intensity and frequency of external causes of extinction found in nature, it is unknown whether the level of mortality influences the temporal pattern of population decline to extinction. For instance, fishing mortality can occur at very high or very low frequency. The main consequence of this varying frequency of perturbations is that the time window available to a population to respond to mortality events differs (Fox and Caldwell 2006, Cowlishaw et al. 2009). A question then arises: can mortality frequency influence the population dynamics of decline to extinction?

1.2. Species Recovery

Fortunately, for a number of species and ecosystems, management practices have been put into place to maintain ecosystem health and enhance species survival. In the last decades, biological corridors (Rosenberg *et al.* 1997) and marine reserves (Hughes et al. 2003), amongst other conservation methods, have been used to enhance species and ecosystem recovery. However, populations released from external mortality due to conservation actions still share many of the same characteristics as populations on the brink of extinction. They have very low abundances and are highly susceptible to environmental and demographic stochasticity.

A species' recovery potential is influenced by a number of life history attributes such as generation time (Beketov et al. 2008, Lobon-Cervia 2008) and population-level characteristics such as initial population size (Jennings 2000). Recovery potential is also

influenced by a number of external factors. For instance, the magnitude of a population's collapse can affect a population's recovery potential (Hutchings 2000). A population's collapse can also alter per capita resource availability and per capita predation risk within an ecosystem (Bundy and Fanning 2005), pushing recovering populations to adapt to an alternate ecosystem state. A better understanding of the dynamics of population recovery following periods of mortality may help to determine the long-term effects of external mortality on population recovery, which may in turn, increase our understanding of the internal and external forces hampering or enhancing recovery.

There exist a number of success stories of species recovery in the wild. Fish stocks have been shown to recover after the closure of fisheries or reductions of quotas (e.g. Murawski et al. 2000, Allen et al. 2007, Pondella III and Allen 2008) and similar case studies exist for a wide range of taxa, from marine mammals (Hucke-Gaete et al. 2004, Prigioni et al. 2007, Recharte Uscamaita and Bodmer 2009) to birds (Whitfield et al. 2008, Sulawa et al. 2010). However, such studies have typically concentrated on the assessment of short-term recovery and surprisingly little is known about the long-term dynamics of the population recovery process itself. Although the literature above highlights the intuitively positive impact of conservation actions on population survival, the accumulation and diversification of criteria used to measure recovery has led ecologists to inadequately assess recovery (Jones and Schmitz 2009). This issue is fundamental to conservation ecology as the concept of recovery can be encountered in almost all conservation plans of national and international organizations for the conservation of species (Species at risk act 2003, National Recovery Working Group 2005, IUCN/SSC 2008, National Marine Fisheries Service 2010) and as a result, much effort, time, and funding are spent on monitoring species survival (Boersma et al. 2001, Clark et al. 2002). By studying the long-term dynamics of recovering species, ecologists may increase their understanding of the ecology of recovering populations, which may, in turn, provide valuable information for the development of appropriate recovery measures.

1.3. Research Approach and Lay-Out of Thesis

While the field of extinction and recovery dynamics has the potential to increase our understanding of the factors regulating population extinction and recovery, predict persistence and stability of populations at low numbers (Inchausti and Halley 2003, Fagan and Holmes 2006, Drake and Griffen 2009, Griffen and Drake 2009), provide valuable information for the development of sustainable management practices, and inform the science community about adequate conservation avenues, it remains a field in its infancy. The main objective of my research was to experimentally explore extinction and recovery dynamics. Although many questions could have been examined related to the dynamics of extinction and recovery, I focused on two primary avenues: the testing of predictions of extinction and recovery dynamics, and the formulation of hypotheses about the potential biological mechanisms responsible for the observed trends.

Most research on extinction and recovery dynamics are theoretical (Dennis et al. 1991, Lande et al. 2003, Watanabe et al. 2005). While some studies of extinction dynamics have used fossil record data or recent extinction data (Fagan and Holmes 2006, Ringsby et al. 2006), for the most part, only a handful of studies have attempted to explore the dynamics of extinction and recovery experimentally (but see Drake and Lodge 2004, Drake and Griffen 2009, Griffen and Drake 2009). While an experimental approach to population ecology, depending on the protocol, does not always take into account the potential influence of environmental parameter fluctuations and the chance of catastrophic events, it can provide valuable information about the intrinsic biology of populations and allow ecologists to manipulate important environmental pressures, both of which represent important pieces of a complex puzzle.

To explore the dynamics of population extinction and recovery dynamics, I varied the rate of external mortality frequency for three meio-invertebrates species; *Daphnia magna*, *Microcyclops varicans*, and *Cypridinae eucypris*. Populations were embedded in laboratory analogues of natural meio-invertebrate communities found in Natural Rocky Shore Microcosms (NRSMs). NRSMs are small contained aquatic habitats populated by

zooplankton and benthic invertebrates. They are ideal systems in which to manipulate population dynamics and food-web structure as they are as complex and biologically realistic as other natural systems, but are easy to sample and contain diverse and tractable food-webs (Srivastava et al. 2004, Bulling et al. 2006). The complete communities can be collected from a wide range of biogeographical locations and can serve as scaled analogues of larger ecological systems (Petersen et al. 2003, Srivastava et al. 2004).

Multi-trophic aquatic communities used to explore the dynamics of population extinction and recovery were collected from NRSMs in Prospect Point, Nova Scotia, Canada, (43°29'26"N, 65°43'10"W). Once collected, communities were transferred to laboratory analogues of outdoor communities and housed in a controlled laboratory room. Microcosms were kept under the most natural biological conditions possible: communities lived in an environment composed of rock pool water and organic substrate. No food or nutrients were added as laboratory rock pool communities are self-sustaining with sufficient light. There are approximately four species of meio-invertebrates in the laboratory analogues of Nova Scotian NRSMs representing four trophic groups. Herbivores/detritivores contain one species of *Alona* sp. and one species of *Alonella* sp. Herbivores are represented by one species, *Daphnia magna*. The omnivore group contains one member of the class Ostracoda, *Cyprinidae eucypris*. Finally, carnivores represent the top predators in the system and are represented by a single species, *Microcyclops varicans*.

The first part of this research (Chapter 2) explores the dynamics of the extinction process itself. I determined the impact of mortality frequency on the duration, growth rate, and stability of populations with different life history traits. I then explored whether it was possible to predict a population's final decline to extinction. To do so, I tested current predictions proposing the existence of relationships between population mean growth rate, temporal variability in abundance, and the dynamics of population decline to extinction.

The second part of this research (Chapter 3) examines the dynamics of populations recovering from a period of strong mortality pressure. I first evaluated differences in the short-term and long-term recovery states of populations. I then determined the influence of varying levels of mortality frequency on the duration, growth rate, and stability of recovering populations. Finally, I examined the potential relationships between the pattern a population takes to collapse to quasi-extinction and the pattern a population takes to recover.

Finally, Chapter 4 discusses the strengths and limitations of my research. It also underscores the relevance of the main results from each chapter in the context of the main goals of my research, which were to gain a deeper understanding of the impact of internal and external factors on population extinction and recovery as well as to determine whether it is possible to predict patterns of population extinction and recovery based on population oscillations prior to extinction and recovery respectively.

CHAPTER 2

Dynamics of Populations on the Edge of Extinction

Véronik Campbell and Tamara N. Romanuk.

2.1 Abstract

The causes and consequences of extinction are well explored. In contrast, little is known about the dynamics of the extinction process itself. Here, we examine the population dynamics of extinction for two species of zooplankton, a herbivorous cladoceran and a carnivorous copepod, in response to different mortality frequencies. High mortality frequency affected the period of final decline to extinction but not the population growth phase prior to the decline. Populations under high mortality frequency had significantly shorter, steeper, and more variable declines to extinction as well as shorter times to extinction. This response to high mortality frequency was consistent across species despite differences in trophic roles and reproductive strategies. Species also responded similarly to low mortality frequency as infrequent removals had no effects on the population dynamics of extinction. It was only under intermediate mortality frequency that differences between species in their responses to mortality frequency emerged. Finally, the growth rate of the increase period was inversely proportional to the duration, growth rate, and temporal variability of the decline to extinction. Our results provide much needed empirical support for theoretical predictions of extinction dynamics

and hold considerable implications for the conservation and management of endangered and exploited species. They demonstrate the persistent effects of mortality frequency on a population's final decline to extinction, highlight the buffering effect of species life history attributes against intermediate levels of external pressure, and show that the duration, rate, and stability of the final decline to extinction can be partially predicted from a species growth rate before it begins its final decline to extinction.

2.2 Introduction

Widespread changes in the global environment have led to a rate of species extinction that is currently a hundred to a thousand times greater than background rates estimated from the fossil record (Pimm *et al.* 1995). The causes of extinction have been extensively studied (Hughes et al. 1997, Pauly et al. 1998, Thomas et al. 2004, Brook et al. 2008) and significant progress has been made in understanding the consequences of diversity loss on the functioning of ecological communities (Chapin III et al. 2000, McCann 2000, Cardinale et al. 2006). Much less attention, however, has been given to the dynamics of the extinction process itself (Benton 2003).

Species extinction is a natural process and is, ultimately, the fate of all species. However, a number of factors influence the probability that a species will be driven to extinction including endogenous factors such as species life history attributes and intrinsic population dynamics (Purvis et al. 2000, Lande et al. 2003, Reynolds et al. 2005, Cardillo et al. 2008, Cowlishaw et al. 2009) as well as exogenous factors such as the type and severity of environmental disturbances (Fisher et al. 2003, Isaac and Cowlishaw 2004, Price and Gittleman 2007, Cowlishaw et al. 2009). The frequency and intensity of external mortality pressure relative to population size and generation time is likely the most significant factor affecting whether or not a species will go extinct. As the time window available for population recovery diminishes, the negative impacts of external mortality on mechanisms such as the maintenance of genetic diversity (Lopes *et al.* 2009) or the expression of density-dependent mechanisms (De Roos et al. 2007, Schröder et al. 2009) are enhanced, therefore increasing a species' probability of extinction. It has also

been proposed that a number of current anthropogenic pressures may reach intensities and frequencies which may not allow a species' biological traits to buffer extinction pressure (Purvis *et al.* 2000). Given the current efforts to unravel the relative roles of internal and external factors on species extinction, it is essential to determine the effects of mortality frequency on the dynamics of populations on the brink of extinction (Cowlishaw *et al.* 2009) independently of life history attributes (Purvis *et al.* 2000).

A complementary approach to predicting extinction from specific internal and external factors is to study the behaviour of population extinction using simple population growth models (Ginzburg et al. 1982, Gilpin and Soulé 1986, Dennis et al. 1991, Lande et al. 2003). In an effort to increase our ability to predict the likelihood of species extinction, predictions stemming from stochastic models of population growth have recently been tested using experimental data (Drake and Griffen 2009, Griffen and Drake 2009). It has been predicted that the duration of a population's final decline to extinction is inversely proportional to the mean growth rate prior to the final decline (Lande et al. 2003, Griffen and Drake 2009) and that populations with greater temporal variability will have shorter times to extinction (Pimm et al. 1988, Inchausti and Halley 2003). If accurate, these predictions suggest that by exploring the population oscillations before the final decline, it may be possible to predict the rate at which populations decline to extinction and therefore which populations will require immediate conservation actions.

Here, we examine the dynamics of population extinction using an experimental framework where we manipulated mortality frequency for two species of zooplankton embedded in laboratory analogues of their natural communities. We first assess the relative importance of external mortality frequency on the persistence, growth rate, and temporal variability of populations with distinct life history attributes (Purvis et al. 2000, Cowlishaw et al. 2009). We then determine whether a population's final decline to extinction can be predicted from the general patterns of population oscillations prior to the final decline. To do so, we test current predictions suggesting the influence of mean growth rate on the duration of the final decline to extinction (Lande et al. 2003, Griffen

and Drake 2009) and the influence of temporal variability in abundance on mean time to extinction (Pimm et al. 1988, Inchausti and Halley 2003).

2.3 Methods

2.3.1 Study species

We examined the population dynamics of *Daphnia magna* (a herbivorous cladoceran) and *Microcyclops varicans* (a predatory copepod) for fifteen weeks to determine whether there were consistent patterns in extinction dynamics between species under three mortality frequency treatments and whether mean growth rate and temporal variability in abundance can be used to predict time to extinction (persistence) as well as the rate and variability of the final decline to extinction. *D. magna* and *M. varicans* were chosen due to their short generation times which makes the study of population response to extinction over several generations tractable (Drake and Lodge 2004, Drake and Griffen 2009, Romanuk et al. 2010).

D. magna and *M. varicans* have a number of different life history attributes including reproductive strategy, body size, trophic position, and generation time. *D. magna* is an herbivore while *M. varicans* is a carnivore. *D. magna* competes for resources with other herbivorous species (e.g. *Alona* sp. and *Alonella* sp.) while *M. varicans* is the top predator in the system. Daphnia reproduce asexually but switch to sexual reproduction through the development of resting eggs when environmental conditions are unfavourable (Bertram 1979) while copepods reproduce sexually. The body size of *D. magna* ranges from 1 to 5 mm while the body size of *M. varicans* ranges from 0.1 to 2 mm. *D. magna* has a generation time of approximately twelve days at 28ºC whereas *M. varicans* has a generation time of approximately sixteen days at 28ºC (Gillooly 2000).

Rock pool meiofauna, water, and detritus were collected from Prospect Point, Nova Scotia, Canada (43°29’26”N, 65°43’10”W) in September 2008. Once collected, communities were transferred to forty 1300 ml transparent plastic containers (12.5 x 12.5

x 20 cm, hereafter referred to as microcosms) and housed in the laboratory. Microcosms were placed in water baths to maintain a constant microcosm water temperature of 28ºC and were exposed to full spectrum light following a regime of 12 hours of day light (07:00 to 19:00). Microcosms were kept under the most natural biological conditions possible: communities persisted in an environment composed of rock pool water and organic substrate. No food or nutrients were added as laboratory rock pool communities are self-sustaining with sufficient light.

2.3.2 Experimental design

Other than the target species, *D. magna* and *M. varicans,* there were three other species of meio-invertebrates in each microcosm: one species of *Alona* sp. and one species of *Alonella* sp*.,* both of which are herbivore/detritivores of the family *Chydoridae*, and an omnivorous ostracod, *Cyprinidae eucypris*. Other biotic components of the community that were present include phytoplankton, periphyton, protists, bacteria, and detritus. The initial relative abundance of non-target meio-invertebrate species and other biotic components differed between microcosms.

Initial abundances of target species were established by concentrating and filtering 1300 ml of rock pool water using a 63 μm sieve net. We then counted the number of *D. magna* and *M. varicans* individuals in each microcosm and added or removed individuals to reach target abundances. For the *D. magna* treatment, twenty microcosms were established with an initial abundance of five adults. For the *M. varicans* treatment, twenty microcosms were established with an initial abundance of six adults. At the start of the experiment, adults did not carry egg sacs or resting eggs (e.g. daphnid eppiphia), and no resting eggs were found throughout the length of the experiment.

The manipulation of mortality frequency started ten days following the establishment of communities and was performed independently on fifteen populations of *D. magna* (Experiment 1) and fifteen populations of *M. varicans* (Experiment 2) from January to April 2009 and March to June 2009, respectively, for a total experimental

length of 100 days for each species. The frequency of mortality was manipulated by removing 20% of the total number of individuals (not considering maturity stage) present every ¼, ½, and total generation time (hereafter referred to as high, medium, and low mortality frequency). These frequencies correspond to removals conducted every 3, 6, and 12 days for *D. magna* and every 4, 8, and 16 days for *M. varicans.* Given that fishing pressure has been shown to reduce a population up to 100% within 15 years, which corresponds to approximately one to two fish generations (Hutchings 2000), our choice of a maximum mortality pressure of 60% within one generation is within the range of mortality pressures currently occurring in natural systems. Each mortality frequency treatment was replicated five times for a total of n=15 microcosms for each experiment. Five populations per experiment were not subjected to mortality for a total of n=5 control microcosms for each experiment.

To estimate how many individuals were to be removed from each microcosm, we counted the total number of individuals of the target species present in the microcosm and then removed 20% of the total populations size. *D. magna* is a large aquatic invertebrate. Removals were therefore done by hand. *M. varicans* cannot be seen with the naked eye. To perform the *M. varicans* removal, we concentrated and filtered each microcosm community using a 63 μm sieve net. The concentrated community was then observed through a dissecting microscope. Total population size was counted and 20% of the total population size was removed. Population abundance was monitored every ¼, ½, and total generation time for the highest frequency treatment, while population abundance was monitored every ½ and total generation time for the remaining treatments and the control.

The abundance of non-target meio-invertebrate species was monitored on a weekly basis using live density counts. To perform live density counts, we gently stirred the microcosm water to ensure that there was a homogeneous distribution of organisms. We then took a 40 ml sub-sample of the microcosm water and counted the number of individuals of each species using a dissection microscope. Numbers of individuals for *Alona* sp. and *Alonella* sp. were combined (*Chydoridae* spp.) as species identification

could not be made on live organisms. Following counts, the organisms were returned to the microcosm. Densities were then extrapolated to the total microcosm water volume (1300 ml) for relative abundance comparisons.

2.3.3 Statistical analysis

To determine the influence of mortality frequency, mean growth rate, and temporal variability on the dynamics of population extinction, we analyzed time series of population decline using a conceptual model inspired by Drake and Griffen (2009) and Griffen and Drake (2009). For each microcosm, we divided time series into time segments according to three temporal values representing the times when each population exceeded important biological thresholds: k_1, the time when populations last exceeded carrying capacity (K, Griffen and Drake 2009), t_{max}, the time when populations reached maximum abundance, and $t_{decline}$, the time when positive growth was no longer observed (Fagan and Holmes 2006). Carrying capacity was defined as the population size around which the population oscillates in a quasi-stationary state (Lande *et al.* 2003) and was calculated as the mean population size from the start of the experiment (t_0) to the time of extinction ($t_{extinction}$). Because a number of other measures could have been used to define carrying capacity such as median population size (Griffen and Drake 2009) we performed analyses using both the mean and median population size. Results did not differ between analyses, thus we only report the results using mean population size.

For each time series, seven temporal segments were established (Fig. 2.1): time to extinction, ΔP (t_0 to $t_{extinction}$), increase to maximum, ΔA (t_0 to t_{max}), increase to final

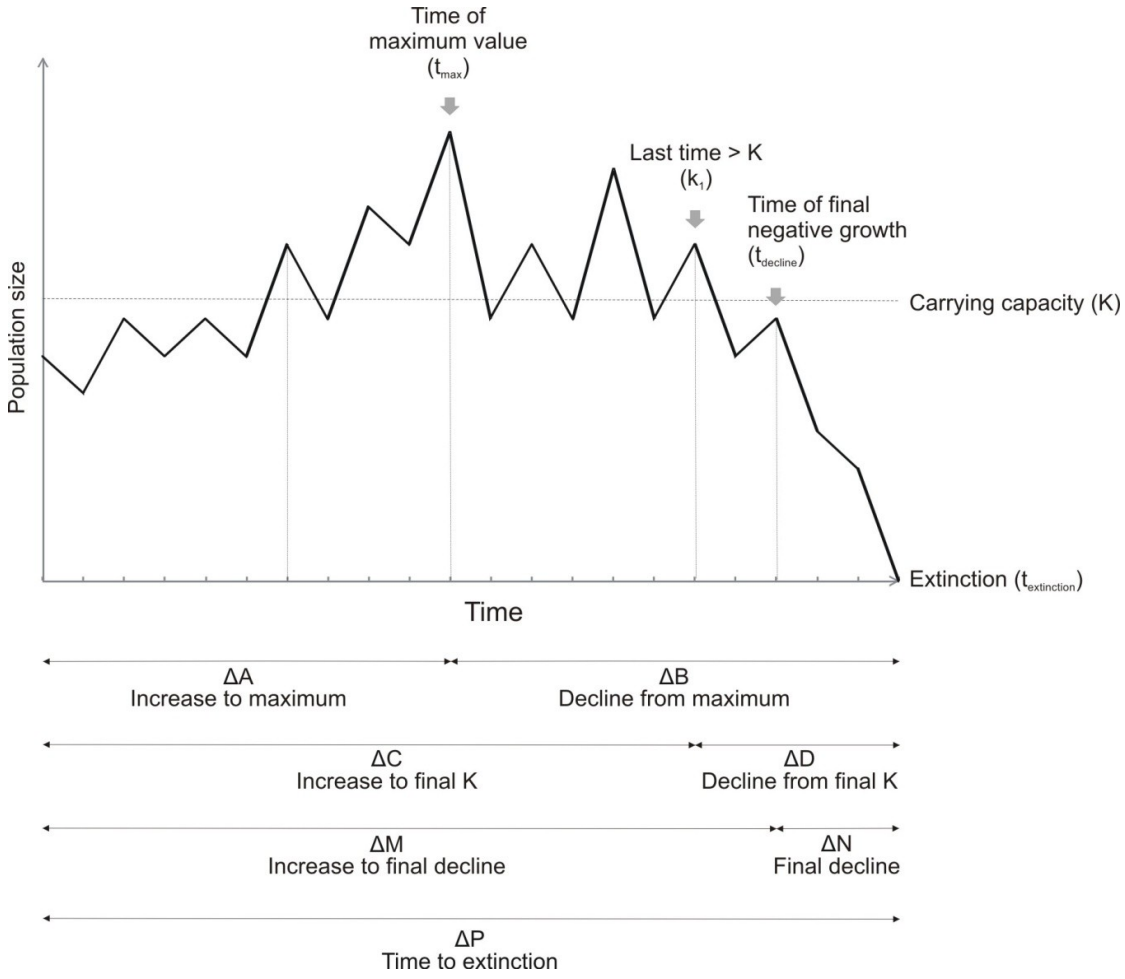

Figure 2.1.Conceptual model of population dynamics of extinction. t_{max} represents the time of maximum value, k_1 represents the last time population size exceeds carrying capacity, $t_{decline}$ represents the time when no future positive growth rate is observed, K represents the carrying capacity, and $t_{extinction}$ represents the time when the population size reaches zero. Time to extinction, ΔP (t_0 to $t_{extinction}$), increase to maximum, ΔA (t_0 to t_{max}), increase to final decline, ΔM (t_0 to $t_{decline}$), increase to final K, ΔC (t_0 to k_1), decline from maximum, ΔB (t_{max} to $t_{extinction}$), final decline, ΔN ($t_{decline}$ to $t_{extinction}$), and decline from final K, ΔD (k_1 to $t_{extinction}$) are shown.

decline, ΔM (t_0 to $t_{decline}$), increase to final K, ΔC (t_0 to k_1), decline from maximum, ΔB (t_{max} to $t_{extinction}$), final decline, ΔN ($t_{decline}$ to $t_{extinction}$), and decline from final K, ΔD (k_1 to $t_{extinction}$). We then calculated the mean growth rate (λ) and temporal variability in abundance (CV) for each time segment. Mean population growth rate (e.g. λΔN) was calculated as the ratio of population abundance change over consecutive time steps (N_{t+1}/N_t). Mean temporal variability in abundance (e.g. ΔN_{CV}) was calculated as the coefficient of variation (CV = standard deviation/mean). If the time segment included fewer than three data points, the coefficient of variation was not calculated (this occurred in 19 of 168 cases). The populations in the high mortality treatment were monitored more frequently to perform removals. The resulting difference in the number of data points across populations from different treatments can bias mean growth rate and temporal variability calculations. We therefore only used ½ and total generation time population size values for estimates of growth rate and temporal variability.

To determine whether mortality frequency affected the population dynamics of extinction, we used general linear models (GLMs) with duration, growth rate, and temporal variability of each time segment as dependent variables (for a total of 21 dependent variables) and mortality frequency (high, medium, and low) and target species (*D. magna* and *M. varicans*) as categorical variables. The mortality regime and target species interaction term was removed for all variables except for ΔB, ΔP_{CV}, log ΔA, and log ΔD (log transformed for normality; y = mortality frequency*target species + mortality frequency + target species). Univariate analyses of variance (ANOVA) were used to determine significant differences between mortality frequency treatments and between target species. The number of control populations per species that did not proceed to extinction was too low to perform statistical analyses, thus these were not included in the GLMs (e.g. *D. magna* n=2). We evaluated normality using the Shapiro-Wilk's test and homogeneity of variance using the Levene's test. Variables showing non-normal distributions were log-transformed (ΔA, ΔD, λΔM, λΔC, ΔB_{CV}; Appendix A). To test the relationship between mean growth rate of population increase (λΔA, λΔM, λΔC) and dynamics of final decline (e.g. ΔB), we scaled mean growth rates to their natural logarithms, as assumed by the original prediction (Lande *et al.* 2003).

The initial prediction of an inverse relationship between growth rate of increase and duration of final decline (Lande *et al.* 2003) applies to specific time spans of increase and decline, namely, the period of increase ends when population size last exceeds carrying capacity (Fig 2.1., ΔC) at which point the period of decline begins and ends at extinction (Fig 2.1., ΔD). The last time a population exceeds carrying capacity is considered an important biological ceiling value as it represents the last time the population oscillates around a quasi-stationary state (Lande *et al.* 2003). Here, in addition to considering dynamics to and from k_1, we expand the original prediction to include time segments of increase based on other biological ceilings of importance, namely ΔA and ΔB, which represent the increase to and decline from the time of maximum population size, t_{max}, as well as ΔM and ΔN, which represent the increase to and decline from the time when final decline to extinction begins, $t_{decline}$ (Fagan and Holmes 2006). In addition to the duration of decline, we also examined whether the growth rate of increase could influence the growth rate and temporal variability of decline. The original prediction of a negative relationship between temporal variability (ΔP_{CV}) and mean time to extinction (ΔP) applies to the entire length of the extinction process (Inchausti and Halley 2003). Given the potential associations between population increase and population decline to extinction with regards to growth rate, we here expand the above to include relationships between the temporal variability of increase and the dynamics of population declines around the biological ceiling values defined above.

To determine whether the duration, rate, and temporal variability of final declines to extinction were inversely proportional to mean growth rates prior to the declines, we used linear regression models with duration, growth rate, and temporal variability of each decline time segment as dependent variables (ΔB, ΔN, ΔD, $\lambda\Delta B$, $\lambda\Delta N$, $\lambda\Delta D$, ΔB_{CV}, ΔN_{CV}, ΔD_{CV}) and the inverse of the natural logarithm of growth rate of increase as the predictor variable ($1/\ln(\lambda\Delta A)$,$1/\ln(\lambda\Delta M)$, $1/\ln(\lambda\Delta C)$). Likewise, to determine whether populations with greater temporal variability in abundance had shorter mean times to extinction (ΔP) and shorter, steeper, and more variable final declines to extinction, we used linear regression models with duration, growth rate, and temporal variability of

mean time to extinction and decline time segments as dependent variables (ΔP, ΔB, ΔN, ΔD, $\lambda\Delta B$, $\lambda\Delta N$, $\lambda\Delta D$, ΔB_{CV}, ΔN_{CV}, ΔD_{CV}) and temporal variability of mean time to extinction and temporal variability of increase time segments as predictor variables (ΔP_{CV}, ΔA_{CV}, ΔM_{CV}, ΔC_{CV}). To determine if there was a significant interaction between mortality frequency and target species and the above relationships, we used a GLM.

Fifteen of 15 *D. magna* and 12 of 15 *M. varicans* populations went extinct. Three *M. varicans* populations (two from the medium and one from the low mortality frequency treatment) did not proceed to extinction. These populations were excluded as it is not possible to calculate time segments of extinction for populations that do not go extinct. Two populations of *D. magna* in the medium mortality frequency treatment and one population of *M. varicans* in the high mortality frequency treatment never showed positive growth rate. These populations were also excluded from the analysis as they collapsed to extinction too rapidly to allow the calculation of extinction time segments. In total, nine high mortality frequency populations, six medium mortality frequency populations, and nine low mortality frequency populations were included in the analysis (n=24). Time series of all populations included in the analysis are shown in Figure 2.2.

Finally, we examined whether species richness, non-target species abundances and growth rates, and community composition were affected by mortality frequency or the identity of the species removed. To determine whether the species removals affected the abundances of each non-target species, we examined the relative change in abundance from the start of the experiment to the time of extinction ((final abundance-initial abundance/total abundance)*100) for each non-target species and for all species pooled using a GLM with relative changes in each species abundance and for total abundance as dependent variables and mortality frequency and target species as categorical variables. To assess whether growth rate of the target species was correlated with the growth rate of each non-target species, we constructed cross-correlations plots by mortality frequency and target species. If the cross-correlations were not significantly different ($p \geq 0.05$), this provided evidence that interspecific interactions were not affected by the mortality frequency treatment or the identity of the species removed. Finally, we examined the

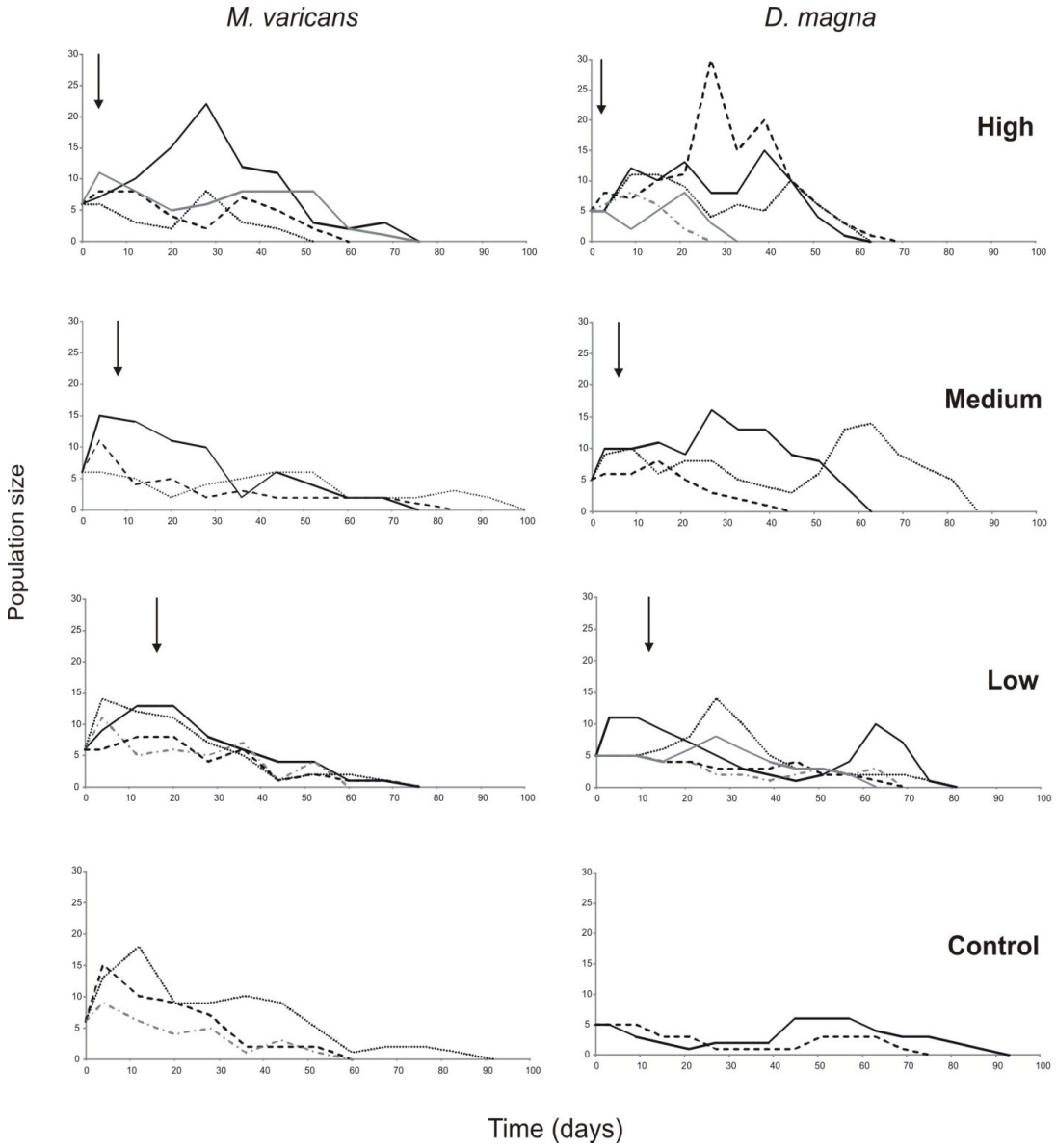

Figure 2.2. Extinction time series of untransformed abundances are shown for *D. magna* and *M. varicans* across high (¼ generation time), medium (½ generation time), low mortality (total generation time), and no mortality (control) frequency treatments. Each line represents one replicate. Frequency of removals is represented by the downward arrow. Each experiment lasted for a total of 100 days. Population abundance was monitored every ¼, ½, and total generation time for the highest frequency treatment and every ½ and total generation time for the medium and low mortality frequency treatments. Only measures of ½ and total generation time of the mortality treatment and control populations are shown here.

changes in relative abundance of each species over time across treatments and target species using non-metric multi-dimensional scaling (NMDS) based on a rank-order resemblance matrix using Bray-Curtis similarity values (Primer-E 2006). NMDS is considered to accurately assess patterns in relative abundance when the stress value is less than 0.15 (Clarke and Warwick 2001). Community composition was considered to change if the trajectories of relative abundance groupings changed over time with mortality frequency treatment, or with the identity of the species removed. Statistica 7.0 was used for all analyses except for the NMDS (StatSoft 2002).

2.4 Results

2.4.1 Impact of external mortality frequency

Of the 21 dependent variables related to duration, growth rate, and temporal variability of declining populations, six were significantly different across mortality frequency treatments across both species (Fig. 2.3; Appendix A). High mortality frequency led to a 21% decrease in time to extinction when compared to lower levels of mortality frequency (ΔP, $p=0.049$). High mortality frequency led to a 33% decrease in the duration of the decline from maximum (ΔB, $p=0.033$) and a 41% decrease in the duration of the decline from final K ($\log\Delta D$, $p=0.015$). High mortality frequency led to a 55% decrease in the mean growth rate of the decline from final K ($\lambda\Delta D$, $p=0.012$). Temporal variability of the final decline (ΔN_{CV}, $p=0.018$) and decline from final K (ΔD_{CV}, $p=0.007$) were 22% and 32% greater for populations under high mortality frequency (Fig. 2.3).

Time to extinction, durations of decline, and the growth rate of decline from final K were always smaller under the high mortality frequency treatment (ΔP, $p=0.034$; ΔB, $p=0.01$; $\log \Delta D$, $p=0.019$; $\lambda\Delta D$, $p=0.021$) and temporal variability of declines were always greater under high mortality frequency (ΔN_{CV}, $p<0.001$; ΔD_{CV}, $p<0.001$; Fig. 2.3). While *D. magna* and *M. varicans* responded similarly to high and low mortality frequency, they did not respond similarly to medium mortality frequency for three of six

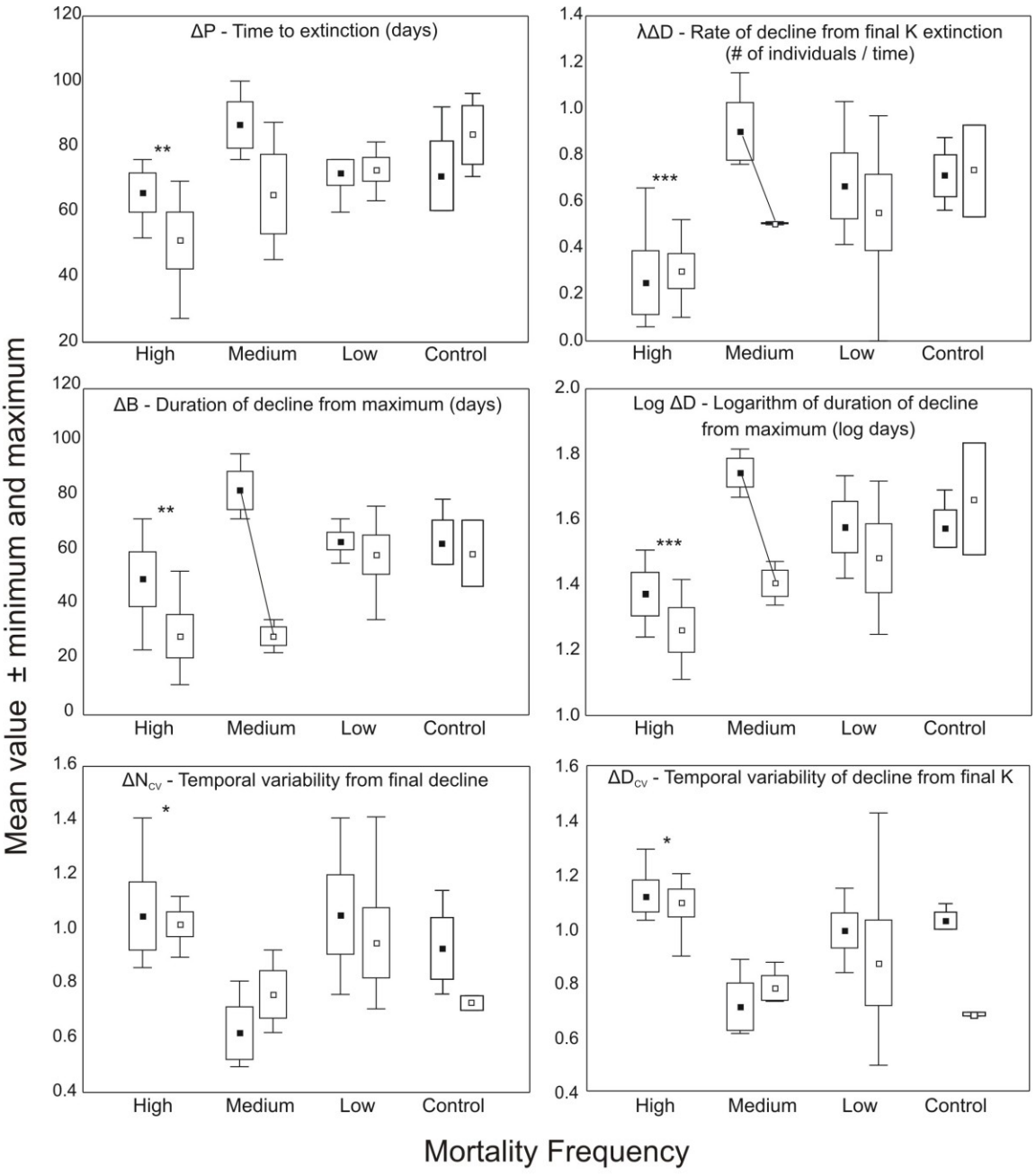

Figure 2.3. General linear model results showing the effects of high, medium, and low mortality frequency on extinction time segments for *D. magna* □ and *M. varicans* ■. Control populations were not included in the analysis as the number of replicate per species was too low. Box plots show the mean values of the duration of time to extinction (ΔP), the duration of the decline from maximum (ΔB), the logarithm of the duration of the decline from final K (log ΔD), the mean decline rate from final K (λΔD), the temporal variability of final decline (ΔN_{CV}), and the temporal variability of decline from final K (ΔD_{CV}). Boxes represent standard error and whiskers represent the minimum and maximum values. Lines connecting mean values show significant differences between the responses of *D. magna* and *M. varicans* to the same mortality frequency treatment. Star symbols (*) located above boxes of the high mortality frequency treatment show significant differences between treatments when species are pooled (high vs. medium *, high vs. low **, and high vs. medium and high vs. low ***).

variables (Fig. 2.3; Appendix B). Under medium mortality frequency, *D. magna* had significantly shorter durations of decline from maximum and from final K than *M. varicans* (ΔB, p=0.003; log ΔD, p=0.003). Likewise, *D. magna* had a significantly lower mean growth rate of decline from final K than *M. varicans* ($\lambda\Delta D$, p=0.029; Fig. 2.3).

2.4.2 Relationships between mean growth rate and dynamics of final decline to extinction

The duration and rate of decline to extinction from maximum were inversely proportional to the mean growth rate of increase to maximum ($1/\ln\lambda\Delta A$; ΔB, r^2=0.34, p=0.004; $\lambda\Delta B$, r^2=0.29, p=0.008; Fig. 2.4). A smaller growth rate of increase to maximum ($\ln\lambda\Delta A$) also predicted a less stable decline from maximum (ΔB_{CV}, r^2=0.2, p=0.032; Fig. 2.4). Mortality frequency and the identity of the target species did not affect the direction and strength of the relationships (mortality frequency, p=0.583; target species, p=0.375).

2.4.3 Relationships between temporal variability and dynamics of extinction

Mean time to extinction (ΔP) was not shorter in populations with higher temporal variability (ΔP_{CV}, r^2=0.02, p=0.551; Fig. 2.5). Temporal variability of increase (ΔA_{CV}, ΔM_{CV}, ΔC_{CV}) was not related to the duration, rate, and temporal variability of declines to extinction (ΔB, ΔN, ΔD, $\lambda\Delta B$, $\lambda\Delta N$, $\lambda\Delta D$, ΔB_{CV}, ΔN_{CV}, ΔD_{CV}). Mortality frequency and the identity of the target species did not affect the direction and strength of the relationships (mortality frequency, p=0.121; target species, p=0.617).

2.4.4 Community level response

Species richness was identical across microcosms and no extinctions of non-target species were observed. The mortality frequency treatment had no effect on relative change in total abundance (p=0.754) whereas the target species treatment had a significant effect on relative change in total abundance (p<0.0001; Appendix C). Total abundance increased by 33% in the *D. magna* treatment, however this increase was not

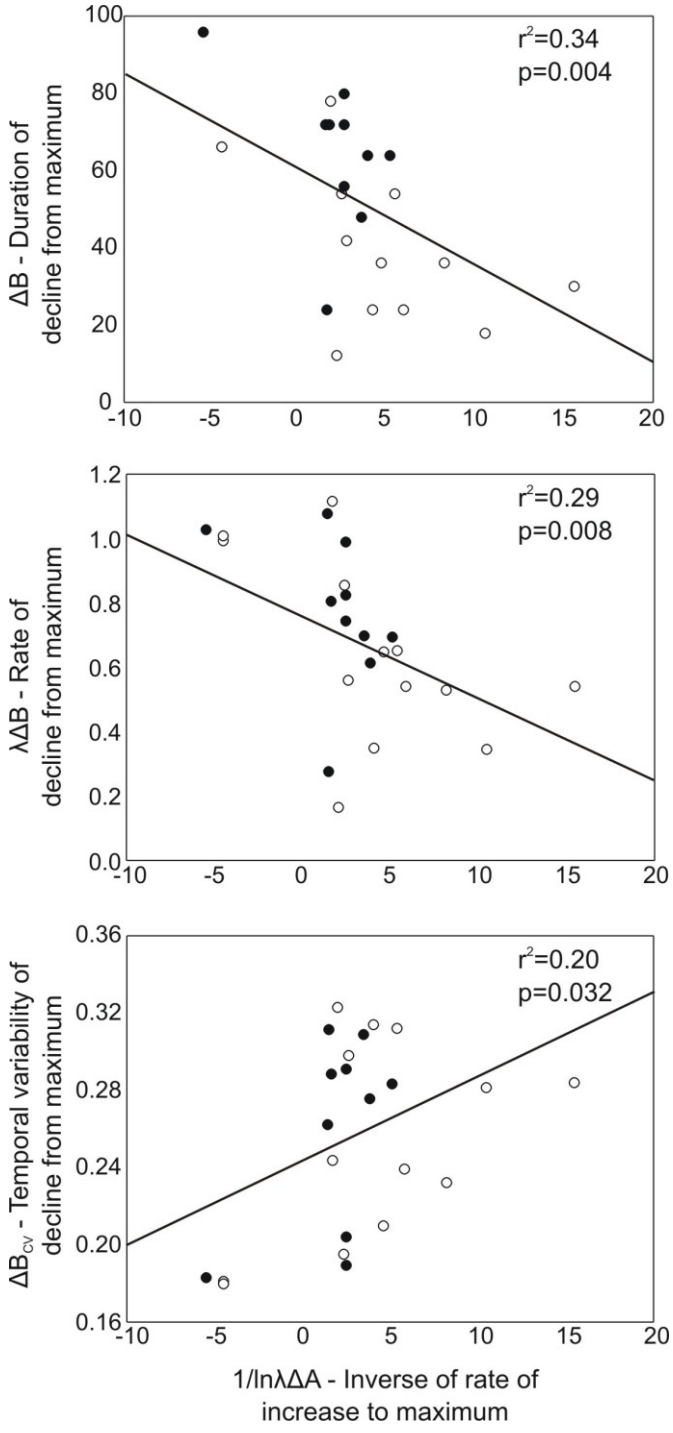

Figure 2.4. Relationship between inverse rate of increase to maximum (1/ln(λΔA)) and duration, rate, and temporal variability of the final decline from maximum (ΔB, λΔB, ΔB_{CV}). Data points for *D. magna* ○ and *M. varicans* ● are shown separately. Mortality frequency and target species treatments did not significantly affect the direction or strength of the relationships.

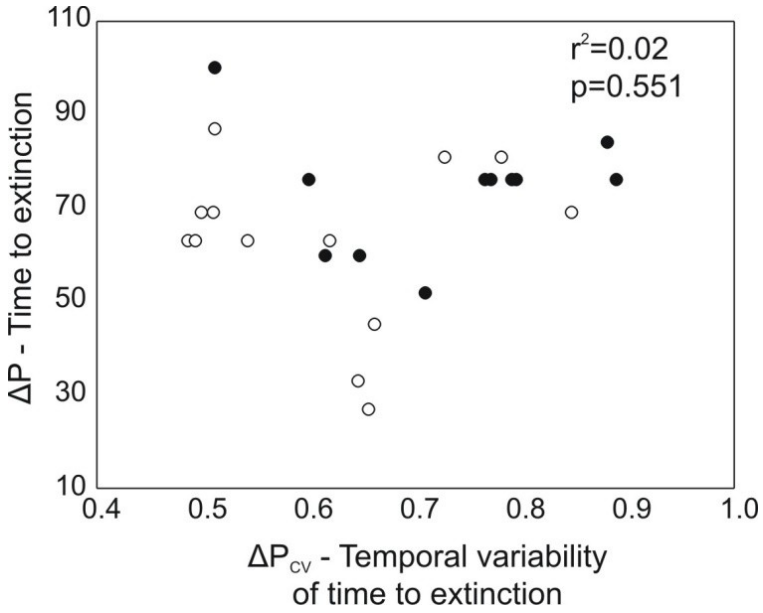

Figure 2.5. No relationship between temporal variability of time to extinction (ΔP_{CV}) and time to extinction (ΔP). Data points for *D. magna* ○ and *M. varicans* ● are shown separately. Mortality frequency and target species treatments did not significantly affect the direction or strength of the relationships.

significant (p=0.091). Total abundance decreased by 53% in the *M. varicans* treatment (p=0.0016). Mortality frequency had no effect on the relative change in abundance for any of the non-target species (all p>0.05; Appendix C). However, the abundance of the *Chydoridae* spp., which are herbivore/detritivores, differed from the initial to final sampling according to the identity of the target species removed (p<0.0001). *Chydoridae* spp. abundance increased by 35% from initial to final sampling in the *D. magna* removal experiment and decreased by 58% from initial to final sampling in the *M. varicans* removal experiment (Appendix C). Despite this difference, growth rates of the target and non-target species did not significantly covary (*D. magna*, r^2=0.001; *M. varicans*, r^2=0.02; Appendix D) and the strength of the cross-correlations was unaffected by mortality frequency (p>0.05). Results from the NMDS analysis show that all microcosms were at least 70% similar across mortality frequency treatments, target species, and over time (initial, halfway to extinction, at extinction; Appendix E). Stress for each target species and mortality frequency treatment was less than 0.15 indicating that the NMDS provided relatively accurate descriptions of the patterns in abundance.

2.5 Discussion

Given the current widespread species loss, a greater understanding of the internal and external factors influencing extinction dynamics is crucial to the development of conservation and management strategies. Here, we examined the population dynamics of extinction of two zooplankton species in response to different levels of mortality frequency. We tested whether external mortality regulates extinction dynamics (Purvis et al. 2000, Cowlishaw et al. 2009), whether there is an inverse relationship between a population's growth rate and the duration of its final decline (Lande *et al.* 2003), and whether a population's temporal variability is related to its mean time to extinction (Inchausti and Halley 2003). To our knowledge, this study is amongst the first to examine the extinction dynamics of populations experimentally and to study extinction dynamics of populations embedded in natural communities, thus allowing species interaction to play a role in the extinction of a population (Griffen and Drake 2008).

Our results suggest a number of patterns that are of considerable interest. First, we found that external mortality frequency influenced extinction risk (Cowlishaw *et al.* 2009). A clear pattern arose in that high mortality frequency affected the period of population decline to extinction but did not affect the period of population increase. Populations under high mortality frequency had the shortest, steepest, and most variable declines to extinction as well as the shortest mean time to extinction (Fig. 2.3). While the more frequent removal of individuals had a direct negative impact on growth rate and temporal stability of decline to extinction, our results suggest that a number of mechanisms which did not influence population oscillations during the increase period may have come in effect as populations neared extinction and therefore contributed to steeper and more variable declines to extinction. Gilpin and Soulé (1986) suggest that a population decreases to extinction in a vortex fashion whereby deleterious mechanisms increase in intensity as extinction nears. Declining populations inevitably become smaller and are therefore increasingly affected by demographic stochasticity whereby the random variations in birth and mortality events increase (Lande *et al.* 2003). As corollaries, temporal variability increases and populations decline at a faster rate as extinction nears (Fagan and Holmes 2006). Finally, a declining density of conspecifics can hamper a number of mechanisms beneficial to population growth such as mate availability, predator dilution, and cooperative predation (Allee effect review, Stephens *et al.* 1999).

The absence of mortality impact on population increase parameters and the significant impact of mortality on population decline parameters have significant implications for harvesting models that attempt to manage populations using criteria such as maximum sustainable yield (Schaefer 1954). A population under mortality pressure can continue to grow in size showing only a weak response to magnitude and frequency of individual removal. At a critical period, however, mortality shifts the population into a relatively steady decline, the speed of which corresponds in part to the frequency and magnitude of mortality and in part to the increased effect of demographic stochasticity and Allee effect mechanisms as extinction nears (Gilpin and Soulé 1986, Stephens et al. 1999). While our results only apply to situations where mortality frequency and magnitude remain constant during the growth period, a population can show healthy

population growth under strong harvesting, indicating a viable stock, despite the fact that it is potentially heading to a sudden collapse.

Our results also indicate that external pressure can influence a species' extinction regardless of its life history attributes (Purvis *et al.* 2000). Despite differences in internal regulating mechanisms, such as resource acquisition and reproductive mode, strong mortality pressure led to similar patterns of extinction dynamics in both species. Species also responded similarly to low mortality frequency as infrequent removals had no effects on the population dynamics of extinction as shown by Figure 2.3 (low vs. control). However, *D. magna* and *M. varicans* responded differently under medium levels of mortality frequency. For instance, the durations of decline from maximum (ΔB) and from final K ($\log\Delta D$) were significantly lower for *D. magna* than for *M. varicans*. Likewise, *D. magna* experienced a significantly lower growth rate under medium mortality frequency than *M. varicans* ($\lambda\Delta D$; Fig. 2.3; Appendix B). These findings underscore the relative importance of life history attributes in buffering intermediate levels of mortality frequency. Our results suggest that species trait differences between *D. magna* and *M. varicans*, such as reproductive mode or trophic role, may have played a key role in buffering extinction risk.

The silver-lining in terms of predicting extinction risk is that it may be possible to predict a population's dynamics of decline to extinction from the dynamics of population increase prior to the final decline. Here, we show that it is possible to predict a population's dynamics of decline to extinction using the mean growth rate prior to the decline (Fig. 2.4). We did not find any significant relationships between the extinction parameters initially proposed by Lande et al. (2003) whereby the duration of decline to extinction from the moment a population last exits the quasi-stationary state around carrying capacity (k_1) is inversely proportional to the rate of population growth during the quasi-stationary state. Instead, our results indicated that the growth rate to maximum ($\lambda\Delta A$), rather than to k_1 ($\lambda\Delta C$), predicted 34%, 29%, and 20% of the variance in the duration, growth rate, and temporal variability of the decline to extinction from maximum, respectively (ΔB, $\lambda\Delta B$, ΔB_{CV}; Fig. 2.4). The lack of significant relationship

between growth rate of increase and duration of decline around k_1 could be explained by a number of factors, the most probable being that our zooplankton time series did not support the assumption of density-independence (Lande *et al.* 2003). While it was possible to predict a population's dynamics of decline to extinction using the mean growth rate prior to the decline, it was not possible to predict a population's dynamics of decline or mean time to extinction using temporal variability in abundance (Fig. 2.5). However, we caution that a number of factors ranging from life history attributes (Pimm *et al.* 1988) to time series length (Inchausti and Halley 2003) can make this relationship difficult to detect. Despite these caveats, the strong predictive power of population increase rates on decline parameters has direct implications for the conservation of endangered species as a population's growth rate may provide key cues as to how quickly a species will decline to extinction.

To date, experimental work on extinction dynamics has been limited to laboratory microcosms using single consumer species and their resources (e.g. Drake and Griffen 2009, Griffen and Drake 2009). Examining extinction dynamics within a community context is crucial as species interactions can play important roles in mediating external pressure (Menge and Sutherland 1987, Griffen and Drake 2008). Here, we show that growth rates of the other meiofaunal species were not significantly related to the growth rate of the target species in any of the mortality frequency treatments (Appendix D) despite the different trajectories in abundance of *Chydoridae* spp. between the *D. magna* and *M. varicans* removal experiments (Appendix C). Additionally, community composition similarity did not change across mortality frequency treatments, across species, or over time (Appendix E) suggesting that the observed trends in extinction dynamics were the result of the direct effects of mortality frequency on population size fluctuations and not the results of indirect effects of mortality frequency on community composition or species interactions.

2.6 Conclusion

We observed three patterns of considerable interest for understanding and predicting how populations decline to extinction. First, external mortality did not affect the period of population increase prior to the decline to extinction but instead negatively affected the duration, growth rate, and temporal stability of the period of decline to extinction. Second, the role of species traits in buffering pressure from external mortality became less important as mortality frequency increased. Finally, our results provide strong support for the prediction of an inverse relationship between growth rate of increase and the dynamics of final decline, suggesting that it may be possible to predict a population's dynamics of final decline to extinction prior to its collapse. Understanding the factors that affect the duration and variability of population decline to extinction is critical for the advancement of theoretical predictions of extinction dynamics and for the conservation and management of endangered and exploited species.

2.7 Acknowledgements

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CHAPTER 3

Recovery and Collapse of Populations under Mortality Pressure

Véronik Campbell and Tamara N. Romanuk.

3.1 Abstract

Given the importance of species survival for ecosystem health and the considerable time and effort investment in species conservation plans, we know astoundingly little about what constitutes a species recovery. Here, we examined the dynamics of population recovery of *Cypridinae eucypris,* a common meio-invertebrate, following a period of induced mortality leading to quasi-extinction. All populations rapidly recovered to high abundances within two to three generations, but then experienced significant declines in abundance within five to six generations. These results suggest that although short-term recovery measures based on abundances and growth rates can indicate signs of recovering populations, sharp declines in abundances can be experienced later in the recover process. This finding also highlights the misleading conclusions of the three-generation time frame criterion commonly used by conservation organizations and suggests that recovery criteria span longer temporal scales. We found that populations under high mortality frequency seem to recover the best, reaching greater abundances than populations under lower mortality frequencies. However, these populations also experienced the greatest recovery-phase declines, suggesting that mortality frequency has a significant impact on long-term population

recovery. Finally, we found strong associations between the rate of population decrease to quasi-extinction and the rate of population increase to recovery, suggesting that it may be possible to predict the dynamics of population recovery from the dynamics of population decline to quasi-extinction.

3.2 Introduction

Worldwide, more than 16,000 species are known to be currently threatened with extinction (Analysis of the 2008 IUCN Red List of Threatened Species, Vié *et al.* 2009). This number will only increase as habitat loss, species invasion, and overexploitation continue to negatively impact biodiversity (Diamond 1989). In response, an incredible amount of time, effort and funding has been expended to reduce the impact of biodiversity loss, monitor species survival, and enhance species recovery (Hughes et al. 1997, Rosenberg et al. 1997). Recovery has become a fundamental concept in all species conservation plans where one must assess recovery feasibility and clearly define long term recovery objectives (Species at risk act 2003, National Recovery Working Group 2005, IUCN/SSC 2008, National Marine Fisheries Service 2010). However, surprisingly little is known about what constitutes long-term species recovery (Gårdmark *et al.* 2003).

Species recovery following declines to low population numbers has been observed in a number of species (e.g. Australian mammals, Short and Smith 1994, Marine fishes, Hutchings 2000, Lake trout, Fabrizio et al. 2001, Antarctic fur seals, Hucke-Gaete et al. 2004, Eurasian otters, Prigioni et al. 2007, Welsh hen harriers, Whitfield et al. 2008, German white-tailed eagles, Sulawa et al. 2010). While these examples highlight the intuitively positive impact of conservation strategies on species survival, they also demonstrate the lack of rigorous recovery criteria (Jones and Schmitz 2009). For example, whether a population has re-established in its historical range (Mech 2005), whether the catch per unit effort of a fish stock has increased over time (Pondella III and Allen 2008), or whether any "measurable condition of population recovery" is met for a period of three generations (National Marine Fisheries Service 2010) are all considered valid recovery criteria. While these measures provide useful information

about the short-term recovery state of populations, whether they predict, and more importantly, assure the long-term viability of recovering populations is unknown. A thorough understanding of the dynamics of the recovery process can highlight the different mechanisms responsible for recovery (Jennings 2000, Lobon-Cervia 2008), provide important insights on the long term viability of recovering populations, and therefore has the potential to contribute to defining accurate and biologically realistic recovery criteria.

The ability to predict the factors that will lead to the most stable recovering growth rates and abundances over long temporal scales is fundamental to conservation ecology. While population recovery can be influenced by internal factors such as life history attributes (e.g. generation time, Beketov et al. 2008) and intrinsic characteristics of populations (e.g. initial population size, Jennings 2000), it can also be affected by external anthropogenic factors (e.g. magnitude of fishing mortality, Hutchings 2000, Hutchings and Reynolds 2004). The type and intensity of external mortality has been predicted to affect population recovery (Connell 1997, Hutchings 2000), with recovery occurring at a rate inversely proportional to the degree to which the external perturbations occurred (Holling 1973, Jones and Schmitz 2009). Two key questions then arise: can mortality frequency leading to population collapse affect the dynamics of population recovery, and is it possible to predict a population's recovery dynamics based on the rate of a population's collapse?

To answer these questions, we manipulated the rate of mortality events in populations of *Cypridinae eucypris,* a common aquatic meio-invertebrate, until populations reached low population size (quasi-extinction) at which time we stopped the mortality pressure and monitored the populations' recovery over time. Here, we first assess the recovery status of populations at different stages in the recovery process to determine whether the measures of high population abundances and high growth rates after three generations adequately represent a population's long-term recovery (National Marine Fisheries Service 2010). We then evaluate the impact of mortality frequency on recovery time and on the long-term population growth rate and temporal stability of

recovering populations (Hutchings 2000). Finally, we explore whether the rate of population recovery is proportional to the rate of population collapse (Holling 1973, Jones and Schmitz 2009).

3.3 Methods

3.3.1 Study system

C. eucypris is an asexual omnivorous Ostracod commonly found in supralittoral rock pools in temperate regions. *It* has a short generation time (sixteen days at 23 °C, Gillooly 2000) which makes the study of population dynamics over several generations tractable. Populations of *C. eucypris* were collected, along with other rock pool meiofauna, water, and detritus, from Prospect Point, Nova Scotia, Canada (43°29'26"N, 65°43'10"W), in September 2009. Communities were transported to the laboratory and transferred to twenty 1300 ml transparent plastic containers (12.5 x 12.5 x 20 cm, hereafter referred to as microcosms). Microcosms were placed in a controlled laboratory room to maintain a constant microcosm water temperature of 23 ºC and were exposed to full spectrum light following a regime of 12 hours of day light (07:00 to 19:00). Microcosms were kept under the most natural biological conditions possible: communities persisted in an environment composed of rock pool water and organic substrate. No food or nutrients were added as laboratory rock pool communities are self-sustaining with sufficient light.

3.3.2 Experimental design

The microcosms were composed of naturally occurring populations of the target species *C. eucypris* and other meiofaunal species including a herbivorous cladoceran, *Daphnia magna,* a carnivore, *Microcyclops varicans,* and two species of herbivore/detrititoves, *Alona* sp. and *Alonella* sp. Other biotic components of the community that were present but not identified to species level include phytoplankton, periphyton, protists, bacteria, and detritus. The initial relative abundance of non-target

species differed between microcosms whereas initial relative abundance of *C. eucypris* was identical across microcosms. Initial abundances of *C. eucypris* were established by concentrating and filtering 1300 ml of rock pool water using a 63 μm sieve net. We then counted the number of *C. eucypris* individuals in each microcosm and added or removed individuals to reach target abundances of six adults.

The manipulation of mortality frequency started in October 2009 and was performed on fifteen populations of *C. eucypris.* When populations reached abundances of two individuals (quasi-extinction), we stopped performing removals. Given the small range of values between extinction and mean population size, we selected a quasi-extinction value of two individuals which corresponds to a slightly lower value than half the initial population size but remains greater than one. The dynamics of recovery were then monitored until March 2010 for a total experimental time of 152 days. The time period allowed for recovery was always as long or longer than the decline to quasi-extinction. The frequency of mortality was manipulated by removing 20% of the total number of *C. eucypris* individuals (not considering maturity stage) present in each microcosm every ¼, ½, and total generation time (hereafter referred to as high, medium, and low mortality frequency). For *C. eucypris*, this corresponds to removals conducted every 4, 8, and 16 days. Given that fishing pressure has been shown to reduce a population up to 100% within 15 years, which corresponds to approximately one to two fish generations (Hutchings 2000), our choice of a maximum mortality pressure of 60% within one generation is within the range of mortality pressures currently occurring in natural systems. Each mortality frequency treatment was replicated five times for a total of n=15 microcosms. Five populations were not subjected to mortality for a total of n=5 control microcosms.

To estimate how many *C. eucypris* individuals were to be removed from each microcosm, we counted the total number of individuals present in the microcosm and then removed 20% of the total population size. To perform the removal, we concentrated and filtered each microcosm using a 63 μm sieve net. The concentrated community was then observed through a dissection microscope. The total population size of *C. eucypris*

was counted and 20% of the total population size was removed every ¼, ½, and total generation time. Population abundance was monitored every ¼, ½, and total generation time for the highest frequency treatment while population abundance was monitored every ½ and total generation time for the remaining treatments and the control.

3.3.3 Statistical analysis

We measured recovery time from when the population declined to two or fewer individuals to 1) the time the population exceeded the carrying capacity of the decline phase (Watanabe *et al.* 2005), 2) the time the population exceeded the maximum abundance of the decline phase, and 3) three generations following quasi-extinction (Fig. 3.1). The three generation time criteria is commonly used in species at risk and recovery plans because it allows the decline or recovery status to be adjusted to a species' life history and because it is considered long enough to be biologically meaningful for conservation actions (IUCN 2010, National Marine Fisheries Service 2010). Carrying capacity was calculated as the mean population size from the start of the experiment to the time of quasi-extinction. Because a number of other measures could have been used to define carrying capacity, such as median population size (Griffen and Drake 2009), we performed analyses using both the mean and median population size. Results did not differ between analyses, thus we only report the results using mean population size.

To determine whether a population's recovery status three generations after quasi-extinction adequately represents a population's long-term recovery status, we first assessed population abundance three generations after quasi-extinction (hereafter referred to as the three generation time threshold; Fig. 3.1) and measured the mean growth rate from quasi-extinction to the three generation time threshold. We then evaluated recovery status when possible six generations following quasi-extinction (hereafter referred to as the six generation time threshold, n=10). We assessed population abundance at the six generation time threshold and measured the mean growth rate from the three to the six generation time thresholds (Fig. 3.1). Mean population growth rate was calculated as the

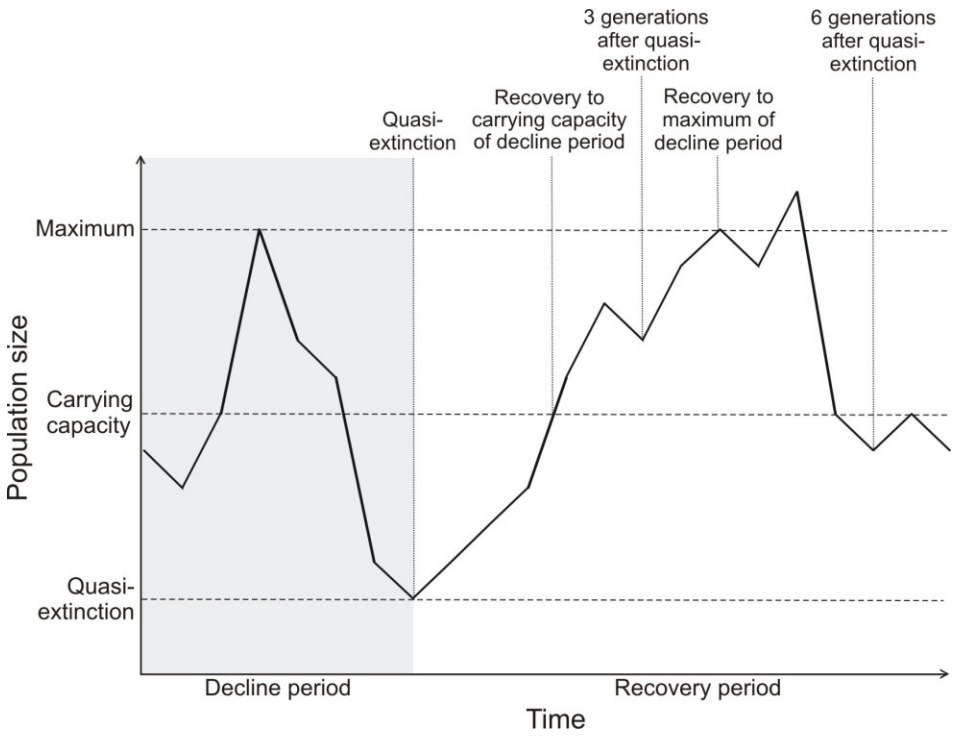


Figure 3.1. Conceptual model of the population dynamics of decline to quasi-extinction (shaded area) and recovery from quasi-extinction. The time when population reaches quasi-extinction population size, the time when population recovers to the carrying capacity of the decline period, the time when population recovers to the maximum abundance of the decline period, the three generation time threshold, and the six generation time threshold are shown.

ratio of population abundance over consecutive time steps as N_{t+1}/N_t. To determine whether there was a relationship between population size at the three and six generation time thresholds, we used a linear regression model with population abundance after six generations as the dependent variable and population abundance after three generations as the independent variable. We also assessed whether the growth rate from quasi-extinction to the three generation time threshold was predictive of the growth rate following the three generation time threshold. To do so, we used a linear regression model with mean growth rate from the three to the six generation time thresholds as the dependent variable and the mean growth rate from quasi-extinction to the three generation time threshold as the independent variable. Relationships were considered significant when $p<0.05$. Normality and homoscedasticity were evaluated using the Shapiro-Wilk's and Levene's test respectively.

To determine whether mortality frequency of the decline period influenced the dynamics of population recovery, we used general linear models (GLM) with recovery time and growth rate to carrying capacity of the decline period, recovery time and growth rate to maximum abundance of the decline period (Fig. 3.1) as well as the mean growth rate, temporal variability, mean population abundance, and maximum population abundance of the recovery period as dependent variables and mortality frequency (high, medium, and low) as the categorical variable. Mean recovery abundance and maximum recovery abundance were transformed to their logarithm to meet the GLM assumption of normality. Temporal variability in abundance was calculated as the coefficient of variation (CV = standard deviation/mean). Given the important declines observed later in the recovery process (Fig. 3.2), we were also interested in testing whether mortality frequency affected the magnitude of the decline following recovery maximum abundance. We first compared the regression slope of decline from recovery maximum abundance to subsequent minimum abundance using a GLM with regression slope value of recovery period declines as the dependent variable and mortality frequency as the categorical variable. We also calculated the magnitude of the recovery period decline as the percent decline from maximum recovery abundance to subsequent minimum abundance.

To determine whether the rate of population recovery was proportional to the rate of population collapse, we examined whether the regression slopes of the decline from maximum abundance to quasi-extinction were similar to the regression slopes of the increase from quasi-extinction to recovery to the decline period maximum abundance using a t-test for dependent samples. Statistica 7.0 was used for all analyses (StatSoft 2002).

One population under low mortality frequency did not decline to quasi-extinction. We therefore excluded this population from the analysis as it was impossible to calculate measures of duration, mean growth rate, and temporal variability related to quasi-extinction. Five high mortality frequency populations, five medium mortality frequency populations, and four low mortality frequency populations were analyzed (n=14). Times series of all populations are shown in Figure 3.2 (n=20).

3.4 Results

3.4.1 Recovery dynamics

All populations recovered to the carrying capacity abundance of the decline period (mean 6 ± 3 S. D. individuals) and to the maximum abundance of the decline period (mean 12 ± 7 S. D. individuals) within two (mean 37 ± 7 S. D. days) to three generations (mean 50 ± 6 S. D. days) respectively. Following the cessation of external mortality, populations recovered to an averaged population size 71% greater than the maximum population size reached during the decline phase. However, after reaching recovery maximum abundances, populations again declined, reaching abundances 67% smaller than recovery maximum abundances. Two of these populations collapsed to the quasi-extinction level while four additional populations declined to the carrying capacity level (Fig. 3.2).

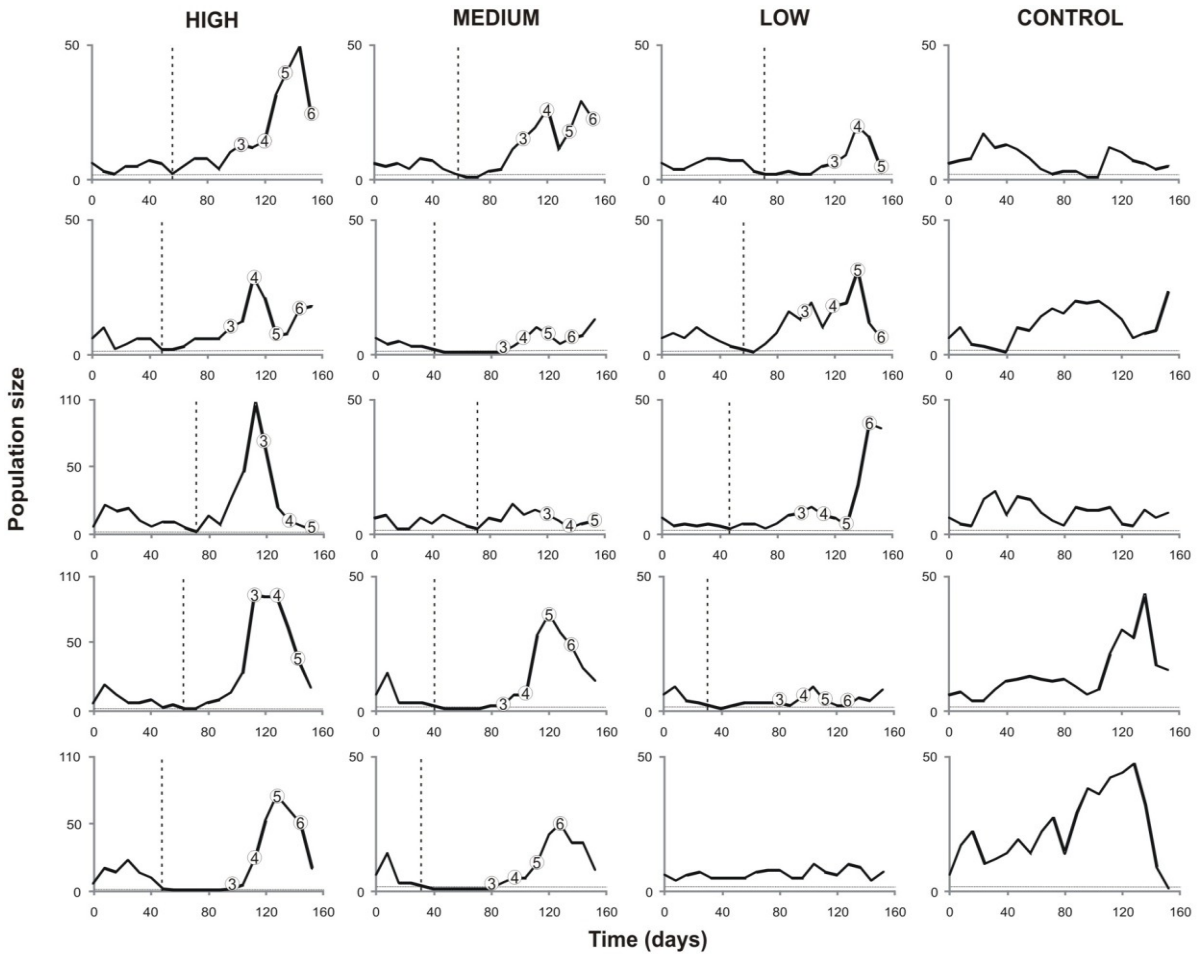


Figure 3.2. Population dynamics of *C. eucypris* decline to quasi-extinction under different mortality frequencies and recovery from quasi-extinction. Time series of untransformed abundances are shown across high (¼ generation time), medium (½ generation time), and low mortality (total generation time) frequency treatments from the start of the experiment to quasi-extinction (dashed vertical line). Quasi-extinction level is indicated by a dashed horizontal line at n=2. Population status after three, four, five, and six generations after quasi-extinction are circled. The experiment lasted for a total of 152 days. Only measures of ½ and total generation time for mortality treatment and control populations are shown here. Populations which did not proceed to quasi-extinction (low mortality frequency, n=1) and controls are included in the figure but were not included in the analysis as it was impossible to calculate decline and recovery measures to and from quasi-extinction. Note that high mortality frequency time series located in the three bottom left panels increased to recovery abundances at least twice as high as the rest of the time series.

Three generations following quasi-extinction, mean population size was 18 individuals, ranging from one to 95 individuals. Six generations following quasi-extinction, mean population size was 22 individuals, ranging from two to 56 individuals. Importantly, population abundance at the three generation time threshold was not significantly predictive of population abundance later in the recovery process (after six generations, r^2=0.04, p=0.574; Fig. 3.3).

Three generations following quasi-extinction, 11 of 14 populations were increasing towards recovery maximum abundances and were experiencing high growth rates. However, six generations following quasi-extinction, nine of the 11 populations that were increasing at the three generation time threshold were showing steep declines in abundance (Fig. 3.2). Population growth from quasi-extinction to the three generation time threshold was negatively related to the mean growth rate following the three generation time threshold (from the three to the six generation time thresholds, r^2=0.44, p=0.037; Fig. 3.3).

3.4.2 Effects of mortality frequency

High mortality frequency led to a significant increase (67%) in the mean population size from the decline to the recovery period (log mean recovery population size, p=0.011) and a significant increase (67%) in the maximum population size attained during the recovery period (log maximum recovery population size, p=0.016; Fig. 3.4). The rate of population decline from recovery maximum abundances was significantly steeper under high mortality frequency (p=0.015; Fig. 3.4). Under high mortality frequency, the average regression slope was -2.28, under medium mortality frequency -0.89, and under low mortality frequency -0.81. This corresponds to populations under high mortality frequency experiencing an average magnitude of decline after recovery maxima of 75% under high mortality frequency (range: 51% to 96%), 62% under medium mortality frequency (range: 24% to 82%), and 60% under low mortality frequency (range: 5% to 80%).

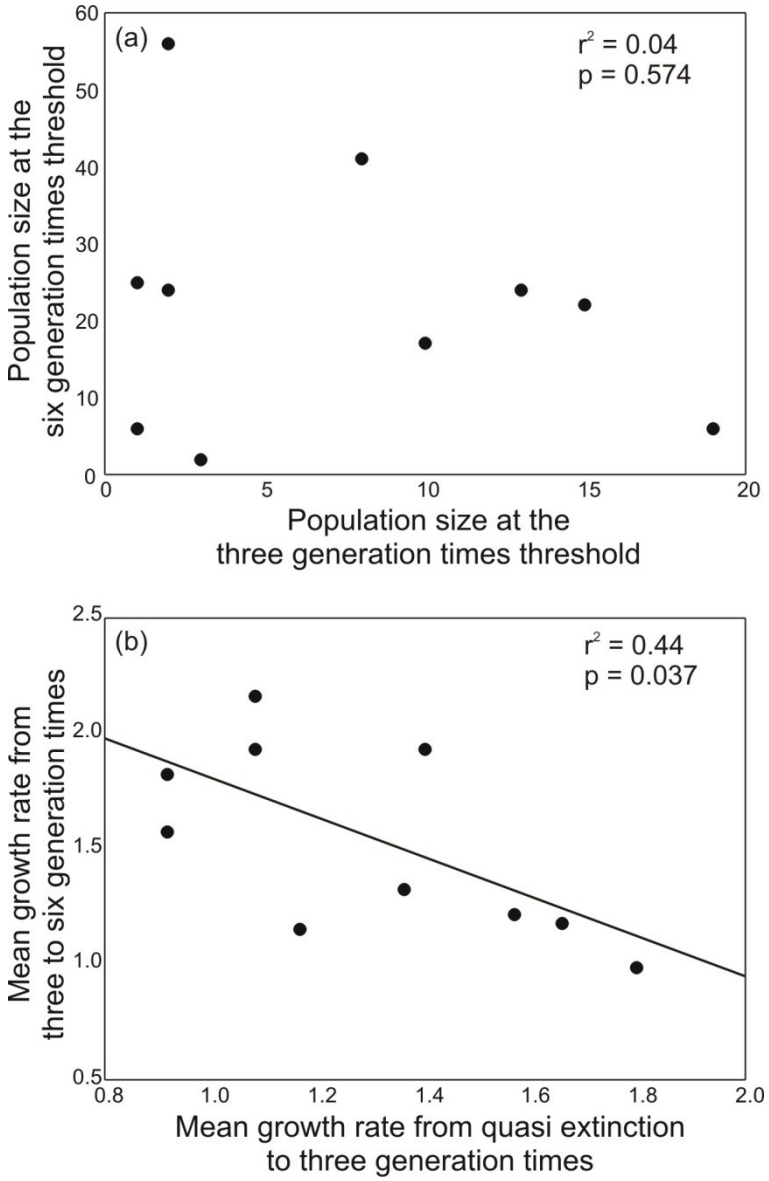


Figure 3.3. Scatter plots showing the relationship between (a) population size three generations after quasi-extinction and population size six generations after quasi-extinction and between (b) mean growth rate from quasi-extinction to the three generation time threshold and mean growth rate between the three and the six generation time thresholds. Mean population growth rate was calculated as the ratio of population abundance (N_{t+1}/N_t).

Mortality frequency during the decline period did not affect recovery time to the carrying capacity (p=0.353) or to the maximum value of the decline period (p=0.419) and the rate of increase to the carrying capacity (p=0.325) or to the maximum value of the decline period (p=0.991). Furthermore, mortality frequency did not affect the mean growth rate (p=0.105) and the temporal variability (p=0.631) of the entire recovery period.

3.4.3 Relationships between the rate of population decline and the rate of population recovery

The absolute rate of population decline to quasi-extinction was similar to the absolute rate of population recovery (p=0.722; Fig. 3.4). Under high mortality frequency, the average slope of decline was -0.38 and the average slope of recovery was 0.48 (p=0.735). Under medium mortality frequency, the average slope of decline was -0.26 and the average slope of recovery was 0.23 (p=0.626). Finally, under low mortality frequency, the average slope of decline -0.18 and the average slope of recovery was 0.2 (p=0.656; Fig. 3.4).

3.5 Discussion

Recovery is the ultimate goal of conservation. All species at risk conservation plans include regulations on the assessment of recovery feasibility and instructions on how to define clear recovery objectives (Species at risk act 2003, National Recovery Working Group 2005, IUCN/SSC 2008, National Marine Fisheries Service 2010). Recovery, however, remains an ill-defined term and its dynamics are relatively unknown. A thorough exploration of the long-term dynamics of population recovery may help to define recovery criteria and may also provide fundamental insights about the long-term viability and behaviour of recovering populations.

The temporal dynamics of *C. eucypris* during a mortality period and a subsequent recovery period show a number of patterns that are of considerable interest. The most

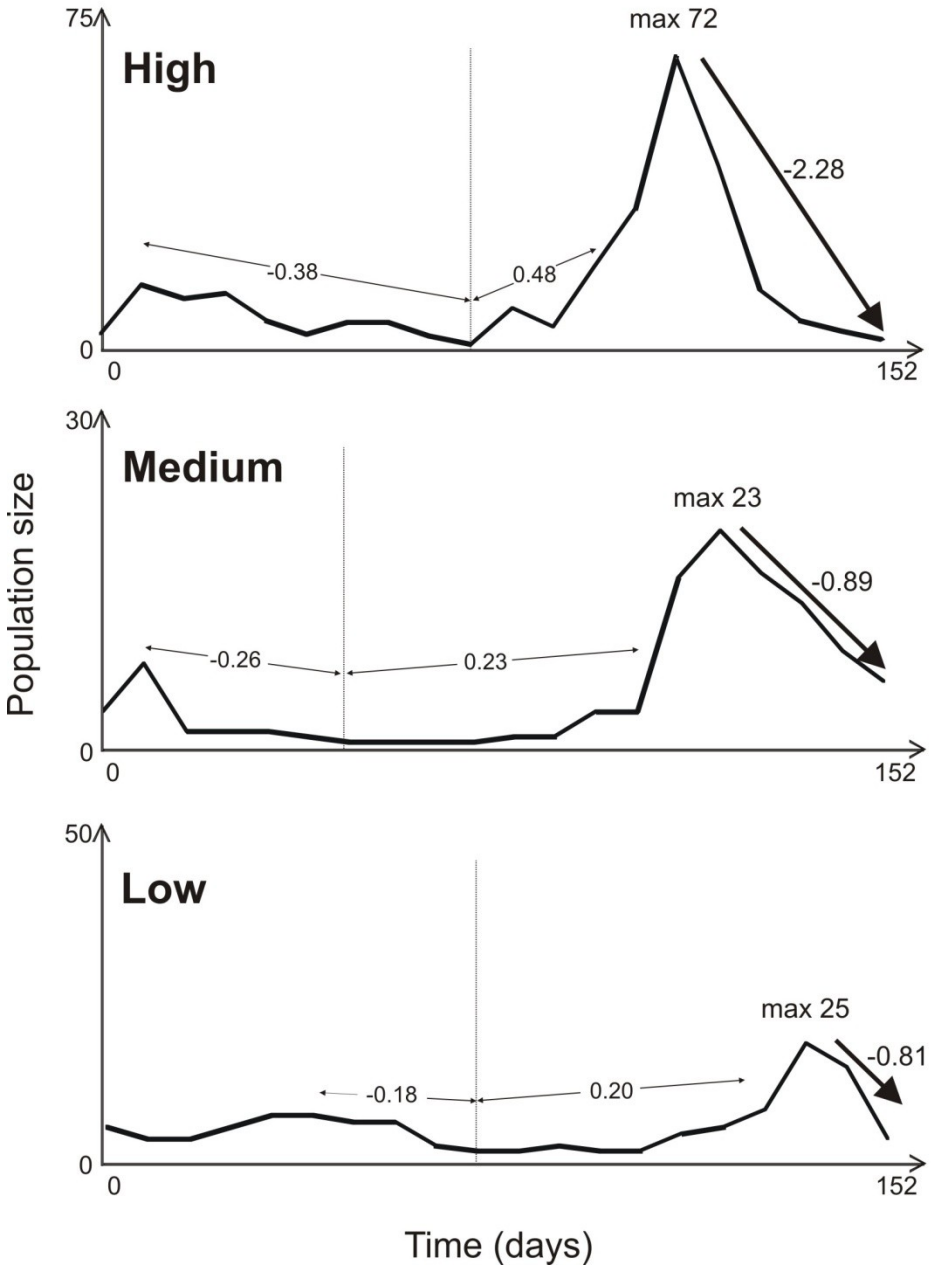


Figure 3.4. Example of *C. eucypris* population time series under high (¼ generation time), medium (½ generation time), and low (total generation time) mortality frequency illustrating the averaged slope of the decline from maximum, averaged slope of recovery to maximum abundance of the decline period, averaged slope from recovery maxima to subsequent minimum, and averaged recovery maximum abundance. The averaged time when populations reached quasi-extinction level (n=2) is indicated by a dashed vertical line.

obvious, and potentially the most important to recovery monitoring and management programmes, is that while populations show rapid short-term recovery to large abundances, they re-collapse to low abundances later in the recovery process (Fig. 3.2). Our results indicate that on average recovering populations reached maximum abundances 71% greater than the maximum population abundances reached during the decline phase. However, following recovery maximum abundance, populations re-declined to levels on average 67% lower than the recovery maximum population size. Two of 14 populations re-collapsed to quasi-extinction and an additional four populations re-collapsed to abundance levels lower than carrying capacity.

Such results have important implications in that recovery criteria such as short-term high population abundances and high reproductive rates should be used with caution. Populations may appear healthy, when indeed, subsequent collapse to low population abundance can occur. Increases in population size following conservation efforts have been observed in a number of case studies (e.g. Hale and Briskie 2009). However, while mortality release remains the main factor leading to such population abundance increases, our results highlight the importance of understanding the role of population responses in the dynamics of recovery. At quasi-extinction per capita resource availability may be high. As a result, a population experiences a high positive growth rate and increases to high abundance. Such signs can certainly be interpreted as signs of recovery; however, once competition for resources comes into play, population growth rate diminishes and a natural population decline ensues. Assessing the recovery status of a species or basing commercial exploitation re-openings on criteria such as increased abundances or high positive reproductive rates in the early stages of recovery is risky as there is a high probability that populations will decline later in the recovery process as a result of density-dependent mechanisms.

Population abundances and mean growth rates three generations after the beginning of the recovery period were not representative of the status of populations later in the recovery process. If recovery had been assessed for a period of three generations after the cessation of mortality, 11 of 14 populations would have been considered as

recovering or having fully recovered as they showed signs of high population abundances as well as positive reproductive rates. However, after a period of six generations after quasi-extinction, our results show that nine of those 11 populations were showing negative population growth rates that steadily led populations to low abundances (Fig. 3.2). Our results also indicate that population' sizes three generations after quasi-extinction were not representative of population' sizes later in the recovery process and that when the growth rate from quasi-extinction to the three generation time threshold was high, the growth rate between the three and the six generation time thresholds was low (Fig. 3.3).

While the three generation time criterion may allow population decline and recovery to be scaled up to a species' life history (IUCN 2010), the choice of a three generation time frame in recovery assessments is questionable. While the generality of our findings may be limited in that they stem from recovery observations of a single small-bodied, short-lived asexual species, they nonetheless warn that assessments of recovery based on the short-term three generation time criterion can be dangerously misleading. Our results further suggest the potential lack of a significant biological basis for the three generation time criterion. We therefore recommend the extension of monitoring recovery programmes to more appropriate temporal scales during the recovery process.

Mortality frequency did not significantly affect the durations of population recovery as well as the mean growth rate and temporal variability of recovering populations. However, we found that populations subjected to high mortality frequency during the decline phase reached greater mean and maximum abundances during the recovery period (Fig. 3.4). Those same populations, however, showed the steepest abundance declines following recovery maxima and the shortest range of magnitudes of decline suggesting that populations subjected to high mortality frequency are more likely to experience a significant decline from recovery maxima than populations subjected to lower levels of mortality frequency. The biological mechanisms responsible for such differences in mean population abundances, maximum population abundances, and

steepness and magnitude of recovery decline across mortality frequency treatments are difficult to assess. However, it has been shown that a population's collapse can significantly alter per capita resource availability and per capita predation risk (Bundy and Fanning 2005), mechanisms which can, in turn, have major impacts on population recovery (Hucke-Gaete et al. 2004). Our understanding of the dynamics of extinction would greatly benefit from further explorations of these recovery mechanisms. For example, it would be useful to determine whether higher mortality frequencies lead to greater per capita resource availability at quasi-extinction which could explain the significant carrying capacity overshoot and subsequent decline observed in *C. eucypris* recovery.

If assessing recovery is a complex endeavour, predicting recovery is also a colossal task. Currently, in order to develop efficient recovery strategies, one must "determine whether the recovery of the listed wildlife species is technically and biologically feasible" (Species At Risk Act 2003). While this task can be partly achieved by examining life history or intrinsic population attributes (Simpfendorfer 2000, Safina et al. 2005, Beketov et al. 2008), our results suggest that it may also be possible to predict the dynamics of population recovery based on the dynamics of population collapse. We found that the rate of decrease to quasi-extinction was inversely proportional to the rate of recovery increase indicating that populations that experience the steepest collapse have the steepest rebound to recovery and that populations that show a shallower decline to quasi-extinction have a gradual increase to recovery (Fig. 3.4). Relationships between patterns of population dynamics during phases of growth and decline have previously been identified (Drake and Griffen 2009, Campbell and Romanuk in revision). The predictive power of such population collapse-recovery connections combined with our current knowledge of the long-term impact of mortality frequency as well as the influence of species life history attributes on recovery may provide a powerful approach to predicting population dynamics of recovery.

3.6 Conclusion

Our study underscores the potentially misleading conclusions of recovery assessments based on criteria such as high abundances and high positive growth rates over short temporal scale (i.e. three generation time criterion). We also demonstrate that while mortality frequency does not impact a population's short-term recovery, it has important negative impacts on a population's long-term recovery. Finally, our study opens the door to a multitude of questions regarding the biological mechanisms responsible for the predictive connection between population dynamics of collapse and population dynamics of recovery. Such findings have wide ranging implications for conservation planning, species management, and policy development.

3.7 Acknowledgements

We thank our volunteers and research assistant Grace Murphy for their help in data collection. We also thank Dave Keith and Stephanie Mogensen for their valuable comments on this manuscript. This work was funded through an NSERC Discovery grant to T.N.R and an NSERC Master's and FQRNT Master's to V.C.

CHAPTER 4

Conclusion: The Study of Extinction and Recovery Dynamics – A Guide to Future Research

Given the current rate of species extinction and the significant effort expended on species recovery, it is imperative to understand the dynamics of population extinction and recovery. The goals of my research were to determine whether there are general patterns associated with species extinction and recovery in response to various levels of mortality pressure and whether it is possible to predict the dynamics of population decline to extinction and the dynamics of population recovery. Here, I summarize the strengths, limitations, results, and implications of my findings.

4.1. An Empirical Basis for the Testing of Extinction and Recovery Hypotheses

My research represents one of the first rigorous experimental sets of observations of the dynamics associated with extinction and recovery over time. My data set is unique and robust in that it illustrates similar dynamics across species with different reproductive strategies and trophic roles. However, the small body sizes, short generation times, and constrained metabolic range (poikilotherms) of the focal species may somewhat limit the generality of my results. Despite this, the observations presented on these model organisms can be used as a baseline to test a number of theoretical hypotheses of population regulation under high environmental stress as well as direct the future development of conservation strategies.

4.2. Patterns of Population Collapse and Recovery Within a Community Context

Among the handful of studies that have experimentally examined the dynamics of population extinction (e.g. Drake and Griffen 2009) and recovery, my thesis represents the first exploration of the dynamics of population extinction within a community context. Species interactions have the potential to hamper or enhance the extinction of a species under environmental stress (Menge and Sutherland 1987, Griffen and Drake 2008). Determining whether a species' extinction is driven solely by the direct effect of external factors or whether it is driven by indirect effect of external factors on inter-specific interactions is therefore crucial to our understanding of extinction dynamics as well as to the development of sound species conservation strategies.

4.3. Extinction – The Negative Influence of Mortality and Connections Between Population Growth and Decline Dynamics

The first part of my research (Chapter 2) yielded three important results for which there exist important theoretical and applied implications. First, I found that high mortality frequency did not affect population increase prior to decline to extinction but instead negatively affected the dynamics of population decline to extinction. Second, my results indicate that while this response was consistent across species, population response to intermediate levels of mortality frequency was not, suggesting that species' life history traits may play a buffering role only against intermediate mortality frequency. Finally, my results indicate that there are strong relationships between a population's growth rate prior to its decline and its dynamics of decline to extinction suggesting that it might be possible to predict the patterns of final decline to extinction from the pre-decline mean growth rate. The field of extinction dynamics is its infancy. It would therefore greatly benefit from further examination of the mechanisms responsible for my observations. I particularly encourage future studies to explore the changes in population age or size structure as well as resource abundance oscillations population size in response to mortality frequency.

4.4. Recovery – The Misleading Conclusions of Short-Term Recovery Measures and Connections Between Population Collapse and Recovery Dynamics

The second part of my research (Chapter 3) yielded three important results. First, I show that high abundance and positive growth rate after three generations, which are typically used to assess the recovery status of a population, can lead to incorrect assessments of species recovery. I demonstrate that this was especially evident for populations under high mortality frequency where recovering populations reached very high maximum abundance but then experienced a significant collapse after recovery maxima. Finally, my results indicate that it may be possible to predict population' recovery dynamics based on the rate at which populations collapse quasi-extinction. Model simulations of recovery dynamics over long temporal scales could greatly advance our understanding of the changes in ecosystem structure occurring following a species' collapse. In particular, simulations would be useful to determine whether the pattern of populations' overshooting their carrying capacity leading to subsequent collapse is regulated by changes in resource availability or changes in predator mortality pressure.

4.5. Conclusion

It was my initial goal and genuine interest to conduct research that advanced both the theoretical study of population extinction and recovery and applied conservation science. The findings described in this thesis provide a strong empirical basis for quantitative theories of extinction and recovery dynamics which, in turn, have implications for the development of sound conservation policies. Moreover, this research encourages a greater understanding of the impact of mortality frequency on the biological mechanisms leading to extinction and recovery, the role of species life history attributes in buffering the effects of environmental perturbations, and the underlying mechanisms of the predictive connection between extinction and recovery dynamics.

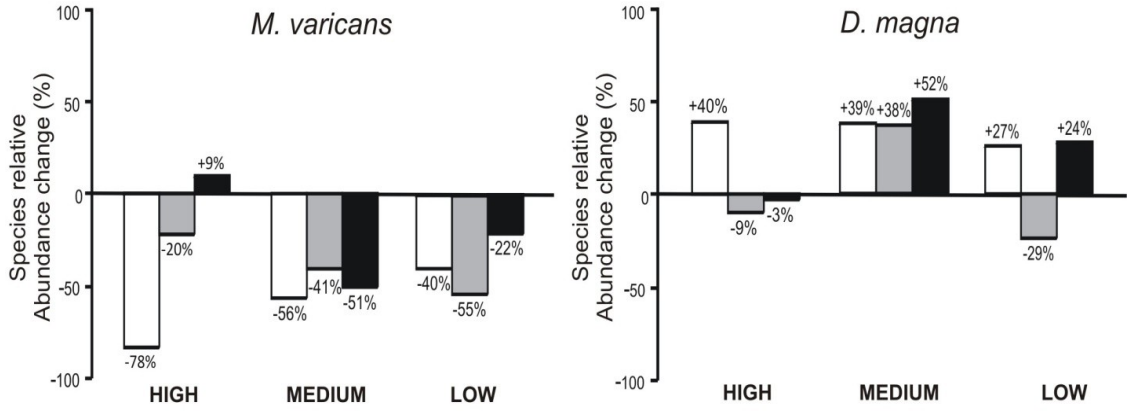
APPENDIX A. General linear model (GLM) results for *D. magna* and *M. varicans* pooled across high, medium, and low mortality frequency treatments (n=24) for all variables. Differences across mortality frequency treatments and target species are considered significant at $p<0.05$ and are indicated by a star symbol *.

Variables		Mortality frequency effect				Target species effect			
		SS	MS	F	p	SS	MS	F	p
Duration	ΔP	1437.90	718.90	3.52	0.0491*	699.90	699.90	3.42	0.0790
	Log ΔA	0.29	0.15	1.29	0.2295	0.66	0.66	5.70	0.0281*
	ΔM	319.50	159.75	0.43	0.6592	259.53	259.53	0.69	0.4155
	ΔC	11.10	5.55	0.02	0.9765	27.23	27.23	0.12	0.7359
	ΔB	2459.42	1229.71	4.05	0.0333*	2945.96	2945.96	9.71	0.0055*
	ΔN	413.51	206.75	0.95	0.4035	107.02	107.02	0.49	0.4912
	Log ΔD	0.27	0.13	5.23	0.0149*	0.21	0.21	8.21	0.0096*
Growth	λΔP	0.02	0.01	0.41	0.6699	0.02	0.02	0.57	0.4611
	λΔA	0.10	0.05	0.41	0.6716	0.26	0.26	2.11	0.1618
	Log λΔM	0.03	0.01	2.13	0.1453	0.00	0.00	0.16	0.6963
	Log λΔC	0.02	0.01	2.36	0.1197	0.00	0.00	0.49	0.4933
	λΔB	0.28	0.14	2.01	0.1607	0.19	0.19	2.68	0.1172
	λΔN	0.26	0.13	2.21	0.1356	0.00	0.00	0.02	0.8996
	λΔD	0.77	0.39	5.55	0.0121*	0.13	0.13	1.84	0.1903
Temporal variability	ΔP_{CV}	0.01	0.00	0.22	0.8049	0.08	0.08	4.95	0.0378*
	ΔA_{CV}	0.08	0.04	1.01	0.3814	0.01	0.01	0.14	0.7169
	ΔM_{CV}	0.00	0.00	0.05	0.9495	0.04	0.04	1.98	0.1743
	ΔC_{CV}	0.04	0.02	1.31	0.2924	0.00	0.00	0.18	0.6756
	Log ΔB_{CV}	0.01	0.00	0.98	0.3921	0.00	0.00	0.03	0.8629
	ΔN_{CV}	0.48	0.24	4.91	0.0184*	0.00	0.00	0.04	0.8481
	ΔD_{CV}	0.48	0.24	6.50	0.0067*	0.02	0.02	0.50	0.4888

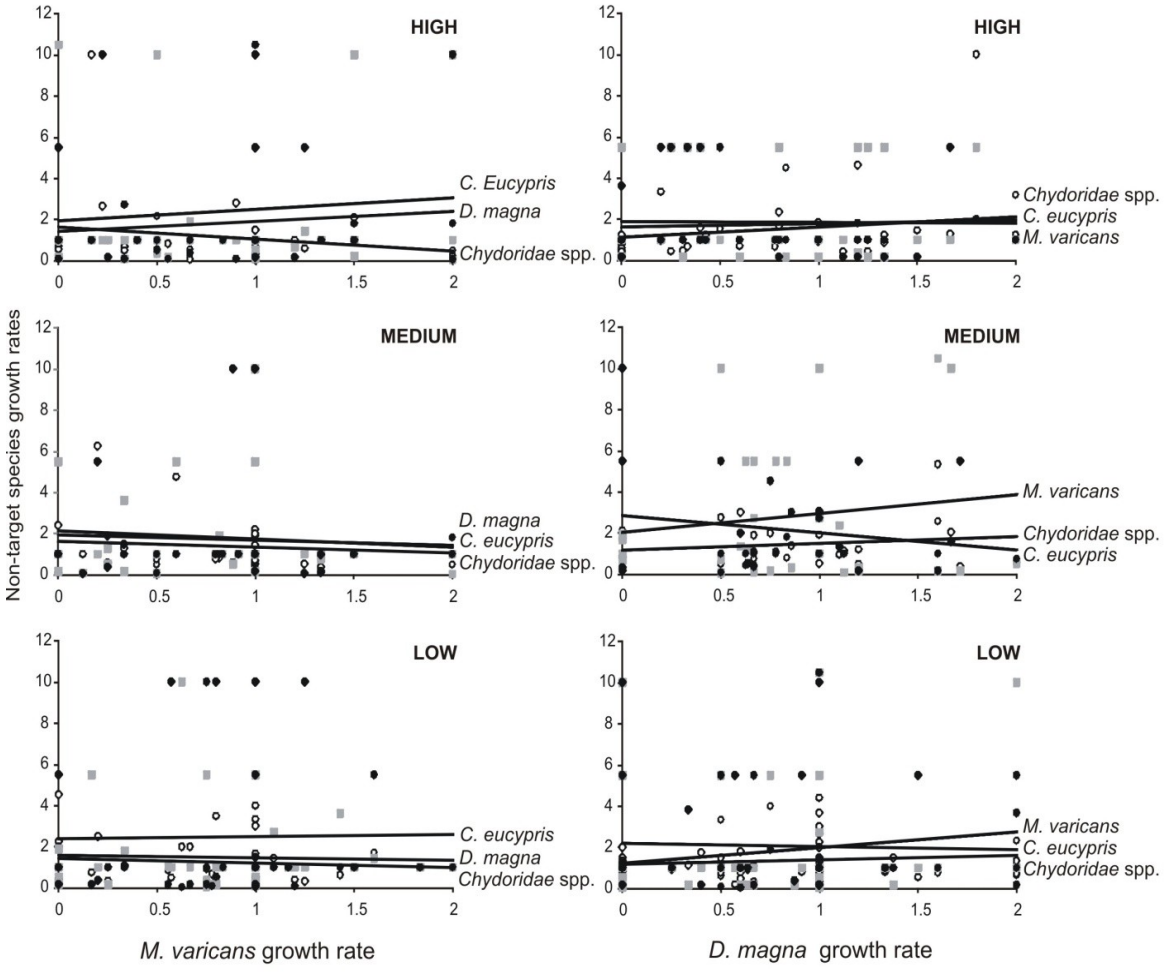
APPENDIX B. Univariate analyses of variance (ANOVA) results indicating differences between mortality frequency treatments for species pooled and differences between species for the time to extinction (ΔP), duration of decline from maximum (ΔB), logarithm of duration of decline from final K (log ΔD), rate of decline from final K (λΔD), temporal variability of final decline (ΔN_{CV}), and temporal variability of decline from final K (ΔD_{CV}). Differences across mortality frequency treatments and target species are considered significant at $p<0.05$ and are indicated by a star symbol *.

Extinction variables	Treatments	n	Sum of squares	Mean square	F ratio	p value
ΔP	High vs. Medium	15	1188.10	1188.10	3.56	0.082
	High vs. Low	18	968.00	968.00	5.35	0.034*
Time to extinction	Medium vs. Low	15	44.10	44.10	0.24	0.629
	Low vs. Control	14	43.21	43.21	0.34	0.569
	High vs. Medium	15	1095.51	1095.50	1.82	0.201
	High vs. Low	18	2357.60	2357.60	8.41	0.01*
ΔB	Medium vs. Low	15	106.71	106.71	0.24	0.63
Duration of decline from maximum	Low vs. Control	14	1.240	1.240	0.01	0.93
	High – *D. magna* vs. *M. varicans*	9	888.89	888.89	2.57	0.153
	Medium – *D. magna* vs. *M. varicans*	6	4160.67	4160.60	44.89	0.003*
	Low – *D. magna* vs. *M. varicans*	9	35.56	35.56	0.22	0.654
	High vs. Medium	15	0.192	0.192	6.18	0.027*
log ΔD	High vs. Low	18	0.212	0.212	6.84	0.019*
	Medium vs. Low	15	0.001	0.001	0.02	0.898
Logarithm of duration of decline from final K	Low vs. Control	14	0.019	0.019	0.59	0.458
	High – *D. magna* vs. *M. varicans*	9	0.040	0.040	1.92	0.209
	Medium – *D. magna* vs. *M. varicans*	6	0.209	0.209	42.76	0.003*
	Low – *D. magna* vs. *M. varicans*	9	0.030	0.030	0.79	0.402
	High vs. Medium	15	0.658	0.658	12.17	0.004*
	High vs. Low	18	0.475	0.475	6.55	0.021*
λΔD	Medium vs. Low	15	0.040	0.040	0.42	0.528
Mean rate of decline from final K	Low vs. Control	14	0.041	0.041	0.53	0.482
	High – *D. magna* vs. *M. varicans*	9	0.002	0.002	0.03	0.857
	Medium – *D. magna* vs. *M. varicans*	6	0.264	0.264	11.23	0.029*
	Low – *D. magna* vs. *M. varicans*	9	0.041	0.041	0.37	0.559
ΔN_{CV}	High vs. Medium	15	0.298	0.298	17.07	<0.001*
	High vs. Low	18	0.042	0.042	2.19	0.162
Temporal variability of final decline	Medium vs. Low	15	0.115	0.115	4.20	0.065
	Low vs. Control	14	0.007	0.007	0.22	0.652
ΔD_{CV}	High vs. Medium	15	0.463	0.463	37.16	<0.001*
	High vs. Low	18	0.144	0.144	3.35	0.086
Temporal variability of decline from final K	Medium vs. Low	15	0.116	0.116	2.30	0.153
	Low vs. Control	14	0.005	0.005	0.08	0.785

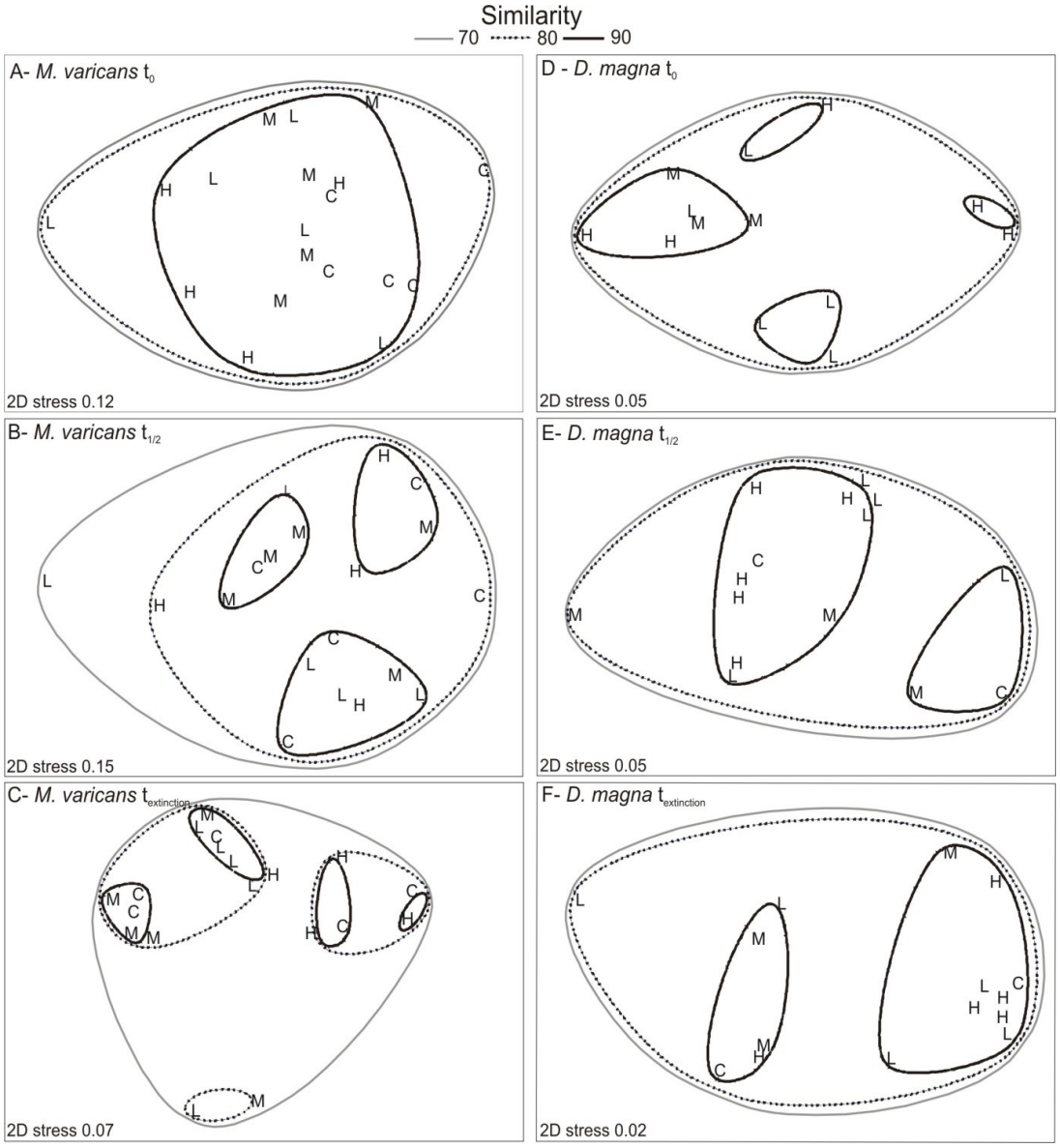
APPENDIX C. Bar plots illustrating the relative change (%) in abundance of *Chydoridae* spp. (white bars), *C. eucypris* (black bars), *D. magna* (gray bars in *M.varicans* plot), and *M. varicans* (gray bars in *D. magna* plot) across mortality frequency treatments for the *D. magna* and *M. varicans* removal experiments. Relative change in abundance was calculated as ((final abundance-initial abundance)/total abundance)*100.



APPENDIX D. Scatter plots showing the cross-correlations between the growth rate of the target species (*D. magna*, *M. varicans*) and the growth rates of the non-target species *Chydoridae* spp. ○, *C. eucypris* ●, *D. magna* ■(for *M. varicans* removal experiment), and *M. varicans* ■ (for *D. magna* removal experiment) across mortality frequency treatments (high, medium, low). Population growth rate was calculated as the ratio of population abundance as N_{t+1}/N_t.



APPENDIX E. Two-dimensional MDS configuration with superimposed clusters at similarity levels of 70% (black bold line), 80% (black dotted line), and 90% (gray line) for *M. varicans* (A) at the start of the experiment, (B) half way to extinction, and (C) at extinction, and for *D. magna* (D) at the start of the experiment, (B) half way to extinction, and (C) at extinction. Plots are based on Bray-Curtis similarity values indicating the similarity of patterns in relative abundance for both target species across mortality frequency treatments. 2D stress below 0.15 indicate an accurate representation of clusters.



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