MECHANISTIC CONCEPTS OF PREDATOR-PREY INTERACTIONS AND THEIR EFFECT ON COMMUNITY DYNAMICS

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December, 2014

A thesis submitted to McGill University in partial fulfillment
of the requirements of the degree of Doctor of Philosophy

DEDICATION

To my friends and family,
thank you for all of your support
in this endeavor and all others.

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ABSTRACT

Trophic interactions, in some form, exist in all natural communities; thus, understanding the factors that allow for the long-term coexistence of predators and their prey is an important first step towards the management of sustainable ecological systems. It is well known that the presence of within-individual and within-species variation for traits that affect trophic interactions can have substantial impacts on community dynamics. However, the generality of such effects has been obscured by disparate sources and trophic locations of traits as well as by the existence of many disparate traits that can exhibit variation. In this thesis, I use mathematical models to compare the effects of distinct phenomena related to within-individual and/or within-species variation. I pay particular attention to their dynamical consequences and abilities to promote the coexistence of trophically linked populations.

First, I assess whether the presence of within-species variation for defense traits can 'rescue' entire communities that are being threatened by environmental change. I compare the potential for evolutionary rescue (through genetic diversity) and plastic rescue (through phenotypic plasticity) by analyzing their differential ability to produce persistence and stable coexistence in model food webs. Furthermore, my analyses consider whether these effects are dependent on the trophic location of variation. I find that within-species variation for defense traits can rescue entire communities. However, both the source and trophic location have significant impacts on the rescue potential of within-species variation. Plasticity promotes persistence and stable coexistence more than genetic diversity; variation at the second highest trophic level promotes stability and persistence more than variation at the autotroph level; and more than variation at two trophic levels.

I then use models of bitrophic systems to investigate how different categories of plastic defenses affect model predictions. In natural systems, there are three major categories of plastic defenses: pre-encounter defenses, post-encounter defenses, and post-consumption defenses. However, most investigations into the effects of plastic defenses (including the second chapter of this thesis) are limited to the dynamical consequences of a single category of defense. Furthermore, the few previous comparative studies that exist have produced conflicting results. I show that plastic defenses can decrease the risk of extinctions due to population oscillations and that clear hierarchies exist at both low and high carrying capacities. Pre-encounter inducible defenses are most likely to promote stable coexistence at low carrying capacities, whereas post-encounter and post-consumption inducible defenses are most likely to promote stable coexistence at high carrying capacities.

Finally, I investigate the dynamical consequences and prevalence of plasticity in predators. The widely used Holling type 2 functional response assumes that the components of predation (i.e. attack rate and handling time) are unaffected by changes in prey density. However, a growing body of empirical and theoretical research suggests that plasticity in predators can allow these components to depend on prey density. In this study, I explore a variety of functional response equations that correspond to situations where prey density-dependent attack rates and/or handling times are likely relevant to natural systems. Using a combination of theory and systematic review of published empirical datasets, I evaluate the prevalence and dynamical implications of these functional responses. Of the 144 datasets that had previously been attributed to the type 2 functional response, AICc analyses indicate that 142 datasets are best fit by consumption equations that incorporate prey density-dependent attack rates and/or handling times. In terms of the community dynamics and stability properties of

systems facing nutrient enrichment, I find that some, but not all, models that incorporate prey density-dependent attack rates and/or handling times are capable of making categorically and fundamentally different predictions than models that incorporate the type 2 functional response. I interpret these findings to mean that predictions of frequent or inevitable destabilization may be overstated. This investigation also highlights the importance of rechecking accepted principles in ecology.

These studies indicate that within-individual and within-species variation for traits that affect trophic interactions may, in general, promote the persistence and stable coexistence of trophically linked populations. However, taken as a whole, this thesis shows that proper evaluation of the dynamical consequences of variation critically depends on its origin, trophic location as well as the specific traits that exhibit variation.

RÉSUMÉ

Les interactions trophiques, de quelque forme, existent dans tous les communautés naturelles; donc, comprendre les facteurs qui permettent la coexistence des prédateurs et leurs proies à long-terme est une première étape vers la gestion de systèmes écologiques durables. Il est bien connu que la présence de variation de trait intra-individuelle et intra-espèce qui affectent les intéractions trophiques peut avoir des impactes considérables sur la dynamique de la communauté. Cependant, la généralité de tels effets a été obscurcie par des sources et locations trophiques de traits disparates et par l'existence de traits qui démontrent ou qui créent de la variation. Dans cette thèse, j'utilise des modèles mathématiques pour comparer les effets de phenomènes distinctes liés à la variation intra-individuelle et/ou intra-espèce. Je prête attention particulièrement à leurs conséquences dynamiques et leurs abilités de promouvoir la coexistence de populations reliées par le réseau trophique.

Premièrement, j'évalue si la présence de variation intra-espèce pour des traits de défense peut 'sauver' d'entières communautés qui sont menacées par des changements environnementaux. Je compare le potentiel de sauvetage par l'évolution (à travers la diversité génétique) et de sauvetage plastique (à travers la plasticité phénotypique) en analysant leur capacité différentielle de produire une stabilité dynamique et persistance dans des réseaux alimentaires modèles. De plus, mes analyses considèrent si ces effets sont dépendent sur la localisation trophique de la variation. Je trouve que la variation intra-espèce pour des traits de défense peut sauver d'entières communautés. Pourtant, la source et la location trophique ont des impactes significatifs sur le potentiel de sauvetage de la variation intra-espèce. La plasticité favorise la stabilité et la persistence plus que la diversité génétique; la variation au deuxième plus

haut niveau trophique favorise la stabilité et persistence plus que la variation au niveau de l'autotrophe, et plus que la variation à deux niveaux trophiques.

Ensuite, j'utilise des modèles avec des systèmes bitrophique pour examiner comment des différentes catégories de défense plastique affectent les prédictions de modèles. Dans des systèmes naturels, il y a trois majeur catégories de défense plastique: des défenses pré-rencontre, des défense post-rencontre, et des défenses post-consommation. Néanmoins, la plupart des examinations dans les effects de défense plastique (incluant le deuxième chapitre de cette thèse) sont limités aux conséquences dynamique d'une seule catégorie de défense. De plus, les quelques études comparatives antérieures qui existent ont produit des résultats contradictoires. Je démontre que les défenses plastiques peuvent réduire les risques d'extinction grâce aux oscillations de populations et que des hiérarchies distinctes existent à une basse et haute capacité limite. Il est plus probable que des défenses pré-rencontres favorisent une stable coexistence à une basse capacité limite, tandis que des défenses post-rencontre et post-consommation ont une tendance de favoriser une stable coexistence à une haute capacité limite.

Finalement, j'ai examiné les conséquences dynamiques et prévélence de plasticité dans les prédateurs. La réponse fonctionnelle Holling type 2, largement utilisée, présume que les composants de prédation (i.e. taux d'attaque et la durée de la manipulation) ne sont pas affectés par des changements dans la densité de proie. Toutefois, un nombre croissant d'études empiriques et théoriques suggère que la plasticité dans les prédateurs peut permettre ces composants de dépendre sur la densité de proie. Dans cette étude, j'explore une variété d'équations de réponse fonctionnelle qui correspond à des situations où le taux d'attaque et/ou la durée de la manipulation dependants de la densité de proie sont pertinent aux systèmes naturels. En utilisant une combinaison de théorie et un examen systématique d'ensemble de données

empirique publiées, j'ai évalué la prévalence et l'implication dynamique de ces réponses fonctionnelles. Parmi les 144 ensembles de données qui ont été attribué à la réponse fonctionnelle de type 2, une analyse de AICc a indiqué que 142 ensembles de données sont mieux ajustées par des équations de consommation qui incluent le taux d'attaque et/ou la durée de la manipulation dependants de la densité de proie. En termes de dynamique de la communauté et de propriétés de stabilité de systèmes faisant face à l'enrichissement en nutrients, je trouve que certains, mais pas tous, modèles qui intègrent le taux d'attaque et/ou la durée de la manipulation dependants de la densité de proie, sont capable de faire de différentes prédictions catégoriques et fondamentales que les modèles que intègre la réponse fonctionnelle type 2. J'interprète que nos résultats veulent dire que les prédictions de déstabilisation fréquente ou inévitable peuvent être surestimé. De plus, ces recherches soulignent l'importance de revérifier des principes acceptés en écologie.

Ces études indiquent que la variation intra-individuelle et intra-expèce de traits qui affectent les interactions trophiques peut, en général, promouvoir la persistence et la coexistence stable de populations reliées par le réseau trophique. Cependant, pris dans son ensemble, cette thèse démontre que l'évaluation appropriée des conséquences dynamiques de variation dépend, de manière cruciale, sur son origine, sa location trophique et les traits spécifiques qui présentent de la variation.

ACKNOWLEDGEMENTS

I thank Gregor Fussmann, my supervisor, for his mentorship over the last six years. His advice, insights, and imperturbable nature have facilitated my growth, both a scientist and as a person. I also thank the two other members of my advisory committee - Andrew Gonzalez and Frederic Guichard-for their invaluable positive and negative feedback. Claire de Mazancourt, although having served on my committee for only a few months before departing McGill, provided pivotal support early in my graduate studies.

Special thanks are due to my co-authors Matthijs Vos and Michael Cortez for their insights and lively discussions. Thanks are due to Ursula Gaedke and her group at the University of Potsdam, Germany, for hosting my research stay and for providing a wonderful academic setting.

My research was made possible through the financial support of the James S. McDonnell Foundation, Quebec Centre for Biodiversity Science (QCBS), Center for Applied Mathematics in Biology and Medicine (CAMBAM), and the Natural Science and Engineering Research Council of Canada (NSERC). I am grateful to Consortium Laval, Universite du Quebec, McGill and Easter Quebec (CLUMEQ) and Compute Canada for the use of the Guillimin Cluster.

I am indebted to the Department of Biology's administrative staff, who facilitated my ability to focus on my research, specially to Ancil Gittens, Lucy Scigliano, Susan Gabe, Luisa Sabaz, Marisa Mastroberardino, Susan Bocti, Carole Smith and Maria Colonna.

I thank my peers in the Fussmann Lab, Biology Department, McGill University, and Montreal for their support, discussions, and friendship. Felipe Dargent, Monica Granados, Romana Limburger, Felipe Perez-Jvostov, Etienne Low-Decarie, Michael Pedruski, Ben

Pyenson, Yony Bresler, Luca Candelori, Allan Brand, Laura Canty, Peter White, Richard Feldman, and Eric Pedersen. I am indebted to Rowshyra Castaneda for the translation of the Abstract. For providing a wonderful weekly escape from the pressures of graduate school, thanks are due to my softball team, Rocket Progress and my ultimate frisbee team, Dirty Deeds, and to the captains who have facilitated and sustained inclusive, fun environments. Claudia Wever, Sami Chaaban, Greg Kramer, Michael Krasser, Michael Collicutt, and Katie Woods.

I am especially grateful to Emily Vogt, who has provided constant support, patience, proof reading, and willingness to go on many adventures! Finally, I thank my brother, Sam, sister, Sarah, and my parents, who have always supported decisions and encouraged my growth.

CONTRIBUTION OF AUTHORS

The research contained in this thesis is predominantly my own. I primarily developed the research questions; designed and performed theoretical experiments; analyzed and interpreted the model output; and wrote the documents contained in this thesis. My co-authors were involved in all or some steps of the research and manuscript writing.

Gregor Fussmann participated in most, if not all, stages of my three research chapters; but most significantly, in the identification of the central questions explored in Chapter 2. Gregor also provided great help structuring certain ideas and sections of all three manuscripts. Michael Cortez was heavily involved with the work presented in Appendices 4.A and 4.B, Specifically, Michael provided significant insights into designing the transformation and discovered valuable analytical solutions for the generalized conditions under which non-saturating functional responses can allow for strong stability. Matthijs Vos provided insights, background knowledge, and assistance in framing the questions addressed in Chapters 3 and 4. My co-authors have given me their permission to include the chapters associated with their names as part of my thesis.

STATEMENT OF ORIGINALITY

The results, analysis, and discussion presented in this thesis contribute to our general knowledge concerning the dynamical consequences of intraindividual and intraspecific variation for traits that affect trophic interactions. While the results presented in this thesis generally corroborate the conclusion that intraspecific and intraindividual variation will promote the stable coexistence of populations, I found that the extent of stable coexistence depends on the source, trophic location, and traits that exhibit or create variation. My work highlights the importance of evaluating stable coexistence in the natural mortality- carrying capacity parameter space. Furthermore, through the framework of weak and strong stability, I have been able to distill complex relationships between stable coexistence and different forms of trait variation. My work has shown that investigations of evolutionary rescue can, and should, consider the rescue of whole communities because there are fundamental similarities between population rescue and community dynamics. Furthermore, I have shown that phenotypic plasticity, in addition to genetic diversity, can rescue populations and communities.

Chapter 2 was published in Philosophical Transactions of the Royal Society B: Biological Sciences, Chapter 3 will soon be submitted to Journal of Animal Ecology, and Chapter 4 will soon be re-submitted to The American Naturalist.

CHAPTER 1

INTRODUCTION

This thesis is focused on the traits that control the interaction of predators and prey; specifically, how allowing these traits to vary within and among conspecific individuals affects the coexistence of trophically linked populations. Throughout this thesis, I use theoretical predator-prey models to explore the boundaries of stable coexistence with respect to increased carrying capacity and natural mortality rate; these model parameters serve as proxies for important, yet extrinsic, forces that threaten many ecological communities. The original research presented in this thesis is divided into three sections. First, I assess how the trophic level and the source of intraspecific variation for anti-predator defensive traits affect persistence and stable coexistence in multi-trophic model communities. I then investigate the dynamical consequences of different forms of inducible anti-predator defense traits. Finally, I use a combination of theory and systematic review of empirical data to look at the prevalence and dynamical consequences of predators that exhibit prey density-dependent attack rates and/or handling times.

1.1 Predator-Prey Interactions and Predictive Ecology

In natural systems, many different biotic and abiotic factors can affect species' abundances and dynamics, such as seasonal temperature fluctuations (Hallett *et al.* 2004), diurnal cycles (Stich & Lampert 1981), disease (Ostfeld & Keesing 2000), intra-and inter-specific competition for resources (Schoener 1983; Chesson 2000), intraspecific competition for mates

(Dobson 1982), predation (Gause, Smaragdova & Witt 1936). Despite the diversity of these forces, from a theoretical ecology standpoint, they can be generalized into two categories: factors that affect births and factors that affect deaths (Lotka 1925; Volterra 1926; Rosenzweig & MacArthur 1963). Predator-prey interactions are particularly interesting because the birth of predators requires the death of prey, and therefore, predation creates a bridge between the aforementioned categories. Furthermore, predators, in some form, exist in virtually all ecological systems (Hairston, Smith & Slobodkin 1960). Thus, it is not surprising that predation has been, and continues to be, a major focus of theoretical ecologists who use models to illuminate key aspects of species interactions (Lotka 1925; Volterra 1926; Rosenzweig & MacArthur 1963; May 1973; McCann, Hastings & Huxel 1998; Fussmann 2008; Abrams 2009). While models have generated great insights (Fussmann *et al.* 2000; Yoshida *et al.* 2003; Yoshida *et al.* 2007; van der Stap *et al.* 2009), the ultimate goal of predictive ecology (i.e. the accurate prediction of dynamics in natural/field conditions), still seems almost impossible (Lawton 1999). Even in simple systems under highly controlled conditions, it is extremely difficult to make accurate predictions of trophic interactions (Fussmann *et al.* 2000; Yoshida *et al.* 2003). It seems that the failures of current models must be due to some overlooked, but crucial, biological phenomena (Roy & Chattopadhyay 2007). Thus, a critical step towards the goals of predictive ecology must be to use theory to identify and resolve the gaps in our knowledge of species interactions. One such underexplored, yet important area, centers on the consequences of phenotype variation within an individual and/or between different individuals (Miner *et al.* 2005; Jones *et al.* 2009). In this thesis, I explore this topic with respect to variation in traits that control the interaction of predators and prey.

1.2 Intraspecific and Intraindividual Variation

Classically, the theoretical ecology framework treats all members of a population as functionally (and mathematically) interchangeable (Miner *et al.* 2005). However, few, if any, naturally occurring systems consist of truly homogenous populations. Instead, the expression of phenotypic traits may vary between different individuals and even within a single individual's lifetime; these phenomena are known as intraspecific and intraindividual variation, respectively (Skulason & Smith 1995; Yamauchi & Miki 2009; Briffa, Bridger & Biro 2013). Ecologists have long been aware that age/stage structure has the capacity to generate intraspecific and intraindividual trait variation, and that such variation can govern population dynamics (Leslie 1945). However, more recently, there has been growing recognition of the profound effects generated by two other sources of trait variation: genetic diversity and adaptive phenotypic plasticity (Shimada, Ishii & Shibao 2010).

Genetic diversity, the occurrence of multiple alleles within a population, can increase the number of phenotypes present in a population. Adaptive phenotypic plasticity occurs when the expression of phenotypic traits is influenced by environmental cues in a way that allows phenotypes to better match environmental conditions, without causing any changes to a population's underlying genetic structure (Via & Lande 1985; Via *et al.* 1995). An important theme of this thesis is the comparative dynamical consequences of genetic diversity vs. adaptive phenotypic plasticity for traits that control the interaction of predators and prey.

Several important distinctions exist between genetic diversity and phenotypic plasticity. First, theory predicts that plasticity can be an exceptionally effective way for populations to adapt to environmental stress (reviewed in (Tollrian & Harvell 1999)) because plasticity can allow faster adaptations than genetic diversity (Chevin, Lande & Mace 2010). Plasticity creates

both intraspecific and intraindividual variation, and therefore, traits may change within a generation. On the other hand, genetic diversity is limited to the production of variation between different individuals, and changes in trait expression are always transgenerational (Cortez 2011; Yamamichi, Yoshida & Sasaki 2011). Nevertheless, genetic diversity is a fundamental requirement for rapid evolution, which can result in phenotypic adaptation, even over fairly short time scales (Bell & Gonzalez 2009; Bell & Gonzalez 2011). Second, for many asexually reproducing populations, plasticity may allow phenotypes that are maladapted to the current environment to reappear when conditions are favorable, whereas phenotypic extinction may be permanent if trait variation comes from genetic diversity (Kovach-Orr & Fussmann 2013). Third, through novel mutations and natural selection, genetic diversity may provide for a wider range of phenotypes than phenotypic plasticity (Barrett & Hendry 2012); furthermore, previous research has shown that genetic diversity can also give rise to wider ranges of phenotypic plasticity (Scheiner 1993).

Despite fundamental differences, both sources of phenotypic expression are highly prevalent (Via & Lande 1985) and can produce rapid phenotypic adaption, which may be especially important given the current period of unprecedented rapid global change. Specifically, many populations and species will be unable to cope 'geographically' with environmental change by adjusting their distributions and must, instead rely on rapid phenotypic adaptation to be 'rescued' from extinction (Chevin, Lande & Mace 2010; Barrett & Hendry 2012). Rescue by phenotypic adaptation occurs when the frequency of traits within a population changes in a way that increases the probability of population persistence. While the role of evolutionary rescue for isolated populations is well known, Kovach-Orr & Fussmann (2013) showed that both genetic diversity and phenotypic plasticity can provide rescue of entire trophic

communities. Rescue was accomplished by allowing for differences in the expression of traits that affect the interaction of predators and prey.

Exploring the consequences of intraspecific and intraindividual variation for defensive and/or offensive traits is especially relevant to our understanding of biological systems because both variation and predator-prey interactions are present in virtually all biotic systems (Hairston, Smith & Slobodkin 1960; Via & Lande 1985). In this thesis, I consider three categories of traits that can affect the interaction of predators and prey: genetic diversity for defense traits in prey (i.e., prey traits that reduce predator feeding ability), phenotypic plasticity for defense traits in prey, and phenotypic plasticity for offense traits in predators (i.e., predator traits that increase predator feeding ability). Due to temporal constraints and because my study of plastic offense traits was largely driven by a systematic review of empirical data sets, none of which contained information on genetic diversity, this thesis does not include an investigation of genetic diversity for offense traits.

Previous research has shown that genetic diversity for defense can alter community dynamics. For example, Yoshida *et al.* (2003) found that traditional models could not account for the long out of phase oscillations observed in their highly controlled chemostat experiments; however, models that incorporated rapid evolution of defense traits in the prey species, *Chlorella vulgaris*, produced predictions that exactly matched experimental observations. Genetic diversity for defense can also allow for increased species' persistence, if genetically distinct populations' abundances fluctuate asynchronously; this phenomenon is a form of compensatory dynamics known as "cryptic dynamics" (Jones & Ellner 2007).

Both theory and experiments have shown that a specific form of phenotypic plasticity, inducible defenses, can significantly enhance the persistence and stable coexistence of small

food webs (Vos et. al. 2004a, van der Stap et. al. 2009). Such stabilizing effects[1] can be attributed to the ability of inducible defenses to create a negative feedback loop between predator density and prey defense: as predators become more abundant, prey exhibit greater defenses, which reduces the likelihood of overexploitation and prey escape (Ramos-Jiliberto & Garay-Narvaez 2007; Ramos-Jiliberto *et al.* 2008; Kovach-Orr & Fussmann 2013).

On the other hand, optimal foraging theory suggests that predators should try to maximize energy intake while minimizing energy output and risk (MacArthur & Pianka 1966). Thus, from an evolutionary perspective, the expression of predator traits should, at least partially, depend on prey (Abrams 1982). Inducible offenses, which allow predator traits to better match environmental conditions, are typically considered in the context of traits that affect predators' ability to consume different prey types (Miner *et al.* 2005; Kopp & Gabriel 2006). For example, the snail *Lacuna variegata* produces sharp teeth when its diet and habitat consist of pure kelp and blunt teeth when its diet includes epiphytes (Padilla 2001). However, predator traits can also depend on the density of a single prey type (Jeschke 2006; Okuyama 2010; Kishida *et al.* 2014). For example, the adult form of the predatory salamander, *Hynobius retardatus* will metamorphose faster, develop a larger gape, and exhibit higher activity levels if, as a larva, it is exposed to high densities of its prey, *Rana pirica* (Kishida *et al.* 2014). Kishida *et al.* (2014) also showed that the induction of these traits results in a 30% decrease in prey survival; therefore, these offenses are likely important factors in natural communities. Nevertheless, previous empirical investigations of resource uptake and mathematical models of predator-prey dynamics, have regularly assumed that predator trait expression is constant and independent of

[1] In this thesis, I adopt a concept of stability that distinguishes between deterministic extinction, cyclic coexistence, and stable coexistence. A detailed introduction follows in section 1.5.

prey density (Murdoch, Briggs & Nisbet 2003; Turchin 2003; Miner *et al.* 2005; Okuyama 2010).

This thesis expands on the aforementioned work and explores the dynamical consequences of genetic diversity for defenses, inducible defenses, and inducible offenses. In my Chapter 2, I compare the effects of inducible defenses and genetic diversity for defense. In Chapter 3, I explore different categories of inducible defense and find that they can have fundamentally different dynamical consequences. In Chapter 4, I combine theory with a systematic review of empirical data to determine the prevalence and dynamical consequences of inducible offenses.

1.3 Descriptions of Predation

Solomon (1949) first proposed the term "functional response" to describe the specific relationship between the number of prey present and the number of prey consumed over a given time interval, per predator. Although early functional response equations could qualitatively describe real predator-prey relationships, they were purely phenomenological (Gause, Smaragdova & Witt 1936; Ivlev 1961; Jassby & Platt 1976). Such phenomenological descriptions eventually gave way to mechanistic models. Mechanistic functional responses incorporate independently measurable components that correspond to specific aspects of the predation process; such as the rate at which predators encounter and successfully attack prey items or the amount of time required to physically manipulate and consume captured prey (Murdoch 1973).

The most commonly used mechanistic functional responses are the Holling type 1, 2, and 3 functional responses (Holling 1959; Holling 1966; Jeschke, Kopp & Tollrian 2002) (Fig. 1.1).

The type 1 functional response $f_1(x)$ equation produces a curve that is monotonic, linear, and saturating (Fig 1.1). This functional response represents the simplest possible relationship between predators and prey: consumption increases linearly with prey density before sharply leveling off at a maximum consumption rate. In terms of community dynamics, the type 1 functional response is widely seen as the most stabilizing of the Holling type functional responses (Turchin 2003). The equation for the increasing region of the type 1 functional response is presented here:

$$f_1(x) = a\,x \qquad (1.1)$$

where x is the density of the prey population. The rate at which predators successfully attack prey, a, is assumed to be constant and independent of prey density (Murdoch 1973); additionally, a determines the slope of the type 1 functional response. The linear nature of this functional response is due to the assumption that processes such as handling or digesting prey can occur without any reduction in time spent on active foraging (Jeschke, Kopp & Tollrian 2002). Although the belief that predators must have a maximum consumption rate is widely accepted (Morin 2011), many dynamical models that use the type 1 functional response, do not incorporate a maximum consumption rate (Turchin 2003). As shown in Chapter 4, the absence of a maximum consumption rate can have meaningful impacts on model predictions; furthermore, this absence is likely responsible for the view that the type 1 functional response is extremely stabilizing[2].

The type 2 functional response $f_2(x)$ equation produces a curve that is monotonic, non-sigmoid, and saturating (Fig 1.1). In other words, consumption eventually approaches a

[2] type 1 functional responses that incorporate a maximum consumption rate would still promote stability more than type 2 functional responses.

maximum but does so at a decelerating rate. The equation for the type 2 functional response is presented here:

$$f_2(x) = \frac{a\,x}{1+a\,h\,x} \qquad (1.2)$$

where *x* and *a* follow the descriptions given above and *h* is the handling time, which is the amount of time required to physically manipulate and consume captured prey before searching for new prey items. In the type 2 functional response, both *a* and *h* are assumed to be constant and independent of prey density (Murdoch 1973). Because the type 2 functional response incorporates handling time, as the rate of consumption increases, predators must spend more total time handling prey and less total time searching for new prey. Furthermore, predators are assumed to be perpetually hungry, but limited by the rate at which they handle prey and the maximum consumption rate is determined by 1/handling time. Additionally, the type 1 and type 2 functional responses are almost identical at very low prey densities (e.g. when x=0, the slope of both curves is equal to *a.* see lower left hand corner of figure 1.1) and/or very low handling times (e.g. when h=0, $f_2(x)$ simplifies to $f_1(x)$). However, in terms of community dynamics, the type 2 functional response is seen the least stabilizing of the Holling type functional responses (Rosenzweig 1971; Murdoch, Briggs & Nisbet 2003; Alexander *et al.* 2012).

The type 3 functional response $f_3(x)$ equation produces a curve that is monotonic, sigmoid, and saturating (Fig 1.1). In other words, as prey density increases from 0, consumption first increases at an increasing rate, then switches to increasing at a decreasing rate, and finally approaches a maximum. The most commonly used formulation for the type 3 functional response that incorporates handling time is:

$$f_3(x) = \frac{a(x)\,x}{1+a(x)\,h\,x} \text{ , where } a(x) = \alpha\,x \qquad (1.3)$$

and where *x* and *h* follow the descriptions given for Equation 1.2 and the attack rate $a(x)$ increases linearly with prey density according to αx. Attack rate may be positively correlated with prey density because as prey density increases, a decreasing proportion of prey are able to utilize refuges; thus, a higher proportion of prey are vulnerable to predation. Additionally, optimal foraging theory suggests that predators may refrain from actively pursuing prey, when those prey are present at low density (MacArthur & Pianka 1966). This is often attributed to narrow-minded predators that are actively pursuing a separate, more abundant, prey species (Heidarian, Fathipour & Kamali 2012); however, it is also possible that predators reduce search effort in an attempt to balance energy expenditure and energy intake (Abrams 1982). Although the type 3 incorporates an increasing attack rate, it also incorporates handling time; therefore, as prey density increases, predators must spend more total time handling prey and less total time searching for new prey. Furthermore, as seen in the type 2 functional response, handling time limitation in the type 3 functional response results in a maximum consumption rate that is equal to 1/handling time. In terms of community dynamics, the type 3 is often seen as stabilizing and may even help prevent the overexploitation and eventual extinction of some species (Alexander *et al.* 2012).

While the type 1, 2, and 3 functional responses appear to have very different functional forms, all three can be described by a single equation:

$$f(x) = \frac{a\,x^p}{1+a\,h\,x^p} \qquad (1.4)$$

where *x, a*, and *h* follow the descriptions given for Equation 1.2 and *p* is a variable that allows for scaling between the type 2 and 3 functional responses (i.e. when $0<p\leq1$, Equation 1.3.4 simplifies to the type 2 functional response; when $p>1$, Equation 1.4 becomes the type 3 functional response). Additionally, when $p=1$ and $h=0$, Equation 1.4 simplifies to the type 1

functional response. Although Equation 1.4 can be transformed into any of the three original Holling type functional responses, it does not address the concerns of many ecologists, who question the biological realism and relevance of the type 1 and 3 functional responses (Abrams & Allison 1982).

Although a large body of empirical work supports the existence of the type 1 functional response (Jeschke 2004), this evidence is often discounted for two reasons. First, many predator prey interactions that are best described by the type 1 functional response, may actually be 'truly' type 2 functional responses; this argument is based on the fact that at low prey densities, the type 1 and 2 functional responses are almost indistinguishable. In fact, one can easily find examples of experiments that produce linearly increasing consumption rates but not saturation (Mohr & Adrian 2000; Kushner & Hovel 2006; Parajulee *et al.* 2006; Long & Whitefleet-Smith 2013). While this is only circumstantial and inconclusive evidence against the type 1 (e.g. the majority of type 2 functional response datasets also fail to test prey densities that cause saturating consumption (Kovach-Orr *et al. unpublished manuscript*), it does cast doubt on type 1 functional responses. Second, only one class of predators, filter feeders, has been shown to exhibit type 1 functional responses (Jeschke 2004). However, less than half of filter feeder species actually exhibit type 1 functional responses (Jeschke 2004). Thus, it seems highly likely that the prevalence and therefore, relevance, of the type 1 functional response has been historically overestimated.

Like the type 1, the type 3 functional response has received support from [an albeit smaller body of] empirical studies (Jeschke 2004). It is widely accepted that sigmoid functional responses (e.g., the type 3) play important roles in complex systems (e.g., multiple prey species, spatial heterogeneity, etc.). However, the type 3 functional response has been criticized as being

unrealistic in more simple systems (i.e. bitrophic food chains in a homogenous environment) (Abrams & Allison 1982). Instead, the type 2 functional response is favored as the most reasonable description of consumption in these systems (Murdoch, Briggs & Nisbet 2003; Turchin 2003). This represents the dominant view that the components of the functional response (i.e. handling time and attack rate) are rigid, unchanging, constants (Murdoch, Briggs & Nisbet 2003; Turchin 2003). Nevertheless, there is growing sentiment that these rigid components are being used for historical reasons or out of convenience, which has lead researchers to question if these components actually are constants (Jeschke 2004; Fussmann, Weithoff & Yoshida 2005; Jeschke 2006; Okuyama 2010; Braza 2012). It is particularly important to address such concerns because terms that can serve equally well as descriptors of species interactions frequently lead to drastically different outcomes in a dynamical context (Fussmann & Blasius 2005). In this thesis, I first investigate the consequences of allowing evolutionary and plastic change to prey traits that control the components of the type 2 functional response. I then combine theoretical analyses with a systematic review of empirical data to determine the prevalence and dynamical consequences of inducible offenses that result in prey density-dependent attack rates and/or handling times.

1.4 Base Model

Ecologists use mathematical models to formalize specific aspects of species interactions- for instance, the uptake of prey by a predator, to predict population dynamics within communities of interacting species, and to gain insight into complex natural processes and systems. Such models are created by making simplifying assumptions that allow for faster, cheaper, and more convenient model analysis. However, extremely simple models, such as the

Lotka-Volterra model of bitrophic food chains, are easy to analyze, but incapable of demonstrating real biological patterns, and therefore, offer only limited insight into real biological systems (Turchin 2003). Thus, the utility of simplifying assumptions must be balanced with the need for models to accurately reflect nature. The simplest predator-prey model that is capable of realistic dynamic behavior, is often believed to be the classical Rosenzweig-MacArthur (R-M) model of bitrophic food chains (Rosenzweig & MacArthur 1963; Turchin 2003; Fussmann & Blasius 2005). The theoretical work presented in this thesis is largely based on the R-M model, which is presented here:

$$\frac{\mathrm{d}\,x}{\mathrm{d}t} = x\,\left(r\left(1-\frac{x}{K}\right)-\frac{a\,y}{1+a\,h\,x}\right) \qquad (1.5a)$$

$$\frac{\mathrm{d}y}{\mathrm{d}t} = y\left(\frac{\varepsilon\,a\,x}{1+a\,h\,x}-m_y\right) \qquad (1.5b)$$

where the prey x, experience a maximum growth rate r, and grow logistically to a carrying capacity K. Predators y, consume prey with a Holling type 2 functional response (i.e. Equation 1.2) (Holling 1966), which has attack rate a and handling time h. Predators convert prey biomass with efficiency ε and experience a natural mortality rate m_y .

From a historical standpoint, the R-M model serves as both the standard for bitrophic analyses and the foundation for more complex community models (McCann & Yodzis 1994; Fussmann & Heber 2002). In addition to being widely used and accepted, the R-M model framework is advantageous because it allows for straightforward evaluation of the relationship between stable coexistence and the extrinsic factors that control prey carrying capacity and natural predator mortality rates (Vos *et al.* 2004).

Carrying capacity and natural mortality rate are particularly relevant for three reasons: First, given the current rate of global environmental change, it is important to explore how

ecological communities may respond to increased environmental stress (Bell & Gonzalez 2009; Barrett & Hendry 2012). Increases in natural mortality rates are the most general population-level effect of increased environmental stress (such as flooding, oil spills, aquatic acidification, pesticides, or any other factors that increase mortality). Carrying capacity is a proxy for nutrient enrichment, a major environmental change in many aquatic ecosystems due to increased fertilizer run-off (Tilman *et al.* 2001). Furthermore, the bottom-up effects of nutrient enrichment can destabilize predator prey dynamics, a phenomenon known as the "paradox of enrichment" (Rosenzweig 1971).

Biologically, destabilization due to enrichment occurs through the mechanism of "prey escape"; whereby top down control is too weak to prevent nutrient enrichment from causing an increase in prey density, which subsequently leads to an unsustainably large predator density; this eventually results in a small predator population that is unable to exert significant top down control on prey (Turchin 2003). Rosenzweig (1971) uncovered this phenomenon using graphical analysis of population zero net-growth isoclines (ZNGI). Such ZNGIs depict how the population densities of predators and prey affect the growth rates of predators and prey. The prey ZNGI approximates an inverted parabola, where predator densities above the prey ZNGI cause a reduction in prey density and predator densities below the prey ZNGI cause an increase in prey density. It is important to note that for any given predator density, increases and decreases in prey density can also potentially affect prey growth. On the other hand, predator density has no effect on the predator ZNGI; predators experience positive growth for all prey densities above the predator ZNGI and negative growth for all prey densities below the predator ZNGI. The intersection of the prey ZNGI and predator ZNGI determines the equilibrium densities of each population. When this intersection occurs to the right of the maximum of the prey ZNGI, the

system will eventually exhibit stable coexistence of predators and prey. However, when the intersection occurs to the left of the maximum of the prey ZNGI, the system will exhibit sustain oscillations or unstable coexistence. One possible explanation is that intersections to the left of the maximum allow for a larger range of predator and prey densities for which predators and prey simultaneously experience positive or simultaneously experience negative growth rates; and that this, in turn, enables predators and prey to more easily "overshoot" their equilibrium densities.

Second, because prey "escape from the control of predators", changes to the background mortality rate of predators can have significant impacts on how systems respond to enrichment (Vos *et al.* 2004). However, this relationship is non-linear and in many cases unintuitive; therefore, it is necessary to evaluate these forces simultaneously (Vos *et al.* 2004; Kovach-Orr & Fussmann 2013).

Third, while the paradox of enrichment was original conceived as the unintuitive result that adding nutrients to a system could result in population oscillations, and therefore, lower minimum densities (Rosenzweig 1971; Roy & Chattopadhyay 2007), researchers have also been puzzled by the significant discrepancy between model predictions and observations of natural systems (Jensen & Ginzburg 2005). Destabilization due to enrichment is readily observed in R-M based models and simple laboratory communities (Fussmann *et al.* 2000; Becks *et al.* 2005), however, more complex natural systems rarely, if ever, exhibit the predicted dynamics (Jensen & Ginzburg 2005; Roy & Chattopadhyay 2007; Becks & Arndt 2008). Instead, natural systems are relatively stable in the face of nutrient enrichment (Murdoch *et al.* 1998; Vos *et al.* 2004; Jensen & Ginzburg 2005).

The mismatch between observations of natural systems and model predictions has received a great deal of attention over the past 40 years (Jensen & Ginzburg 2005) and it is now accepted that discrepancies must be due to certain phenomena which are absent in highly simplified systems (Roy & Chattopadhyay 2007). In fact, many theoretical and experimental model systems that more accurately reflect field observations do so by allowing certain traits to vary within and/or between individuals. However, these advancements have relied on incorporating diverse phenomena, such as spatial heterogeneity (Scheffer & Deboer 1995), inducible defenses (Vos *et al.* 2004), genetic diversity (Mougi & Iwasa 2011), inedible prey (Grover 1995), unpalatable prey (Genkai-Kato & Yamamura 1999), and predator interference (Arditi *et al.* 2004) into the R-M model. Because these phenomena represent distinct biological processes, it is highly likely that they may promote stable coexistence to different degrees and over different ranges of conditions. Therefore, in order to understand natural systems, we must thoroughly explore each of these phenomena. While ecologists have made great strides in some areas, research is only beginning in others; furthermore, we still know very little about their comparative effects. This is especially true in multitrophic models; however, even in bitrophic systems, many phenomena remain underexplored. In thesis, I first compare the effects of different sources (i.e., genetic diversity *vs.* phenotypic plasticity) and trophic locations of defense traits that vary between conspecific individuals. I then use bitrophic models to explore how different categories of plastic defenses (i.e. inducible defenses) affect the stable coexistence of predators and prey. In my final research chapter, I explore the prevalence and dynamical consequences of intraindividual inducible predator offenses.

1.5 Notions of Stability

One of the principal concerns of ecologists is the stability of populations and communities (MacArthur 1955). This interest often stems from the perception that in natural settings, instability is correlated with negative ecosystem effects such as species invasions (Elton 1958), loss of biodiversity (Tilman 1996), extinctions (Inchausti & Halley 2003), and reduced ecosystem productivity (Lehman & Tilman 2000). However, creating a unified definition of "stability" has proven to be extremely difficult because stability can exist at different levels (e.g., population *vs.* community stability) and in many different forms (Ives & Hughes 2002). Previous studies have used stability metrics based on ecosystem productivity (Tilman 1996), the persistence, resilience, and/or coefficient of variation of genotypes and/or species abundances (Huisman & Weissing 1999; McGrady-Steed & Morin 2000; Descamps-Julien & Gonzalez 2005; Rooney *et al.* 2006; Ives & Carpenter 2007), and the range of environmental conditions where communities exhibit different system behaviors (i.e., stable coexistence[3], cyclic coexistence, chaos, and deterministic extinction) (Gonzalez-Olivares & Ramos-Jiliberto 2003; Vos *et al.* 2004; Ramos-Jiliberto, Duarte & Frodden 2008; Ramos-Jiliberto *et al.* 2008).

Conceptually, it is easy to understand that these metrics are not interchangeable and a system can be classified as "stable" by one metric and "unstable" by another. For example, Huisman & Weissing (1999) showed that under some conditions, chaotic population dynamics can lead to the coexistence of many different competing species of phytoplankton. Using definitions based solely on community level persistence or ecosystem productivity, this system would likely be classified as "stable"; however, using definitions that consider system behaviors and/or species abundance, this system would be classified as "unstable". Therefore, one must

[3] In Chapter 2, I use the term "dynamic stability" in place of "stable coexistence". Prior to the publication of Chapter 2, I was not aware of the ambiguity associated with the term "dynamic stability".

cautiously chose the definition of stability that is most appropriate for the system and questions at hand.

In this thesis, I consider stability and instability in terms of the range of environmental conditions that allow communities of predators and prey to exhibit different system behaviors. I pay particular attention to stable coexistence[4] because previous research indicates that in natural settings, temporally stable populations tend to have less of an extinction risk than oscillating populations (Pimm, Jones & Diamond 1988; Inchausti & Halley 2003). I focus on the community level because organisms rarely exist in single, isolated populations; instead, they exist in assemblages of populations and trophic levels. While it is important to understand the theoretical basis for the persistence and stability of single populations (e.g. evolutionary rescue), exploring stability at the community level more realistically reflects the conditions that organisms face in the wild. As detailed in Section 1.4, currently, nutrient enrichment and increased natural mortality are major issues facing ecological systems. Understanding the mechanisms that can allow communities to be robust to a wider range of these environmental stressors is extremely important given our current rate of global environmental change.

1.5.1 Evaluation of Stability (Bifurcation Theory)

In this thesis, I am primarily interested in three system behaviors: deterministic extinction, cyclic coexistence, and stable coexistence. Bifurcations are simply the boundaries between these regions of fundamentally different dynamics (Vos *et al.* 2004). For example, the region of stable coexistence is separated from the region of deterministic predator extinction by the transcritical bifurcation. On the other hand, the region of stable coexistence is separated

[4] In Chapter 2, I also consider "persistence" which I define as the cyclic coexistence of populations that maintain minimum densities significantly above 0.

from the region of cyclic coexistence by the supercritical Hopf bifurcation. Bifurcation diagrams offer a way to visualize how these boundaries change across multiple dimensions.

Recently, there has been growing interest in the relationship between carrying capacity and the breadth of stability, i.e. the range of mortality rates where stable coexistence occurs at a given carrying capacity (Kretzschmar, Nisbet & McCauley 1993; Vos et al. 2004; van Voorn et al. 2008; Kovach-Orr & Fussmann 2013) (Fig. 1.2). The breadth of stability can also be thought of as the distance between certain bifurcations (e.g., the Hopf and the transcritical bifurcations), at a given carrying capacity. Previous research has shown that the specific nature of this relationship can be used to classify models into fundamentally different categories of model stability known as "weak", "strong", and "complete" stability (Kretzschmar, Nisbet & McCauley 1993; van Voorn *et al.* 2008). Weak stability occurs when the breadth of stability decreases, and eventually approaches 0, as carrying capacity approaches infinity (Fig. 1.2 a, b). It is well known that the classic Rosenzweig-MacArthur model predicts weak stability for bitrophic food chains (Rosenzweig 1971; Vos *et al.* 2004; van der Stap, Vos & Mooij 2007; Kovach-Orr & Fussmann 2013).

Strong stability occurs when the breadth of stability is unaffected, or even increases, as carrying capacity is increased (Fig. 1.2 c, d) (Kretzschmar, Nisbet & McCauley 1993; Vos *et al.* 2004; van Voorn *et al.* 2008). Strong stability has been predicted by models that incorporate phenomena such as inedible prey where edible prey maintain a constant percentage of total prey density (Kretzschmar, Nisbet & McCauley 1993), inducible defenses (Vos *et al.* 2004), and invulnerable prey (Abrams & Walters 1996). Complete stability can be seen as the limit case of strong stability, in that complete stability occurs when systems do not exhibit limit cycles anywhere in the mortality-carrying capacity parameter space (Fig. 1.2 e). Complete stability has

been predicted by models that incorporate constant proportion prey refuges (Maynard Smith 1974) but see (McNair 1986); wasteful killing, where predators kill prey with a type 1 functional response, but consume prey with a type 2 functional response (Turchin 2003); and inedible prey, where edible prey maintain a fixed population density and all nutrients above some value are absorbed by the inedible prey (Grover 1995).

While stable coexistence in systems exhibiting complete stability is generally more robust to parameter choice (i.e., cyclic coexistence does not exist in the mortality-carrying capacity parameter space) than stable coexistence in systems that exhibit strong stability (although see (McNair 1986)), both forms of stability allow for stable coexistence as carrying capacity approaches infinity. On the other hand, the distinction between weak and strong/complete stability is of special interest because the classic R-M model predicts weak stability for trophic systems, whereas stable coexistence in natural communities is generally robust to changes in carrying capacity (Murdoch *et al.* 1998). Finding and understanding the mechanisms that may have allowed natural systems to tolerate past and present environmental stress is a crucial first step towards mitigating the consequences of future environmental change.

Bifurcation analysis is a powerful tool that can provide information about ecologically important attributes such as the strength and endpoint of stabilization as well as the parameter ranges for which stabilization occurs. Through bifurcation analysis, previous research has identified phenomena that may promote stable coexistence in natural systems, such as genetic diversity (Yamamichi, Yoshida & Sasaki 2011), reciprocal plasticity (Mougi & Kishida 2009), negative density dependence (Ramos-Jiliberto 2003), in addition to the aforementioned phenomena that produce strong and/or complete stability. In my second and third chapters, I use bifurcation analysis to show that different types of defense traits, as well as different sources and

trophic locations of variation for those traits, can have considerable influence on stable coexistence in the mortality-carrying capacity parameter space. I highlight some of the conditions where these different traits, sources, and trophic locations are most and least effective at promoting stable coexistence. In my fourth chapter, I use bifurcation analysis to show that empirically supported inducible offenses can promote stable coexistence and even strong stability in the mortality-carrying capacity parameter space.

1.6 Chapter Overview

Although we are beginning to understand how genetic diversity and phenotypic plasticity influence community dynamics, we still know very little about the comparative effects of their many possible manifestations. Previous work on the dynamical consequences of intraspecific variation for prey defenses often focused on the effects of a single source of variation and single type of prey defense; while this practice may be suitable for the description of a specific empirical predator-prey interaction, it cannot be employed to establish some degree of generality. Furthermore, the few studies that have directly investigated the comparative effects of genetic diversity and phenotypic plasticity have focused on variation at a single level in simple food chains and webs. On the other hand, inducible offenses have received far less attention and while theory predicts that such offenses should be relatively common, no study has used empirical data to address the general prevalence of this phenomenon. Throughout this thesis, I investigate the dynamical consequences of intraindividual and intraspecific variation for traits that affect the interaction of predators and prey. Specifically, I compare trophically linked communities that have homogenous populations *vs.* communities consisting of populations that possess: different sources and trophic locations of variation for prey defenses (Chapter 2); a

single source of variation (phenotypic plasticity) but different categories of prey defenses (Chapter 3); inducible offenses (Chapter 4). Additionally, in Chapter 4, I use empirical data taken from a wide range of systems to investigate the prevalence of inducible offenses.

In Chapter 2, I compare the potential for evolutionary rescue (through genetic diversity) and plastic rescue (through phenotypic plasticity) by analyzing their differential ability to produce persistence and stable coexistence in model food webs. I also evaluate how the trophic location of variation affects rescue potential. I find that plasticity promotes stable coexistence and persistence more than genetic diversity. Furthermore, stable coexistence of genetically diverse populations occurs for a limited subset of defense-cost combinations. Contrary to the conclusions of previous studies, variation at the second highest trophic level promotes stable coexistence and persistence more than variation at the lowest trophic level; and more than variation at two trophic levels. Our study shows that proper evaluation of the rescue potential of intraspecific variation critically depends on its origin and trophic location.

In Chapter 3, I assess whether different categories of inducible defenses produce disparate dynamical consequences for bitrophic communities. This study is a result of the emergence of different mathematical formulations of inducible defenses that reflect different mechanistic views about how defenses affect predator-prey interactions. Although two previous studies had partially addressed this topic, they produced conflicting results. Nevertheless, our results indicate predictable, definitive, and consistent relationships between the category of inducible defense, the effectiveness of defense, and stable coexistence in the mortality-carrying capacity parameter space. Furthermore, we show that inducible defenses can decrease the risk of extinctions due to population oscillations and that clear hierarchies exist at both low and high carrying capacities. This study does not address the dynamical consequences of genetic diversity

for different categories of defenses. As I show in Chapter 2, for most cost-benefit combinations, genetic diversity for defense results in the cyclic coexistence of predators and prey; however, for some cost-benefit combinations, genetic diversity can produce high levels of stable coexistence. Therefore, a true comparison must test many different cost-benefit combinations; this was determined to be too computationally and temporally expensive.

In Chapter 4, I explore inducible offenses using a variety of functional response equations that incorporate prey density-dependent attack rates and/or handling times. Using a combination of theory and systematic review of published empirical datasets, I evaluate the prevalence and dynamical implications of these functional responses. Of the 144 datasets that had previously been attributed to the type 2 functional response (a model that does not incorporate inducible offenses), AICc analyses indicate that 142 datasets are best fit by consumption equations that incorporate prey density-dependent attack rates and/or handling times. In terms of the community dynamics and stability properties of systems facing nutrient enrichment, I find that some, but not all, models that incorporate prey density-dependent attack rates and/or handling times are capable of making categorically and fundamentally different predictions than models that incorporate the type 2 functional response. I interpret these findings to mean that predictions of frequent or inevitable destabilization may be overstated. This study also highlights the importance of rechecking accepted principles in ecology.

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1.8 Figures

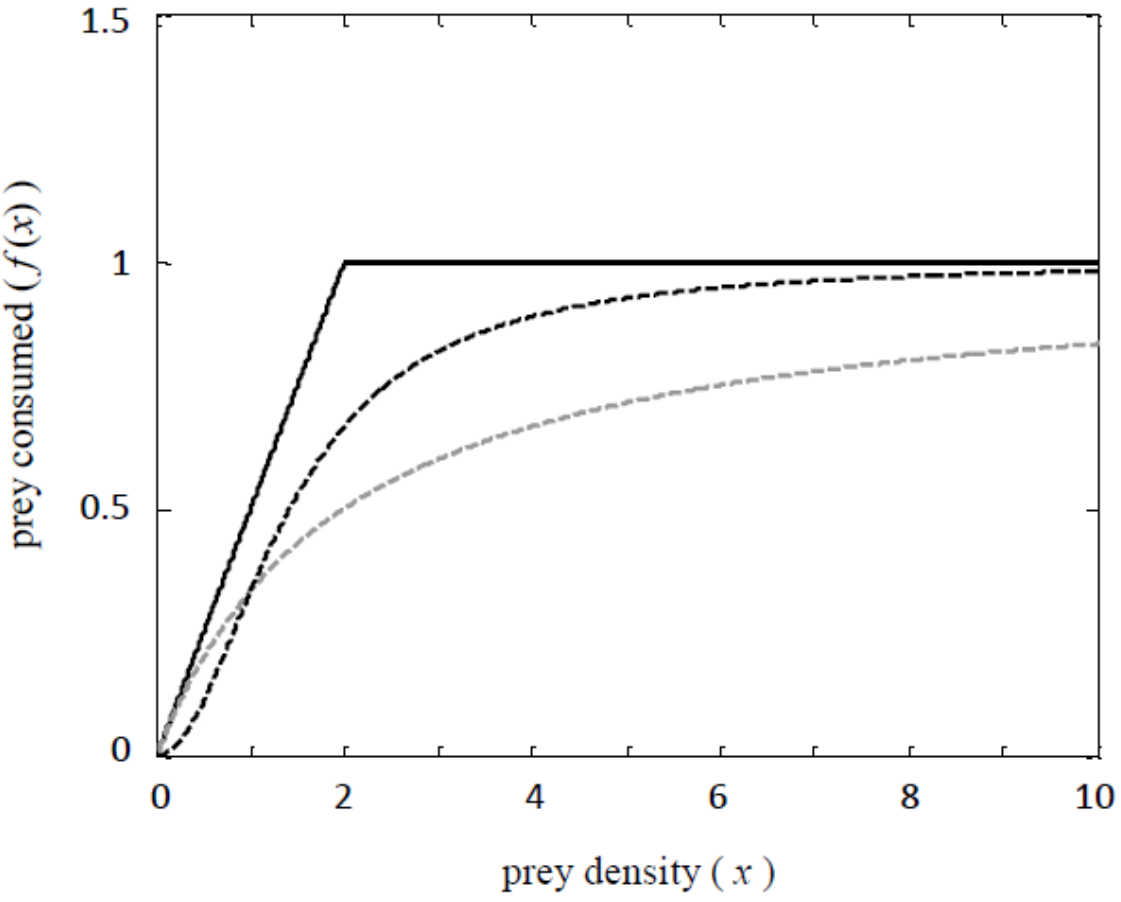

Figure 1.1. The Holling type 1 (*black-solid line*), 2 (*grey-dashed curve*), and 3 (*black-dashed curve*) functional responses. Prey density (x) is shown on the x-axis. Consumption ($f(x)$) is shown on the y-axis. Parameterizations are as follows a=0.5, h=1.

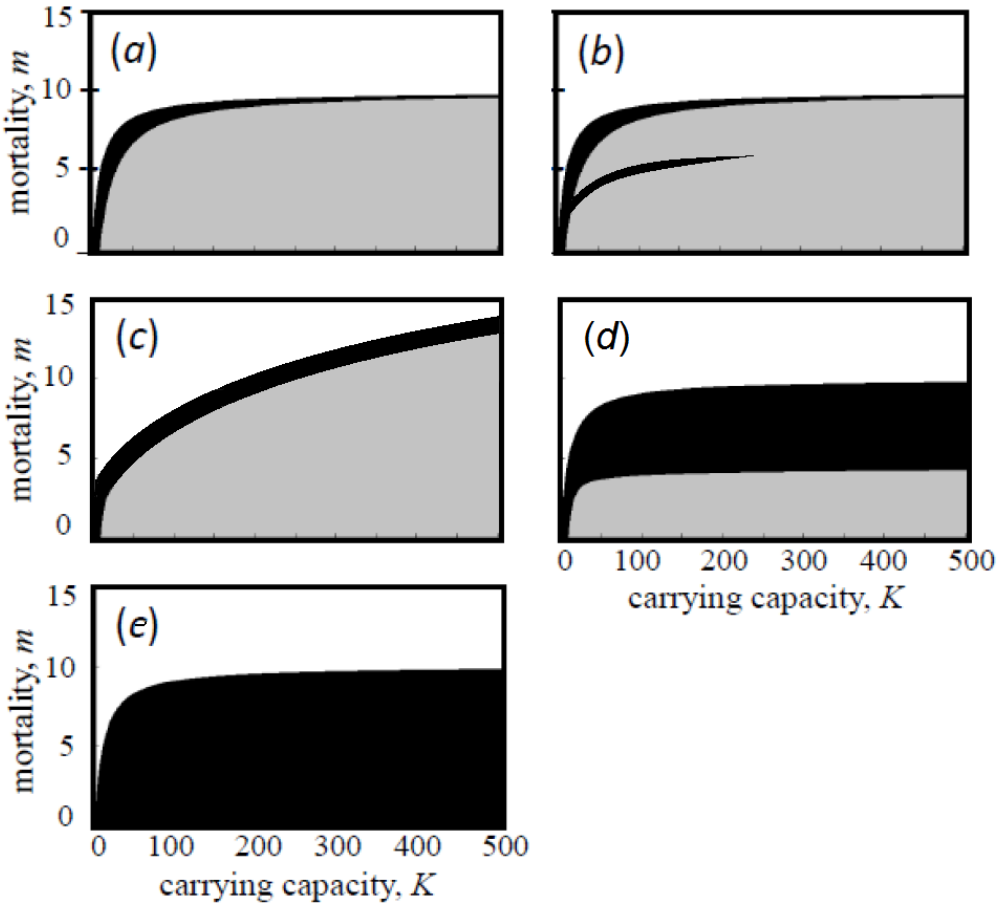

Figure 1.2. Bifurcation diagrams of a bitrophic food web. Carrying capacity (*K*) is shown on the *x*-axis, and mortality (m_y) is shown on the *y*-axis. The *white area* is the region of deterministic predator extinction. The *black area* is the region of stable coexistence; it is separated from the region of predator extinction by the transcritical bifurcation; above the transcritical bifurcation, the predator cannot maintain positive growth. The *grey area* is the region of cyclic coexistence (i.e. limit cycles); it is separated from the region of stable coexistence by the Hopf bifurcation. The range of mortality rates where stable coexistence occurs (i.e. the size of the *black area*) at a given carrying capacity is known as the "breadth of stability". (*a*) Weak stability: as carrying capacity approaches infinity, the breadth of stability approaches 0. (*b*) Weak stability: the presence of genetic diversity has lead to a second band of

stable coexistence: however, as carrying capacity approaches infinity, the breadth of stability for both bands approaches 0. (*c*) Strong stability: as carrying capacity approaches infinity, the breadth of stability approaches a value greater than 0, however, limit cycles exist for some parameter values. Furthermore, for any given mortality rate, increasing carrying capacity will eventually cause sustained oscillations. (*d*) Strong stability: like figure 1.2*c*, the breadth of stability approaches a value greater than 0; however, for some mortality rates, increasing carrying capacity cannot cause sustained population oscillations. (*e*) Complete stability: limit cycles are absent for all combinations of carrying capacity and mortality.

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CHAPTER 2

EVOLUTIONARY AND PLASTICITY RESCUE IN MULTITROPHIC MODEL COMMUNITIES

Caolan Kovach-Orr & Gregor F. Fussmann (2013). Evolutionary and plasticity rescue in multitrophic model communities. Philosophical Transactions of the Royal Society B: Biological Sciences. (368) 1610. DOI: 10.1098/rstb.2012.0084

Note: Chapter 2 was originally published in a British scientific journal; in order to maintain continuity with that publication, British spellings are used throughout this chapter.

2.1 Abstract

Under changing environmental conditions, intraspecific variation can potentially rescue populations from extinction. There are two principal sources of variation that may ultimately lead to population rescue: genetic diversity and phenotypic plasticity. We compared the potential for evolutionary rescue (through genetic diversity) and plastic rescue (through phenotypic plasticity) by analysing their differential ability to produce dynamical stability and persistence in model food webs. We also evaluated how rescue is affected by the trophic location of variation. We tested the following hypotheses: (***i***) Plastic communities are more likely to exhibit stability and persistence than communities in which genetic diversity provides the same range of traits. (***ii***) Variation at the lowest trophic level promotes stability and persistence more than variation at higher levels. (***iii***) Communities with variation at two levels have greater probability of stability and persistence than communities with variation at only one level. We found that (***i***) plasticity promotes stability and persistence more than genetic diversity, that (***ii***) variation at the second highest trophic level promotes stability and persistence more than variation at the autotroph level and (***iii***) more than variation at two trophic levels. Our study shows that proper evaluation of the rescue potential of intraspecific variation critically depends on its origin and trophic location.

Key words: Evolutionary rescue, phenotypic plasticity, genetic diversity, inducible defense, rapid evolution, multitrophic communities.

2.2 Introduction

In the current period of unprecedented rapid global change, ecological communities are experiencing abrupt and sustained environmental stress. Many populations and species will be unable to cope "geographically" with environmental change by adjusting their distributions and must rely on rapid phenotypic adaptation to be rescued from extinction (Chevin, Lande & Mace 2010; Barrett & Hendry 2012). Rescue by phenotypic adaptation occurs when the frequency of traits in a population changes in a way that increases the probability of population persistence. In nature, rapid phenotypic change can be based on two principal sources of trait variation: genetic diversity and phenotypic plasticity (Shimada, Ishii & Shibao 2010). In the present theoretical study, we compare these two sources of intraspecific variability for their differential ability to preserve populations that face changing environmental conditions and interact with one another in food webs.

Theory predicts that rapid evolution can rescue populations that are threatened by significant and sustained environmental change (Gomulkiewicz & Holt 1995). In this evolutionary rescue scenario, natural selection changes allele frequencies in populations so that phenotypes are better adapted to novel conditions, thereby maintaining or restoring a positive population growth rate. Genetic diversity – the raw material for natural selection and, ultimately, evolutionary rescue – arises either from pre-existing variation or de novo mutations that occur during rescue. Evolution must be rapid because allele frequencies need to be re-adjusted before the expression of maladaptive alleles leads to the demise of the population. A recent experimental study using yeast showed that evolutionary rescue could be accomplished over as few as 25 generations (Bell & Gonzalez 2009; Bell & Gonzalez 2011).

The second source of intraspecific variation, adaptive phenotypic plasticity, leaves a population's genetic structure unchanged but allows the environment to influence the development of individuals' phenotypes (Via & Lande 1985; Via *et al.* 1995). Plasticity is found in many different ecosystems and at all trophic levels (Skúlason & Smith 1995; Lass & Spaak 2003). Theory predicts that plasticity can be an exceptionally effective way for populations to adapt to environmental stress (Barrett & Hendry 2012); reviewed in (Tollrian & Harvell 1999). Plasticity can occur within a generation, while evolution is always transgenerational; therefore, plasticity will likely allow faster adaptations than genetic diversity (Chevin, Lande & Mace 2010). In many cases, plasticity may be capable of providing most or all of the adaptive trait change required for populations to avoid extinction (Barrett & Hendry 2012).

Our study of "rescue" differs from previous research in two ways. First, many studies on rescue evaluate variability in the traits that explicitly determine an individual's fitness given a specific environmental stress (Bell & Gonzalez 2009; Chevin *et al.* 2013; Kirkpatrick & Peischl 2013; Martin *et al.* 2013). However, in nature, environmental change may often produce several novel stressors, and only a small fraction of populations' variability will be pre-adapted to any of them (Barrett & Hendry 2012). Therefore, we decided to look at how variability, in general, increases the chance of rescue in the face of the general effects of environmental stress, which we believe are an increase in mortality and bottom-up effects of changes in carrying capacity. In particular, we focus on variability in a specific class of traits: those involved in defence against predation. Variation in defensive traits can occur prior to environmental change, and therefore, may contain pre-adaptations that help populations persist following a change (Vos *et al.* 2004a; Vos *et al.* 2004b). Defensive traits can mitigate mortality effects by reducing the impact of predation and, through costs, mitigate the bottom-up effects of nutrient enrichment.

Furthermore, defensive traits occur at multiple levels and have been shown to have very different effects depending on their trophic location (Vos *et al.* 2004a; Vos *et al.* 2004b; Ramos-Jiliberto *et al.* 2008).

Secondly, we consider rescue in the framework of food web dynamics; that is, rather than focussing on the persistence of single, isolated populations or species, we evaluate the potential for rescue of ensembles of populations in specific model food web assemblages. While it is important to establish theoretical and empirical benchmark values of selection strength, critical population size, etc. that are necessary for single population rescue, we feel that our approach more realistically reflects the natural community context under which population rescue commonly happens in the wild. Previous studies have shown that within species diversity affects food web dynamics (Fussmann & Heber 2002; Yoshida *et al.* 2003; Jones *et al.* 2009); here we examine how different sources of this horizontal diversity contribute to the persistence and rescue of whole communities.

We use two criteria to assess the likelihood of rescue: community persistence and dynamic stability. Persistence means that at least one phenotype/genotype from each trophic level maintains positive abundance over the period of model evaluation (i.e., the opposite of extinction). Persistence is in obvious ways related to evolutionary rescue: if, for instance, a community containing plasticity displays persistence over a wider range of parameter values than a community with genetic diversity, we would conclude that phenotypic plasticity enhances the likelihood of survival and that the potential for plastic community rescue is greater than that of evolutionary community rescue. Within the set of persistent dynamics, we use "dynamic stability" as a secondary, more refined criterion for rescue potential. Dynamic stability refers to dynamics characterised by stable equilibria as opposed to intrinsically unstable dynamics

characterised by regular or irregular population oscillations. We believe that the relative frequency of stable equilibria vs. oscillations correlates with the likelihood of community rescue. Our major argument is that in natural settings, temporally stable populations tend to have less of an extinction risk than oscillating populations (Pimm, Jones & Diamond 1988; Inchausti & Halley 2003).

Both genetic diversity and phenotypic plasticity have been shown to increase the likelihood of persistence of trophic communities in theoretical and experimental systems. Most of the evidence derives from predator-prey studies in which prey express traits that provide different degrees of protection from predation. Trait variability arises either from the existence of several genotypes with different defence levels or from a plastic, inducible defence. Genetic diversity can increase population persistence if one or more of the phenotypes are well suited to the new environment (e.g., (Reusch *et al.* 2005)) and/or if genotypes fluctuate asynchronously; the latter phenomenon is a form of compensatory dynamics known as "cryptic dynamics" (Jones & Ellner 2007; Yoshida *et al.* 2007). Similarly, inducible defences can significantly enhance persistence and dynamic stability of small communities when subjected to nutrient and mortality stress (Verschoor, Vos & van der Stap 2004; Vos *et al.* 2004a; van der Stap *et al.* 2009). The impact on community persistence seems to be greatest when plasticity is expressed at the autotroph level (van der Stap *et al.* 2007; Ramos-Jiliberto *et al.* 2008).

Although we are beginning to understand how genetic diversity and plasticity affect community persistence, we know little about their comparative effects and interaction. Studies comparing the effect of the two sources on communities have focused on variation at a single level in simple food chains and webs (Abrams 2009; Cortez 2011; Yamamichi, Yoshida & Sasaki 2011). These studies have found that plasticity is more stabilising than genetic diversity

(Cortez 2011; Yamamichi, Yoshida & Sasaki 2011), that plasticity can increase the maximum mortality that predators can withstand (Abrams 2009) and that plasticity cannot produce all of the dynamics seen with genetic diversity (Cortez 2011). Despite these findings, no studies have attempted to generalise how the trophic level of variation affects persistence and stability by comparing the two sources at multiple trophic levels.

In our study, we explored the potential for rescue in community models with alternative sources (genetic vs. plastic) and different trophic locations (autotroph vs. herbivore) of variability (Fig. 2.1). We specifically tested the following hypotheses: (***i***) Plastic communities are more likely to exhibit stability and persistence than communities where genetic diversity provides the same range of traits. (***ii***) Variation at the lowest trophic level promotes stability and persistence more than variation at higher levels. (***iii***) Communities with variation (either of plastic or genetic origin) at two levels have greater likelihoods of stability and persistence than communities with variation at only one level.

The results of our food web analyses did not allow us to reject our first hypothesis (***i***): plasticity promotes persistence and stability more than genetic diversity. However, we reject hypotheses (***ii***) and (***iii***) by showing that variation at the herbivore level is more stabilising than variation at the autotroph level or at multiple levels. These results question the generality of previous findings which implicate variation for defence in the autotroph as the key driver of community dynamics (van der Stap *et al.* 2007; Ramos-Jiliberto *et al.* 2008).

2.3 Methods

We analysed ten model food webs that vary in the degree, origin and trophic location of intraspecific variation for morphological defences (Fig. 2.1). These webs are based on, and

parameterised for a rotifer-algal system (*Scenedesmus* sp., autotrophic phytoplankton; *Brachionus* spp., herbivorous rotifers; and *Asplanchna* sp., carnivorous rotifers (Verschoor, Vos & van der Stap 2004; Vos *et al.* 2004a)). This system allows for phenotypic variation at the autotroph and herbivore levels. Newly produced autotroph algae can be induced by the herbivore to form defensive colonies (Verschoor, Vos & van der Stap 2004), and newly produced herbivorous rotifers can be induced by the carnivorous rotifer to grow long posterolateral spines (Gilbert 1966; van der Stap *et al.* 2007). In our model food webs, genetic variation covers the same trait range (undefended or fully defended) as plasticity, but phenotypes are produced by genotypes that are fixed for either trait value.

Webs 1 through 4 (Fig. 2.1) have no intraspecific variation and serve as controls with either no defence (web 1) or constitutive, fixed defences (webs 2-4). Webs 5 ("autotroph genetic") and 6 ("autotroph plastic") incorporate genetic diversity and plasticity, respectively, at the autotroph level. Webs 7 ("herbivore genetic") and 8 ("herbivore plastic") incorporate the same sources of variation at the herbivore level. Finally, webs 9 ("two levels genetic") and 10 ("two levels plastic") have variation at both herbivore and autotroph levels.

We adapted the equations for our model from Vos *et al.* (2004a). Yamamichi, Yoshida, & Sasaki (2011) analysed a related equation system for a flow-through chemostat scenario. Our formulation for food webs with plastic variation ("plastic webs") represents the effects of phenotypic plasticity by allowing plastic individuals to produce individuals in one of two discrete phenotypic populations (one population is undefended and the other is defended; both are linked by a "reproductive switching term" S). Plastic webs are derived from food webs with genetic diversity ("genetic webs") by adding S and a term to describe the production of new individuals by the non-focal phenotype (see Appendix 2.A for detailed equation systems for all

ten model food webs). Genotypic diversity is modelled for asexual reproduction and is therefore mathematically equivalent to having two distinct species at each of the respective trophic levels. In contrast, phenotypic plasticity allows a phenotype to channel part of its future reproductive output into the opposite phenotype. The most complex food web model with phenotypic plasticity at the autotroph and herbivore levels (web 10) consists of five coupled differential equations:

$$\frac{dP_u}{dt} = r\left(1-\frac{P}{K}\right)S_P(H)P - [f_H(P)\,H + \,m]P_u \tag{2.1a}$$

$$\frac{dP_d}{dt} = (1-\mu)\,r\left(1-\frac{P}{K}\right)[1-S_P(H)]P - [(1-\beta)\,f_H(P)\,H + \,m]P_d \tag{2.1b}$$

$$\frac{dH_u}{dt} = \epsilon[P_u + \,(1-\beta)\,P_d]\,f_H(P)\,S_H(C)\,H - \,[f_C(H)C + m]H_u \tag{2.1c}$$

$$\frac{dH_d}{dt} = (1-\mu)\,\epsilon\,[P_u + \,(1-\beta)\,P_d\,]\,f_H(P)\,[1-S_H(C)]\,H - \,[(1-\beta)\,f_C(H)C + m]H_d \tag{2.1d}$$

$$\frac{dC}{dt} = \{\gamma\,[H_u + \,(1-\beta)\,H_d\,]\,f_C(H) - m\,\}C \tag{2.1e}$$

with $P = P_u + P_d$; $H = H_u + H_d$

with switching functions S and functional response functions f:

$$S_P(H) = \left[1+\left(\frac{H_u+H_d}{g_H}\right)^{b_H}\right]^{-1} \tag{2.1f}$$

$$S_H(C) = \left[1+\left(\frac{C}{g_C}\right)^{b_C}\right]^{-1} \tag{2.1g}$$

$$f_H(P) = \frac{v_{PH}}{1+h_{PH}v_{PH}P_u+h_{PH}v_{PH}P_d} \tag{2.1h}$$

$$f_C(H) = \frac{v_{HC}}{1+h_{HC}v_{HC}H_u+h_{HC}v_{HC}H_d} \tag{2.1i}$$

P_d and P_u are the densities of the defended and undefended autotrophs, H_d and H_u are the densities of the defended and undefended herbivores, and C is the carnivore density. P_d and P_u grow logistically to a joint carrying capacity K, which applies to the sum of the two phenotypes.

S is a decreasing, sigmoid function that takes values between 1 and >0; it describes the reproductive investment towards producing undefended types (as a fraction of the whole population's growth rate r); this investment decreases with increasing densities of "inducers" at the next trophic level up; conversely, the investment towards producing defended types increases with inducer densities according to (1 - S). g_H and b_H determine the shape of $S_P(H)$; g_H is the density of herbivores that produces equal investment in defended and undefended phenotypes, and b_H describes the steepness of the function (analogously for g_C and b_C). ε and γ are the herbivores' and carnivore's conversion efficiencies. β is the defence level; as β increases, (1 - β) decreases and therefore the functional response of predators on defended types is diminished. μ reflects the cost of defence, such that a high μ corresponds to high costs and a low growth rate. (See Table 2.1 for a full list of parameters and their values).

Our model formulation differs in three ways from Vos *et al.'s* (2004a). First, we implement defence by multiplying attack rate by (1 - β) and dividing handling time by (1 - β) [note: the (1 - β) terms in the denominator cancel each other]. We believe this to be the most correct way to implement defence because in our system, defences in the herbivore center around the production of long posterolateral spines, these spines decrease the likelihood of successful attack (decrease attack efficiency) and also increase handling time by both wasting time on unsuccessful attacks (Jeschke, Kopp & Tollrian 2002) and by requiring more time to manipulate the herbivore into a position where it can be consumed. Vos *et al.* (2004a) implemented defence through increasing handling time of predators on defended prey; however, handling time is only present in the denominator of the functional response equation, which is identical for both prey types; thus, defended and undefended prey obtain the same benefit under Vos *et al.'s* (2004a) formulation (Kovach-Orr *et al. unpublished manuscript*). Additionally, defences bases entirely

on attack efficiency are not particularly applicable to our algal / rotifer system (Vos *et al.* 2004a). While it is true that decreasing attack efficiency by the same rate we increase handling time is arbitrary, it seems no less arbitrary than any other relationship between handling time and attack rate. We prefer our formulation because defended prey receive a greater benefit from defence than undefended prey, although, the presence of defended prey does benefit the undefended prey through the direct interaction of the defended prey and predator (Kretzschmar, Nisbet & McCauley 1993). Secondly, our mortality term represents increased environmental stress and affects all species equally, as opposed to Vos *et al.* (2004a), who manipulated mortality of the carnivore while leaving other trophic levels' mortalities static. Our analyses show that this assumption produces results that are qualitatively similar to those for a system where each species has its own mortality and only carnivore mortality is affected by environmental change (data not shown). Finally, Vos et al. used a switching function that mimics direct transfer or migration between populations of defended and undefended types. In contrast, our switching function distributes total population growth rate among types. This is a more realistic description of the mechanism of defence induction of aquatic invertebrates that cannot switch between defended and undefended states during their lifetime (Yamamichi, Yoshida & Sasaki 2011).

2.3.1 Model Analysis

We mathematically analysed the potential for community persistence in the context of varying environmental carrying capacity and natural mortality. We chose carrying capacity because it is a proxy for nutrient enrichment, a major environmental change in many aquatic ecosystems due to increased fertiliser run-off (Tilman *et al.* 2001) , and because the bottom-up

effects of nutrient enrichment can destabilise predator-prey dynamics (Rosenzweig 1971). We chose to manipulate mortality because increased natural mortality is the most general population-level effect of increased environmental stress (such as flooding, oil spills, aquatic acidification, pesticides, or any other factors that increase mortality). The production of defensive traits is typically costly for the prey and incorporated into models as a trade-off between defence β and cost μ. We followed this convention but evaluated all biologically relevant trade-off combinations rather than limiting our analysis to predetermined categories of trade-off curves (concave up, linear, and concave down).

We used two mathematical/computational approaches to analyse our model food webs: numerical simulations and numerical bifurcation analysis.

2.3.1.1 Numerical Simulations

At all parameter combinations, we evaluated our models for persistence and dynamic stability. Initial population densities were: undefended and defended autotrophs each equal to half of carrying capacity, undefended and defended herbivores each equal to 0.5 mg C L^{-1}, and carnivores equal to 0.25 mg C L^{-1}. To avoid transient dynamics, we evaluated simulations from time $t = 1000$ until $t = 21{,}000$. For persistence vs. extinction, a population was considered extinct if, at any time $1000 \leq t \leq 21{,}000$ its density was below 1.6×10^{-4} mg C L^{-1} (~ 1 carnivore individual per 3,000 ml (Verschoor, Vos & van der Stap 2004)). We considered a community to persist, if at least one population from every trophic level was always above this threshold. Note that extinction can occur either because the carnivore has a zero equilibrium (trivial equilibrium) or as a consequence of extinction through extreme oscillations (Fussmann 2007). For stability, we used the same numerical simulations as above, but also evaluated the maximum and

minimum population density from t = 14,000 to t = 21,000. If the difference between the maximum and minimum was less than the equivalent of 1 individual carnivore per 300 ml (1.6×10^{-3} mg C L^{-1}), the food web was considered to be stable. Note that this operational evaluation of stability scores oscillatory dynamics with minute amplitude as factually stable.

We used parallel computing (Guillimin cluster at CLUMEQ: 160 parallel threads) for numerical simulation of food web dynamics with MATLAB (MATLAB 2009). We quantified differences between food webs by calculating the frequency of persistence and dynamic stability over 296,960 combinations of mortality m (29 intervals), carrying capacity K (40), defence level of defended phenotype β (16), and costs for the defended phenotype μ (16) [See Table 2.1 for ranges and interval sizes].

2.3.1.2 Bifurcation Analysis

Bifurcation analysis can provide deeper insights into the mechanisms underlying the distributions of persistent and stable dynamics. For instance, bifurcation analysis can reveal why variation at a certain level increases the potential for stability more than variation at a different level. All bifurcation diagrams were produced using numerical bifurcation software (PyDSTool 2008). While our numerical simulations were performed for K = 0.25 to 19.75 mg C L^{-1}, our numerical bifurcations were evaluated for K=0 to 50 mg C L^{-1}.

2.4 Results

Table 2 shows the average frequencies of persistence and stability for all food webs (see Fig. 2.1) and across all combinations of carrying capacity K, mortality m, cost of defence μ, and defence value β. We found a positive correlation between the two measures that we used to

quantify the potential for community rescue ($R^2 = 0.57$, for $n = 10$ food web types in Table 2.2). That is, food webs with a high frequency of persistence also tended to display higher frequencies of dynamic stability. Residual variation was almost entirely due to the differences between food webs with genetic vs. plastic variation. In "plastic webs" (webs 6, 8, 10) more than half of the food web parameterisations that persisted in our simulations did so at dynamic equilibria, while oscillatory dynamics dominated in persisting "genetic webs" (webs 5, 7, 9; Table 2.2).

In our model simulations, food webs with constitutive, fixed defences were generally less persistent and less dynamically stable than webs that did not feature defences at any trophic level (webs 2-4 vs. web 1). Food webs with intraspecific variation were more persistent and stable than their specific counterparts with no variation, i.e., variable defences at the autotroph level led to increased persistence and stability over webs that had permanently fixed defences for autotrophs (webs 5 & 6 vs. web 2); the same was true for defences introduced at the herbivore level (webs 7 & 8 vs. web 3) or at both levels (webs 9 & 10 vs. web 4). Variability did not generally increase persistence but did on average increase stability in comparison to webs that were entirely undefended (webs 5-10 vs. web 1) (note: webs 5 and 9 showed reduced persistence and stability when compared to web 1); both the source (genetic or plastic) and trophic location of the variation critically influenced the magnitude of stability and persistence, and therefore the potential for rescue.

2.4.1 Effect of the Source and Trophic Location of Variation

In general, plasticity was more effective than genetic diversity at increasing the frequencies of persistence and stability in our model food webs (Table 2.2). Plasticity yielded 1.1 times more persistence and 3.8 times more stability than genetic diversity. In genetic

diversity webs (webs 5, 7, 9), almost all persistence was in the form of unstable, oscillatory dynamics; only 17.8% - 21.9% of persistence manifested as stability (Table 2.2). Plastic, persistent webs tended to be much more stable with 60.9%-80.6% of persistent dynamics being equilibria (Table 2.2). Using a specific simulation example, Fig. 2.2 demonstrates how this observed effect can contribute to population and community rescue. In web 1, with no variation for defence, the community exhibits regular oscillations at low carrying capacity (Fig. 2.2a) ; when carrying capacity is increased, the carnivore is quickly eliminated. In web 7, the community is at equilibrium at low carrying capacity, demonstrating an effect of diversity (Fig. 2.2b); when carrying capacity is increased, the web shows damped oscillations and slowly approaches equilibrium. In web 8, increased carrying capacity causes damped oscillations that quickly reach equilibrium (Fig. 2.2c).

We found that variation at the herbivore level had the greatest impact on persistence and stability, regardless of the source of variation (Table 2.2). Webs 7 and 8 are the only webs that increased in both persistence and stability when compared to web 1. This means, interestingly, that variation at the herbivore level alone is more conducive to persistence and stability than when the same variation is introduced in combination with variation at the autotroph level.

At the autotroph level, we found that plasticity was less likely to promote persistence than genetic diversity (Table 2.2); however, plasticity was far more likely to promote stability. On the contrary, we found that plasticity at the herbivore level promoted both stability and persistence compared to genetic diversity. Finally, we found that genetic and plastic variation at two trophic levels promoted approximately equal levels of persistence, however, plastic variation promoted stability far more than genetic variation.

2.4.2 Bifurcation Analyses and Strong versus Weak Stabilization

So far, we have presented the results of our numerical simulations as frequencies of persistent and stable dynamics. We now turn to bifurcation analysis, which can provide deeper insights into the mechanisms underlying the distributions of persistent and stable dynamics. Figure 2.3 illustrates our general framework of evaluation, using a simpler version of our model: the classic bitrophic Rosenzweig and MacArthur (Rosenzweig & MacArthur 1963) predator-prey system (Fig. 2.3a), the bitrophic system with genetic diversity in the autotroph (Fig. 2.3b), and with plasticity (Fig. 2.3c). The carrying capacity-mortality diagram shows two bifurcation lines: a transcritical bifurcation (black line), above which persistence is not possible, since the predator cannot maintain a positive growth rate; and a Hopf bifurcation (red line), which denotes the transition from stable equilibria (above) to oscillatory dynamics (below). We analysed how plasticity and genetic diversity affect the location of these bifurcation lines. Figure 2.3 also demonstrates two categorically distinct patterns of stabilisation ("weak" and "strong" (Kretzschmar, Nisbet & McCauley 1993)) which we introduce because they turn out to be strongly associated with the source of variability that we apply in our model food webs. Weak stabilisation occurs when the transcritical and Hopf bifurcations are moved apart, but still approach the same asymptote; thus the area of stable equilibrium is larger, but still finite (Fig 2.2 b). Strong stabilisation occurs when the transcritical and Hopf bifurcations approach different asymptotes (Fig 2.2 c) (Kretzschmar, Nisbet & McCauley 1993). Only strong stabilisation allows stable dynamics at some infinite parameter values (e.g. infinite carrying capacity). In the genetic web, we were only able to find weak stabilisation (through the presence of a new finite set of Hopf bifurcations). In the plastic web, we found strong stabilisation.

We now present the results of the bifurcation analysis for the tritrophic model. Because we cannot graphically present the results for all combinations of defence level β and cost of defence μ that we analysed, figure 2.4 shows typical bifurcation diagrams for each of the ten food web configurations ($\mu = 0.5, \beta = 0.5$, K: x-axis, m: y-axis). While all bifurcations in figure 2.4 are qualitatively typical, these parameters maximize the stability of webs 6 and 10 (Appendix 2.B-2). The bifurcation plot for the control web without variation (web 1) shows a single finite stability band ("weak stability"; Fig. 2.4 (1)). As carrying capacity increases, mortality must also increase in order for the community to maintain equilibrium. This continues until $m = 0.533$, at which point the transcritical and Hopf bifurcations converge and stability becomes nearly impossible. Bifurcation plots for food webs with fixed defences (webs 2-4) are qualitatively the same but the transcritical bifurcation is shifted downwards (i.e., in the presence of the defended autotrophs or herbivores, the carnivore can tolerate less mortality), in agreement with results given in Table 2.2. We were unable to find strong stabilisation in any of the food webs with genetic diversity, i.e., the transcritical and Hopf bifurcations always converged at high carrying capacity resulting in finite areas of stable dynamics (Fig. 2.4 (5), 2.4 (7), 2.4 (9)). On the contrary, we found strong stabilisation was possible in all of the food webs with plastic variability (Fig. 2.4 (6), 2.4 (8), 2.4 (10)).

Specifically, genetic variation at the autotroph level (web 5) produces a single finite stability band (Fig. 2.4 (5)); its area and position are nearly identical to those in web 1 because the defended type is eliminated. Plastic variation at the autotroph level (web 6) can produce a narrow infinite stability band (indicating strong stabilisation), which exists between $m = 0.345$ and $m = 0.365$ (Fig. 2.4 (6)).

Genetic variation at the herbivore level (web 7) can produce one or two finite stability bands (Fig. 2.5); the second band only exists in 14% of the μ-β parameter range. Outside of this parameter range, this web looks very similar to web 1 (Fig. 2.4 (7)) because the defended type is eliminated. Between the two stability bands, the community exhibits stable limit cycles; therefore, the three lower bifurcations are Hopf bifurcations (supported by numerical simulation analysis). The lower band contains both defended and undefended types and is possible because of predator-mediated coexistence. Plastic variation at the herbivore level (web 8) produces a single, broad and infinite stability band (Fig. 2.4 (8)); the transcritical bifurcation reaches its asymptote at $m = 0.533$ and the Hopf bifurcation reaches its asymptote at $m = 0.305$. When we consider variation at both the autotroph and herbivore levels, the genetic web (web 9) produces a single finite stability band (Fig. 2.4 (9)); this band is nearly identical to web 1 because the defended types are eliminated. Plastic variation (web 10) produces one infinite stability band (Fig. 2.4 (10)); this band occurs at lower mortalities than the band in web 8.

2.5 Discussion

The principal concern of our study was to investigate how genetic diversity and phenotypic plasticity differentially contribute to the rescue of communities facing environmental change. "Evolutionary rescue" through rapid evolution or through the use of standing adaptive genetic variation (Barrett & Schluter 2008) is currently receiving a lot of attention (Orr & Unckless 2008; Bell & Gonzalez 2009; Bell & Gonzalez 2011; Bell 2013; Gienapp *et al.* 2013; Kirkpatrick & Peischl 2013; Martin *et al.* 2013; Osmond & de Mazancourt 2013), but rescue by evolution only considers one of the two potential sources of phenotypic diversity. Our ultimate goal must be to evaluate the likelihood of species survival through local adaptation in a changing

environment. A realistic assessment of rescue potential will allow for a combination of genetic and plastic sources of phenotypic variation.

A few recent theoretical studies have compared how population dynamics change when either genetic or plastic variation is introduced (Abrams 2009; Cortez 2011; Yamamichi, Yoshida & Sasaki 2011). In our study, we adopted a similar framework of analysing dynamic stability, but placed an emphasis on persistence vs. extinction, i.e. on dynamic consequences that can be interpreted in relation to evolutionary or plastic rescue of populations and whole communities. We also paid particular attention to the trophic level at which genetic or plastic diversity are introduced into food webs and how this affects the potential for rescue.

The results support our hypothesis (***i***) and confirm previous findings that plasticity is significantly more likely to promote stability than genetic diversity (Abrams 2009; Cortez 2011; Yamamichi, Yoshida & Sasaki 2011). There are three possible explanations for why plasticity promotes stability: (1) Plasticity allows phenotypes that are maladapted to the environment to reappear when conditions are favourable, whereas in genetic webs, phenotype extinction is permanent. This would apply to our study if the abiotic environment was actively changing during simulations (it is not) or if there was a strong effect of initial conditions. We took steps to limit the impact of initial conditions and believe the reintroduction of phenotypes could only be minimally responsible for the disparity between genetic and plastic webs. (2) Plasticity allows prey to adapt to predator density changes faster than genetic diversity (Cortez 2011; Yamamichi, Yoshida & Sasaki 2011). Yamamichi, Yoshida, & Sasaki (2011) showed that the relative abundances of prey phenotypes in plastic webs are able to change much faster than in genetic webs and that slowing the plastic response speed reduces the likelihood of stability. Although speed certainly plays a role in the observed differences between plasticity and genetic diversity,

we believe there is an even greater driver for the observed differences: (3) The reproductive switching function, *S,* in plastic webs creates an obligatory negative feedback loop between predator density and predator efficiency (Ramos-Jiliberto & Garay-Narvaez 2007) that buffers predator and prey populations from extinction. If the predator becomes very abundant, prey defence increases, causing predator efficiency, and ultimately, predator density to decrease (thus stabilising the food web by moving it further from the Hopf bifurcation); on the other hand, if predators approach extinction, prey defence decreases, causing predator efficiency, and ultimately, predator density to increase (thus reducing the risk of predator starvation). In plastic webs, the relative abundances of the prey phenotypes are entirely controlled by the density of the predator. At sufficiently high predator density, the switching function will cause the defended prey type to persist, even if the costs outweigh the benefits. Genetic webs also have a feedback loop, but it is not obligatorily "negative", because changes in the relative abundances of prey phenotypes are driven by a combination of costs, natural mortality, and predation. The defence is adaptive if the per-capita growth rate of the defended type is higher than that of the undefended type; that is (for the autotroph) $\frac{1}{P_d}\frac{dP_d}{dt} > \frac{1}{P_u}\frac{dP_u}{dt}$, which leads to $\frac{\beta}{\mu} > \frac{r\left(1-\frac{P}{K}\right)}{Hf_H(P)}$. As a special case, we can consider the situation of a rare defended mutant invading a population containing only undefended prey (i.e. a situation with $P_u = P$ and P_d approaches 0). Assuming the community is at a stable equilibrium (otherwise the analysis becomes more difficult), we can set $\frac{dP_u}{dt} = 0$, from which follows $H^* f_H(P^*) = r\left(1 - \frac{P^*}{K}\right) - m$. Plugging this into the above result yields $\mu < \beta\left(1 - \frac{m}{r\left(1-\frac{P^*}{K}\right)}\right)$. [Here, * denotes equilibrium densities in the absence of defended prey; note that at equilibrium $\frac{m}{r\left(1-\frac{P^*}{K}\right)}$ must be between 0 and 1.] In general, whether

the defence is adaptive depends not only on predator but also on prey density (because the effect of costs is reduced due to the density-depended regulation term). Therefore, our interpretation of effects of costs is limited.

While maladaptive phenotypes persist in the plastic webs, they are eliminated from the genetic webs; thus, the parameter range where negative feedback loops are possible is much smaller for genetic webs. If genetic diversity were constrained to cost-benefit combinations that fall along continuous trade-off curves due to adaptive evolution prior to the rescue event, such that only beneficial adaptations exist, the potential for evolutionary rescue would, on average, actually decrease for webs 5 and 9, but increase for web 7 (Appendix 2.B). Webs 5 and 9 are only stable when the defended type is eliminated, adaptive evolution would allow the defended type [s] to persist, which, in turn, could cause the extinction of the carnivore. On the other hand, because both genotypes can persist in web 7, adaptive evolution can increase stability.

Furthermore, we found that plasticity, but not genetic diversity, can lead to strong stabilization and therefore, plasticity can resolve the paradox of enrichment. Strong stabilisation has been shown to occur in model food webs exhibiting ecological attributes such as inducible defences, inedible prey, predator feeding thresholds, or predator interference (see (van Voorn *et al.* 2008; Bontje *et al.* 2009)). Predator interference works in a similar fashion to inducible defenses; the transcritical bifurcation remains unaffected because at very low predator density, there are not enough predators to induce prey/to interfere with each other. As the system approaches the Hopf bifurcation, the additional predators reduce predator efficiency, either through interfering with other predators or by inducing defended prey. While these are fundamentally different processes, they both operate under the framework of creating negative feedback loops with changes in predator density.

A word of caution needs to be added when applying our results to natural communities. Genetic diversity in our study was only allowed to operate within the same confines of trait space as phenotypic plasticity, i.e. genotypes could not exceed the trait variation that was provided through plasticity. Our finding that plastic rescue is more likely than evolutionary rescue is valid within this limit of evolvability. It is, of course, conceivable that at some point, evolution will generate genetic variation that exceeds the limits of plastic response (Barrett & Hendry 2012). A possible scenario is that phenotypic plasticity takes the role of provisional response, rescuing populations from initial and fast environmental change of low to intermediate intensity, while an evolutionary, genotype-based response needs to take over if stress is severe and long lasting.

The second important result of our study is that the trophic location of variation can constrain the potential for rescue of populations and communities. Both phenotypic plasticity and genetic diversity can occur at any trophic level (from the autotroph to the top-carnivore). Contrary to previous findings that variation will generally contribute to rescue (Vos *et al.* 2004a; Vos *et al.* 2004b; Jones & Ellner 2007; Yamamichi, Yoshida & Sasaki 2011), we found that variability at the herbivore is particularly likely to contribute to community rescue, and reject hypothesis (***ii***) that attributes the highest rescue potential to variability at the basal, autotroph level (van der Stap *et al.* 2007; Ramos-Jiliberto *et al.* 2008).

Variation in the herbivore is special because the presence of two herbivore phenotypes allows for two distinct relationships between carnivore dynamics and mortality/carrying capacity. In web 7 (genetic variation in the herbivore), this can create two finite bands of stability separated by an area of oscillations (Fig. 2.5b): in the upper band, only undefended herbivores are present in the population, and in the lower band, both undefended and defended herbivores are present. In web 8 (plastic variation in the herbivore), the additional herbivore

phenotype allows for a wide, strongly stable band: at the upper boundary of the band, all herbivores are undefended, whereas at the lower boundary, all herbivores are defended; Vos *et al.* (2004a) obtained a similar result for a bitrophic plastic web. Variation at the herbivore level is able to create these special types of stability through a negative feedback loop that offsets changes in carnivore density with changes in herbivore defence.

Variation in the autotroph operates under the same fundamental principles; it allows the autotroph to offset changes in herbivore density with changes in autotroph defence. Why, then, doesn't variation at the autotroph level generate the same degree of rescue potential as variation at the herbivore level (compare webs 5, 6 vs. webs 7, 8)? The reasons are different for each source of variation. In the genetic case, the increased rescue potential of web 7 (genetic variation in herbivores) derives from equilibrium coexistence of defended and undefended genotypes and carnivore (the lower stability band in Fig. 2.5b), which is possible due to the diamond configuration of the food web (McCann, Hastings & Huxel 1998). At the autotroph level, equilibrium coexistence of the two genotypes, herbivore, and carnivore is impossible because of apparent competition (Holt 1977), so that the stability properties of web 5 are essentially the same as those of the tri-trophic food chain (web 1). In the case of plasticity, trophic cascade dynamics prevent autotrophic plasticity from having the same impact as herbivore plasticity. Plasticity in the autotroph lowers the transcritical and Hopf bifurcations, whereas plasticity in the herbivore only lowers the Hopf bifurcation. The transcritical bifurcation defines the minimal productivity of a system, at a given mortality, that is necessary to support the carnivore. However, productivity must be transferred "up" the food web, and so one can think of the transcritical bifurcation defining the minimal herbivore density of a system, at a given mortality, that is necessary to support the carnivore. When we consider plasticity in the herbivore, as the

food web approaches the transcritical bifurcation, carnivore density goes to zero, so all herbivores become undefended, carnivore efficiency increases, thus decelerating carnivore extinction. When we consider plasticity in the autotroph, as carnivore density goes to zero, herbivore density increases, which causes increased defences in the autotroph, which decreases herbivore density and accelerates extinction of the carnivore. Furthermore, the Hopf bifurcation for web 6 occurs at higher mortality values, and is therefore closer to the transcritical bifurcation, than the Hopf bifurcation for webs 8 or 10. The Hopf bifurcation for web 6 is so high because web 6 is less effective at countering increases in carnivore density. This being said, plasticity in the autotroph still enhances stability compared to the undefended tri-trophic chain (web 1) because it makes herbivore density more robust to changes in carnivore density.

Contrary to our expectations in hypothesis (***iii***), variation at the herbivore level alone produces more stability and persistence than variation at two levels (Table 2.2). For the case of genetic variation, the defence and cost values that allow predator-mediated herbivore coexistence in web 7 cause the extinction of the defended herbivore when they are also present in the autotroph (web 9). Therefore, stability for web 9 is only possible when web 9 simplifies to the tri-trophic food chain (webs 1 or 2) (Fig 2.4); furthermore, this implies that stability in web 9 is only possible under the same carrying capacities and mortalities where webs 1 and/or 2 are stable. For the case of plastic variation, the addition of plasticity at the autotroph level affects web 10 through the same mechanisms described for web 6: plasticity in the autotroph effectively lowers the transcritical and Hopf bifurcations of web 8 to produce web 10.

The effect of two-level plasticity is sensitive to the concrete parameterisation of the food web model. Table 2.2 shows the two-level plastic web has less stability than the herbivore-plasticity-only web. Nevertheless, for 75% of the parameter combinations, plasticity at two

levels was nearly as stable as plasticity at the herbivore level alone (Fig. 2.4 (8), 2.4 (10), Appendix 2.B); however, for the remaining 25% of parameter combinations, observed at moderate and high defence levels, web 8 is far more stable than web 10 (Appendix 2.B). In this range, the Hopf bifurcation of web 10 approaches the x-axis; however, there is little stability because the transcritical bifurcation is also very close to the x-axis (data not shown). Similarly, the Hopf bifurcation of web 8 approaches the x-axis; however, there is greater stability because the transcritical bifurcation is not affected by plasticity at the herbivore level (data not shown).

Furthermore, our assumption that μ and β are identical for both trophic levels probably restricts the range of outcomes, however, the authors feel that testing different trade-offs at different levels was outside the scope of this study.

In summary, we found that variation did not generally promote rescue; rather, rescue potential depended on the trophic location and source of variation. While plasticity increased stability regardless of trophic location, genetic diversity only had this effect when present exclusively at the herbivore level. Our results suggest that variation for defence at the second highest trophic level should have a greater impact on rescue than variation at lower levels. Variation for defence traits creates negative feedback loops with changes in predator density: when defence is present at the second highest level, the negative feedback loop works to maintain the top-level. When defence is present two levels below the top, it can create a positive feedback loop with changes in top-level density. Defence could also potentially create a negative feedback loop for changes in top-level density, if present three or five levels below the top; however, we suspect that this effect would diminish with trophic distance.

We acknowledge that some of our interpretations and conclusions are specific to introducing variation in the form of an inducible defence trait; this is particularly true for

arguments involving indirect top-down and bottom-up effects in food webs. However, only a small fraction of populations' variability will typically be pre-adaptive to novel stress (Barrett & Hendry 2012) and environmental change may produce more than one novel stressor. Defence is a general, variable trait, occurs at multiple trophic levels and may or may not serve as a pre-adaption that helps populations persist following a change (Vos *et al.* 2004a; Vos *et al.* 2004b). Overall, we feel that by modelling defensive traits we were able to provide a more general treatment of evolutionary and plastic rescue of communities than by concentrating on traits that are directly adaptive to environmental change or stress.

2.6 Acknowledgements

We acknowledge support by the James S. McDonnell Foundation. We thank Matthijs Vos, Yony Bresler, Emily Vogt, the two anonymous reviewers, and the Fussmann lab for their suggestions and comments on this manuscript. We are grateful to CLUMEQ (Consortium Laval, Université du Québec, McGill and Eastern Québec) and Compute Canada for the use of the Guillimin Cluster. We also thank Andrew P. Hendry and Rowan D.H. Barrett for the term "plastic rescue".

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2.8 Tables

Table 2.1. Definitions and default values of model variables and parameters (obtained from Vos et al. (Vos *et al.* 2004b))

parameter	value	interpretation
r	1.42 d^{-1}	intrinsic rate of increase of autotrophs
K	0.25 to 19.75 mg C L^{-1} by [0.5]*	carrying capacity
m	0.025 to 0.725 d^{-1} by [0.025]*	mortality
v_{PH}	0.77 L d^{-1} mg C^{-1}	herbivore search rate on autotrophs
v_{HC}	2.71 L d^{-1} mg C^{-1}	carnivore search rate on herbivores
h_{PH}	0.5 d^{-1}	handling time of herbivores on autotrophs
h_{HC}	0.83 d^{-1}	handling time of carnivores on herbivores
ε	0.36	conversion efficiency of herbivores
γ	0.5	conversion efficiency of carnivores
β	0 to 0.9375 by [0.0625]*	defence level of defended phenotypes
μ	0 to 0.9375 by [0.0625]*	cost of defence as decrement in growth rate
$S(H)$	$1 \geq S(H) > 0$	plastic reproductive switching function for autotrophs
$S(C)$	$1 \geq S(C) > 0$	plastic reproductive switching function for herbivores
g_H	0.06 mg C L^{-1}	half saturation constant for plastic switching in autotroph
g_C	0.02 mg C L^{-1}	half saturation constant for plastic switching in herbivore
b_H	2.05	shape of plastic switching in autotroph
b_C	1.5	shape of plastic switching in herbivore

* marks the interval size for β, μ, m and K.

Table 2.2. Frequencies of persistence and equilibrium dynamics across 286,720 food web simulations. Average percentages for ten food web categories that differ in degree, trophic location and source of variability. Persistence: percent of simulations with ≥ 1 population persisting at each trophic level. Equilibria: percent of simulations with stable, non-oscillatory dynamics. Only simulations exhibiting persistence were analysed for stability but percent values are given as the fraction of all 286,720 simulations.

web #	trophic level of defence	trophic level of variation	source of variation	persistence [%]	equilibria [%]
1	none	none	none	24.05	4.74
2	autotroph	none	none	4.06	1.27
3	herbivore	none	none	5.02	0.91
4	both levels	none	none	2.37	0.58
5	autotroph	autotroph	genetic	19.63	3.66
6	autotroph	autotroph	plastic	14.06	8.56
7	herbivore	herbivore	genetic	25.49	5.59
8	herbivore	herbivore	plastic	37.74	23.89
9	both levels	both levels	genetic	20.13	3.58
10	both levels	both levels	plastic	20.45	16.49

2.9 Figures

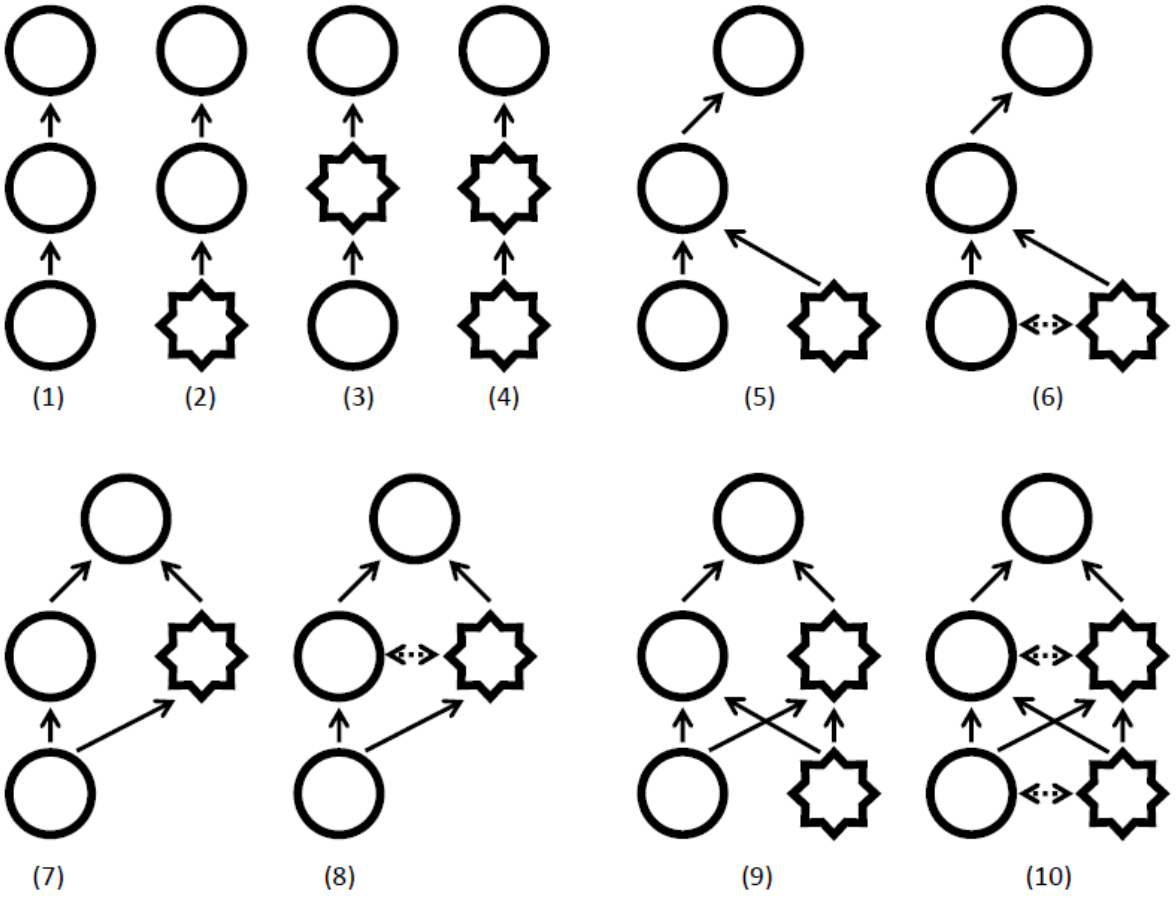


Figure 2.1. The ten tri-trophic model food webs used in the analysis of the effects of the source and trophic location of variation. Each individual circle represents a population expressing a single phenotype. Autotroph populations are at the bottom level of each food web, herbivores at the intermediate level and the carnivore at the top. The autotrophs' and herbivores' shape identifies the presence of defence (smooth circles are undefended, jagged circles are defended). A dashed horizontal arrow denotes the presence of plasticity. Solid lines indicate feeding links. Webs 1-4 have one genotype with fixed phenotypic expression; web 1 has no defence, web 2 has defence at the autotroph level, web 3 has defence at the herbivore level, web 4 has defence at both the autotroph and herbivore levels. Webs 5 and 6 have genetic and plastic variation,

respectively, at the autotroph level. Webs 7 and 8 have genetic and plastic variation, respectively, at the herbivore level. Webs 9 and 10 have genetic and plastic variation, respectively, at both the autotroph and herbivore levels.

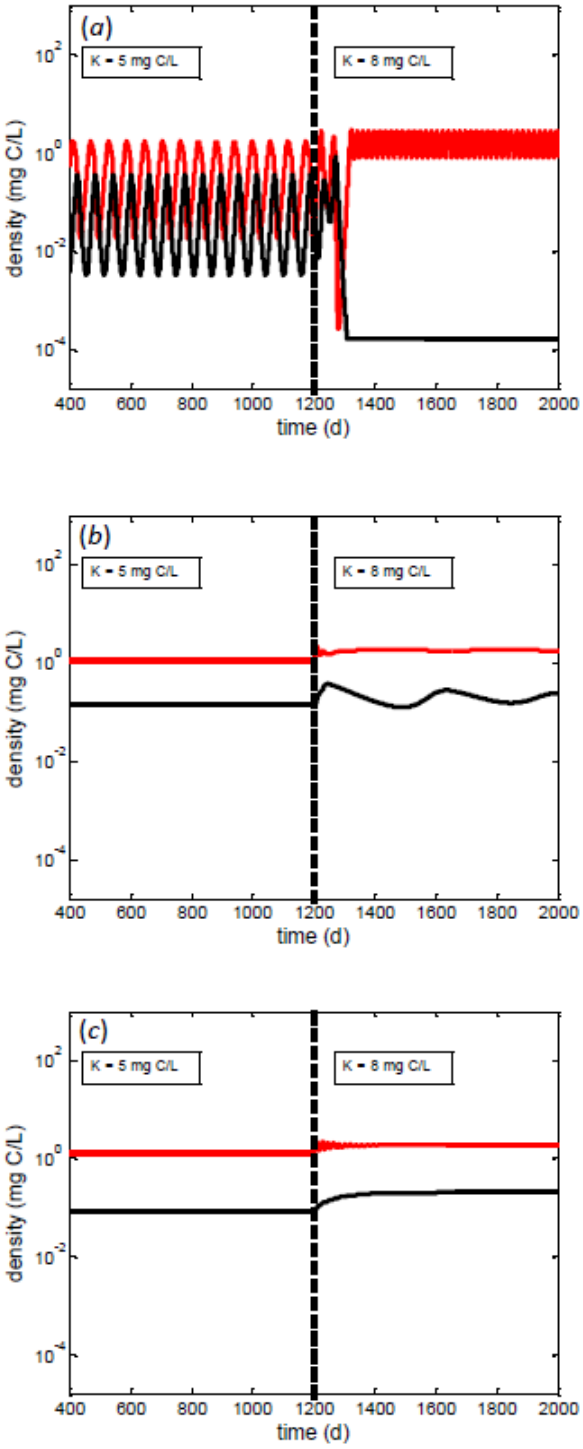


Figure 2.2. Time series of (*a*) web 1 with no variation and no defence (*b*) web 7 with genetic variation and (*c*) web 8 with plastic variation at the herbivore level. At the *vertical dashed line*, carrying capacity increases during the simulation, mimicking the sudden onset of environmental change. Black line denotes carnivore; red line denotes sum density of herbivores. Web 1 displays non-equilibrium dynamics before the change, both webs with variation display stable equilibrium dynamics before the change (K = 5); the increase in carrying capacity (K = 8) causes the extinction of the carnivore in web 1 (*a*), slowly damped oscillations of herbivores and carnivore in web 7 that eventually return to equilibrium (not shown) (*b*), while web 8 quickly returns to equilibrium (*c*). m = 0.25, β = 0.5, m = 0.5.

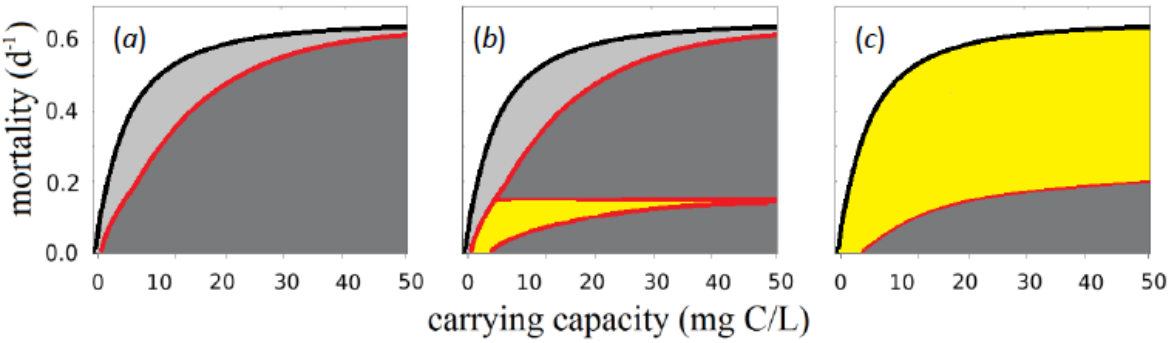


Figure 2.3. Bifurcation diagrams of a bitrophic food web. Carrying capacity (*K*) is shown on the X-axis, and mortality (*m*) on the Y-axis. The *higher (black) line* is the transcritical bifurcation; above the transcritical bifurcation (*white area*), the herbivore cannot maintain positive growth. The *lower (red) line* is the Hopf bifurcation; below the Hopf (*dark grey area*), population dynamics are unstable. Between the transcritical and Hopf bifurcations (*light grey area*), the undefended autotroph and carnivore populations are persistent and stable. In the *yellow area*, both autotroph populations and the herbivore are persistent and stable. (*a*) The classical Rosenzweig-MacArthur (Rosenzweig & MacArthur 1963) model. (*b*) Weak stabilisation: a new set of Hopf bifurcations are produced, but still reach the same asymptote at high carrying capacity. (*c*) Strong stabilisation: the transcritical and Hopf bifurcations are moved apart and approach different asymptotes, thus creating a region of stability extending to infinite carrying capacity.

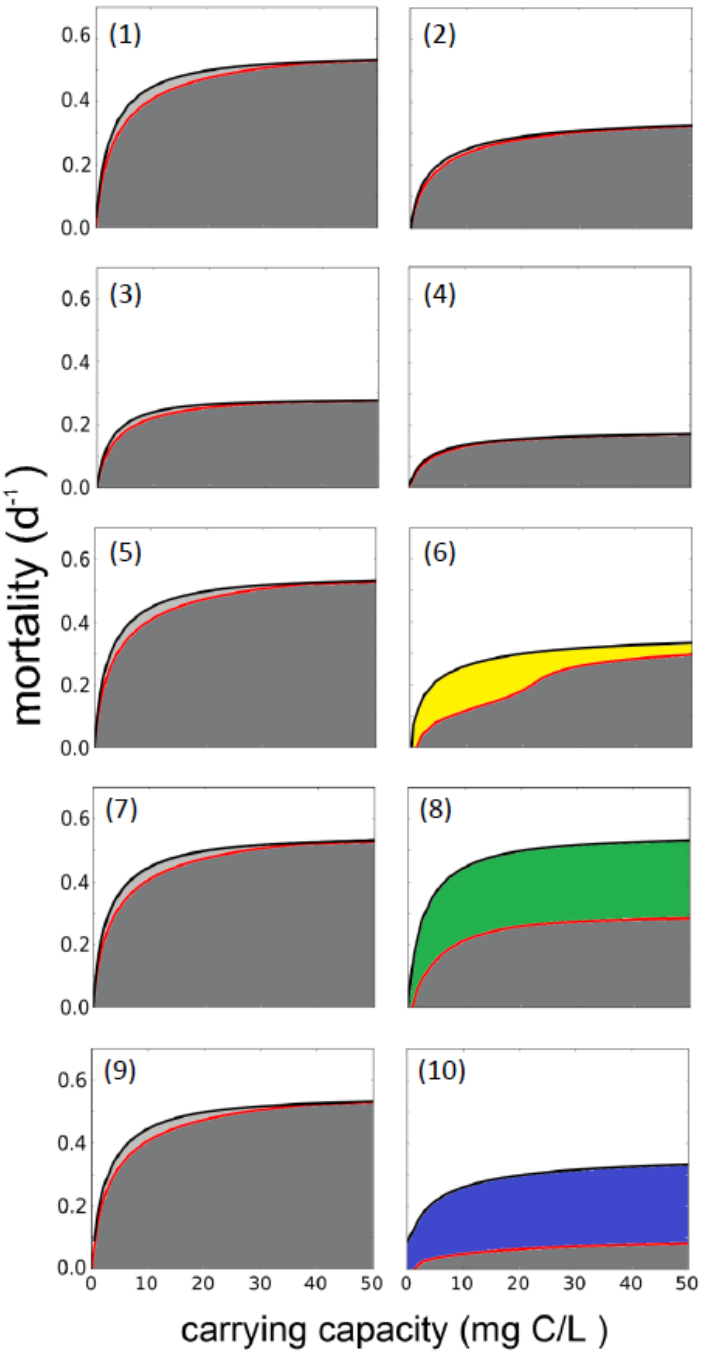


Figure 2.4. Bifurcation diagrams of all ten food webs. Each numbered plot refers to its corresponding numbered food web in Fig. 2.1. The *higher (black) line* is the transcritical bifurcation; the *lower (red) line* is the Hopf bifurcation. In the *light grey area*, the carnivore persists, at equilibrium, with one herbivore population and one autotroph population; in the *yellow area*, both undefended and defended autotrophs exist at equilibrium with herbivores and carnivores; in the *green area*, both herbivore populations persist, at equilibrium, with the autotroph and carnivore; in the *blue area*, both herbivore populations and both autotroph populations persist, at equilibrium, with the carnivore; see Fig. 2.3 for description of other colours stability properties. Note that the transcritical and Hopf bifurcations converge at high carrying capacity for all webs except 6, 8, and 10 (the plastic webs). Note the similarity of webs 5, 7, and 9 to web 1. $\beta = 0.5$, $\mu = 0.5$

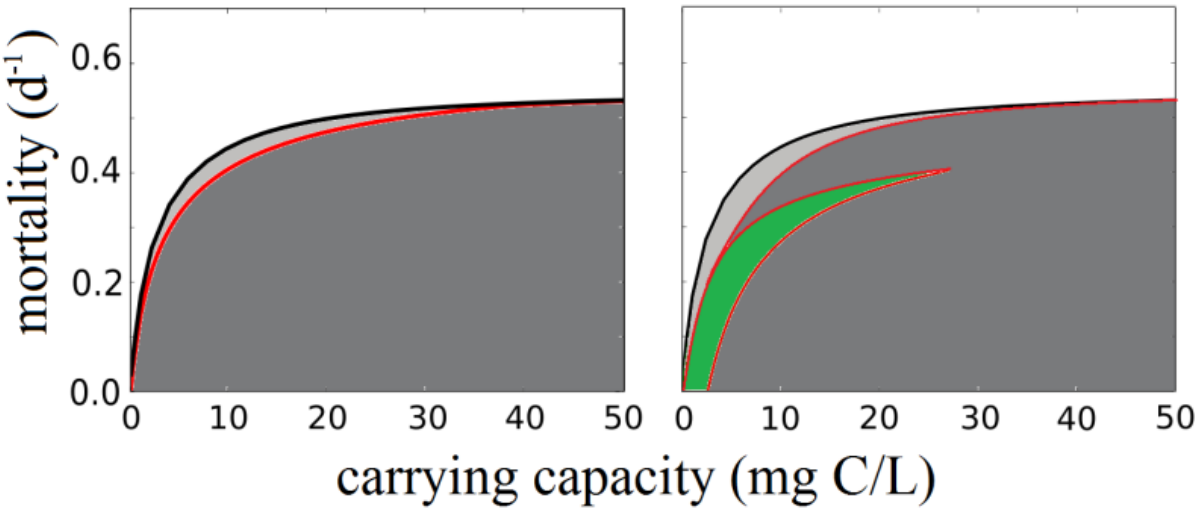


Figure 2.5. Bifurcation diagrams of web 7 (with genetic variation at the herbivore level). Description and parameter values as in Fig. 2.4, except for cost μ in Fig. 2.5.b . (*a*) In the stable band, the defended herbivore is extinct. (*b*) μ = 0.15. Note the presence of a new second set of Hopf bifurcations; the upper band is identical to the band in (*a*); however, the lower band corresponds to an equilibrium with predator-mediated coexistence of defended and undefended herbivores. Between the upper and the new lower stability band, dynamics are characterised by oscillations

2.10 Appendices

Appendix 2.A- Differential Equation Systems for the Ten Food Web Models Listed in Table 2.1 and Figure 2.1

Web 1

No variation. Autotroph undefended. Herbivore undefended.

$$\frac{dP_u}{dt} = r\left(1-\frac{P_u}{K}\right)P_u - \left[f_H(P_u)H_u + m\right]P_u$$
$$\frac{dH_u}{dt} = \varepsilon P_u f_H(P_u)H_u - \left[f_C(H_u)C + m\right]H_u$$
$$\frac{dC}{dt} = \left[\gamma H_u f_C(H_u) - m\right]C$$

with:

$$f_H(P_u) = \frac{v_{PH}}{1 + h_{PH}v_{PH}P_u}$$
$$f_C(H_u) = \frac{v_{HC}}{1 + h_{HC}v_{HC}H_u}$$

Web 2

No variation. Autotroph defended. Herbivore undefended.

$$\frac{dP_d}{dt} = (1-\mu)r\left(1-\frac{P_d}{K}\right)P_d - \left[(1-\beta)\, f_H(P_d)\, H_u + m\right]P_d$$

$$\frac{dH_u}{dt} = \varepsilon\left[(1-\beta)\, P_d\right] f_H(P_d) H_u - \left[f_C(H_u)C + m\right]H_u$$

$$\frac{dC}{dt} = \left[\gamma H_u\, f_C(H_u) - m\right]C$$

with:

$$f_H(P_d) = \frac{v_{PH}}{1 + h_{PH} v_{PH} P_d}$$

$$f_C(H_u) = \frac{v_{HC}}{1 + h_{HC} v_{HC} H_u}$$

Web 3

No variation. Autotroph undefended. Herbivore defended.

$$\frac{dP_u}{dt} = r\left(1-\frac{P_u}{K}\right)P_u - \left[\, f_H(P_u)H_d + m\right]P_u$$

$$\frac{dH_d}{dt} = (1-\mu)\varepsilon P_u\ f_H(P_u)\,H_d - \left[\left(1-\beta\right) f_C(H_d)C + m\right]H_d$$

$$\frac{dC}{dt} = \left\{\gamma\left[\left(1-\beta\right)H_d\right] f_C(H_d) - m\right\}C$$

with:

$$f_H(P_u) = \frac{v_{PH}}{1+h_{PH}v_{PH}P_u}$$

$$f_C(H_d) = \frac{v_{HC}}{1+h_{HC}v_{HC}H_d}$$

Web 4

No variation. Autotroph defended. Herbivore defended.

$$\frac{dP_d}{dt} = (1-\mu)r\left(1-\frac{P_d}{K}\right)P_d - \left[(1-\beta)\, f_H(P_d)H_d + m\right]P_d$$

$$\frac{dH_d}{dt} = (1-\mu)\varepsilon\left[(1-\beta)P_d\right] f_H(P_d)H_d - \left[(1-\beta)\, f_C(H_d)C + m\right]H_d$$

$$\frac{dC}{dt} = \left\{\gamma(1-\beta)H_d \;\; f_C(H_d) - m\right\}C$$

with:

$$f_H(P_d) = \frac{v_{PH}}{1 + h_{PH}v_{PH}P_d}$$

$$f_C(H_d) = \frac{v_{HC}}{1 + h_{HC}v_{HC}H_d}$$

Web 5

Genetic variation. Autotroph defended. Herbivore undefended.

$$\frac{dP_u}{dt} = r\left(1-\frac{P}{K}\right)P_u - \left[f_H(P)H_u + m\right]P_u$$

$$\frac{dP_d}{dt} = (1-\mu)r\left(1-\frac{P}{K}\right)P_d - \left[(1-\beta)\, f_H(P)H_u + m\right]P_d$$

$$\frac{dH_u}{dt} = \varepsilon\left[P_u + (1-\beta)P_d\right] f_H(P)H_u - \left[f_C(H_u)C + m\right]H_u$$

$$\frac{dC}{dt} = \left[\gamma H_u f_C(H_u) - m\right]C$$

with:

$$P = P_u + P_d$$

$$f_H(P) = \frac{v_{PH}}{1 + h_{PH}v_{PH}P_u + h_{PH}v_{PH}P_d}$$

$$f_C(H_u) = \frac{v_{HC}}{1 + h_{HC}v_{HC}H_u}$$

Web 6

Plastic variation. Autotroph defended. Herbivore undefended.

$$\frac{dP_u}{dt} = r\left(1-\frac{P}{K}\right)S_P(H_u)P - \left[f_H(P)\,H_u + m\right]P_u$$

$$\frac{dP_d}{dt} = (1-\mu)r\left(1-\frac{P}{K}\right)\left[1-S_P(H_u)\right]P - \left[\left(1-\beta\right)f_H(P)H_u + m\right]P_d$$

$$\frac{dH_u}{dt} = \varepsilon\left[P_u + \left(1-\beta\right)P_d\right]f_H(P)\,H_u - \left[f_C(H_u)C + m\right]H_u$$

$$\frac{dC}{dt} = \left[\gamma H_u\ f_C(H_u) - m\right]C$$

with:

$$P = P_u + P_d$$

$$S_P(H_u) = \left[1+\left(\frac{H_u}{g_H}\right)^{b_H}\right]^{-1}$$

$$f_H(P) = \frac{v_{PH}}{1+h_{PH}v_{PH}P_u + h_{PH}v_{PH}P_d}$$

$$f_C(H_u) = \frac{v_{HC}}{1+h_{HC}v_{HC}H_u}$$

Web 7

Genetic variation. Autotroph undefended. Herbivore defended.

$$\frac{dP_u}{dt} = r\left(1-\frac{P_u}{K}\right)P_u - \left[f_H(P_u)H + m\right]P_u$$

$$\frac{dH_u}{dt} = \varepsilon P_u f_H(P_u) H_u - \left[f_C(H)C + m\right]H_u$$

$$\frac{dH_d}{dt} = (1-\mu)\varepsilon P_u f_H(P_u) H_d - \left[\left(1-\beta\right) f_C(H)C + m\right]H_d$$

$$\frac{dC}{dt} = \left\{\gamma\left[H_u + \left(1-\beta\right)H_d\right] f_C(H) - m\right\}C$$

with:

$$H = H_u + H_d$$

$$f_H(P_u) = \frac{v_{PH}}{1 + h_{PH} v_{PH} P_u}$$

$$f_C(H) = \frac{v_{HC}}{1 + h_{HC} v_{HC} H_u + h_{HC} v_{HC} H_d}$$

Web 8

Plastic variation. Autotroph undefended. Herbivore defended.

$$\frac{dP_u}{dt} = r\left(1-\frac{P_u}{K}\right)P_u - \left[\, f_H(P_u)H + m\right]P_u$$

$$\frac{dH_u}{dt} = \varepsilon P_u \; f_H(P_u)\, S_H(C)\, H - \left[\, f_C(H)C + m\right]H_u$$

$$\frac{dH_d}{dt} = (1-\mu)\varepsilon P_u \; f_H(P_u)\left[1 - S_H(C)\right]H - \left[\left(1-\beta\right) f_C(H)C + m\right]H_d$$

$$\frac{dC}{dt} = \left\{\gamma\left[H_u + \left(1-\beta\right)H_d\,\right] f_C(H) - m\right\}C$$

with:

$$H = H_u + H_d$$

$$S_H(C) = \left[1+\left(\frac{C}{g_c}\right)^{b_C}\right]^{-1}$$

$$f_H(P_u) = \frac{v_{PH}}{1+h_{PH}v_{PH}P_u}$$

$$f_C(H) = \frac{v_{HC}}{1+h_{HC}v_{HC}H_u + h_{HC}v_{HC}H_d}$$

Web 9

Genetic variation. Autotroph defended. Herbivore defended.

$$\frac{dP_u}{dt} = r\left(1-\frac{P}{K}\right)P_u - \left[f_H(P)H + m\right]P_u$$

$$\frac{dP_d}{dt} = (1-\mu)r\left(1-\frac{P}{K}\right)P_d - \left[(1-\beta)\, f_H(P)H + m\right]P_d$$

$$\frac{dH_u}{dt} = \varepsilon\left[P_u + (1-\beta)P_d\right] f_H(P)\, H_u - \left[f_C(H)C + m\right]H_u$$

$$\frac{dH_d}{dt} = (1-\mu)\varepsilon\left[P_u + (1-\beta)P_d\right] f_H(P)H_d - \left[(1-\beta)\, f_C(H)C + m\right]H_d$$

$$\frac{dC}{dt} = \left\{\gamma\left[H_u + (1-\beta)H_d\right] f_C(H) - m\right\}C$$

with:

$$H = H_u + H_d$$

$$P = P_u + P_d$$

$$f_H(P) = \frac{v_{PH}}{1 + h_{PH}v_{PH}P_u + h_{PH}v_{PH}P_d}$$

$$f_C(H) = \frac{v_{HC}}{1 + h_{HC}v_{HC}H_u + h_{HC}v_{HC}H_d}$$

Web 10

Plastic variation. Autotroph defended. Herbivore defended.

$$\frac{dP_u}{dt} = r\left(1-\frac{P}{K}\right)S_P(H)P - \left[f_H(P)H + m\right]P_u$$

$$\frac{dP_d}{dt} = (1-\mu)r\left(1-\frac{P}{K}\right)\left[1-S_P(H)\right]P - \left[\left(1-\beta\right)f_H(P)H + m\right]P_d$$

$$\frac{dH_u}{dt} = \varepsilon\left[P_u + \left(1-\beta\right)P_d\right]f_H(P)S_H(C)H - \left[f_C(H)C + m\right]H_u$$

$$\frac{dH_d}{dt} = (1-\mu)\varepsilon\left[P_u + \left(1-\beta\right)P_d\right]f_H(P)\left[1-S_H(C)\right]H - \left[\left(1-\beta\right)f_C(H)C + m\right]H_d$$

$$\frac{dC}{dt} = \left\{\gamma\left[H_u + \left(1-\beta\right)H_d\right]f_C(H) - m\right\}C$$

with:

$$H = H_u + H_d$$

$$P = P_u + P_d$$

$$S_P(H) = \left[1+\left(\frac{H_u + H_d}{g_H}\right)^{b_H}\right]^{-1}$$

$$S_H(C) = \left[1+\left(\frac{C}{g_c}\right)^{b_C}\right]^{-1}$$

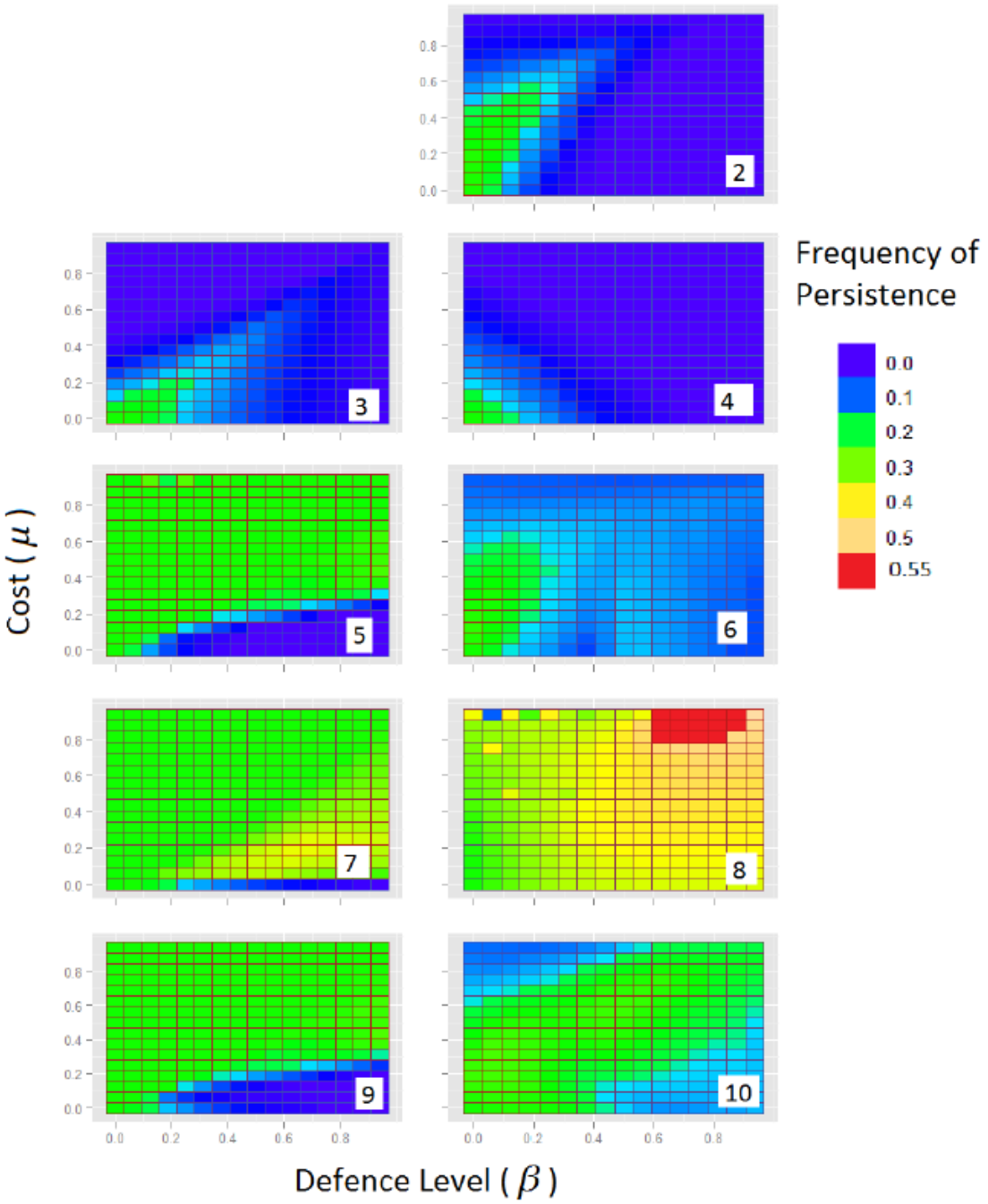
$$f_H(P) = \frac{v_{PH}}{1 + h_{PH}v_{PH}P_u + h_{PH}v_{PH}P_d}$$

$$f_C(H) = \frac{v_{HC}}{1 + h_{HC}v_{HC}H_u + h_{HC}v_{HC}H_d}$$

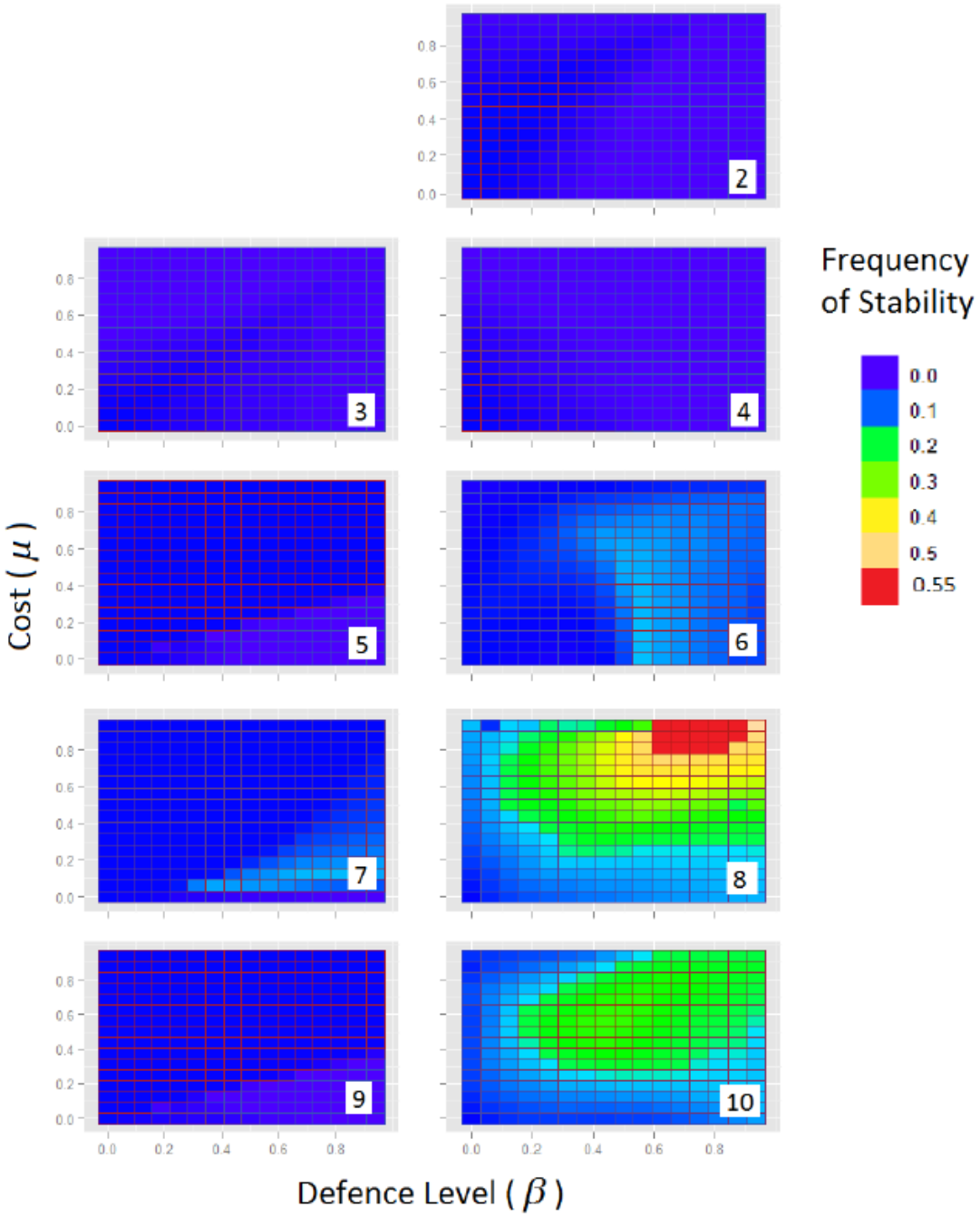
Appendix 2.B- Supplementary Figures

The frequency of (1) Persistence; (2) Dynamic stability; (3) Stability given persistence across different costs (μ:y axis) and benefits (β:x axis). Warmer colours signify higher frequencies of persistence or equilibria, respectively [note: the scale changes in (2.B.3)]. The number in the lower right hand corner corresponds to the number of the food web.

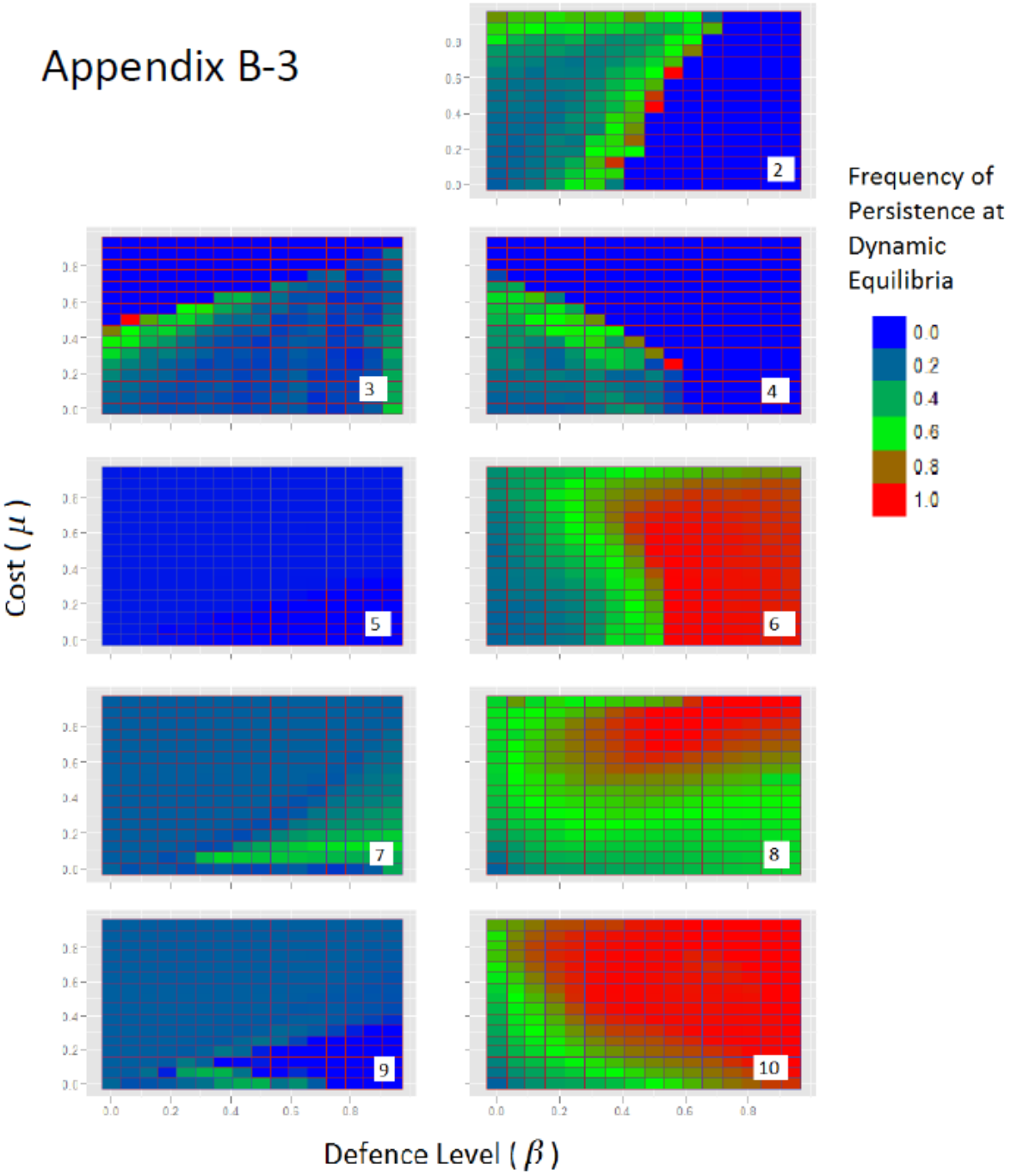
Appendix B-1


2
3
4
5
6
7
8
9
10
Frequency of Persistence
0.0
0.1
0.2
0.3
0.4
0.5
0.55
Cost (μ)
Defence Level (β)

Appendix B-2


2
3
4
5
6
7
8
9
10
Frequency of Stability
0.0
0.1
0.2
0.3
0.4
0.5
0.55
Cost (μ)
Defence Level (β)

Appendix B-3


2
3
4
5
6
7
8
9
10
Frequency of Persistence at Dynamic Equilibria
0.0
0.2
0.4
0.6
0.8
1.0
Cost (μ)
Defence Level (β)

Appendix 2.C-Expanded Details for Table 2.2

Frequencies of persistence and equilibrium dynamics across food web simulations where μ>β. Average percentages for ten food web categories that differ in degree, trophic location and source of variability. Persistence: percent of simulations with ≥ 1 population persisting at each trophic level. Equilibria: percent of simulations with stable, non-oscillatory dynamics. Only simulations exhibiting persistence were analysed for stability but percent values are given as the fraction of all simulations where μ>β.

Mu < Beta

Web Number	Trophic Level of Defense	Trophic Level of Variation	Source of Variation	Persistence %	Equilibrium %
1	None	None	None	24.05	4.74
2	Autotroph	None	None	1.05	0.34
3	Herbivore	None	None	6.56	1.01
4	Both Levels	None	None	1.94	0.42
5	Autotroph	Autotroph	Genetic	14.69	2.56
6	Autotroph	Autotroph	Plastic	12.62	9.86
7	Herbivore	Herbivore	Genetic	27.05	6.80
8	Herbivore	Herbivore	Plastic	39.22	21.06
9	Both Levels	Both Levels	Genetic	15.66	2.56
10	Both Levels	Both Levels	Plastic	19.38	16.11

CONNECTING STATEMENT

The work presented in Chapter 2 used tritrophic models to discern the comparative effects of different sources and trophic locations of variation. I considered stable coexistence (i.e., dynamic stability) and persistence (i.e., non-extreme amplitude cyclic coexistence) as proxies for 'rescue'. In Chapter 3, I deeply explore the dynamical consequences of inducible defenses by looking at how the category of defense affects model predictions. In order to accomplish my goals, I use a simpler bitrophic system and consider community dynamics in the more traditional framework of stable coexistence instead of 'rescue'. However, the results presented in Chapter 3 could very easily be applied to a study of community rescue.

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CHAPTER 3

DYNAMICAL CONSEQUENCES OF INDUCIBLE DEFENSE TRAITS

Caolan Kovach-Orr, Matthijs Vos, & Gregor F. Fussmann (soon to be submitted to *Journal of Animal Ecology*). Dynamical Consequences of Inducible Defense Traits.

3.1 Abstract

In natural systems, there are three major categories of inducible defense: pre-encounter defense, post-encounter defense, and post-consumption defense. However, most investigations into the effects of plastic defenses are limited to the dynamical consequences of a single category of defense. We use models of bitrophic systems to investigate how different categories of inducible defense affect model predictions in the predator mortality-carrying capacity parameter space. Our findings help to harmonize the conflicting results produced by previous comparative studies. We show that plastic defenses can decrease the risk of extinctions due to population oscillations and that clear hierarchies exist at both low and high carrying capacities. Pre-encounter inducible defenses are most likely to promote stable coexistence at low carrying capacities, whereas post-encounter and post-consumption inducible defenses are most likely to promote stable coexistence at high carrying capacities.

Key words: inducible defenses, model specification, strong stability, functional response, predator-prey

3.2 Introduction

Inducible defense is a special form of adaptive phenotypic plasticity; it allows prey that have encountered predators to express defense traits without any changes to the underlying genetic structure of the prey population (Harvell 1990). Inducible defenses have been documented across a wide range of habitats and taxa (reviewed in (Havel 1987; Karban 1997; Tollrian & Harvell 1999)); and defenses can arise from changes in morphological (Gilbert & Waage 1967), behavioral (Lampert 1989), physiological (Harvell 1990), or chemical traits (Rhoades 1985).

In this paper, we are concerned with how inducible defenses affect predator-prey population dynamics. It is intuitively understandable that inducible defenses can promote the stable coexistence of predators and prey because such defenses create negative feedback loops; i.e., when predators are abundant, the induction of defenses provides prey with refuge from predation, and when predators are sparse, the decay of defenses provides predators with resources (Vos *et al.* 2004a; Miner *et al.* 2005; DeAngelis 2013; Kovach-Orr & Fussmann 2013). This general reasoning has been confirmed by empirical and theoretical studies that found inducible defenses to be stabilizing, either by preventing extinction of the prey or by dampening oscillatory predator-prey dynamics to small amplitude fluctuations and equilibria (Ramos-Jiliberto 2003; Verschoor, Vos & van der Stap 2004; Vos *et al.* 2004a; Ramos-Jiliberto, Frodden & Aranguiz-Acuna 2007; Ramos-Jiliberto & Garay-Narvaez 2007; van der Stap *et al.* 2009; Cortez 2011; Yamamichi, Yoshida & Sasaki 2011; Kovach-Orr & Fussmann 2013). On the other hand, theoretical studies have used many different mathematical formulations to incorporate inducible defenses into ecological models, reflecting different mechanistic views about how the defense affects the predator-prey interaction. This paper explores whether the use

of alternative inducible defense formulations leads to different predictions in dynamical predator-prey models.

Categorizing inducible defenses by changes in morphological, behavioral, physiological, or chemical traits is of great operational value but is not a key distinction when it comes to translating the defense mechanism into mathematical language (Miner *et al.* 2005). For instance, a behavioral change in a fish that results in hiding behind ocean rocks may have an impact that is very similar to that of a physiological color change in an octopus that results in being camouflaged as a rock. Conversely, the same morphological defense (e.g., the long posterolateral spines of the rotifer *Brachionus calyciflorus* that can be spread apart upon physical contact with a predator (Gilbert 1966) may have very different effects depending on whether the specific predator experiences increased handling time or reduced attack rate due to the defense – or varying degrees of both. In the dynamical context of our study, we therefore classified inducible defenses based on how they affect the predator-prey interaction, rather than adopting the traditional scheme. Broadly speaking, we distinguish three categories of inducible defense: (1) Pre-encounter defenses, which decrease the predator's attack rate (Ramos-Jiliberto, Frodden & Aranguiz-Acuna 2007). (2) Post-encounter defenses, which increase the predator's handling time (Ramos-Jiliberto, Frodden & Aranguiz-Acuna 2007). (3) Post-consumption defenses, which decrease the predator's conversion efficiency. Inducible defenses that are combinations of these categories are also possible. Some commonly observed effects of inducible defenses emerge as special cases of these three categories. Defenses can allow prey to completely avoid predation (i.e., the attack rate on defended individuals drops to 0); prey can completely disrupt the predators consumption process (i.e.; handling time on defended individuals effectively goes towards infinity); prey can be nutritionally worthless (i.e., conversion efficiency on defended

individuals drops to 0), or even be toxic to predators (i.e., conversion efficiency on defended individuals becomes negative, as is seen with cyanobacteria and *Daphnia* (Lampert 1981)).

In our study we link biologically plausible defense mechanisms to commonly used mathematical formulations. Our goal was to systematically compare the dynamical consequences of incorporating these mechanisms into the same benchmark predator-prey model. Although all inducible defenses will stabilize the dynamics in some way, we expected differences in many ecologically important attributes such as the strength and endpoint of stabilization or the parameter ranges for which stabilization occurs. Some preliminary work on this topic has been done prior to our study (Vos *et al.* 2004a; Ramos-Jiliberto, Frodden & Aranguiz-Acuna 2007; Garay-Narvaez & Ramos-Jiliberto 2009; Kovach-Orr & Fussmann 2013), but a comparative investigation is needed because many theoretical studies claim to study the general effect of inducible defenses in a given dynamical model context, but offer only one specific mechanistic implementation (Vos *et al.* 2004a; Vos *et al.* 2004b; Cortez 2011; Yamamichi, Yoshida & Sasaki 2011; Kovach-Orr & Fussmann 2013). It appears that this practice may be suitable for the description of a specific empirical predator-prey interaction but cannot be employed to establish some degree of generality.

To evaluate how different categories of inducible defenses affect the stable coexistence of predators and prey, we used mathematical models of bitrophic systems based on Vos *et al.* (2004a). We performed bifurcation analyses to establish the relative presence of stable coexistence *vs*. oscillatory dynamics with respect to ranges of two model parameters: predator mortality and carrying capacity. We used predator mortality because it determines the force of top-down control on the system, and carrying capacity because it affects bottom-up control (Vos *et al.* 2004a). As our results will show, it is important to consider both of these parameters

simultaneously (i.e., investigate the mortality-capacity parameter plane) because the region of stable coexistence has a complex, interactive relationship with these parameters, and evaluating stable coexistence in only one dimension can produce potentially misleading predictions.

3.3 Models

In our analyses, the prey species can either be constitutively undefended (i.e. all prey are undefended, regardless of predator density) or inducibly defended (i.e. the proportion of defended individuals is directly dependent on predator density). These analyses of inducible defense are based on, and parameterized for, a "real" rotifer-algal system that exhibits inducible defenses (Hessen & Vandonk 1993; Verschoor *et al.* 2004; Vos *et al.* 2004a; van der Stap *et al.* 2009). Constitutively undefended prey can be modeled using parameter values, based on units of carbon per liter, and rates, measured in time related units, that correspond to the undefended prey state (Table 3.A.1). This system can then be investigated using a Rosenzweig-MacArthur (Rosenzweig & MacArthur 1963) model of bitrophic predator-prey systems that has been modified to include density-independent prey mortality (Vos *et al.* 2004a) :

$$\frac{\mathrm{d}x}{\mathrm{d}t} = x\left(r\left(1-\frac{x}{K}\right) - \frac{a\,y}{1+a\,h\,x} - m_x\right) \tag{3.1a}$$

$$\frac{\mathrm{d}y}{\mathrm{d}t} = y\left(\frac{\varepsilon\,a\,x}{1+a\,h\,x} - m_y\right) \tag{3.1b}$$

where the prey x experience a maximum growth rate r, grow logistically to a carrying capacity K, and have a natural mortality rate m_x. Predators y, consume prey with attack rate a, handling time h, convert prey biomass with efficiency ε, and experience a natural mortality rate m_y. This classic model is particularly well known for its tendency to destabilize from stable coexistence to

sustained oscillations as carrying capacity, *K*, increases, a dynamic behavior known as the "paradox of enrichment" (Rosenzweig 1971).

Inducible defenses can be incorporated into system (3.1) by the addition of a second prey population and induction/decay functions that allow prey to switch between undefended and defended phenotypes (Vos *et al.* 2004a):

$$\frac{\mathrm{d}x_1}{\mathrm{d}t} = x_1\left(r_1\left(1-\frac{x_1+x_2}{K}\right)-\frac{a_1 y}{1+a_1h_1x_1+a_2h_2x_2}-m_{x1}\right)-$$

$$i\,x_1\left(1-\left(1+\left(\frac{y}{g}\right)^b\right)^{-1}\right)+i\,x_2\left(1+\left(\frac{y}{g}\right)^b\right)^{-1} \qquad (3.2a)$$

$$\frac{\mathrm{d}x_2}{\mathrm{d}t} = x_2\left(r_2\left(1-\frac{x_1+x_2}{K}\right)-\frac{a_2 y}{1+a_1h_1x_1+a_2h_2x_2}-m_{x2}\right)+$$

$$i\,x_1\left(1-\left(1+\left(\frac{y}{g}\right)^b\right)^{-1}\right)-i\,x_2\left(1+\left(\frac{y}{g}\right)^b\right)^{-1} \qquad (3.2b)$$

$$\frac{\mathrm{d}y}{\mathrm{d}t} = y\left(\frac{\varepsilon_1 a_1 x_1+\varepsilon_2 a_2 x_2}{1+a_1h_1x_1+a_2h_2x_2}-m_y\right) \qquad (3.2c)$$

where index 1 relates to the undefended population and index 2 relates to the defended population. Prey grow logistically to a joint carrying capacity *K*, which applies to the sum of the density of the two phenotypes. The induction and decay of defenses follow sigmoid functions with a maximum rate of induction or decay *i*. The predator density *g* at which induction or decay reaches half of its maximum rate and a scaling parameter *b* control the shape of these functions. As *y* approaches 0, the rate of induction approaches 0, while the rate of decay approaches *i*; as *y* approaches infinity, the rate of induction approaches *i* , while the rate of decay approaches 0.

In system (3.2), prey are considered defended if $a_1 > a_2$ (pre-encounter defense), $h_1 < h_2$ (post-encounter defense), or $\varepsilon_1 > \varepsilon_2$ (post-consumption defense) [note: it is also possible that prey exhibit a combination of these defenses]. The original rotifer-algal system, on which our parameterization is based, only exhibits post-encounter inducible defenses (colony formation) that result in increased mortality for the defended prey (Table 3.A.3). To investigate pre-encounter and post-consumption inducible defenses we standardized the relative change in the defense trait and applied this change to the respective parameter for each category of inducible defense (Tables 3.A.2 and 3.A.4). Although this represents just one parameterization for each category of inducible defense, our results provide insights into the kind of changes produced by inducible defenses; additional analyses are provided in Appendix 3.B. For each category of defense, we also investigated the limit case where the defense is completely effective. For pre-encounter inducible defenses this occurs when $a_1 > a_2$ and $a_2 = 0$ (representing a defended prey population that can completely avoid predators), for post-encounter inducible defenses this occurs when $h_1 < h_2$ and $h_2 \to \infty$ (representing a defended prey population that fully disrupts the predation process), and for post-consumption inducible defenses this occurs when $\varepsilon_1 > \varepsilon_2$ and $\varepsilon_2 \leq 0$ (representing a defended prey population that is nutritionally worthless or even toxic to predators).

3.4 Model Analysis

We analyzed a model that lacks inducible defenses, three different categories of inducible defenses (pre-encounter, post-encounter, and post-consumption inducible defenses), as well as their limit cases, for their impact on the stable coexistence of predators and prey. In the m_y - K parameter space, the region of stable coexistence is separated from the region of deterministic

predator extinction by the transcritical bifurcation. Specifically, at any given *K*, predator mortality rates above the transcritical bifurcation cause deterministic predator extinction. The region of stable coexistence is separated from the region of coexistence on stable limit cycles by the supercritical Hopf bifurcation. Specifically, at any given *K*, predator mortality rates below the Hopf bifurcation cause oscillatory dynamics. The relationship between carrying capacity and the breadth of stability, i.e. the change in distance between the transcritical and Hopf bifurcations over a range of *K* values, can be used to classify models into fundamentally different categories of model stability known as "weak" , "strong", and "complete" stability (Kretzschmar, Nisbet & McCauley 1993; van Voorn *et al.* 2008). Weak stability occurs when the Hopf bifurcation approaches the same asymptotic limit as the transcritical bifurcation, as carrying capacity approaches infinity (i.e. the size of the breadth of stability approaches 0) (Fig. 3.1 a, b). Strong stability occurs when the Hopf bifurcation approaches a lower asymptotic limit than the transcritical bifurcation, as carrying capacity is increased (i.e. the size of the breadth of stability never approaches 0) (Fig. 3.1 c, d) (Kretzschmar, Nisbet & McCauley 1993; Vos *et al.* 2004a). Complete stability can be seen as the limit case of strong stability, in that complete stability occurs when systems do not exhibit limit cycles anywhere in the m_y -*K* plane (i.e. the Hopf bifurcation is absent) (Fig. 3.1 e) (van Voorn *et al.* 2008).

Important differences in system behavior can also arise at finite values of *K* for model systems that exhibit weak or strong stability. For instance, models may differ in the carrying capacity that is required to cross the Hopf bifurcation as $m_y \rightarrow 0$ (i.e., stable coexistence gives way to sustained oscillations); when predators consume prey with a type 2 functional response (as in systems (3.1) and (3.2)), this model property corresponds to the minimum carrying capacity that can produce sustained population oscillations. Additionally, because models may

differ in their specific relationship between carrying capacity and breadth of stability, it is important to identify the conditions that allow different models to have relatively larger or smaller breadths of stability. Therefore, in order to visualize the breadth of stability at finite values of *K*, we created bifurcation diagrams, which can illustrate how the breadth of stability changes at biologically plausible parameter values. Because of the complex nature of inducible defense systems, it is not possible to generate exact analytical solutions for finite values of *K*. Therefore, we used MATLAB (2013) and the associated package MatCont (Govaerts & Kuznetsov 2013) for numerical bifurcation analysis of all bitrophic models.

At extreme values of *K*, bifurcation analyses become computationally expensive; therefore, in order to show the dynamical consequences of inducible defenses at extreme values of *K*, we turned to numerical simulations of time series which show dynamics for a single parameter combination. These simulations were performed using MATLAB (2013). Initial population densities were $x_1 = x_2 = 10^5$ mg C L^{-1}, and y=100 mg C L^{-1}. Carrying capacity was set equal to 10^6 mg C L^{-1}. To avoid transient dynamics, we evaluated stable coexistence in numerical simulations from t=900,000 to t=1,000,000. If there was less than a 1% difference between the maximum and minimum populations' densities over this range, the system was considered to exhibit stable coexistence.

3.5 Results

We start by showing that the presence and nature of inducible defenses can affect model predictions of stable coexistence at low (i.e. finite) carrying capacities. Table 3.1 shows the carrying capacity that is required to cross the Hopf bifurcation as $m_y \rightarrow 0$ (i.e., equilibrium dynamics give way to stable limit cycles) for models with no defenses as well as models that

incorporate inducible defenses. When compared to the no defense model, it is immediately clear that pre-encounter inducible defenses allow for stable coexistence at higher carrying capacities and, therefore, have the potential to mitigate the destabilizing effects of nutrient enrichment (Table 3.1; Fig. 3.1, intersection of K axis with the blue curve). On the other hand, post-encounter inducible defenses can intensify the impact of enrichment by allowing the Hopf bifurcation to occur at lower values of K than the model that lacks defenses (Table 3.1). Incorporating post-consumption inducible defenses has very little effect on the Hopf bifurcation as $m_y \rightarrow 0$ (Table 3.1); in fact, the slight difference between models with no defenses and post-consumption inducible defenses is almost entirely due to the costs associated with defended phenotypes (i.e. increased mortality) (Appendix 3.B).

The K value for which the Hopf bifurcation occurs as $m_y \rightarrow 0$ is an initial assessment of stabilization through induced defenses; bifurcation analyses offer a more complete picture of the boundaries of stable coexistence across the whole m_y-K parameter space. (Note: the transcritical bifurcation is identical for all models because as equilibrium predator density approaches 0, the decay function approaches its maximum, and all prey become undefended.) As K increases, model predictions diverge rapidly and no longer predict similar boundaries of stable coexistence for the cases of no defenses and post-consumption inducible defenses (Fig. 3.1 a, d). In fact, the breadth of stability for the model with no defenses rapidly shrinks relative to any of the inducible defense models (Fig. 3.1); the values of m_y and K where the Hopf bifurcations intersect are given in Table 3.2.

Important distinctions exist between the different classes of inducible defenses. As carrying capacity increases, the breadth of stability for pre-encounter inducible defenses decreases and eventually becomes smaller than the breadth of stability for post-encounter and

post-consumption inducible defenses (Table 3.2; Fig. 3.1 b, c, d). For values of $K > 14.2$ mg C L^{-1}, the breadth of stability is larger for post-encounter than pre-encounter inducible defenses, and vice versa for smaller values of K (Table 3.2). A similar critical point exists at $K = 10.9$ mg C L^{-1} for pre-encounter vs. post-consumption inducible defenses, whereas the Hopf bifurcations for post-encounter and post-consumption inducible defenses never intersect (Table 3.2; Fig. 3.1 c, d); the breadth of stability is always greater for post-consumption inducible defenses.

In addition to the differences in model predictions at finite values of K, the presence and nature of inducible defenses also affects stable coexistence at infinite values of K. It is well known that system (3.1) is incapable of producing strong stability (Rosenzweig 1971; Kretzschmar, Nisbet & McCauley 1993; Vos *et al.* 2004a; Kovach-Orr & Fussmann 2013). Vos *et al.* (2004a) derived that post-encounter and post-consumption inducible defenses allow for strong stabilization as K approaches infinity, as long as $\frac{\varepsilon_2}{h_2} < m_y < \frac{\varepsilon_1}{h_1}$. Our results support that very strong post-consumption inducible defenses (i.e. $\varepsilon_2 \leq 0$) will lead to stable coexistence for all $m_y < \frac{\varepsilon_1}{h_1}$, as K approaches infinity (Fig. 3.2 b); however, only trivial stable coexistence occurs when h_2 equals infinity (Fig. 3.2 c). Specifically, when h_2 equals infinity, the presence of any defended prey will cause the predators' consumption of both prey types to equal 0. This, combined with the fact that in system (3.2) the presence of the predator causes at least some prey to be defended, makes it intuitively understandable that if h_2 equals infinity and $m_y > 0$, predators and inducibly defended prey cannot coexist.

Our analyses have also identified that the limit case of pre-encounter inducible defense, where defended prey completely avoid predators (i.e. $a_2=0$) , allows for not just strong, but complete, stabilization. Numerical time series (Fig. 3.3 a, b) show that when $a_2=0$, system (3.2) does not exhibit the Hopf bifurcation for any $m_y \geq 0$, even at extreme values of K (Figs. 3.2 a, 3.3

a, b); although Fig. 3.3 only includes two predator mortality rates and one extreme value for *K*, we have confirmed that other predator mortality rates and higher carrying capacities also produce stable coexistence (data not shown). On the other hand, even very small positive values of a_2 cannot produce complete, or even strong, stability (Fig. 3.3 c).

3.6 Discussion

Previous work has shown that inducible defenses have the potential to promote stable coexistence in the m_y -*K* parameter space (Vos *et al.* 2004b; Ramos-Jiliberto, Frodden & Aranguiz-Acuna 2007; Ramos-Jiliberto & Garay-Narvaez 2007; van der Stap, Vos & Mooij 2007; Cortez 2011; Yamamichi, Yoshida & Sasaki 2011; Kovach-Orr & Fussmann 2013). Furthermore, inducible defenses have been shown to offer a potential resolution to the paradox of enrichment by promoting stable coexistence at extreme levels of *K*; a result that has been corroborated by laboratory studies (Verschoor, Vos & van der Stap 2004; van der Stap *et al.* 2009). However, previous work has also shown that under some conditions, the presence of inducible defenses can impede stable coexistence, even when compared to systems without inducible defenses (Yamamichi, Yoshida & Sasaki 2011; Kovach-Orr & Fussmann 2013).

Surprisingly, little attention has been given to the effect of different categories of inducible defenses. The few comparative studies that do exist have indicated that systems without inducible defenses are less likely to exhibit stable coexistence than systems with pre-encounter (Vos *et al.* 2004a; Ramos-Jiliberto, Frodden & Aranguiz-Acuna 2007), post-encounter (Vos *et al.* 2004a; Ramos-Jiliberto, Frodden & Aranguiz-Acuna 2007), or post-consumption (Vos *et al.* 2004a) inducible defenses. However, these studies have produced conflicting predictions of which category of inducible defense is most likely to promote stable coexistence.

Vos *et al.* (2004a) concluded that post-encounter and post-consumption inducible defenses are more likely to promote stable coexistence than pre-encounter inducible defenses. Furthermore, Vos *et al.'s* (2004a) analytical solutions imply that increasing the effectiveness of post-encounter and post-consumption inducible defenses will further promote stable coexistence. On the other hand, Ramos-Jiliberto, Frodden & Aranguiz-Acuna (2007) compared pre-encounter and post-encounter, but not post-consumption, inducible defenses and found that post-encounter inducible defenses create more rich (i.e., variable) dynamics than pre-encounter inducible defenses. Additionally, Ramos-Jiliberto, Frodden & Aranguiz-Acuna (2007) found finite increases to the effectiveness of pre-encounter and/or post-encounter inducible defenses will first promote and then potentially impede stable coexistence.

Our analyses indicate that two factors contributed to the discrepancies between the conclusions of Vos *et al* .(2004a) and Ramos-Jiliberto, Frodden & Aranguiz-Acuna (2007). First, unlike Ramos-Jiliberto, Frodden & Aranguiz-Acuna (2007), Vos *et al.'s* (2004a) comparative study of different forms of inducible defenses did not consider dynamical consequences at low levels of enrichment; instead, Vos *et al.'s* (2004a) analyses were limited to the effects of inducible defenses as carrying capacity approached(s) infinity. This is especially important in light of our results, which indicate pre-encounter inducible defenses transition from most likely to least likely to promote stable coexistence as carrying capacity increases (Fig. 3.1 b, c, d. Table 3.1).

Second, Ramos-Jiliberto, Frodden & Aranguiz-Acuna (2007) used a fundamentally different approach towards natural mortality rates and incorporated negative density dependence in both predators and prey. Specifically, in Ramos-Jiliberto, Frodden & Aranguiz-Acuna's (2007) model, the natural mortality rate was equal to 0.1 × population density. Such self-

limitation may make sense for some systems (Agusti 1991; Kirk 1998; Burns 2000); however, we feel Vos *et al.'s* (2004a)/our model is more appropriate for the study of stable coexistence and enrichment for three reasons. First, imposing self-limiting mortality rates constrains the biological relevance of model predictions. Not all populations experience increased mortality in the face of increased conspecific density; in fact, many populations have been shown to exhibit the exact opposite phenomenon: positive density dependence (Milinski 1984; Getz *et al.* 1993; Kohler & Huth 1998; Kie 1999). On the other hand, some populations do exhibit self-limiting mortality rates; however, the intensity of self-limitation can differ between populations (Harvell 1990; van der Stap, Vos & Mooij 2006). This is especially important because models with high levels of self-limitation may not capture certain phenomena – for example, if the prey population is self-limited to low density, prey may be unable to escape predator control.

Second, negative density dependent mortality rates are generally stabilizing (Ramos-Jiliberto 2003); however, we are more interested in the role that different forms of inducible defenses may play in promoting the stable coexistence of more 'at risk' populations that do not have the benefit of self-limiting mortality rates.

Third, because mortality rate is directly dependent on only population density in Ramos-Jiliberto, Frodden & Aranguiz-Acuna's (2007) model, one cannot isolate the effects of environmental changes that directly result in increased natural mortality rates. Natural mortality rates can be increased indirectly, but only if environmental changes result in increased resource availability and/or decreased predation. Furthermore, because the predator mortality rate increases with predator population density, incorporating self-limitation creates a 'bizarre' phenomenon where the frequency of defended prey is always positively correlated with the

predator mortality rate. While these phenomena may co-occur, they are fundamentally separate processes, and therefore deserve individual consideration.

Although predator mortality rate is not explicitly tied to the frequency of defended prey, these forces do interact in the Vos *et al.* (2004a)/our model framework. For example, because increased predator mortality results in fewer predators, fewer prey exhibit defenses. In fact, for high levels of enrichment, this relationship can allow for stable coexistence. Specifically, it allows inducibly defended prey to limit the predator population to very low densities (data not shown); this mitigates the destabilizing effects of enrichment because predators are unable to overexploit prey, and therefore, prey escape does not result in sustained oscillations. This phenomenon is the basis for the ability of all three categories of inducible defense to promote stable coexistence at high levels of enrichment. Both post-encounter and post-consumption inducible defenses reduce the maximum resource uptake rate of predators; therefore, they also lower the asymptotic limit of the Hopf bifurcation, as K approaches infinity (Vos *et al.* 2004a) this, in turn, allows post-encounter and post-consumption inducible defenses to create strong stability (Vos *et al.* 2004a). Furthermore, at high K values, as post-encounter and post-consumption inducible defenses become completely effective (i.e., approach their limit cases), the band of cyclic coexistence at low m_y becomes increasingly small and the band of strong stability becomes increasingly large. On the other hand, pre-encounter inducible defenses (where $a_2 \geq 0$) cannot create strong stability because they do not affect the asymptotic limit of the Hopf bifurcation as K approaches infinity (Vos *et al.* 2004a).

A different mechanism governs the stable coexistence of predators and prey at lower predator mortality rates. Low mortality rates can allow predators to grow to high density, even when all prey are defended; this, in turn, causes predators to overexploit prey, a subsequent

reduction in predator density, which is followed by prey escape, and ultimately, sustained population oscillations. Upon visual inspection of figure 3.1 (a, b, c, d), it becomes immediately clear that post-encounter, but not pre-encounter or post-consumption inducible defenses can result in population oscillations at lower values of *K* than the model without inducible defenses (also see Table 3.1). To the best of our knowledge, no previous work has shown that incorporating inducible defenses may promote or impede stable coexistence [compared to no defenses**]** depending on the implementation of defense.

At low predator mortality rates and carrying capacities, post-encounter inducible defenses accelerate destabilization due to enrichment because increased handling time forces predators to approach their maximum consumption rate at lower prey densities; thus, increases in prey density are not met with increases in consumption and prey can more easily escape predator control (Turchin 2003; Kovach-Orr *et al. unpublished manuscript*). Furthermore, the low predator mortality rate allows unsustainably high predator densities, which perpetuates cyclic coexistence. On the other hand, reducing the attack rate (i.e. pre-encounter inducible defense) causes predators to approach their maximum consumption rate at higher prey densities; thus, increases in prey density are bet with increases in consumption and prey can less easily escape predator control at low levels of enrichment. While reducing the conversion efficiency (i.e., post-consumption inducible defense) does affect the absolute resource uptake rate, it has no effect on the relationship between prey density and consumption rate.

Furthermore, in Appendix 3.B, we show that increasing the effectiveness of pre-encounter and/or post-encounter inducible defenses will continuously intensify these effects. Therefore, post-encounter inducible defenses cannot promote stability at high *K* values without impeding stability at low values of *K*. On the other hand, fact, our results indicate that as $a_2 \rightarrow 0$,

the K value (of the Hopf as $m_y \rightarrow 0$) approaches infinity (Fig. 3.3, Appendix 3.B). Because the Hopf bifurcation never enters the positive m_y-K parameter space, the system will not exhibit sustained oscillations for any combinations of predator mortality rate and carrying capacity (Fig 3.3): a phenomenon known as complete stability (van Voorn *et al.* 2008). Surprisingly, this implies that strong stability is not a pre-requisite for complete stability.

Our analyses also show that for $m_y > 0$, stable coexistence is impossible for the limit case of post-encounter inducible defense (i.e. $h_2 \rightarrow \infty$), where it is all but guaranteed for the limit cases of pre-encounter and post-consumption inducible defenses (Fig 3.3 a, b). Interestingly, all three of the limit cases (a_2=0, $h_2 \rightarrow \infty$, and $\varepsilon_2 \leq 0$) result in predators being unable to absorb biomass from defended prey; however, very strong post-encounter defenses (i.e. $h_2 \rightarrow \infty$) also prevent predators from absorbing biomass from undefended prey.

We acknowledge that our analyses consider simplified systems and natural communities are much more complex. For instance, it is possible that prey exhibit a combination of inducible defenses (Van Buskirk & McCollum 2000; Kovach-Orr & Fussmann 2013) or different forms of costs (Via & Lande 1985; Via *et al.* 1995); however, the specific nature of costs has been shown to have little effect (Ramos-Jiliberto 2003) and we believe that it is important to understand simple systems before investigating more complex communities and processes. Nevertheless, our results indicate predictable, definitive, and consistent relationships between the form of inducible defense, the effectiveness of defense, and stable coexistence in the m_y -K parameter space (Appendix 3.B). Furthermore, we showed that inducible defenses can decrease the risk of extinctions due to population oscillations and that clear hierarchies exist at both low and high carrying capacities (Table 3.1; Fig. 3.1).

From a biological perspective, all three categories of inducible defense are likely relevant to natural communities. The effectiveness of pre-encounter inducible defenses at finite carrying capacities, and the realization that pre-encounter inducible defenses include behavioral changes in addition to morphological, chemical, and physiological changes, suggest that this form of inducible defense may have the largest impact on the stable coexistence of predators and prey. Nevertheless, the ability of inducible defenses to promote stable coexistence will ultimately depend on both the biology of the organisms in question, as well as the abiotic factors of carrying capacity and predator mortality rate.

3.7 Acknowledgements

G.F.F and C.K.O. acknowledge support from the James S. McDonnell Foundation and NSERC. G.F.F was also supported by the EU project FEMMES (MTKD-CT-2006-042261). G.F.F and C.K.O. thank Ursula Gaedke and her group at the University of Potsdam, Germany for hosting our research stays and for discussions. We are grateful to Consortium Laval, Université du Québec, McGill and Eastern Québec (CLUMEQ) and Compute Canada for the use of the Guillimin Cluster.

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3.9 Tables

Table 3.1. Model predictions of stable coexistence for inducible defense (ID) food webs. Column 2: the parameter and value of the defense trait. Column 3: the K value required to cross the supercritical Hopf bifurcation as $m_y \rightarrow 0$. For these bitrophic systems, this represents the minimum carrying capacity required for the community to transition from stable coexistence to sustained cyclic coexistence. Column 4: model predictions of the breadth of stability as K approaches infinity. Additionally, pre-encounter ID* refers to the limit case where attack rate on defended individuals is equal to 0. Additional Parameter values are given in Tables 3.A.1, 3.A.2, 3.A.3, and 3.A.4.

Web	defense term	K value of Hopf as $m_y \rightarrow 0$	stable coexistence as K approaches infinity
undefended	- NA -	2.893 mg C L^{-1}	weak stability
pre-encounter ID	$a_2 = 0.370$	6.188 mg C L^{-1}	weak stability
post-encounter ID	$h_2 = 1.040$	1.431 mg C L^{-1}	strong stability
post-consumption ID	$\varepsilon_2 = 0.173$	2.976 mg C L^{-1}	strong stability
pre-encounter ID*	$a_2 = 0$	- NA -	complete stability

Table 3.2. . Location of intersections of supercritical Hopf bifurcations in the m_y-K parameter space in terms of pair-wise comparisons. Location is given with respect to carrying capacity (Column 2) and predator mortality rate (Column 3). Because all models have the same transcritical bifurcation, the intersection of the Hopf bifurcations signifies a change in which model produces a larger breadth of stability. Three of the models never intersect. Pre-encounter and post-consumption inducible defenses always generate a larger breadth of stability than undefended webs. Additionally, post-consumption inducible defenses always generate a larger breadth of stability than post-encounter inducible defenses. Parameter values are given in Tables 3.A.1, 3.A.2, 3.A.3, and 3.A.4.

models being compared	K value	m_y rate
undefended ID *vs* pre-encounter ID	- NA -	- NA -
undefended ID *vs* post-encounter ID	4.99 mg C L^{-1}	0.192 d^{-1}
undefended ID *vs* post-consumption ID	- NA -	- NA -
pre-encounter ID *vs* post-encounter ID	14.19 mg C L^{-1}	0.283 d^{-1}
pre-encounter ID *vs* post-consumption ID	10.86 mg C L^{-1}	0.197 d^{-1}
post-encounter ID *vs* post-consumption ID	- NA -	- NA -

3.10 Figures

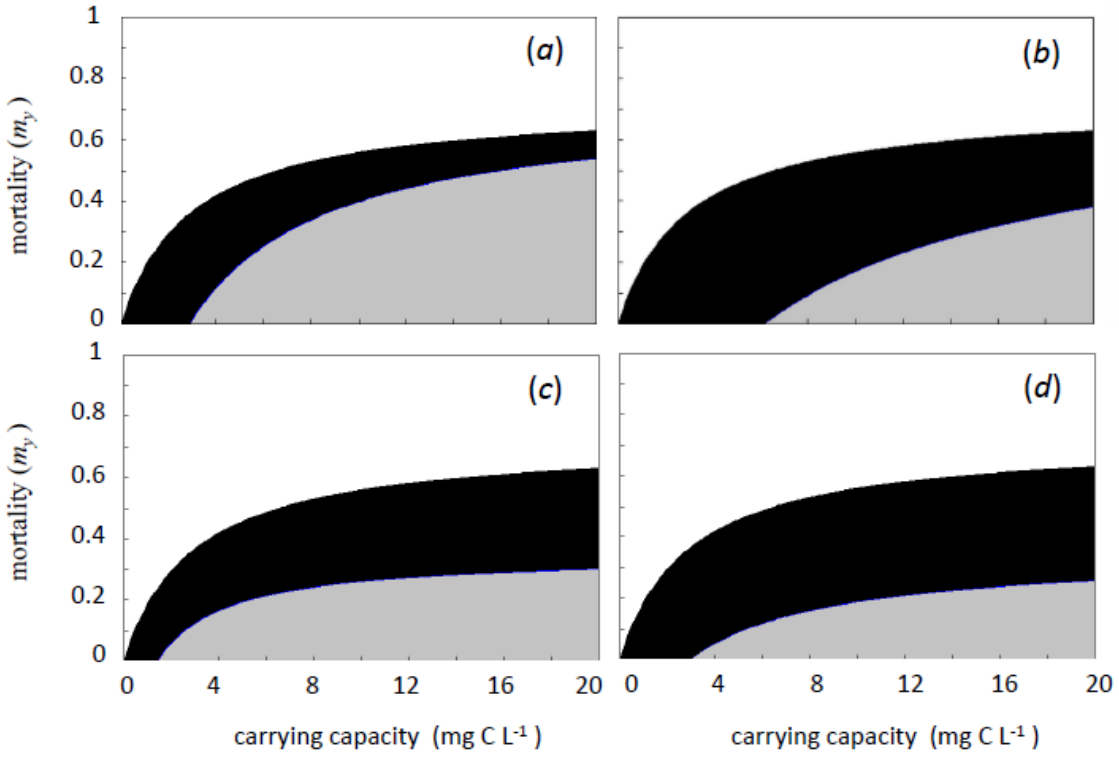


Figure 3.1. Bifurcation diagrams of (*a*) system (3.1) with constitutively undefended prey. (b-d) system (3.2) with inducibly defended prey. (*b*) pre-encounter inducible defense (i.e. attack rate). (*c*) post-encounter inducible defense (i.e. handling time). (*d*) post-consumption inducible defense (i.e. conversion efficiency). Carrying capacity (K) is shown on the x-axis, and predator mortality rate (m_y) is shown on the y-axis. The *white area* is the region of deterministic predator extinction. The *black area* is the region of stable coexistence; it is separated from the region of predator extinction by the transcritical bifurcation; above the transcritical bifurcation, the predator cannot maintain positive growth. The *grey area* is the region of cyclic coexistence (i.e. limit cycles); it is separated from the region of stable coexistence by the supercritical Hopf

bifurcation. The range of mortality rates where stable coexistence occurs (i.e. the size of the *black area*) at a given carrying capacity is known as the "breadth of stability". (*a*,*b*) Weak stability: as carrying capacity approaches infinity, the breadth of stability approaches 0. (*c*,*d*) Strong stability: as carrying capacity approaches infinity, the breadth of stability approaches a value greater than 0, however, limit cycles exist for some parameter values.

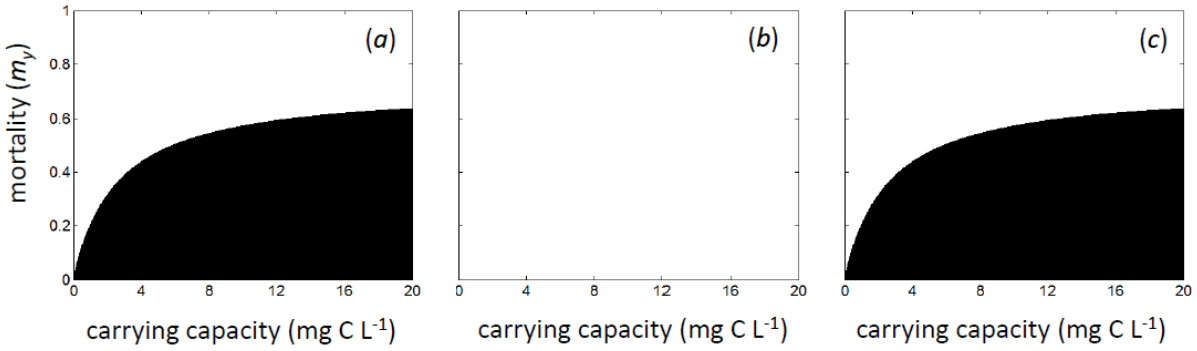
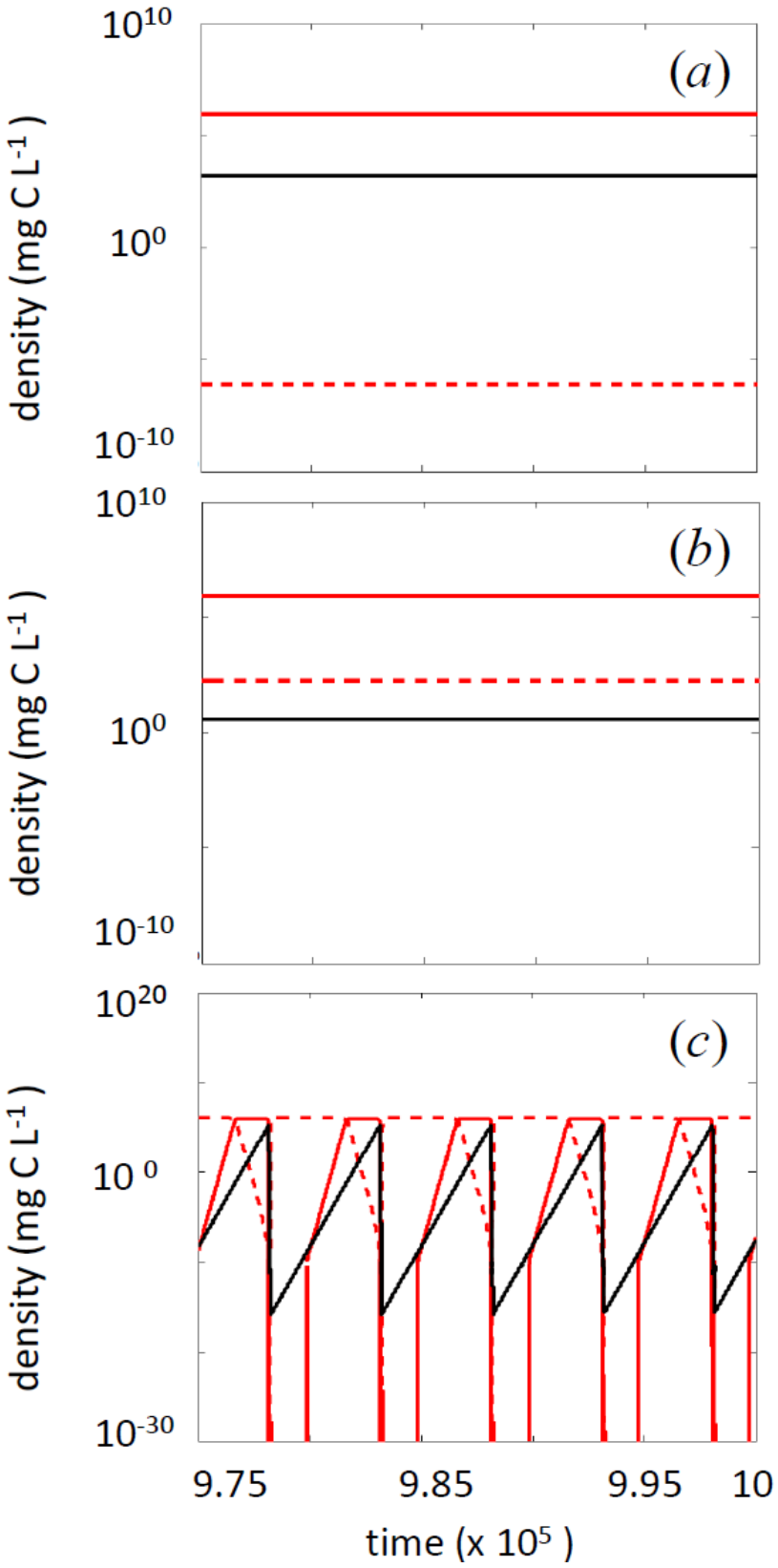


Figure 3.2- Bifurcation diagrams of system (3.2) with the limit cases of inducibly defended prey. (*a*) pre-encounter inducible defense (i.e. $a_2 = 0$). (*b*) post-encounter inducible defense (i.e. $h_2 \rightarrow$infinity). (*c*) post-consumption inducible defense (i.e. $\varepsilon_2 = 0$). Carrying capacity (*K*) is shown on the *x*-axis, and predator mortality rate (m_y) is shown on the *y*-axis. Description of colors as in Figure 3.1. (*a*, *c*) Complete stability: increasing carrying capacity does not cause sustained population cycles, regardless of predator mortality rate. (*b*) Trivial stability: predators cannot survive for any predator mortality rate greater than 0.

Figure 3.3- time series for system (3.2) at extreme values of K (K=1,000,000). *dashed red*- undefended prey. *solid red* - defended prey. *solid black*- predators. (a, b) $a_2 = 0$ (a limit case of pre-encounter inducible defense), which exhibits complete stability. (a) $m_y = 0$ (which shows that the Hopf bifurcation never enters the system). (b) $m_y = 0.71$. (c) $a_2 = 0.01$, $m_y = 0.71$, stable coexistence is not possible. Compare panels (b) and (c) to show that strong/complete stability is not possible if $a_2 > 0$. Note that the y-axis is in log scale, and the y-axis in (c) covers a larger range than in (a) or (b) .



3.11 Appendices

Appendix 3.A – *Supplementary Tables with Parameter Values for Each Category of Inducible Defense*

Table 3.A.1- definitions and values of model variables and parameters for the bitrophic system with no defenses (obtained from Vos *et al.* (2004*b*))

parameter	value	units	interpretation
r	1.42	d^{-1}	intrinsic rate of increase of prey
K	Free	mg C L^{-1}	carrying capacity
a	0.77	L d^{-1} mg C^{-1}	predator attack rate on prey
h	0.5	d · mg C^{-1} · mg C^{-1}	predator handling time on prey
m_x	0.145	d^{-1}	prey mortality rate
ε	0.36	mg C /mg C	predator conversion efficiency
m_y	free	d^{-1}	predator mortality rate

Table 3.A.2- definitions and values of model variables and parameters for the bitrophic system with pre-encounter inducible defenses (based on values obtained from Vos *et al.* (2004*b*))

parameter	value	units	interpretation
r_1	1.42	d^{-1}	intrinsic rate of increase of undefended prey
r_2	1.42	d^{-1}	intrinsic rate of increase of defended prey
K	free	mg C L^{-1}	joint carrying capacity of prey
a_1	0.77	L d^{-1} mg C^{-1}	predator attack rate on undefended prey
a_2	0.37019	L d^{-1} mg C^{-1}	predator attack rate on defended prey
h_1	0.5	d · mg C^{-1} · mg C^{-1}	predator handling time on undefended prey
h_2	0.5	d · mg C^{-1} · mg C^{-1}	predator handling time on defended prey
m_{x1}	0.145	d^{-1}	mortality rate of undefended prey
m_{x2}	0.18	d^{-1}	mortality rate of defended prey
ε_1	0.36	mg C/mg C	predator conversion efficiency on undefended prey
ε_2	0.36	mg C/mg C	predator conversion efficiency on defended prey
m_y	free	d^{-1}	predator mortality rate

Table 3.A.3- definitions and values of model variables and parameters for the bitrophic system with post-encounter inducible defenses (based on values obtained from Vos *et al.* (2004*b*))

parameter	value	units	interpretation
r_1	1.42	d^{-1}	intrinsic rate of increase of undefended prey
r_2	1.42	d^{-1}	intrinsic rate of increase of defended prey
K	free	mg C L^{-1}	joint carrying capacity of prey
a_1	0.77	L d^{-1} mg C^{-1}	predator attack rate on undefended prey
a_2	0.77	L d^{-1} mg C^{-1}	predator attack rate on defended prey
h_1	0.5	d · mg C^{-1} · mg C^{-1}	predator handling time on undefended prey
h_2	1.04	d · mg C^{-1} · mg C^{-1}	predator handling time on defended prey
m_{x1}	0.145	d^{-1}	mortality rate of undefended prey
m_{x2}	0.18	d^{-1}	mortality rate of defended prey
ε_1	0.36	mg C/mg C	predator conversion efficiency on undefended prey
ε_2	0.36	mg C/mg C	predator conversion efficiency on defended prey
m_y	free	d^{-1}	predator mortality rate

Table 3.A.4- definitions and values of model variables and parameters for the bitrophic system with post-consumption inducible defenses (based on values obtained from Vos *et al.* (2004*b*))

parameter	value	units	interpretation
r_1	1.42	d^{-1}	intrinsic rate of increase of undefended prey
r_2	1.42	d^{-1}	intrinsic rate of increase of defended prey
K	free	mg C L^{-1}	joint carrying capacity of prey
a_1	0.77	L d^{-1} mg C^{-1}	predator attack rate on undefended prey
a_2	0.77	L d^{-1} mg C^{-1}	predator attack rate on defended prey
h_1	0.5	d · mg C^{-1} · mg C^{-1}	predator handling time on undefended prey
h_2	0.5	d · mg C^{-1} · mg C^{-1}	predator handling time on defended prey
m_{x1}	0.145	d^{-1}	mortality rate of undefended prey
m_{x2}	0.18	d^{-1}	mortality rate of defended prey
ε_1	0.36	mg C/mg C	predator conversion efficiency on undefended prey
ε_2	0.173077	mg C/mg C	predator conversion efficiency on defended prey
m_y	free	d^{-1}	predator mortality rate

Appendix 3.B – Effectiveness vs *K* value required to cross Hopf bifurcation as $m_y \rightarrow 0$

Relationship between inducible defense effectiveness (*y* axis) and the *K* value (*x* axis) required to cross the supercritical Hopf bifurcation as $m_y \rightarrow 0$ for models built using system (3.2). (1) Pre-encounter inducible defenses; (2) Post-encounter inducible defenses; (3) Post-consumption inducible defenses. Effectiveness and carrying capacity increase from the bottom left hand corner to the top right hand corner. For all three figures, the minimum effectiveness shown corresponds to the parameterization used to model undefended prey (given in Table 3.A1). Carrying capacity- defense effectiveness parameter combinations that fall into the *black shaded area* produce stable coexistence, parameter combinations that fall into the *grey shaded area* produce cyclic coexistence. Note that increasing effectiveness of pre-encounter inducible defenses will increase the *K* value required to cross the Hopf bifurcation as $m_y \rightarrow 0$; the opposite is true for post-encounter inducible defenses. The effectiveness of post-consumption inducible defenses has little impact on the *K* value required to cross the Hopf bifurcation as $m_y \rightarrow 0$; however, as effectiveness becomes extremely effective (i.e., $\varepsilon_2 \rightarrow 0$), post-encounter inducible defenses are shown to increase the *K* value required to cross the Hopf bifurcation as $m_y \rightarrow 0$.

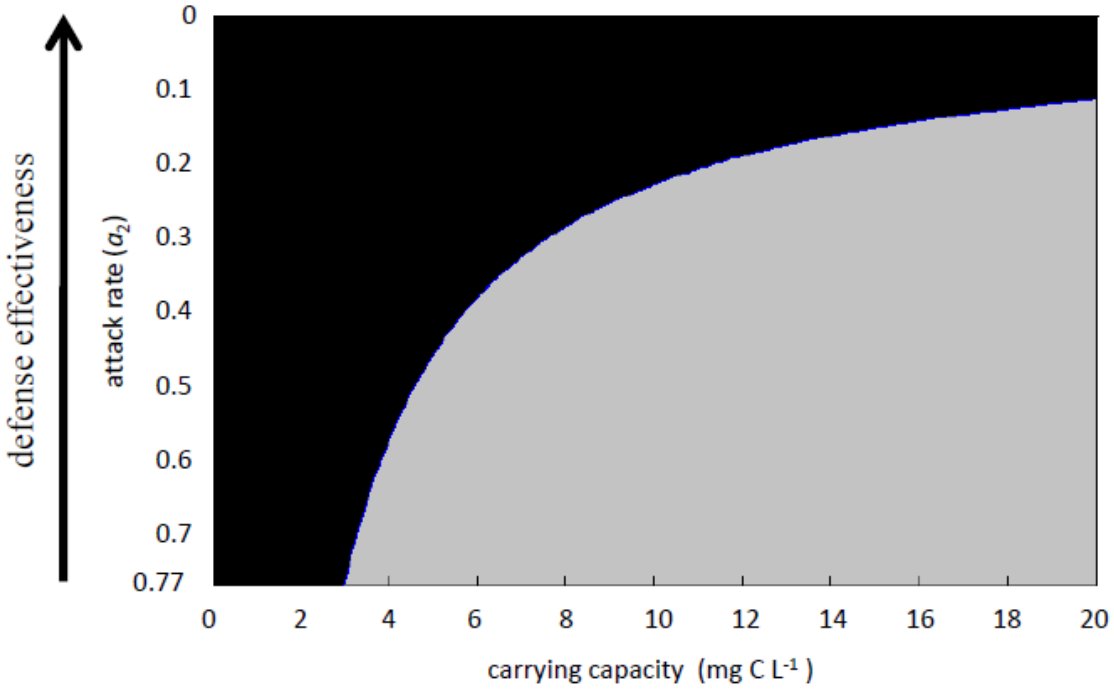


Figure 3.B.1 – pre-encounter inducible defenses

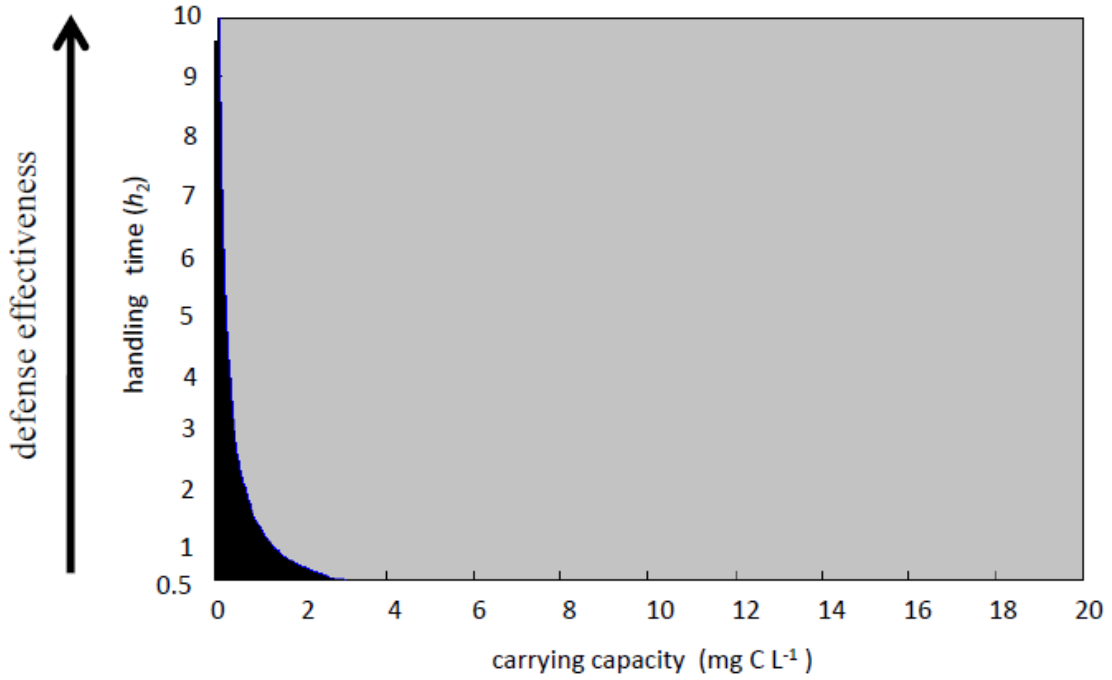


Figure 3.B.2 – post-encounter inducible defenses

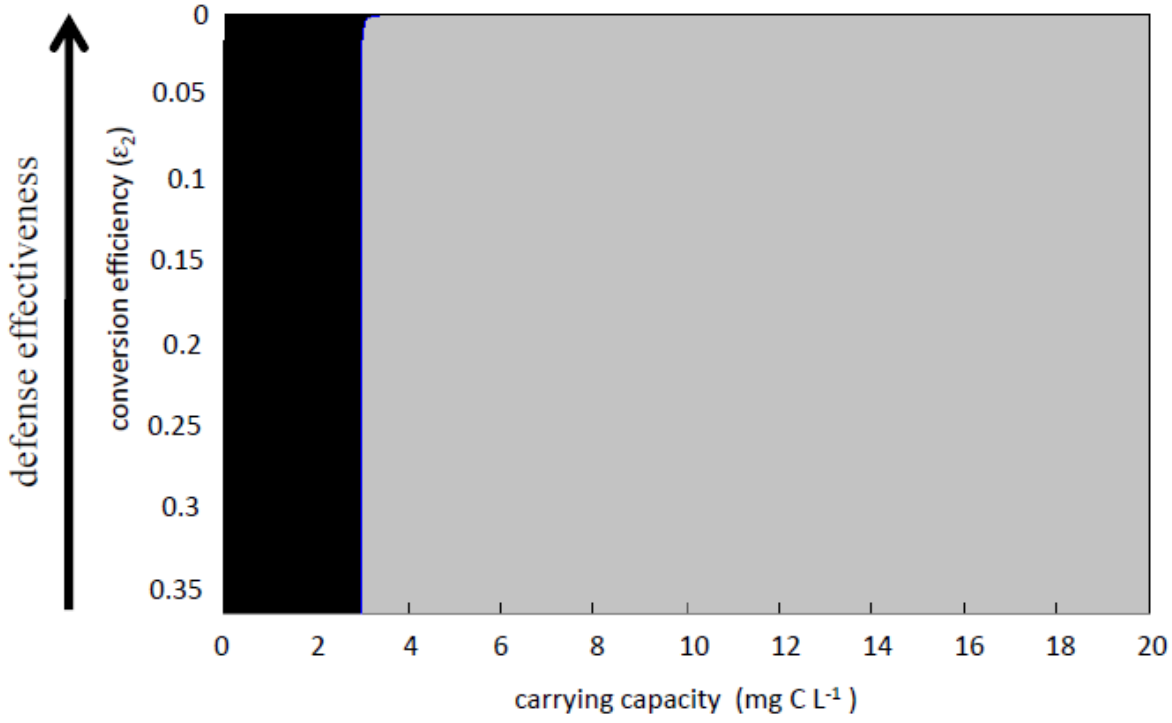


Figure 3.B.3 – post-consumption inducible defenses

CONNECTING STATEMENT

In the previous two chapters, I have shown the dynamical consequences of intraspecific variation for prey defenses, and that model predictions are sensitive to the origin, trophic location, and the category of defense traits. I now turn my attention to the another type of variation for traits that affect the interactions of predators and prey: inducible predator offenses. In Chapter 4, I use a combination of theory and systematic review of published empirical data to investigate the dynamical consequences and prevalence of intraindividual variation in predators. Specifically, I consider inducible offenses that allow predator attack rate and/or handling time to become functions of prey density.

CHAPTER 4

PREY-DENSITY DEPENDENT ATTACK RATE AND HANDLING TIME: PREVALENCE AND COMMUNITY LEVEL CONSEQUENCES

Caolan Kovach-Orr, Michael Cortez, Matthijs Vos, & Gregor F. Fussmann (soon to be re-submitted to *The American Naturalist*). Prey-density dependent attack rate and handling time: prevalence and community-level consequences

4.1 Abstract

The widely used Holling type 2 functional response assumes that the components of predation (i.e. attack rate and handling time) are unaffected by changes in prey density. However, a growing body of empirical and theoretical research suggests that these components should depend on prey density. In our study, we explore predator inducible offenses using a variety of functional response equations that correspond to situations where prey density-dependent attack rates and/or handling times are likely relevant to natural systems. Using a combination of theory and systematic review of published empirical datasets, we evaluate the prevalence and dynamical implications of these functional responses. Of the 144 datasets that had previously been attributed to the type 2 functional response, AICc analyses indicate that 142 datasets are best fit by consumption equations that incorporate prey density-dependent attack rates and/or handling times. In terms of the community dynamics and stability properties of systems facing nutrient enrichment, we find that some, but not all, models that incorporate prey density-dependent attack rates and/or handling times are capable of making categorically and fundamentally different predictions than models that incorporate the type 2 functional response. We interpret our findings to mean that predictions of frequent or inevitable destabilization may be overstated. Our study also highlights the importance of rechecking accepted principles in ecology.

Key words: inducible offense, functional response, predator-prey, strong stability, paradox of enrichment

4.2 Introduction

Ecologists use mathematical models to formalize specific aspects of species interactions – for instance, the uptake of prey by a predator. In many cases, a number of mathematical expressions can satisfactorily describe the process of species interactions and ecologists have settled on using one or a few of them for historical reasons or out of convenience. The same set of descriptors of species interactions is typically put to use in dynamical models of interacting populations, where they form the interaction terms linking differential or difference equations; for example, the type 1, 2, and 3 functional response equations (Holling 1959; Myerscough, Darwen & Hogarth 1996). As previously shown, the mathematical description of the species interaction becomes crucially important if the task at hand is prediction of community dynamics because terms that can serve equally well as mechanistic descriptors of species interactions frequently lead to drastically different outcomes in a dynamical context (Fussmann & Blasius 2005).

In this paper, we are primarily concerned with the attack rate and handling time components of predation. Attack rate is defined as the rate at which a predator encounters and successfully captures prey (Murdoch 1973). Handling time is the amount of time required to physically manipulate and consume captured prey (Murdoch 1973). Optimal foraging theory suggests that predators should try to maximize energy intake while minimizing energy output and risk (MacArthur & Pianka 1966). Thus, from an evolutionary perspective, the components of the functional response (attack rate and handling time) should, at least partially, depend on prey density (Abrams 1982); however, it is often assumed that these components are constant and independent of prey density (Murdoch, Briggs & Nisbet 2003; Turchin 2003). Such "static" parameterizations occur in a number of popular mathematical predator-prey descriptions, such as

the "type 2" functional response, and are implicitly employed in classical and basal community models, such as the widely used Rosenzweig-MacArthur (R-M) model for bitrophic predator-prey interactions (Rosenzweig & MacArthur 1963). From a theoretical standpoint, the R-M model serves as both the standard for bitrophic analyses and the foundation for more complex community models (Fussmann & Heber 2002; Rooney *et al.* 2006); however, the R-M model rarely, if ever, accurately describes the dynamics of predator-prey interactions under natural field conditions (Jensen & Ginzburg 2005). Specifically, the R-M model predicts that as the carrying capacity of the prey increases, stable coexistence will become practically impossible, a phenomenon known as the "paradox of enrichment" (Rosenzweig 1971); instead, natural systems are relatively stable under enrichment (Murdoch *et al.* 1998; Vos *et al.* 2004; Jensen & Ginzburg 2005). Despite the lack of empirical support for the R-M model, it remains a paradigm of community ecology, and theorists have tried to resolve the mismatch between theory and observation by expanding the R-M model (Jensen & Ginzburg 2005). Advancements have relied on incorporating phenomena such as inducible defenses, reciprocal plasticity, genetic diversity, inedible prey, unpalatable prey, predator interference, and spatial heterogeneity into basal (more simple) predator-prey models (Grover 1995; Scheffer & Deboer 1995; Jansen 2001; Yoshida *et al.* 2003; Arditi *et al.* 2004; Vos *et al.* 2004; Mougi & Iwasa 2011; Yamamichi, Yoshida & Sasaki 2011); however, the disagreement between theory and natural systems remains a debated topic because many of these solutions are considered system specific (Roy & Chattopadhyay 2007).

On the other hand, many of these phenomena can be modeled using functional responses that incorporate prey density-dependent components (PDCs), e.g. functional responses where the handling times and/or attack rates change with prey density. For instance, under certain

circumstances, the presence of prey refuges (spatial heterogeneity) can cause attack rate to become an increasing function of prey density because as prey density increases, a decreasing proportion of prey can hide from predators (Murdoch & Oaten 1975); this, in turn, can produce a sigmoid functional response (Oaten & Murdoch 1975). A positive correlation between prey density and attack rate is also seen in systems with inducible defenses (Hammill, Petchey & Anholt 2010).

However, phenotypic plasticity within a single predator can create inducible offenses that allow for PDC functional responses in the absence of spatial heterogeneity or multiple prey types (Kishida *et al.* 2014). For instance, handling time can become a decreasing function of prey density when predators abandon and/or only partially consume prey (Okuyama 2010). Such "wasteful killing" can confer an evolutionary advantage (Maupin & Riechert 2001; de Mazancourt & Schwartz 2012) and has been observed across a wide range of systems (Sandness & McMurtry 1972; Johnson, Akre & Crowley 1975; Samu & Biro 1993; Tripler *et al.* 2002; Fantinou *et al.* 2008; Trubl, Blackmore & Johnson 2011); for example, during salmon runs, brown bears discard the majority of the carcass and selectively consume brain tissue for its high nutritional value (Gende *et al.* 2004). Although wasteful killing causes per unit mass conversion efficiency to decrease, it allows increases in total predator energy intake and the strength of top down control. Only a handful of experiments have directly looked at the relationship between handling time and prey density, but the majority have found a negative correlation (Okuyama 2010). Additionally, inducible offenses can affect a wide range of other traits. For example, the adult form of the predatory salamander *Hynobius retardatus* will metamorphose faster, develop a larger gape, and exhibit higher activity levels if, as a larva, it is exposed to high densities of its prey, *Rana pirica* (Kishida *et al.* 2014). Kishida *et al.* (2014) also showed that the induction of

these traits results in a 30% decrease in prey survival; therefore, these offenses are likely important factors in natural communities. Our investigation focuses on inducible offenses that create PDC functional responses in bitrophic systems.

How might functional responses that incorporate PDCs affect dynamics in simple communities? Okuyama (2010) showed that incorporating decreasing handling times into the R-M model can extend the range of carrying capacity where stable coexistence exists, for finite values of carrying capacity. Sigmoid functional responses are predicted to promote stable coexistence by reducing the likelihood of overexploitation and prey escape at low prey densities (Oaten & Murdoch 1975). Despite these important findings, no previous study has analyzed the effects of multiple PDCs across a wide range of functional response shapes; a surprising fact, given that model predictions are so sensitive to the shape of the functional response curve (Armstrong 1976; Williams & Martinez 2004; Fussmann & Blasius 2005) and that incorporating PDCs can create functional response curves that are saturating, non-saturating, monotonic, sigmoid, unimodal or some combination (Fig. 4.1).

In our study, we employed a variety of functional response equations that correspond to situations where PDCs are likely relevant to natural systems; using a combination of theory and systematic review of empirical studies, we analyzed their potential to help resolve the discrepancy between model predictions and field data. As part of this analysis, we explored the dynamical effects of type 2 functional responses (i.e., with prey density-***independent*** components) versus functional responses that contain PDCs (referred to as "PDC functional responses"); using published studies, we also investigated the occurrence and prevalence of type 2 and PDC functional responses over a wide range of predator-prey interactions that have been previously classified as "type 2".

Through AICc analyses, we find that the vast majority (99%) of empirical datasets are best fit by PDC equations; although, the type 2 functional responses can serve as an adequate descriptor of predator uptake for 61% of the empirical cases we consider. In terms of community dynamics and stability properties, our results indicate that some, but not all, models that incorporate PDCs are capable of making categorically and fundamentally different predictions than models that incorporate the type 2 functional response. Because the shapes of functional responses play an important role in the dynamical stability of ecological systems, we interpret the prevalence of PDC functional responses to mean that predictions of frequent or inevitable destabilization may be overstated.

4.3 Models

Here we present a generalized Rosenzweig-MacArthur type model (Rosenzweig & MacArthur 1963):

$$\frac{dx}{dt} = r\,x\,\left(1-\frac{x}{K}\right) - f(x)\,y \tag{4.1a}$$

$$\frac{dy}{dt} = \varepsilon\,f(x)\,y\;-\;m_y\,y \tag{4.1b}$$

where x and y are the densities of prey and predator populations, respectively, r is the maximum growth rate of the prey, and K is the prey carrying capacity. The predator consumes prey at a per capita rate determined by the functional response $f(x)$, converts prey biomass into new predator biomass with efficiency ε, and experiences a natural mortality rate m_y. The conversion efficiency ε, can be scaled out of the system by a rescaling of time and predator density (see Appendix 4.A), hence we set ε equal to 1 in our analyses.

The R-M model is traditionally evaluated using a mechanistic version of the Holling type 2 functional response (Oksanen *et al.* 1981):

$$f_2(x) = \frac{x\ a}{x\ a\ T_h + 1} \tag{4.2a}$$

where a is the attack rate and T_h is the handling time; both of these variables are assumed to be constant and independent of prey density (Figs. 4.2*b, 4*.2*c*) (Murdoch 1973) . The type 2 functional response is a monotonic, non-sigmoid, saturating curve (Fig. 4.2*a*); for these functional responses, as prey density increases, consumption is strictly increasing, but at a decreasing rate, and approaches a limit (Fig. 4.2*a*) (Holling 1959).

Incorporating PDCs into functional response models can have a variety of impacts on the shape of the functional response curve; these effects can depend on which component is prey-dependent as well as the specific relationship between the component and prey density. Due to the breadth of this variety, we focus on specific mathematical formulations that can give rise to different classes of functional response shape. Broadly speaking, we identified five classes of functional responses that represent the situations that are likely relevant to biological systems.

Functional Responses that are Monotonic, Non-Sigmoid, & Saturating (Fig. 4.2*d*) - Although PDCs are incorporated, these curves are very similar to type 2 curves in that consumption is strictly increasing, non-sigmoid, and approaches an asymptote. Furthermore, the type 2 can be considered a degenerate case of this class where the components of the functional response are constants. On the other hand, PDC curves of this class can be created by incorporating a decreasing attack rate and/or handling time that approaches a value greater than 0 at high prey densities. Because our systematic review indicated that curves of this class that incorporate a decreasing attack rate fit more datasets than a decreasing handling time (data not shown), we used a curve based on decreasing attack rate:

$$f_3(x) = \frac{x\ a\ (x)}{x\ a\ (x)\ T_h + 1}\ ,where\ a(x) = \alpha\ (e^{-x} + 1) \quad (4.2b)$$

and where $a(x)$ is the attack rate function and α is a constant (Fig. 4.2*e*). In the attack rate function, exp(-*x*) is offset by a value of 1 so that the attack rate does not approach 0 at very high densities. Handling time is held constant (Fig. 4.2*f*).

Functional Responses that are Monotonic, Non-Sigmoid, & Non-Saturating (Fig. 4.2*g*) – Like the previous functional responses, these consumption curves are still concave down, increasing functions of prey density; however, unlike the previous functions, these curves have no upper limit (Rosenzweig 1971; Abrams 1982; Okuyama 2010; Braza 2012); this is phenomenologically similar to the curves described by log(*x*) or $\sqrt{x}$. Non-saturating behavior occurs when handling time is allowed to asymptotically decrease towards 0 at high prey densities. While, in the limit, infinite consumption is impossible, non-saturating functional responses can provide valuable insights into situations where saturation occurs at such high prey densities that saturation becomes irrelevant (see discussion). We used a natural logarithm-based function to describe handling time:

$$f_4(x) = \frac{x\ a}{x\ a\ T_h(x) + 1}\ ,where\ T_h(x) = \frac{c}{\ln(x+1)+1} \quad (4.2c)$$

and where $T_h\ (x)$ is the handling time function and *c* is the maximum handling time (which occurs at low prey densities) (Fig. 4.2*i*). Prey density, in the logarithm function, is offset by a value of 1 so that the curve can pass through the origin, and the logarithm function is offset by a value of 1 to prevent handling time from approaching infinity at very low prey densities. Attack rate is held constant (Fig. 4.2*h*).

Functional Responses that are Monotonic, Sigmoid, & Saturating (Figs. 4.2 *j, m*) – In contrast to the previous functional responses, at low prey densities, these curves are concave

up, increasing functions (i.e. consumption increases at an increasing rate); however, as prey density increases, these curves become concave down, increasing functions that approach asymptotic limits (i.e. they start to resemble $f_2(x)$ at high prey densities). These curves can be created by incorporating increasing attack rates or decreasing handling times. The most widely known equation of this class is the Holling type 3 functional response (Fig. 4.2*j*):

$$f_{5A}(x) = \frac{x\ a\ (x)}{x\ a\ (x)\ T_h + 1}\ , where\ \ a(x) = \alpha\ x \tag{4.2d}$$

and where the attack rate increases linearly with prey density according to $\alpha\, x$ (Fig. 4.2*k*), and handling time is held constant (Fig. 4.2*l*).

On the other hand, these curves can also be obtained for some decreasing handling time models. We used an exponential-based function to describe handling time (Fig. 4.2*m*):

$$f_{5B}(x) = \frac{x\ a}{x\ a\ T_h(x) + 1}\ , where\ T_h(x) = e^{-x} + \ c \tag{4.2e}$$

and where c is a constant that is greater than 0, but less than, or equal to, exp(-2). In $T_h(x)$, c+1 is the maximum handling time and c is the minimum handling time (Fig. 4.2*o*). Attack rate is held constant (Fig. 4.2*n*). We include this formulation because it offers a novel mechanism for the production of a sigmoid functional response.

Functional Responses that are Monotonic, Sigmoid, & Non-Saturating (Fig. 4.2*p*) – Responses of this class are concave up, increasing curves at low prey density and switch to concave down, increasing functions at higher densities, but do not approach an asymptotic limit. These curves can be created by incorporating an attack rate that increases with prey density and a handling time that decreases towards an asymptotic limit of 0 at high prey densities. We used a combination of $f_4(x)$ and $f_{5A}(x)$ to create this function:

$$f_6(x) = \frac{x\ \ a(x)}{x\ a(x)\ T_h(x) + 1}\ \ where\ T_h(x) = \frac{c}{\ln(x+1)+1}\ and\ a(x) = \alpha\ x \tag{4.2f}$$

and where attack rate follows the description given for $f_{5A}(x)$ (Fig. 4.2*q*) and handling time follows the description given for $f_4(x)$ (Fig. 4.2*r*).

Functional Responses that are Unimodal (Fig. 4.2*s*) - Like the type 2, these curves are concave down, increasing functions at low densities; however, at some positive prey density, consumption peaks and becomes a decreasing function of prey density. These curves can be created by incorporating an attack rate that decreases towards an asymptotic limit of 0 (very quickly) or a handling time that increases as prey density increases. Because our systematic review indicated that curves of this class that incorporate an increasing handling time fit more datasets than a decreasing attack rate (data not shown), we used a curve based on increasing handling time. Nevertheless, while certain mechanisms can allow handling time to approach 0, the authors are unaware of any mechanisms that force handling time to increase towards infinity with increasing prey density; therefore, we chose a logarithm-based function that allows handling time to increase towards an upper limit:

$$f_7(x) = \frac{x\ a}{x\ a\ T_h(x) + 1}\ , where\ T_h(x) = \frac{c}{\frac{1}{\ln(x+1)}+1} \tag{4.2g}$$

and where the attack rate is held constant (Fig. 4.2*t*), c is the maximum handling time (which occurs at high prey density), and 0 is the minimum handling time (which occurs at low prey density) (Fig. 4.2*u*). Prey density, in the logarithm function, is offset by a value of 1 so that the curve can pass through the origin. The inverse of the logarithm function is then offset by a value of 1 to prevent handling time from approaching infinity. Note that at high prey densities, this curve becomes a concave up, decreasing function that approaches an asymptotic limit greater than 0, but less than the maximum consumption rate (which occurs at low to moderate prey densities).

4.4 Methods

4.4.1 Ecological Consequences of PDC Functional Responses (Theory)

We analyzed how the different functional response equations affect community level dynamics; specifically, we were interested in three system behaviors: deterministic predator extinction, stable coexistence of predators and prey, and cyclic coexistence of the two species. Our analysis focused on the dynamics that arise for different combinations of mortality, m_y, and carrying capacity, K. We chose m_y and K, because m_y represents the force of top-down control on the system, and K represents enrichment and hence the force of bottom-up control (Vos *et al.* 2004). Recently, there has been growing interest in the relationship between carrying capacity and the breadth of stability, i.e. the range of mortality rates where stable coexistence occurs at a given K (Kretzschmar, Nisbet & McCauley 1993; Vos *et al.* 2004; Kovach-Orr & Fussmann 2013) (Fig. 4.3). Previous research has shown that the specific nature of this relationship can be used to classify models into fundamentally different categories of model stability known as "weak" , "strong", and "complete" stability (Kretzschmar, Nisbet & McCauley 1993; van Voorn *et al.* 2008). Weak stability occurs when the breadth of stability decreases, and eventually approaches 0, as carrying capacity is increased (Fig. 4.3*a*). Strong stability occurs when the breadth of stability is unaffected, or even increases, as carrying capacity is increased (Fig. 4.3*b*)(Kretzschmar, Nisbet & McCauley 1993; Vos *et al.* 2004). Complete stability can be seen as the limit case of strong stability, in that complete stability occurs when systems do not exhibit limit cycles anywhere in the m_y-K parameter space (Fig. 4.3*c*)(van Voorn *et al.* 2008). The distinction between weak and strong/complete stability is of special interest because the classic R-M model predicts weak stability for bitrophic systems, whereas stable coexistence in natural systems is generally unaffected by changes in carrying capacity (Murdoch *et al.* 1998). To

explore if simple bitrophic systems with type 2 vs. PDC functional responses produce qualitatively similar regions of stable coexistence, we analyzed how the breadth of stability changes when the R-M model is modified with different formulations of $f(x)$.

We provide analytical solutions for the breadth of stability as K approaches infinity for the functional response equations, where possible. However, because of the complex nature of some PDC functional responses, some of our results are limited to partial analytical solutions or are in the form of implicit functions that must be solved numerically to find the breadth of stability. In order to visualize the breadth of stability at finite values of K, we created bifurcation diagrams, which can offer deep insights into how and why the breadth of stability changes at biologically plausible parameter values. Because it is not possible to use analytical methods to produce bifurcation diagrams for our PDC functional response models, we used MATLAB (2012) and the associated package MatCont (Govaerts & Kuznetsov 2013) for numerical bifurcation analysis of all of our R-M based models. To produce bifurcation diagrams, we chose a particular parameterization of $f_2(x)$ (*attack rate*=1, *handling time*= 0.1); we then used nonlinear least squares methods to maximize the phenomenological similarity of the PDC functional responses to $f_2(x)$ over the range x=0 to x=90 (Fig. 4.1, see Table 4.C1 for parameter values) (Fussmann & Blasius 2005). Note that given the parameter values chosen, $f_2(x)$ approaches an asymptotic limit of 10 and that at a prey density of 90, $f_2(x)$ is equal to 9.

4.4.2 Systematic Review of PDC Functional Responses

In order to establish the prevalence of PDC functional responses, we performed a systematic review of empirical data. This review analyzed 144 experimental functional response datasets from 47 peer-reviewed articles published between January 1, 2010 and December 31,

2012 (see Appendix 4,D). These datasets come from a wide variety of bitrophic systems, including aquatic, marine, terrestrial, microbial, arthropod, fish, bird, and mammal communities. To obtain articles, we searched the ISI Web of Science website using the terms "functional response" AND ["type II" OR "type 2"]. Datasets were included only if data were obtained from pairwise predator-prey interactions, the authors had fit monotonically increasing curves to the data (i.e. type 2, Ivlev, Rogers, etc), and the authors had not concluded that alternative models (i.e. type 3, decreasing handling time, etc.) were viable. Additionally, we excluded approximately 25 articles/datasets because they included transformed data, non-original data, data that were behind a pay wall we could not access at McGill University, and/or datasets from experiments with non-ecological objectives, such as experiments that investigated the effects of drug toxicity on functional response.

We used Plot Digitizer v 2.5.1 (Huwaldt 2011) to digitize and extract functional response data from PDF versions of published, peer-reviewed articles. Each data point was counted only once. We used the statistical program R (R-Core-Team 2013) and the associated package minpack.lm (Elzhov *et al.* 2013) to perform nonlinear least squares tests on the fit of the functional response equations to the extracted data. Model selection was performed using modified Akaike Information Criterion analyses (AICc), which corrects for small sample size, on the output of the nonlinear least squares tests (Akaike 1974; Hurvich & Tsai 1989). To determine the best-fitting equation for each dataset, we identified the equation that produced the most negative AICc value for that dataset. In order to rectify the number of PDC functional response equations compared to the type 2, we used AIC weights analyses (Johnson & Omland 2004; Wagenmakers & Farrell 2004). This allowed us to infer how much more likely the best fitting equation is than other equations. Using two datasets as examples, figure 4.4 demonstrates

the ability of our methodology to determine the best fitting model in situations where PDC and type 2 functional responses both provide adequate fits (Fig. 4.4*a*) and in situations where the best fitting model is clearly superior to other models (Fig. 4.4*b*). Additional data analyses are provided in Appendix 4.B.

4.5 Results

4.5.1 Systematic Review of PDC Functional Responses

Our systematic review provided an estimate of the prevalence of PDC functional responses among the 144 datasets that had previously been categorized as type 2 functional responses. Using AICc analysis of the fits produced by nonlinear least squares methods, we determined that only 2 datasets (1.4%) are best described by $f_2(x)$ and only 41 datasets (28%) are best described by 'true' monotonic, non-sigmoid equations (Table 4.1). Additionally, although increasing handling time models produce unimodal consumption curves, over the range of prey densities tested in each dataset, the functional response was strictly increasing for 35 of the 36 datasets best fit by $f_7(x)$. Therefore, over the range of prey densities considered, 76 datasets (53%) are best fit by 'monotonic, non-sigmoid' curves. Surprisingly, 67 datasets (47%) are best fit by monotonic, sigmoid functional responses, and $f_{5A}(x)$, the type 3 functional response, nearly tied with $f_7(x)$, the 'unimodal' functional response, for having the lowest AICc value for the most number of datasets (Table 4.1). Furthermore, 58 datasets (40%) are best fit by functions that have no upper limit (Table 4.1). When AIC weights are considered, no dataset is more than twice as likely to be truly a type 2 than truly a PDC functional response, whereas 56 datasets (39%) are more than twice as likely to be truly a PDC functional response than truly a type 2 functional response (Table 4.1), with major contributions from $f_4(x)$, $f_{5A}(x)$, and $f_6(x)$

(Table 4.1). In other words, although the type 2 was an adequate model for 61% of datasets, PDC functional responses were adequate or more than adequate for 100% of datasets. Additional analyses are provided in Appendix 4.B.

4.5.2 Ecological Consequences of PDC Functional Responses (Theory)

The R-M model based on $f_2(x)$, the type 2 equation, is only capable of producing "weak stability" because, at high carrying capacities, the mortality rate for which dynamics switch between limit cycles and stable coexistence approaches the same asymptotic value as the maximum mortality rate that the predator can withstand (Figs. 4.5 *a, b*). In other words, stable coexistence of predator and prey becomes effectively impossible as K increases because the breadth of stability shrinks towards zero (see Appendix 4.A). On the other hand, replacing the type 2 with PDC functional responses, even those that appear almost indistinguishable from the type 2 at low to moderate prey densities (Fig. 4.1), can have significant impacts on model predictions (Fig. 4.5). Specifically, the use of equations $f_4(x)$, $f_{5A}(x)$, $f_{5B}(x)$, and $f_6(x)$ can produce strong stability (Figs. 4.5 *f, h, j, l*). In Appendix 4.A, we derive sufficient conditions under which the R-M model has a non-narrowing breadth of stability for our classes of functional responses. We show that saturating functional responses always lead to weak stability at high mortality rates. We also show that many different formulations of monotonic, non-saturating functional responses can yield strong stability; however, the mere absence of saturation does not prevent weak stabilization from occurring. Instead, a non-narrowing breadth of stability arises at high mortality rates when a monotonic, non-saturating functional response satisfies either:

$$\lim_{x \to \infty} f'(x) \to c > 0 \qquad (4.3a)$$

or

$$\lim_{x \to \infty} x f'(x + A) \to c > 0 \text{ , where } A = A(x) = \frac{x f(x)}{f(x) - x f'(x)} \quad (4.3b)$$

Both $f_4(x)$ and $f_6(x)$ fulfill these conditions. See Appendix 4.A for additional discussion.

Replacing $f_2(x)$ with $f_4(x)$ results in a smaller region of stable coexistence at low carrying capacities and predator mortality rates, which indicates that models built on $f_4(x)$ are more sensitive to enrichment at these parameter combinations than models built on $f_2(x)$ (Figs. 4.5 *a, e*). However, at higher K and m_y, systems based on $f_4(x)$ exhibit a greater breadth of stability than those built on $f_2(x)$ (Figs. 4.5 *b, f*). In fact, as K approaches infinity, models built on $f_4(x)$ are capable of producing strong stability, with the breadth of stability approaching the constant: $\left[\frac{\varepsilon}{c} \ln(2) \right]$ (Fig. 4.5 *f*) (Appendix 4.A).

On the other hand, R-M models built on $f_{5A}(x)$ and $f_{5B}(x)$ exhibit weak stability at high m_y (Figs. 4.5 *h, j*); however, at low m_y, these models can produce strong stability through a separate band of stable coexistence (Figs. 4.5 *g, h, i, j*). For $f_{5A}(x)$ (the type 3 functional response), the breadth of stability approaches the constant: $\frac{1}{2 T_h}$ (Fig. 4.5 *h*) (Appendix 4.A). Because of its complex mathematical nature, the implicit analytical solution of $f_{5B}(x)$ must be evaluated numerically; results are given in Table 4.C2. Note that as a increases and/or c decreases, the breadth of stability increases. Despite these findings of strong stability, models built on $f_{5A}(x)$ and $f_{5B}(x)$ do not promote stable coexistence across all parameter combinations. Specifically, at high m_y, models built on $f_{5A}(x)$ predict a smaller breadth of stability than models built on $f_2(x)$ (Figs. 4.5 *a, b, g, h*). At very low m_y, systems that incorporate $f_{5B}(x)$ exhibit a separate band of cyclic coexistence (Figs. 4.5 *i, j*); at low K, this band reduces the breadth of

stability and results in predictions of destabilization at lower K than predicted by models that incorporate $f_2(x)$ (Figs. 4.5 *a, i*).

The monotonic, sigmoid, and non-saturating functional response, $f_6(x)$, is capable of producing two bands of strong stability, one at high m_y and another at low m_y (Figs. 4.5 *k, l*). As K approaches infinity, the breadth of stability of the upper band approaches the constant: $\left[\frac{\varepsilon}{c} \ln(2) \right]$ (Fig. 4.5 *l*) (Appendix 4.A). Due to the complex nature of $f_6(x)$, the solution for the breadth of the lower band of stable coexistence must be found numerically using very high values of K; results are given in Table 4.C3. Note that as a and/or c decreases, the breadth of the lower band of stability increases. Because the breadth of the upper band of stability also increases as c decreases (but is unaffected by changes in a), the total breadth of stability increases as a and/or c decreases.

In spite of the strong stability produced by sigmoid and non-saturating equations that satisfy Equation 4.3, unimodal and monotonic, non-sigmoid equations that do not satisfy Equation 4.3 (i.e. $f_3(x)$ and $f_7(x)$), are only capable of weak stability. Therefore, the predictions of models based on these equations are not fundamentally different from the predictions of models based on the type 2 (Figs. 4.5 *a-d, m, n*). Although, from a quantitative point of view, predictions based on these PDC functional responses may differ from those based on the type 2; for instance, when one considers models based on $f_7(x)$ at very low m_y, stable coexistence is present over a greater range of K than in models built on $f_2(x)$ (Figs. 4.5*a, m*). The opposite is true for models based on $f_3(x)$ (Figs. 4.5 *a, c*).

4.6 Discussion

It seems that the type 2 functional response, in addition to the R-M model, has become an ecological axiom: "an assumed property that can only be overturned by proof it does not exist" (Jensen & Ginzburg 2005); however, a growing body of literature indicates that the components of the functional response may depend on prey density (Abrams 1982; Abrams 1989; Hammill, Petchey & Anholt 2010; Okuyama 2010) and that prey density-dependent components may play important roles in ecological communities (Lundberg & Astrom 1990; Jeschke 2006; Abrams 2010; Okuyama 2010). Furthermore, through adaptive phenotypic plasticity, predators may exhibit inducible offenses in the presence of a single prey population (Kishida *et al.* 2014). We used a combination of theory and systematic review to investigate the dynamical consequences and prevalence of PDC functional responses across a wide variety of bitrophic predator-prey interactions.

Our systematic review provides little support for the type 2 functional response (which uses prey density-***independent*** components) among datasets that had been specifically categorized as type 2. Although the type 2 is an adequate descriptor of predation for 88 of the 144 datasets (61%) tested, only 2 datasets (1.4%) are best described by the type 2 equation, and neither dataset is more than twice as likely a type 2 than a PDC functional response (Table 4.1). There are three possible explanations for why the type 2 is so rare: (1) Given how biotic systems are so prone to environmental noise and experiments to error, the shape of monotonic, non-sigmoid PDC functional responses is so similar to the shape of the type 2 that they are functionally indistinguishable. Although this may be occurring in some cases, it applies to a minority of datasets. Of the 74 datasets (51%) that are best fit by "monotonic, non-sigmoid" PDC functional response curves, 19 are more than twice as likely a PDC than a type 2 functional

response; therefore, only 55 datasets (38%) are potentially affected by this issue. (2) Due to problems with experimental procedures, the prevalence of sigmoid functional responses is greatly overestimated. As Rogers (1972) and Juliano (2001) have shown, most experimental studies of predation underestimate predator efficiency at low prey densities because consumed prey are not replaced. Although this might explain the high frequency of sigmoid functional responses, the Roger's Random Predator Equation (1972), which accounts for the effects of prey depletion, had a lower (better) AICc score than the type 2 equation for only 8 datasets (6%) (data not shown). (3) The type 2 is rare because it is actually rare. Despite the fact that evolutionary biology suggests that the components of the functional response should depend on prey density (Abrams 1982) and that the predictions of models that incorporate the type 2 are incongruent with observations of natural systems, the type 2 has remained prominent. Our analyses suggest that the perceived prevalence of the type 2 functional response is at least partially due to biases within researchers themselves. Almost half (47%) of "type 2" datasets are actually best fit by monotonic, sigmoid functional responses ($f_{5A}(x)$, $f_{5B}(x)$, and $f_{6}(x)$) (Table 4.1). Some of this bias is expected; the type 2 does provide an adequate, although inferior, description of uptake for 61% of datasets and even those datasets that are fit much better by other descriptors can easily be mistaken for type 2 upon visual inspection (Fig. 4.4 *b*). Furthermore, given the overwhelming number of possible functional response equations (Jeschke, Kopp & Tollrian 2002), it is understandable that authors may only consider a small percentage of potential models. However, had authors considered $f_{5A}(x)$ (i.e. the type 3, which is a widely accepted and common sigmoid functional response) in addition to the type 2, they would have found the type 3 was a better model for 47 datasets (33%) coming from 25 publications (data not shown).

On the other hand, the type 3 is only one of many possible models. While 99% of datasets are best fit by PDC functional response equations, our results do not indicate a single PDC functional response that best describes all datasets; nor do we expect that the diversity of the natural world would allow such a "dominant" equation to exist. In addition to inducible offenses (Padilla 2001; Mougi & Iwasa 2011), previous studies have identified a long list of phenomena that may [or may not] influence consumption, such as: genetic diversity (Fussmann, Ellner & Hairston 2003), inducible defenses (Hessen & Vandonk 1993), the ratio of predator to prey density (Arditi & Ginzburg 1989), epigenetics (Agrawal, Laforsch & Tollrian 1999), etc. Even if one only considers PDCs in pairwise predator-prey interactions, numerous functional relationships are possible (Fig. 4.1); furthermore, limiting analyses to a single phenomenon, such as decreasing handling time, will not exempt one from considering multiple functional response shapes and equations (Figs. 4.2 *g*, *m*, Table 4.1). Therefore, it is highly likely that any given functional response equation or shape will only apply to a subset of predator-prey interactions; a conclusion which is also supported by our data (Table 4.1). Given that multiple viable models exist, the use of traditional statistical methods, such as pairwise comparisons and null hypothesis testing, can lead to suboptimal results (Johnson & Omland 2004; Wagenmakers & Farrell 2004). Instead, we suggest that researchers performing functional response experiments fit a wide range of alternative models to their data and use model selection criteria (i.e. AIC, AICc, BIC, Schwarz criterion, etc.) to determine the best descriptor of consumption.

Through our own use of model selection criteria, we found widespread support for PDC functional responses, which is especially important in light of the fundamentally different predictions of models that incorporate PDC functional responses and those that incorporate the type 2. It is well known that in models that incorporate type 2 functional responses, as *K*

approaches infinity, the breadth of stability always approaches 0, and thus, destabilization due to enrichment (i.e. the paradox of enrichment) is almost certain (Kretzschmar, Nisbet & McCauley 1993; Vos *et al.* 2004; Yamamichi, Yoshida & Sasaki 2011). Our results indicate that inevitable destabilization also occurs for unimodal functional responses and monotonic, non-sigmoid functional responses that do not satisfy Equation 4.3 (see Appendix 4.A). However, for models using sigmoid and/or non-saturating equations (that do satisfy Eq. 4.3), it is possible to avoid destabilization in the m_y-K parameter space. Even in simple bitrophic systems, incorporating these functional responses can allow the breadth of stability to remain constant with increasing carrying capacity, a concept known as "strong" stability (Kretzschmar, Nisbet & McCauley 1993). These results confirm previous findings that incorporating sigmoid functional responses can counteract the destabilizing effects of enrichment (McNair 1986; Yodzis & Innes 1992).

On the other hand, while previous studies have shown that monotonic, non-sigmoid, non-saturating equations (that satisfy Eq. 4.3) can promote stable coexistence (Rosenzweig 1971; Okuyama 2010), ours is the first to show that these functional responses can produce strong stability. However, four important distinctions exist: (1) Strong stability is distinct from "complete" stability (the total absence of limit cycles). PDC functional responses cannot eliminate the potential for limit cycles; instead, they can reduce the extent of this instability. In contrast, some phenomena can produce complete stabilization in enriched systems: constant proportion prey refuges (Maynard Smith 1974) but see (McNair 1986); wasteful killing, where predators kill prey with a type 1 functional response, but consume prey with a type 2 functional response (Turchin 2003); and inedible prey, where edible prey maintain a fixed population density and all nutrients above some value are absorbed by the inedible prey (Grover 1995). However, while systems exhibiting complete stability are more robust to parameter choice (i.e.,

cyclic coexistence does not exist in the m_y–K parameter space) than those that exhibit strong stability (although see (McNair 1986)), the dynamic stability of natural systems facing enrichment can be explained without invoking phenomena that produce complete stability (Abrams & Walters 1996; Vos *et al.* 2004; Roy & Chattopadhyay 2007; Kovach-Orr & Fussmann 2013). Many previously identified solutions to the paradox of enrichment, such as inedible prey where edible prey maintain a constant percentage of total prey density (Kretzschmar, Nisbet & McCauley 1993), inducible defenses (Vos *et al.* 2004), and invulnerable prey (Abrams & Walters 1996) also fail to eliminate the potential for limit cycles. While these phenomena can prevent destabilization due to enrichment for some range of mortality rates (or transition rates, in the case of invulnerable prey), limit cycles and deterministic extinction exist outside of these ranges.

(2) In systems that utilize sigmoid and/or non-saturating functional responses, two separate mechanisms can allow for strong stabilization. Sigmoid functional responses produce strong stabilization because at low predator mortality rates, top down control forces prey to densities that correspond to the "concave-up/accelerating" section of the functional response. The effects of accelerating predation mimic the effects of linear predation (i.e. the type 1 functional response) by buffering the system against overexploitation and prey escape (Oaten & Murdoch 1975). On the other hand, non-saturating functional responses result in increased consumption at high prey densities, which allows predators to maintain higher population densities compared with models that use equivalent saturating functional responses. Increased consumption is especially important at high mortality rates because it can allow predators to avoid deterministic extinction and increased predator density strengthens top-down control, which buffers the system against prey escape. The combination of these effects results in strong

stability at high mortality rates (Figs. 4.5 *f, l*). While it is true that for any given mortality rate, increasing carrying capacity will eventually lead to destabilization for models using monotonic, non-sigmoid, non-saturating functional responses, our results show that for any given carrying capacity, there is a non-negligible range of mortality rates where stable coexistence is possible (Figs. 4.5 *e, f, k, l*). This is distinct from the predictions of models that use type 2 functional responses, where the breadth of stability quickly decreases and approaches a value of 0, with increasing carrying capacity (Figs. 4.5 *a, b*).

(3) Sigmoid functional responses can be produced by incorporating an increasing attack rate and/or decreasing handling time (Figs. 4.2 *j, m*); in fact, the type 3 functional response can be rearranged so that the sigmoid shape comes from a prey density-dependent handling time instead of attack rate (see Appendix 4.B). On the other hand, non-saturating functional responses can only be produced if handling time asymptotically approaches 0 as prey density increases; therefore, strong stabilization, at high mortality rates, can only occur if handling time approaches 0 (Figs. 4.5 *f, l*). (4) The inclusion of PDCs does not unilaterally promote stable coexistence. For certain combinations of predator mortality and low carrying capacity, some PDC functional responses result in limit cycles while stable coexistence is observed with the type 2 functional response (Fig. 4.5); although, as carrying capacity increases, the strongly stabilizing PDC functional responses ($f_4(x)$, $f_{5A}(x)$, $f_{5B}(x)$, and $f_6(x)$) do have more stable coexistence (Fig. 4.5). Despite the fact that under some conditions, PDC functional responses can hinder stable coexistence, we found that the majority of datasets (69%) are best fit by PDC functional responses that promote strong stability (Table 4.1, Fig. 4.5). Thus, PDC functional responses may help bridge the gap between theoretical predictions and observations of natural systems.

Some of the conclusions drawn from our work rely on the assumption that handling time can approach 0 for two PDC functional responses, $f_4(x)$ and $f_6(x)$. Although this phenomenon is possible under some circumstances – for instance, many filter feeders exhibit linear (i.e., type 1) functional responses (Jeschke 2004) – for our equations, as prey density approaches infinity, consumption will also approach infinity. We do not wish to imply that "true" non-saturating functional responses can exist in real predator-prey systems; at some high prey density, predators will become unable to increase consumption with increasing prey density and consumption will approach an asymptote. However, we believe that the use of $f_4(x)$ and $f_6(x)$ is justified because they provide useful examples of situations where saturation occurs at such high prey densities and consumption rates that imposing a limit becomes unnecessary. In an attempt to fully understand the 32 datasets that are best fit by $f_4(x)$, we tested many different equations where handling time decreases towards an asymptotic limit greater than 0 (data not shown). For these 32 datasets, $f_4(x)$ was superior to all but one alternative: a functional response model built on $f_4(x)$, but where handling time increased by a constant, given by:

$$f_{4B}(x) = \frac{x\ a}{x\ a\ T_h(x) + 1}\ \text{, where } T_h(x) = \frac{c}{\ln(x+1)+1} + \beta \qquad (4.4)$$

and where β is a constant and all other variables are the same as $f_4(x)$. However, it is not until β is less than, or equal to, 10^{-20}, that the statistical fit of this alternative surpasses that of $f_4(x)$. For these 32 datasets, the maximum consumption rate predicted by $f_{4B}(x)$ is, on average, 19 orders of magnitude (2.8×10^{19}) times higher than the maximum predicted by the type 2 (median $=1.7 \times 10^{18}$). Over biologically plausible prey densities, model predictions are virtually identical for $f_4(x)$ and $f_{4B}(x)$ (Fig. 4.6). Therefore, the distinction between a functional response that saturates at implausibly high values and one that never truly saturates is far less meaningful than the distinction between these functional responses and a functional response that saturates at

relatively low values; nevertheless, this does indicate that non-saturating predation cannot create "true" strong stability.

We acknowledge that our analysis of the prevalence of inducible offenses that create PDC functional responses is limited because we excluded datasets where the functional response had been categorized as something other than type 2 (i.e. type 1, 3, or 4 functional responses). However, we believe that type 1 functional responses fall outside the scope of our analyses because type 1 functional responses are exclusive to a specific class of predators: filter feeders (Jeschke 2004). Furthermore, the type 3 and 4 functional response equations often incorporate prey density-dependent components (Holling 1966; Gentleman *et al.* 2003). On the other hand, our analyses were constrained to specific biological relationships and therefore, a small subset of potential functions; yet, despite these constraints, our results overwhelmingly support PDC functional responses. Furthermore, our goal was not an exhaustive analysis of every possible phenomenon, but rather to determine if PDC functional responses, and therefore inducible offenses, are likely a common occurrence and to evaluate how incorporating PDCs affects model predictions.

In summary, we found that PDC functional responses outperform the type 2 in two important ways: first, almost all "type 2" datasets (99%) are best fit by PDC functional responses; second, for the majority of datasets (69%) , models built on the best fitting functional response produce strong stability, which is more consistent with observations of natural systems. Our findings are especially exciting because many previous solutions to the mismatch between theoretical predictions and field observations have widely been seen as system specific (Roy & Chattopadhyay 2007). While our study does not fully resolve the paradox of enrichment, our results suggest that inducible offenses that create PDCs are a common, if not pervasive, property

of predator-prey interactions, and that predictions of frequent or wide spread instability are overstated. In order to better describe predator-prey interactions, we recommend that statistical evaluations of functional response data involve model selection criteria (e.g., AIC analysis) of a wide variety of alternatives to the type 2. Additionally, our study highlights the importance rechecking accepted principles in ecology. All ecological models are built upon other models; if we do not fully comprehend low complexity models, how can we expect to predict dynamics in natural communities?

4.7 Acknowledgements

G.F.F. and C.K.O. acknowledge support from the James S. McDonnell Foundation and NSERC; G.F.F. was also supported by the EU project FEMMES (MTKD-CT-2006-042261). M.H.C. was supported by the National Science Foundation under Award No. DMS- 1204401. We thank editor Kevin Gross, two anonymous reviewers, and the Fussmann Laboratory for suggestions and comments that have improved the manuscript. G.F.F. and C.K.O. thank Ursula Gaedke and her group at the University of Potsdam, Germany, for hosting our research stays and for discussions

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4.9 Tables

Table 4.1. Fit of functional response models to empirical datasets. Rows 1 and 2: The number (and percentage) of datasets best fit by each equation. Rows 3 and 4: The number (and percentage) of datasets for which the AIC weights analysis indicates a fit to one of the PDC models ($f_3(x)$ through $f_7(x)$) that is at least twice as likely as the fit to the type 2 equation ($f_2(x)$).

functional response	$f_2(x)$	$f_3(x)$	$f_4(x)$	$f_{5A}(x)$	$f_{5B}(x)$	$f_6(x)$	$f_7(x)$
# of datasets best fit	2	7	32	35	6	26	36
% of datasets best fit	1.4%	4.9%	22.2%	24.3%	4.2%	18.1%	25.0%
# of best fit datasets where model is 2 (or more) times as likely as the type 2	-	1	13	24	0	13	5
% of best fit datasets where model is 2 (or more) times as likely as the type 2	-	0.7%	9.0%	16.7%	0.0%	9.0%	3.5%

4.10 Figures

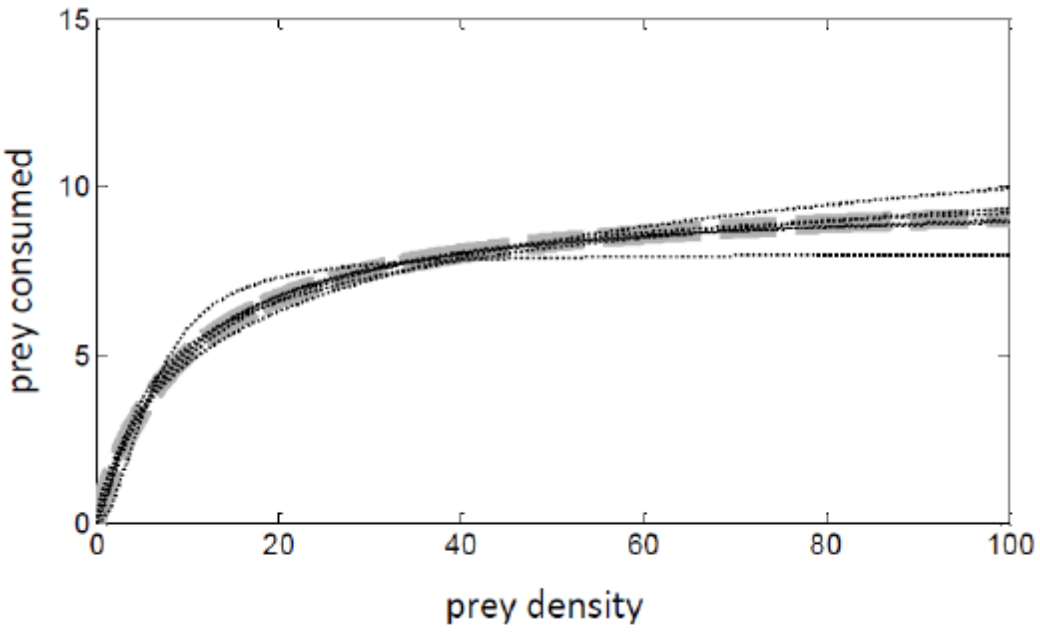

Figure 4.1: Holling type 2 (*grey- dashed curve*) and prey density-dependent component (PDC) (*black- dotted curves*) functional responses. Prey density (x) is shown on the x-axis. Consumption ($f(x)$) is shown on the y-axis. Type 2: a =1, T_h = 0.1. The six PDC functional response curves depicted represent the curves with maximum phenomenological similarity to the type 2 curve over the range of x=0 to x=90. Under the current parameterization, $f_2(x)$ approaches an asymptotic limit of 10 and that at a prey density of 90, $f_2(x)$ is equal to 9. Note that only five PDC curves are discernible due to the strong similarity of two curves. Parameter values for PDC functional responses are given in Table 4.C1.

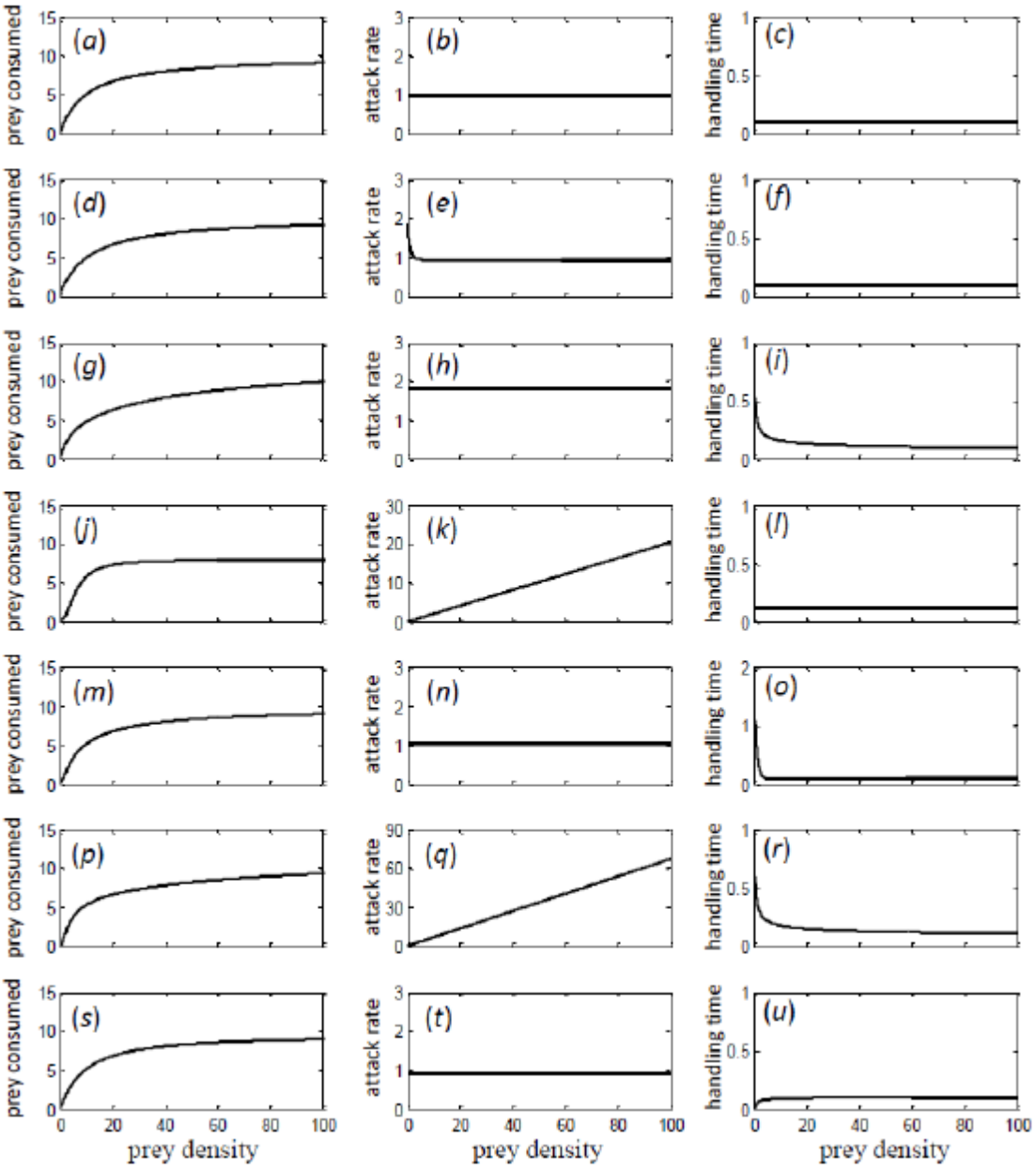

Figure 4.2: Holling type 2 and prey density-dependent component (PDC) functional responses. Prey density (x) is shown on the x-axis. Functional response curves (left column), attack rates (middle column), and handling times (right column) are shown on the y-axis. (*a-c*) $f_2(x)$, the type 2 - a monotonic, non-sigmoid, saturating functional response. (*d-f*) $f_3(x)$ - a PDC monotonic, non-sigmoid, saturating functional response. (*g-i*) $f_4(x)$ - a PDC monotonic, non-

sigmoid, non-saturating functional response. (*j*-*l*) $f_{5A}(x)$, the type 3 - a PDC monotonic, sigmoid, saturating functional response. (m_y-*o*) $f_{5B}(x)$ - an alternative PDC monotonic, sigmoid, saturating functional response. (*p*-*r*) $f_6(x)$ - a PDC monotonic, sigmoid, non-saturating functional response. (*s*-*u*) $f_7(x)$ - a PDC non-sigmoid, unimodal functional response. Note that because $f_7(x)$ is based on the curve produced by $f_2(x)$ (a strictly increasing function), the unimodal behavior (decreasing section) occurs at prey densities that are much higher than presented in figure 4.2. Parameter values as in figure 4.1.

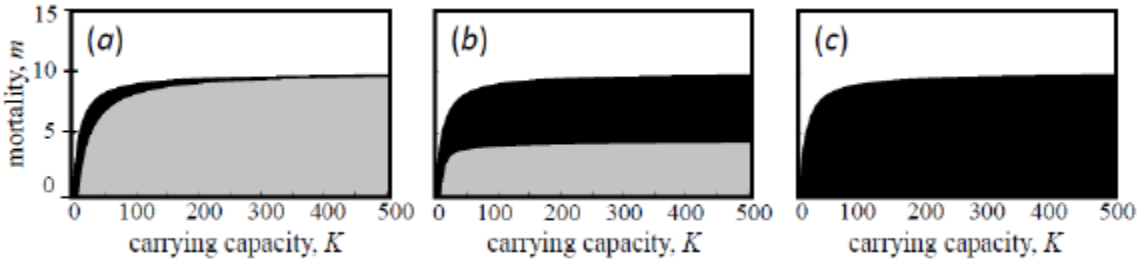

Figure 4.3: Bifurcation diagrams of a bitrophic food web. Carrying capacity (K) is shown on the x-axis, and mortality (m_y) is shown on the y-axis. The *white area* is the region of deterministic predator extinction. The *black area* is the region of stable coexistence; it is separated from the region of predator extinction by the transcritical bifurcation; above the transcritical bifurcation, the predator cannot maintain positive growth. The *grey area* is the region of cyclic coexistence (i.e. limit cycles); it is separated from the region of stable coexistence by the Hopf bifurcation. The range of mortality rates where stable coexistence occurs (i.e. the size of the *black area*) at a given carrying capacity is known as the "breadth of stability". (*a*) Weak stability: as carrying capacity approaches infinity, the breadth of stability approaches 0. (*b*) Strong stability: as carrying capacity approaches infinity, the breadth of stability approaches a value greater than 0, however, limit cycles exist for some parameter values. Also see figure 1.2c. (*c*) Complete stability: limit cycles are absent for all combinations of carrying capacity and mortality

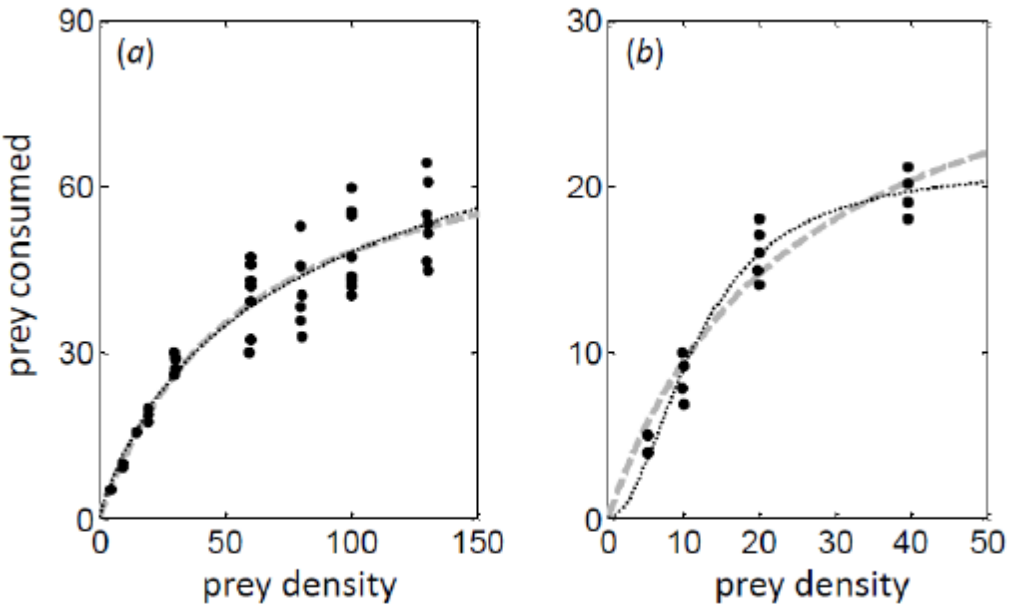

Figure 4.4: Examples of empirical consumption data (*black- filled circles*) with best fit PDC (*black- dotted curves*) and type 2 (*grey- dashed curves*) functional responses. (*a*) Although both $f_2(x)$ and $f_4(x)$ can provide adequate descriptions of uptake, $f_4(x)$ is 1.13 times more likely than $f_2(x)$ to be the correct functional response (Jalali, Tirry & De Clercq 2010). (*b*) The PDC functional response, $f_{5A}(x)$, is 29.68 times more likely than $f_2(x)$ to be the correct functional response (Fathi & Nouri-Ganbalani 2010).

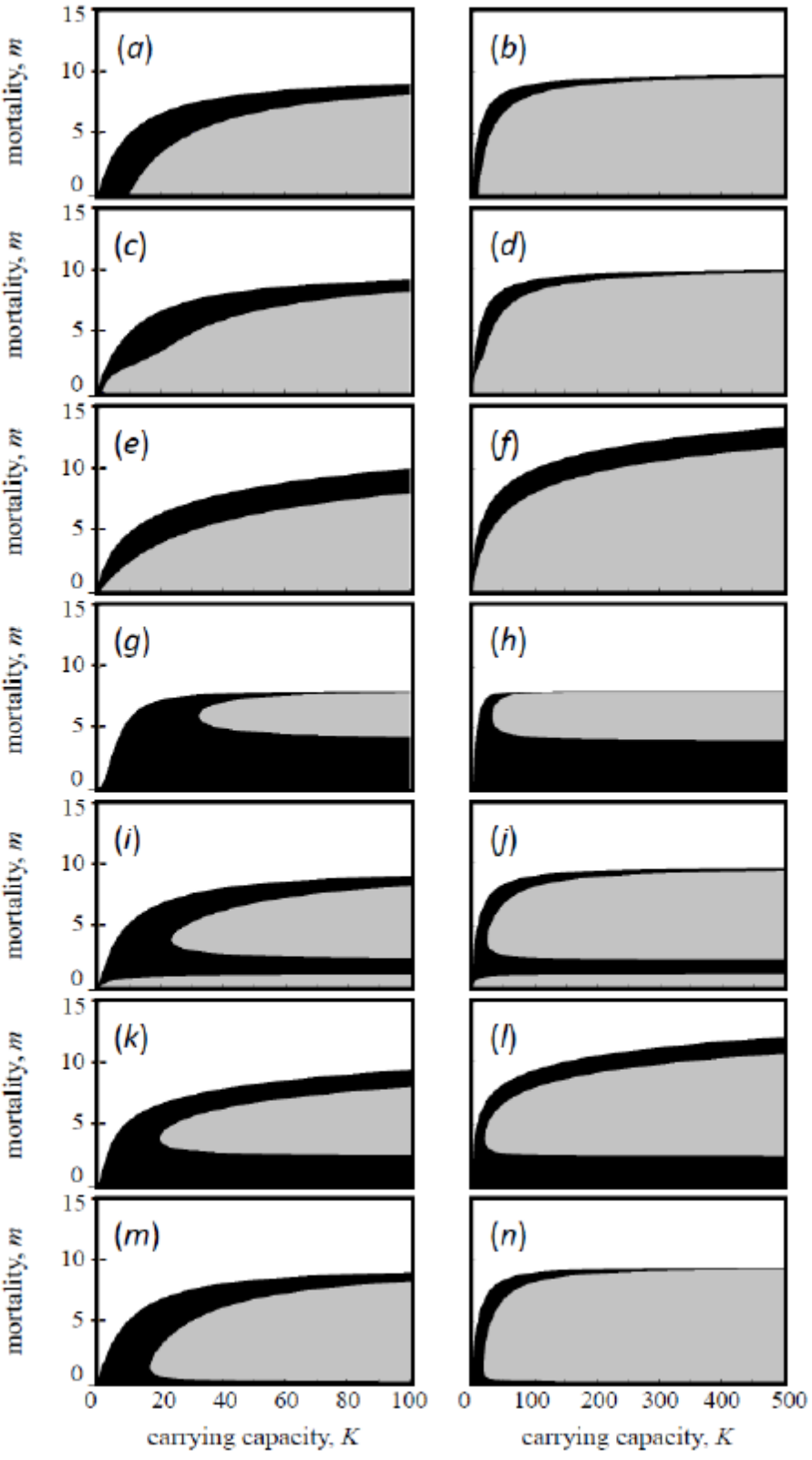

Figure 4.5: Bifurcation diagrams of Rosenzweig-MacArthur models based on different formulations of functional response. Carrying capacity (K) is shown on the x-axis, and mortality (m_y) on the y-axis. The left column shows bifurcations for K values between 0 and 100, the right column for K values between 0 and 500. For description of colors, see figure 4.3. (a, b) The classic R-M model based on $f_2(x)$ exhibiting weak stability: the breadth of stability is negatively

correlated to carrying capacity, K. (c, d) The R-M model based on $f_3(x)$ exhibiting weak stability. (e, f) The R-M model based on $f_4(x)$ exhibiting strong stability at high mortality rates. (g, h) The R-M model based on $f_{5A}(x)$ exhibiting strong stability at low mortality rates. (i, j) The R-M model based on $f_{5B}(x)$ exhibiting strong stability at low mortality rates. (k, l) The R-M model based on $f_6(x)$ exhibiting strong stability at both high and low mortality rates. (m_y, n) The R-M model based on $f_7(x)$ exhibiting weak stability. Parameter values as in figure 4.1.

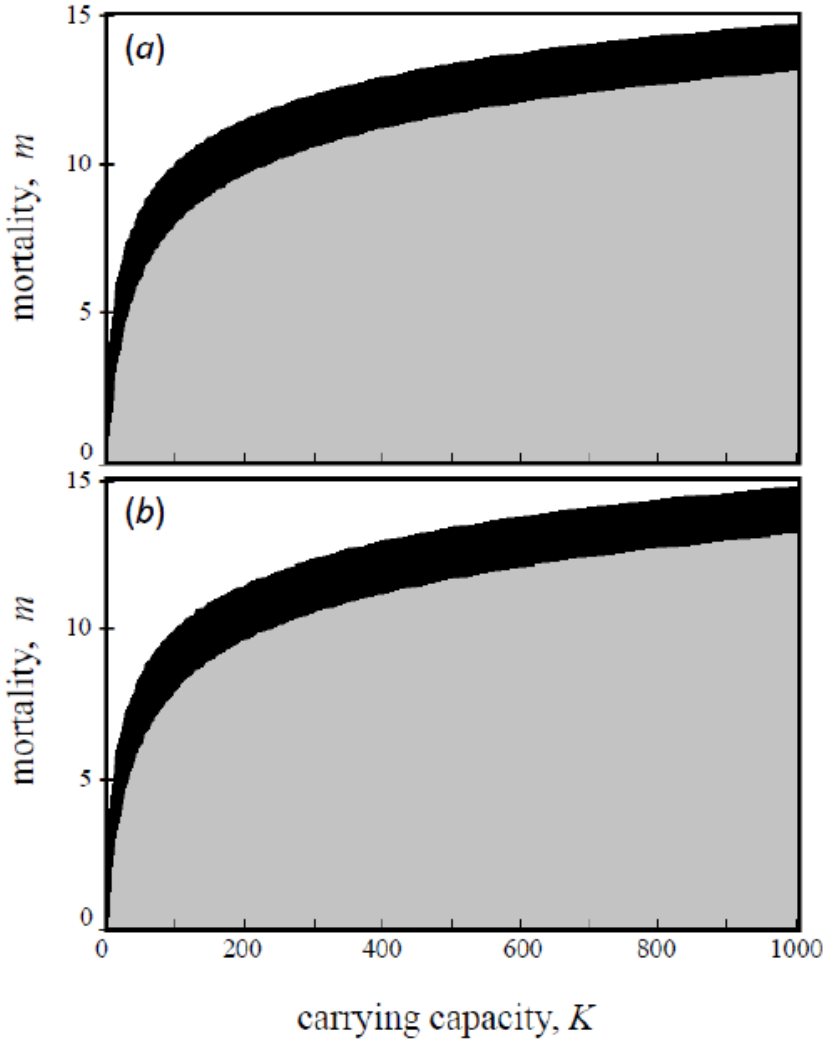

Figure 4.6: Bifurcation diagrams of Rosenzweig-MacArthur models based on (*a*) $f_4(x)$ – a PDC monotonic, non-sigmoid, non-saturating functional response and (*b*) $f_{4B}(x)$ – a PDC monotonic, non-sigmoid, functional response that approaches a maximum at "extreme" values. For description of colors, see figure 4.3. Parameter values of $f_{4B}(x)$ are given in Table 4.C1; parameter values of $f_4(x)$ as in figure 4.1.

4.11 Appendices

Appendix 4.A – Computing the breadth of stability

Appendix A.1 Model and definition of bifurcation curves

The model is

$$\begin{aligned}\frac{dx}{dt} &= rx\left(1-\frac{x}{K}\right) - yf(x)\\ \frac{dy}{dt} &= \epsilon yf(x) - my.\end{aligned} \tag{A1}$$

Via the rescaling $\bar{y} = y/\epsilon$, $\bar{t} = \epsilon t$, $\bar{r} = r/\epsilon$ and $\bar{m} = m/\epsilon$, we can scale out ϵ. Thus, throughout we set $\epsilon = 1$. Let primes denote derivatives, e.g., $f'(x) = df/dx$. We assume f has a continuous second derivative and satisfies $f(0) = 0$, $f'(x) > 0$, $f''(x) < 0$ for all x. We also assume that $f(x)/x$ is a decreasing function of x, i.e., $\frac{\partial}{\partial x}\left(\frac{f(x)}{x}\right) = [f'(x)x - f(x)]/x^2 < 0$.

Let $g = f^{-1}(m)$. The boundary equilibrium of system (A1) is $(K, 0)$. For parameters values satisfying $f(K) = m$, the boundary equilibrium undergoes a transcritical bifurcation. In parameter space, the transcritical bifurcation curve is

$$K_T(g) = g. \tag{A2}$$

The coexistence equilibrium is

$$(x^*, y^*) = \left(f^{-1}(m), \frac{rf^{-1}(m)}{m}\left[1 - \frac{f^{-1}(m)}{K}\right]\right). \tag{A3}$$

Note that $x^* = g$. The Jacobian evaluated at the coexistence equilibrium is

$$J|_{(x^*,y^*)} = \left.\begin{pmatrix} r\left(1-\frac{x}{K}\right) + \frac{-rx}{K} - yf'(x) & -f(x) \\ y\frac{\partial}{\partial x}f(x) & 0 \end{pmatrix}\right|_{(x^*,y^*)}. \tag{A4}$$

A Hopf bifurcation occurs when the trace of the Jacobian equals zero,

$$0 = r\left(1-\frac{x}{K}\right) + \frac{-rx}{K} - yf'(x)\Big|_{(x^*,y^*)}. \tag{A5}$$

Solving equation (A5) for K yields the equation for the Hopf bifurcation curve,

$$K_H(g) = g + \frac{gf(g)}{f(g) - gf'(g)}. \tag{A6}$$

Note that our assumption $\frac{\partial}{\partial x}\left(\frac{f(x)}{x}\right) < 0$ implies $f(g) - gf'(g) > 0$.

In equation (A6), the Hopf curve is defined as a function of g. We now take the inverse of equation (A6) and write the Hopf curve as a function of K. In general, the resulting function of K will be multivalued. For those cases, we focus on the upper branch of the function and denote the branch by $K_H^{-1}(K)$, or equivalently, $m_H(K) = f(K_H^{-1}(K))$. Because we only focus on the upper branch, our analytical results only address when weak and strong stability arise for high predator mortality rates. Note that in this notation the transcritical bifurcation curve is $m_T(K) = f(K)$.

Appendix A.2 Computing the distance between bifurcation curves

We are interested in the conditions under which the Hopf and transcritical bifurcation curves do and do not converge as K tends to infinity. Using the above notation, we are interested in the value of $\lim_{K\to\infty} m_T(K) - m_H(K)$. We first derive the distance between the Hopf and transcritical bifurcation curves for a particular value of K. Choose K sufficiently large such that there exists a Hopf bifurcation for some value of m. Let $[K, m_1]$ denote the point on the Hopf bifurcation curve with this value of K. Via equation (A6), this point can be written as $[K_H(g_1), m_1]$ where $g_1 = f^{-1}(m_1)$. Since the transcritical bifurcation curve is defined by $m_T(K) = f(K)$, the corresponding point on the transcritical bifurcation curve is $[K, f(K)] = [K_H(g_1), f(K_H(g_1))]$. Dropping the subscript 1 ($_1$) notation, yields the distance between the Hopf and transcritical bifurcation curves for any value of K,

$$m_T(K) - m_H(K) = f(K_H(g)) - m_1 = f(K_H(g)) - f(g). \tag{A7}$$

We use this formula to compute $\lim_{K\to\infty} m_T(K) - m_H(K)$.

We first consider non-sigmoid functional responses that are increasing and have an upper limit, e.g., the type 2 functional response. Such functional responses satisfy $\lim_{x\to\infty} f(x) = c < \infty$. The following shows that these functional responses always yield weak stability in Rosenzweig-MacArthur types models.

Theorem 1. *Assume the continuous function f satisfies $f'(x) > 0$, $f''(x) < 0$, $\frac{\partial}{\partial x}(f/x) < 0$, and $\lim_{x\to\infty} f(x) = c < \infty$. Then, $f(K_H(g)) - f(g) \to 0$.*

Proof. Let $g = f^{-1}(m_1)$. Note that $\lim_{m_1\to c} g = \infty$. Also, $\lim_{x\to\infty} f(x) = c < \infty$, $f' > 0$, and $f'' < 0$ imply that $\lim_{x\to\infty} f'(x) = 0$. Thus

$$\begin{aligned}
\lim_{m_1\to c} f(K_H(g)) - f(g) &= \lim_{m_1\to c} f\left(g + \frac{gf(g)}{f(g) - gf'(g)}\right) - m_1 && \text{(A8)}\\
&= f\left(\lim_{m_1\to c} g + \lim_{m_1\to c} \frac{gf(g)}{f(g) - gf'(g)}\right) - c && \text{(A9)}\\
&= f\left(\lim_{g\to\infty} g + \lim_{g\to\infty} \frac{gf(g)}{f(g) - gf'(g)}\right) - c && \text{(A10)}\\
&\leq f\left(\lim_{g\to\infty} g + \lim_{g\to\infty} \frac{gf(g)}{f(g)}\right) - c && \text{(A11)}\\
&= f\left(\lim_{g\to\infty} g + \lim_{g\to\infty} g\right) - c && \text{(A12)}\\
&= c - c = 0. && \text{(A13)}
\end{aligned}$$

In the above we make use of $\frac{\partial}{\partial x}(f/x) < 0$, $f(g) > f(g) - gf'(g)$, and that f is a continuous function. □

We now consider functional responses that do not have an upper limit. These functions satisfy $\lim_{x\to\infty} f(x) = \infty$, which implies $\lim_{g\to\infty} K_H(g) = \infty$. For this class of functions it is convenient to write $\lim_{K\to\infty} m_T(K) - m_H(K)$ in a different form. Our goal is to compute

$$\lim_{g\to\infty} f(K_H(g)) - f(g) = \lim_{g\to\infty} f\left(g + \frac{gf(g)}{f(g) - gf'(g)}\right) - f(g) \tag{A14}$$

Because f has a continuous first derivative, we can rewrite the right hand side as

$$\lim_{g\to\infty} f(K_H(g)) - f(g) = \lim_{g\to\infty} \int_g^{g+A(g)} f'(x)dx \tag{A15}$$

where $A(g) = gf(g)/[f(g) - gf'(g)]$. We now use the above to determine the breadth of stability for functional responses that do not have an upper limit.

Theorem 2 shows that if the derivative of f is bounded from below, then the breadth of stability is infinite. Thus, these functions yield strong stability. Theorem 3 gives one sufficient condition for weak stability and one sufficient for strong stability. Note that theorem 3 does not address functions where $xf'(x) \to c > 0$ and $xf'(x + A) \to 0$. It is unclear whether the bifurcation curves converge in this case or not.

Theorem 2. *Assume the continuous function f satisfies $f'(x) > 0$, $f''(x) < 0$, $\frac{\partial}{\partial x}(f/x) < 0$, and $f'(x) \to c > 0$. Then $\lim_{g\to\infty} f(K_H(g)) - f(g) = \infty$.*

Proof. $f''(x) < 0$ and $f'(x) \to c > 0$ imply $f'(x) > c > 0$ for all x, which implies

$$\lim_{g\to\infty} \int_g^{g+A(g)} f'(x)dx \geq \lim_{g\to\infty} \int_g^{g+A(g)} cdx = \lim_{g\to\infty} cA(g) \geq \lim_{g\to\infty} g = \infty.$$

The last inequality follows from $A(g) = gf(g)/[f(g) - gf'(g)] > gf(g)/f(g) = g$ since $-gf'(g) < 0$ and $f(g) - gf'(g) > 0$. □

Theorem 3. *Assume the continuous function f satisfies $f'(x) > 0$, $f''(x) < 0$, $\frac{\partial}{\partial x}(f/x) < 0$, and $\lim_{x\to\infty} f'(x) = 0$. Let $A(x) = xf(x)/[f(x) - xf'(x)]$.*
(i) If $\lim_{x\to\infty} xf'(x) = 0$, then $\lim_{g\to\infty} f(K_H(g)) - f(g) = 0$.
(ii) If $\lim_{x\to\infty} xf'(x + A(x)) = c > 0$, then $\lim_{g\to\infty} f(K_H(g)) - f(g) \geq c$.

Proof. Proof of (i): Since $f''(x) < 0$ and $A(g) > 0$, $f'(g) \geq f'(x)$ for all $x \in [g, g + A(g)]$. Thus,

$$\int_g^{g+A(g)} f'(x)dx \leq \int_g^{g+A(g)} f'(g)dx = A(g)f'(g). \tag{A16}$$

Since $\lim_{x\to\infty} xf'(x) = 0$, we have that

$$\lim_{x\to\infty} A(x)f'(x) = \lim_{x\to\infty} \frac{xff'}{f - xf'} = \lim_{x\to\infty} xf' \lim_{x\to\infty} \frac{f}{f - xf'} = 0 \cdot 1 = 0 \tag{A17}$$

Hence, $0 \geq \lim_{g\to\infty} \int_g^{g+A(g)} f'(x)dx \leq \lim_{g\to\infty} A(g)f'(g) = 0$.

Proof of (ii): Since $f'' < 0$ and $A(g) > 0$, $f'(g+A) \leq f'(x)$ for all $x \in [g, g+A(g)]$. Recall from the proof of theorem 2 that $A(g) > g$. Thus,

$$\int_g^{g+A(g)} f'(x)dx \geq \int_g^{g+A(g)} f'(g+A(g))dx = A(g)f'(g+A(g)) > gf'(g+A(g)) \quad \text{(A18)}$$

Hence, if $\lim_{x\to\infty} xf'(x + A(g)) = c > 0$, then $\lim_{g\to\infty} \int_g^{g+A(g)} f'(x)dx \geq c$. □

Appendix A.3 Interpretation and application of results

Our results about the occurrence of weak and strong stability at high mortality rates are summarized as follows:

(1) If $f''(x) < 0$, $f'(x) \to 0$ and $f(x) \to c > 0$, then weak stability occurs.

(2) If $f'(x) \to c > 0$, then strong stability occurs.

(3) Assume $f''(x) < 0$, $f(x) \to \infty$ and $f'(x) \to 0$. If $xf'(x) \to 0$, then weak stability occurs. If $xf'(x + A(x)) \to c > 0$ where $A(x) = xf(x)/[f(x) - xf'(x)]$, then strong stability occurs.

Condition (1) says that weak stability occurs for all non-sigmoid saturating functional responses, e.g., the type 2 functional response, $f_2(x) = ax/(1 + hax)$. Conditions (2) and (3) tell us that strong stability can occur for non-sigmoid non-saturating functional responses at high mortality rates, but non-saturation is not enough to guarantee that strong stability will occur; we need to know additional information about $f'(x)$. Condition (2) says that strong stability will occur if the functional response looks linear (with nonzero slope) eventually, e.g., a hybrid of the type 1 and 2 functional responses, $f(x) = bx + ax/(1 + hax)$. Condition (3) implies that if the slope of the functional response does approach 0, but the functional response does not saturate, then weak or strong stability is possible. We do not currently have a biological interpretation for condition (3).

Conditions (1) through (3) provide a checklist for determining if strong or weak stability will occur in Rosenzweig-MacArthur type models with non-sigmoid functional responses. Begin by asking, 'is the function bounded from above, i.e., does it saturate?' If yes, then weak stability will occur. If not, then ask, 'is the derivative bounded from below?' If yes, then strong stability will occur. If not, then check the conditions on xf' and $xf'(x + A(x))$. By going through these conditions, one can determine if the bifurcation curves converge or not. Below we apply our results to the functional forms considered in the main text.

f_2 yields weak stability: f_2 is bounded above by $1/h$, hence via theorem 1 it yields weak stability for large mortality rates.

f_3 yields weak stability: f_3 is bounded above by $1/h$, hence via theorem 1 it yields weak stability for large mortality rates.

f_4 yields strong stability: f_4 is unbounded and $f_4' \to 0$ as $x \to \infty$. Computing the leading order terms of the numerator and denominator of $xf'(x + A(x))$ yields $\lim_{x\to\infty} xf'(x + A(x)) = 1/2c$. Hence, via theorem 3, f_4 yields strong stability for large mortality rates.

f_{5A} yields strong stability: f_{5A} is bounded above by $1/T_h$, hence, via theorem 1, only weak stability occurs for large mortality rates. The Hopf bifurcation has three branches which are solutions to the third order polynomial (in K) defined by equation (A5). The upper branch converges to the transcritical bifurcation curve (which is the result from theorem 1) and the lower branch only exists for negative values of m. The middle branch approaches the constant $1/2T_h$ from above, implying that the system is stable for all values of K if $m \leq 1/2T_h$. Hence, f_{5A} allows for strong stability for low mortality rates.

f_{5B} yields strong stability: f_{5A} is bounded above by $1/T_h$, hence, via theorem 1, only weak stability occurs for large mortality rates. The branches of the Hopf bifurcation are defined by solutions to equation (A5), or equivalently

$$K_H(g) = g + \frac{gf(g)}{f(g) - gf'(g)} = g + \frac{1}{a}\left[\frac{1 + agc + age^{-1}}{c + (1 - g)e^{-g}}\right]. \tag{A19}$$

There are three branches of interest but we cannot compute the branches analytically because equation (A19) is a transcendental equation. However, we can still compute the breadths of stability. We know, via theorem 1, that one branch converges to the transcritical bifurcation curve. Hence, the breadth of stability for large m (or large g) values is zero. The other two branches exist for intermediate values of m. The breadth of stability for intermediate mortality rates is related to the distance between the vertical asymptotes of $K_H(g)$. The asymptotes are defined by solutions to $0 = c + (1 - g)e^{-g}$. If g_1 and g_2 denote the locations of the asymptotes, then the breadth of stability for intermediate m values is $|f(g_1) - f(g_2)|$. Table C2 presents numerically computed breadths of stability for different parameter values. Because the distance between the asymptotes is nonzero, f_{5B} allows for strong stability for intermediate values of m.

f_6 yields strong stability: f_6 is unbounded and $f_4' \to 0$ as $x \to \infty$. Computing the leading order terms of $xf'(x + A(x))$ yields $\lim_{x\to\infty} xf'(x + A(x)) = 1/2c$. Hence, via theorem 3, f_6 yields strong stability for large mortality rates.

f_7 yields weak stability: f_7 is bounded above by $1/h$, hence via theorem 1 it yields weak stability for large mortality rates. Note that there exists a lower branch of the Hopf bifurcation curve, but it converges to $m = 0$, implying weak stability for low mortality rates as well.

Appendix 4.B – Signatures of density-dependent components

Our fits of different functional responses to the datasets suggests that density dependent attack rates and handling times are common and widespread. In addition to this statistical support, our functional response equations also suggest that qualitative signatures of density dependent attack rates and handling times can be seen in the data. To see this, consider the ratio of the prey density to the predator functional response, $x/f(x)$. For the type 2 functional response, the ratio is a line with constant (positive) slope, $x/f_2(x) = xT_h + 1/a$. For functional responses with density dependent attack rates and handling times, the ratio is a curve with non-constant slope, $x/f(x) = xT_h(x) + 1/a(x)$. These differences strongly suggest that the shape of plots of the ratio $x/f(x)$ versus prey density x can be used to identify situations where the components of the functional response depend on prey density. In particular, linearity suggests a type 2 functional response whereas nonlinearity suggests a functional response with density dependent parameters.

We fit both linear and quadratic models to the transformed data and then used AIC analyses (Akaike 1974) to determine which model best fit the data. Greater support for a linear model supports the type 2 functional response. Greater support for a quadratic model supports the functional responses with density dependent parameters. For 72 of our data sets, there was more support for the quadratic models than the linear models. As an illustrate example, figure *A1a* and *A1b* presents the original and transformed data, respectively, from the Fathi and Nouri-Ganbalani (2010) study. In figure *A1b*, the data has a concave up shape. This corroborates our statistical analysis which identifies $f_{5A}(x)$, a function that incorporates an increasing attack rate, as the best fitting functional response (solid curve in figure *A1a*).

It is important to note that while this methodology can help identify if components of the functional response depend on prey density, it faces two significant limitations. (1) The transformation requires the exclusion of important data in two settings. First, one must exclude data points where predators do not consume any prey because including these data causes a division by zero in the ratio $x/f(x)$. Second, because the ratio $x/f(x)$ is very sensitive to small values of $f(x)$, data where consumption is low can be very noisy, often requiring the removal of the data. When fitting the untransformed data with the functional responses as in the main text, neither of these

issues arise, and hence data does not need to be excluded.

(2) While the deviations from linearity are easy to test for, the methodology does not allow one to distinguish between density dependent handling times and density dependent attack rates. This model identifiability problem arises because one cannot tease apart the density dependent terms in the ratio $x/f(x) = xT_h(x) + 1/a(x)$ with just functional response data. Consequently, fitting the transformed data does not allow one to identify the mechanism driving the nonlinearities in the transformed data.

Mathematically, this identifiability problem can be seen by rewriting our functional response with density dependent attack rates and handing times as

$$\begin{aligned} f(x) &= \frac{a(x)x}{1 + a(x)T_h(x)x} = a_0 x \left(\frac{a(x)}{a_0 + a_0 a(x) T_h(x) x} \right) \\ &= a_0 x \left(\frac{1}{\frac{a_0 + a_0 a(x) T_h(x) x}{a(x)}} \right) = a_0 x \left(\frac{1}{1 - \frac{x}{x} + \frac{a_0 + a_0 a(x) T_h(x) x}{a(x)}} \right) \\ &= a_0 x \left(\frac{1}{1 - \frac{x}{x} + \frac{a_0}{a(x)} + a_0 T_h(x) x} \right) \\ &= \frac{a_0 x}{1 + a_0 x \left[\frac{1}{a_0 a(x) x} + T_h(x) - \frac{1}{a_0 x} \right]} \end{aligned} \tag{B1}$$

where a_0 is any constant that represents the new density independent attack rate and the new density dependnet handling time is $\left[\frac{1}{a_0 a(x) x} + T_h(x) - \frac{1}{a_0 x} \right]$. This transformation shows that mathematically a density dependent attack rate can be turned into a density dependent handling time. As a more concrete example, consider the type 3 functional response $f_3(x) = ax^2/(1 + aT_h x^2)$ where the density dependent attack rate is an increasing function of prey density, $a(x) = ax$. The type 3 functional response can be rewritten as

$$f(x) = \frac{ax^2}{1 + aT_h x^2} = \frac{ax}{1 + ax \left[\frac{1}{ax^2} + T_h - \frac{1}{ax} \right]} \tag{B2}$$

where the new handling time, $\left[\frac{1}{ax^2} + T_h - \frac{1}{ax} \right]$ is a decreasing function of prey density. This implies that the shape of the type 3 functional response can be driven by (i) an increasing attack rate and a constant handling time or (ii) a decreasing handling time and a constant attack rate.

In total, our analysis suggests that curvature (or deviations from linearity) in the transformed data are a signature of density dependent attack rates and handling times. The nonlinear shapes observed in 72 of the transformed data sets lends additional support to our claim that density dependent attack rates and handling times are common. However, we note that this methodology can only lend qualitative support to our statistical analysis because of its sensitivity to small consumption values.

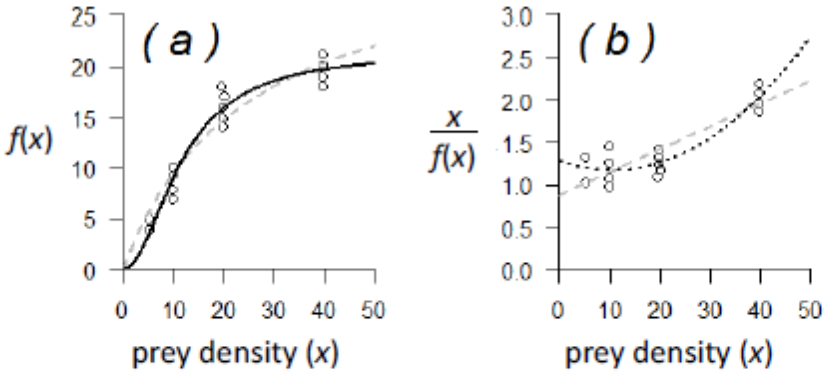

Figure 1: Untransformed and transformed functional response data from the Fathi and Nouri-Ganbalani (2010) study. *(a)* Untransformed data fit by the best fitting type 2 functional response (dashed gray curve) and the best fitting type 3 functional response ($f_{5A}(x)$; solid black curve). The type 3 functional response fits the data significantly better than the type 2 functional response. *(b)* Transformed data fit by the best fitting linear (dashed gray line) and quadratic (dotted black curve) functions. The quadratic function fit the data significantly better than the linear function. Note that after using the transformation $x/f(x)$, the type 2 functional response in *(a)* and the linear function in *(b)* are the same.

Appendix 4.C – Figure parameters

Table C1: Parameter values for Holling type 2 and PDC functional responses in figures 1, 2, 5 and 6.

Figures	Function	a	α	T_h	c
1, 2a-c, 5a-b	$f_2(x)$	1.00000	-	0.10000	-
1, 2d-f, 5c-d	$f_3(x)$	-	0.93755	0.09810	-
1, 2g-i, 5e-f, 6a	$f_4(x)$	1.82745	-	-	0.53377
1, 2j-l, 5g-h	$f_{5A}(x)$	-	0.20387	0.12488	-
1, 2m-o, 5i-j	$f_{5B}(x)$	1.06805	-	-	0.10182
1, 2p-r, 5k-l	$f_6(x)$	-	0.66926	-	0.60022
1, 2s-u, 5m-n	$f_7(x)$	0.89206	-	-	0.12251
6b	$f_{4B}(x)$	1.82745	-	-	0.53377

Table C2: Numerical solutions for the breadth of stability of the R-M type models with $f_{5B}(x)$

	breadth of stability	
c	$a = 1$	$a = 2$
0.13	0.373	0.555
0.1	0.555	1.72
0.05	1.115	4.09
0.01	1.720	9.73
0.005	2.436	11.96
0.001	4.090	16.47

Table C3: Numerical solutions for the breadth of the lower band of stable coexistence and the total breadth of stability at $K = 10^6$ for R-M type models with $f_6(x)$

	breadth of lower band		total breadth of stability	
c	$a = 1$	$a = 2$	$a = 1$	$a = 2$
0.1	17.61	15.79	24.55	22.72
0.5	2.70	2.37	4.09	3.76
1	1.19	1.03	1.88	1.72
2	0.51	0.45	0.86	0.79
10	0.08	0.07	0.15	0.14
100	0.0058	0.0055	0.0127	0.0125

Appendix 4.D – Data set references

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CHAPTER 5

CONCLUSIONS

5.1 Overview

Although the impact of intraindividual and intraspecific variation on ecological dynamics has been recognized for some time now, early considerations of these phenomena were limited to the investigation of variation created by age/stage structure (Leslie 1945). While age/stage structure may be widespread, on its own, age/stage structure cannot account for much of the variation seen in natural populations. More recent theoretical and empirical investigations have illuminated the existence and profound effects of trait variation that allows for rapid phenotypic adaptation (Fussmann, Ellner & Hairston 2003; Yoshida *et al.* 2003; Jones *et al.* 2009; Kishida *et al.* 2014). Many studies have found that rapid phenotypic adaptation can significantly enhance the persistence of populations and communities facing environmental stress (van der Stap, Vos & Mooij 2007; Mougi & Kishida 2009; Bell & Gonzalez 2011). However, the disparity of the sources, trophic locations, and traits that can exhibit or create variation has impeded our ability to draw general conclusions about the ecological consequences of rapid phenotypic adaptation. The need to understand the comparative effects of such disparate phenomena has led to the development and evaluation of mathematical models presented in this thesis. In Chapter 2, I compared the dynamics and rescue potential generated by genetic diversity for defenses and plasticity for defenses; using multitrophic models, I was able to discern how the trophic location of variation for defenses can affect stable coexistence and, therefore, rescue. In Chapter 3, I investigated the effects of different categories of inducible defenses. In Chapter 4, I used a combination of theory and a systematic review of empirical data to determine the dynamical consequences and prevalence of inducible offenses. The key conclusions of these chapters are:

Chapter 2: Both the source (genetic or plastic) and trophic location of variation for defenses critically influence the magnitude of persistence and stable coexistence, and therefore the potential for rescue. Specifically, plastic defenses are more likely than genetic defenses to promote these properties. Furthermore, our results suggest that variation for defense traits at the penultimate highest trophic level should have the greatest impact on stable coexistence, persistence, and rescue.

Chapter 3: Inducible defenses can decrease the risk of extinctions due to population oscillations and clear hierarchies exist at both low and high carrying capacities. Furthermore, our results indicate predictable, definitive, and consistent relationships between the form of inducible defense, the effectiveness of defense, and stable coexistence in the predator mortality – carrying capacity parameter space. Pre-encounter inducible defenses are most likely to promote stable coexistence at low carrying capacities, whereas post-encounter and post-consumption inducible defenses are most likely to promote stable coexistence at high carrying capacities. Ultimately, the ability of inducible defenses to promote stable coexistence will depend on the biology of the organisms in question, as well as the abiotic factors of carrying capacity and predator mortality rate.

Chapter 4: Nearly all predator-prey interactions that had been previously attributed to the type 2 functional response are best described by models that incorporate inducible offenses in the form of prey density-dependent attack rates and/or handling times. In terms of the community dynamics and stability properties of systems facing nutrient enrichment, we found that some, but

not all, models that incorporate prey density-dependent attack rates and/or handling times are capable of making categorically and fundamentally different predictions than models that incorporate the type 2 functional response. Our findings indicate that predictions of frequent or inevitable destabilization may be overstated. This study also highlights the importance of rechecking accepted principles in ecology.

5.2 Contributions & Outlook

This thesis identifies and explores specific forms of intraindividual and intraspecific variation that have the potential to promote the persistence and stable coexistence of trophically linked populations. Because these phenomena may play a role in mitigating the effects of global environmental change, understanding their different mechanisms is an important first step towards the sustainable management of ecological systems. In this sense, my work expands our general knowledge concerning these phenomena. My specific contributions are detailed below.

While the results presented in this thesis generally corroborate the conclusion that intraspecific and intraindividual variation will promote the persistence of ecological populations and communities, I found that the extent of stable coexistence depends on the source, trophic location, and specific traits that exhibit or create variation. My work highlights the importance of evaluating stable coexistence in the mortality – carrying capacity parameter space. Furthermore, through the framework of weak and strong stability, I have been able to distill complex relationships between stable coexistence and different forms of trait variation.

In spite of strong theoretical arguments against the use of the Holling type 2 functional response (Abrams 1982; Jeschke, Kopp & Tollrian 2002; Abrams 2010; Okuyama 2010), and despite the fact that even in simple systems, under highly controlled conditions, it is extremely

difficult to make accurate predictions using models that incorporate type 2 functional responses (Fussmann *et al.* 2000; Yoshida *et al.* 2003; Jensen & Ginzburg 2005), the type 2 functional response is often hailed as the "true" functional response for most systems (Jeschke, Kopp & Tollrian 2002; Turchin 2003; Jeschke 2004). In Chapter 4, we provide empirical evidence that corroborates previous theoretical concerns with the use of the type 2 functional response. Furthermore, through my own theoretical analyses, I show that the predictions of models that incorporate some, but not all, forms of inducible offense are more in line with observations of natural systems with respect to nutrient enrichment.

The concept of 'rescue' has typically been thought of in terms of adaptive traits that allow the survival of single, isolated populations facing harsh environmental conditions. While community dynamics have typically been investigated in terms of the mechanisms that allow whole communities to persist in harsh environmental conditions, rapid phenotypic adaptation generated through intraspecific and intraindividual variation has been incorporated into such analyses relatively recently (Yoshida *et al.* 2003; Vos *et al.* 2004). My work has shown that investigations of rescue can, and should, consider the rescue of whole communities because there are fundamental similarities between population rescue and community dynamics. Furthermore, through our study, Gregor Fussmann and I were able to show that plasticity, in addition to genetic diversity, can rescue populations and communities; at the suggestion of Rowan Barrett and Andrew Hendry, Kovach-Orr & Fussmann (2013) coined the term "plasticity rescue".

5.3 References

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