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Toward a Theory of Trophic Cascades in Complex, Adaptive and Open Ecological Systems

A Dissertation submitted in partial satisfaction
of the requirements for the degree of

Doctor of Philosophy

in

Evolution, Ecology and Organismal Biology

by

Ashkaan Kevin Fahimipour

August 2015

Dissertation Committee:
- Dr. Kurt Anderson, Chairperson
- Dr. Darrel Jenerette
- Dr. Helen Regan
- Dr. William Walton

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The Dissertation of Ashkaan Kevin Fahimipour is approved:

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To my parents.

ABSTRACT OF THE DISSERTATION

Toward a Theory of Trophic Cascades in Complex, Adaptive and Open Ecological Systems

by

Ashkaan Kevin Fahimipour

Doctor of Philosophy, Graduate Program in Ecology, Evolution and Organismal Biology
University of California, Riverside, August 2015
Dr. Kurt E. Anderson, Chairperson

Trophic cascades occur when changes in an ecosystem's top trophic level indirectly drive changes in biomass at lower levels. Cascades of varying magnitudes have now been documented in every ecosystem, but explaining this variation remains a fundamental challenge in ecology; predictions from mathematical models have received equivocal experimental support. This is in

part due to the mismatch between the closed-system food chain models that underlie most cascade theory and the complex food webs they aim to abstract. In the following chapters I report the results of field and laboratory experiments, and computational and analytical models that extend cascade theory toward scenarios involving complex, adaptive and open ecological systems. In chapter two I combine models and experiments to demonstrate that feedbacks between animal movement and the foraging behavior of adaptive omnivores in food webs controls the strength and sign of emergent cascades in freshwater habitats. This study reveals that a deeper understanding of how spatial processes modify behavior, and how this scales up to communities, will be required to predict cascades. With my third chapter, I report results of simulations of complex *in silico* food web networks. By using allometric scaling rules derived from a global species dataset to parameterize models of energy flow, I demonstrate that basic features of network architecture influence the probability that cascades will emerge and the magnitudes of those cascades. In chapter four, I use analytical models to show that bottom-up and top-down cascades can interact to yield non-intuitive patterns in emergent community structure. Namely I demonstrate that under certain conditions, consumption of basal species by top predators (i.e.,

omnivory) can lead to a greater facilitation of basal species' biomasses than if predators did not consume producers at all. This result contends with current theory, which expects cascades to be weaker in omnivory systems because of direct consumption of producers by predators. Overall, I demonstrate that cascades are strongly influenced by processes that operate on very different scales: from animal movement at the landscape scale, to community architecture at the network scale, gross diet at the population scale and behavior at the scale of the individual.

Contents

List of Figures **xii**

List of Tables **xiii**

1 Introduction

1.1 Trophic Cascades 1
1.2 A Brief Summary of the Dissertation 3

2 Colonization Rate and Adaptive Foraging Control the Emergence of Trophic Cascades **6**

2.1 Abstract 6
2.2 Introduction 7
2.3 Materials and Methods 10
2.3.1 Experimental Design and Sampling Protocol of Field Experiment 10
2.3.2 Laboratory Feeding Experiment 14
2.3.3 Statistical Analyses 16
2.3.4 Mathematical Model 17
2.4 Results 21
2.4.1 Effects of Colonization Rate on Trophic Cascades in Mesocosms 21
2.4.2 Effects of Colonization Rate on Cascades Dynamics in Mesocosms 25
2.4.3 Mathematical Models and Diet Information Reveal Mechanism 27
2.5 Discussion 30

3 Network Size and Topology Regulate Trophic Cascades in Size-Structured Food Webs **41**

3.1 Abstract 41

3.2 Introduction 42
3.3 Methods 46
3.3.1 The Niche Model 47
3.3.2 The Allometric Model 48
3.3.3 Analyses 52
3.4 Results 55
3.5 Discussion 57

4 Strong Trophic Cascades in Enriched Omnivory Systems 68

4.1 Abstract 68
4.2 Introduction 69
4.3 Model Motivations 72
4.3.1 Food Chains and Fixed Omnivory Models 73
4.3.2 Quantifying Net Interspecific Interactions 77
4.3.3 Adaptive Omnivory 79
4.4 Results and Discussion 81
4.4.1 Analyses of Food Chain and Fixed Omnivory Cascades . . 81
4.4.2 Simulations of Food Chains and Adaptive Omnivory Cascades 83
4.5 Conclusions 85

5 Conclusions 95

5.1 Conclusions 95

Bibliography 98

A List of Taxa Encountered in Irvine, CA 106

B Supplementary Methods and Analyses 108

B.1 Model Details and Extensions 108
B.2 Supplementary Analyses of Field Experiment 112

C Additional Food Web Simulations 114

List of Figures

2.1 Effects of colonization rate on trophic cascades. 38
2.2 The dynamics of experimental ponds 39
2.3 Results of laboratory feeding experiments 40

3.1 Frequency distributions of cascade magnitudes 63
3.2 Predator invasions prompt different types of dynamical behavior 64
3.3 Estimated probabilities of cascade emergences 65
3.4 Cascade magnitudes depend on network size and complexity . . . 66
3.5 Compensation explains hidden cascades 67

4.1 Comparative analyses of cascades in omnivory and food chain systems 90
4.2 Effects of press perturbations on producer facilitation 91
4.3 Prey conversion efficiencies alter enrichment effects 92
4.4 High prey conversion efficiencies reverse enrichment patterns . . . 93
4.5 Results of adaptive foraging simulations 94

B.1 Parameter impacts 111
B.2 Results of simulations with Type II responses 113
C.1 Cascade magnitudes across all simulations 115
C.2 Cascade magnitudes as a function of covariates 116

List of Tables

2.1 Results of statistical analyses 37

4.1 Summary of analytical models 88
4.2 Parameter ranges used for adaptive foraging simulations 89

A.1 Taxa encountered during field experiment 107

B.1 Parameter value distributions 109

Chapter 1

Introduction

1.1 Trophic Cascades

Human activity is disproportionately affecting Earth's top predators [25, 45, 85], causing changes in top-down regulation of ecosystems that cascade down food webs and impact plants and microbes [7, 19, 26, 25, 32, 41, 45, 65, 68, 79, 80]. This phenomenon was famously articulated by Hairston, Smith and Slobodkin's [39] "green world" hypothesis and, two decades later, dubbed the *trophic cascade* by Paine [66].

Trophic cascades are the indirect facilitation of predators on producers via control of intermediate species [19]. Examples of cascades in freshwater, marine and terrestrial systems abound; predators indirectly regulating communities by

controlling prey have now been observed in every type of ecosystem [7, 79, 80]. Despite their ubiquity however, the strength of trophic cascades – the emergent biomass response of one component of a food web to forced changes elsewhere in the web – is highly variable between ecosystems [68, 80]. Synthesis of our understanding of the mechanisms that underlie this observed variation is in its nascency [80], and remains one of the great fundamental questions in ecology [84]. The simple food chain models that explicate most cascade theory [32, 41, 62, 64, 65, 85] have so far failed to explain this extensive variation [7, 32, 41]. The oft-ignored complexities of real ecosystems that are necessarily eschewed by simple mathematical models are a strong candidate for explaining this mismatch between theory and data. Many factors that are thought to influence the strength of trophic cascades have received little empirical or theoretical attention [45].

Most cascade models make biologically unrealistic assumptions such as very few species lumped into discrete trophic levels [62, 64], non-saturating consumer functional responses [8] and implausible combinations of parameter values [90, 94]. Moreover, most cascade theories assume closed systems [27, 62] and ignore variation in animal behavior, which is known to influence interspecific interactions [1] and therefore trophic cascades. I do not wish to detract from

these studies; the simple models that make these assumptions are tractable, can describe the gross dynamics of some systems (e.g., [27]) and provide analytical insight into ecological phenomena [15]. But, new food web data amassed over the last decade [3, 8, 9, 15, 19, 23, 28, 47, 86] are now strongly challenging these assumptions [80]. New models and experiments that extend current cascade theories toward more complex, open and adaptive ecological systems are sorely needed [27, 65, 80]. The importance of fundamental ecology notwithstanding [15], a predictive cascade theory has consequences for ecosystem restoration [25], biodiversity maintenance [85], the spread of zoonotic diseases [25], biogeochemical cycling [41] and agroecosystem pest management strategies [75], which combine to make this a vital gap in ecological knowledge.

1.2 A Brief Summary of the Dissertation

In the present dissertation, I combine analytical and computational models with laboratory and landscape-scale field experiments in order to extend cascade theory to include several ubiquitous features of ecological systems that are largely unexplored by current cascade models and experiments [27, 45, 80]. In chapter two I combine experiments in the field and laboratory with computational

simulations to demonstrate that a feedback between prey movement at the landscape scale and the local foraging behavior of adaptive predators in food webs controls the strength and direction of emergent cascades in experimental freshwater ponds. While prior work has demonstrated that animal movement can modify cascades through effects on the composition and body masses of the predator guild [13] and local extinction frequencies [16], the results of Chapter 2 demonstrate that changes in local predator foraging behaviour driven by exogenous prey supplies can have equally dramatic effects on cascade strengths.

In chapter three, I use an ecological niche model to generate realistic food web skeletons [92], and use empirical allometric scaling rules [8, 9, 90, 94] to parameterize a model of energy flows between species. I demonstrate that basic features of network architecture − the network's size and average degree − influence the probability that a cascade will emerge, and the magnitude of that cascade. Namely, I demonstrate that cascades in these empirically-parameterized network models are stronger and more likely to emerge in small and highly connected food webs.

With my fourth chapter, I use analytical models to show that bottom-up and top-down regulating forces in webs can interact to yield non-intuitive trophic

cascade patterns. The key result is that under certain conditions, a predator that also consumes basal resources (i.e., an omnivore) prompts stronger cascades than strictly carnivorous predators in analogous food chains. This result is in discrepancy with current theory, which expects cascades to be weaker in the presence of omnivory [65, 80] because of direct consumption of basal species by omnivorous predators.

Overall, the present dissertation demonstrates that cascades may be influenced by many factors that have so far received little empirical or theoretical attention. The models and experiments analyzed here share a common theme in that they link cascades to processes that operate on different scales: from animal movement at the landscape scale, to the whole-network, population and individual scales. Although such multi-scale ecological problems can be difficult to synthesize [52, 85], my hope is that the present extension of cascade theory will encourage researchers to study trophic cascades in other complex ecosystems.

Chapter 2

Colonization Rate and Adaptive Foraging Control the Emergence of Trophic Cascades

2.1 Abstract

Ecological communities are assembled and sustained by colonization. At the same time, predators make foraging decisions based on the local availabilities of potential resources, which reflects colonization. We combined field and laboratory experiments with mathematical models to demonstrate that a feedback between these two processes determines emergent patterns in community structure. Namely, our results show that prey colonization rate determines the strength of trophic cascades – a feature of virtually all ecosystems – by prompting

behavioural shifts in adaptively-foraging omnivorous fish predators. Communities experiencing higher colonization rates were characterized by higher invertebrate prey and lower producer biomasses. Consequently, fish functioned as predators when colonization rate was high, but as herbivores when colonization rate was low. Human land use is changing habitat connectivity worldwide. A deeper quantitative understanding of how spatial processes modify individual behavior, and how this scales to the community level, will be required to predict ecosystem responses to these changes.

2.2 Introduction

Anthropogenic habitat alteration is changing the spatial context in which species interactions occur. Human land use in particular is eroding historical patterns of habitat connectivity [31, 36], resulting in altered exogenous supplies of resources (e.g., prey resources for predators) to food webs [28, 43, 67, 82]. An important goal for efforts to predict and manage human effects on ecosystems is to understand how changes in spatial habitat features that govern resource supplies affect direct and indirect interactions in food webs, and how this in turn impacts emergent patterns of community structure [22, 45]. Trophic cascades are

the archetypical example of emergent outcomes of food web interactions, and occur when predators directly reduce prey populations, indirectly facilitating populations at lower trophic levels. But, despite the fact that cascades have been documented in virtually every type of ecosystem [80], both conceptual and mathematical theories have been unable to fully explain widespread variation in observed cascade strength [7, 32, 41, 45, 68, 80]; predictions from mathematical models have received equivocal experimental support [32, 41, 80], and the mechanisms underlying spatial variation in cascade strength in nature remain largely unexplored [45].

Traditionally, cascade models that have provided predictions for experiments lump species into discrete trophic levels, and assume that species interactions are fixed and occur in closed systems [41, 62, 64, 85]. These assumptions are now being challenged [80], however, by evidence that suggests many if not most predators feed across multiple trophic levels (i.e., omnivory) and forage adaptively or flexibly to some degree [3, 34, 48, 86]. Moreover, ecological communities are open systems governed by exogenous spatial flows of colonists [46, 52, 53]. In many communities, predator foraging behavior and prey colonization rates may be linked, because variation in the influx of different

resources can influence predator foraging strategies, thereby altering direct and indirect food web interactions. It is therefore conceivable that these ubiquitous features of real food webs – omnivory, adaptive foraging and colonization – influence one another through feedbacks that could help explain empirical deviations from cascade theory [45, 68, 80]. How- ever, to our knowledge, no studies have investigated their joint effects on trophic cascades.

Here, we combine field and laboratory experiments with mathematical models to demonstrate that colonization rate determines foraging strategies of top omnivorous predators and, therefore, how cascades manifest in food webs. Specifically, we manipulated colonization rate in an array of experimental pond communities, and studied variation in the strength of cascades induced by an adaptively forag- ing omnivorous fish predator, *Gambusia affinis*, in these communities. We report that variation in colonization rate among habitats strongly altered the strength and direction of cascades. We observed a traditional cascade, with fish strongly depressing prey (i.e., primary consumers) and facilitating producer biomass densities when colonization rate was high. However, the facilitation effect was overridden by direct consumption of producers when colonization rate was low. Mathematical models, gut content analyses and

laboratory feeding experiments reveal the underlying mechanism: experimentally reducing colonization rate caused differences in the relative biomass of prey and producer resources at the community scale, which prompted an adaptive shift in the foraging effort of individual fish increasingly toward producers.

2.3 Materials and Methods

2.3.1 Experimental Design and Sampling Protocol of Field Experiment

On 12 June 2012, we deployed an array of 32 experimental pond mesocosms downwind from a permanent lake, which acted as a colonization source, at the San Joaquin Marsh Reserve (SJMR) in Irvine, CA, USA. In this system, small natural ponds are seasonally filled with water and rapidly colonized by primary producers (e.g., wind-dispersed and hitch-hiking phytoplankton) and invertebrate prey (e.g., flying aquatic insects, oviposited insect larvae, wind-dispersed zooplankton; species list provided in Appendix A) that immigrate from the nearby permanent lake. Increasing the distance between ponds and the nearest permanent lake reduces species' colonization rates [28, 43]. Thus, through a

distance treatment, we created *high* and *low* colonization rate communities by placing ponds either 30 or 300 m from the colonization source, respectively.

All mesocosms began the experiment at a state in which no species were present. We scrubbed with bleach, washed and placed 16 1-m diameter plastic wading pools at each of the two distances, filled them with a 25 mm layer of heat sterilized sand, 25 g of rabbit food (Small World, Manna Pro, St. Louis, Missouri, USA) to provide an initial source of nutrients and 70 L of filtered and treated tap drinking water. Tap water is the only available clean water source at the SJMR, and was treated for chloramines and chlorine; the absence of these and other contaminants was confirmed using water testing kits prior to the experiment.

We allowed communities to naturally assemble for four weeks, at which point we added two individual *Gambusia affinis* mosquitofish approximately 30 mm in length, collected from the permanent lake, to eight randomly-selected mesocosms in each distance treatment (i.e., half of the ponds at each distance). *G. affinis* is a generalist adaptive omnivore that readily consumes resources in multiple trophic levels – both prey (e.g., zooplankton, aquatic macroinvertebrates) and producers (e.g., phytoplankton, diatoms). *Gambusia* have dorsally-oriented mouths and dorsoventrally flattened heads, and are known

to feed primarily on pelagic resources or near the surface, while largely avoiding the unpalatable $CaCO_3$-encrusting plankton that comprise benthic mats [35, 70]. For these reasons our sampling focused on the pelagic phytoplankton-zooplankton-insect community. Fish abundance and predation pressure were kept constant throughout the experiment by removing offspring in the event of a male-female pairing, and replacing dead individuals ($< 18\%$ of all individuals) or individuals who underwent significant growth with a new individual approximately 30 mm in length, following surveys every three to five days. We also replaced evaporated water during surveys, to maintain pond volume at approximately 70 L and control for variation in habitat size. Communities were sampled two, four, six, eight, ten and twelve weeks after the establishment of ponds; the average natural pond at the SJMR continuously contains water for two to eight weeks (P.A. Bowler, *pers. comm.*). Thus, the length of our experiment is sufficient to capture relatively long-term changes in this system over 4-12 generations of an average member species, and 2-4 generations of the longest-lived taxa [43].

During each sampling event we counted all macroinvertebrates by sweeping a 1- mm mesh net through the water and benthos until no individuals

were detected on three consecutive sweeps. We measured the body lengths of the first 20 individuals of each species with digital calipers before returning them to mesocosms. We then took four 250-mL zooplankton samples using an integrated depth sampler. Plank- ton samples were combined and filtered through 1-mm nitex mesh, anesthetized with carbonated water, preserved with 10% acid Lugol's solution and enumerated and measured in the laboratory. All zooplankton and macroinvertebrates (i.e., prey) were identified to the highest possible taxonomic resolution (Appendix A), and population biomass densities (mg $\times$ mL^{-1}) were calculated as B_i/V, where $B_i = \mathrm{M}_i \times \mathrm{N}_i$; M_i and N_i are the average body mass (mg) and abundance of species i, respectively and V is the measured habitat volume (mL). Body masses were estimated from measured lengths using a set of taxon-specific conversions for aquatic invertebrates compiled by Fahimipour & Hein [28]. Producer (e.g., phytoplankton, diatoms) biomass densities were estimated by measuring chlorophyll a fluorescence values from five vertical water samples collected with an integrated depth sampler, using a handheld AquaFluor fluorometer (Turner Designs, Sunnyvale, California, USA).

Following the field experiment's conclusion, we sacrificed fish in order to perform gut content analyses and determine whether fish diets depended on

habitat colonization rate. Gut contents were preserved in 70% ethanol and stained with 5% Lugol's solution before identifying and measuring food items using a stereomi- croscope. The relative volumetric quantity of food in the gut was estimated by assuming the gut was cylindrical, measuring its dimensions, calculating total gut volume and then visually estimating the fraction of the gut occupied by food as a proxy for total food biomass. We then separated gut contents into two components, prey and producers, and estimated the fraction of prey in the gut by measuring body lengths and using taxon-specific length-volume conversions ([59]; see also references in Appendix S1 in Fahimipour & Hein [28]). The fraction of prey in fish diets sacrificed from mesocosms experiencing different colonization rates were compared using *ANOVA*.

2.3.2 Laboratory Feeding Experiment

To characterize the foraging strategies of *G. affinis*, we performed laboratory feeding trials with individual mosquitofish in replicate 3.5 L aquaria. On 10 December 2013, we starved twelve fish for five hours to establish a consistent intermediate hunger level [70], at which point we exposed them to approximately 20 mg (dry biomass) of total food, varying only the ratio of

producer (the algae, *Spirogyra spp.*; Carolina Biological Supply) to prey (mosquito larvae, *Culex quinquefasciatus* from laboratory stocks) biomass densities. We experimentally altered food availabilities to *c.a.* 1:3, 1:1 and 3:1 producer:prey biomass ratios. However, variation in the masses of algae filaments caused variation about the precise ratios. Following a 24-hour period, we measured the amount of prey and producer biomass consumed by each fish by calculating the difference between remaining (after 24 hours) and initial prey and producer biomass densities. We used a second-order polynomial regression of the ratio of prey:producers eaten on the ratio available to determine whether fish foraged in a manner consistent with adaptive foraging models during laboratory feeding experiments, with a significant quadratic term being consistent with the hypothesis of adaptive foraging. Conversely, if *Gambusia* forages in a manner in which producers and prey comprise a fixed proportion of the total diet, then we would expect a linear relationship between the ratio eaten and the environmental ratio (q_R and q_N in eq. 1 are constants for fixed foragers, so the ratio of producers to prey eaten $q_R/q_N \times R/N = wR/N$, where w is a constant).

2.3.3 Statistical Analyses

To quantify trophic cascade strength, we calculated effect sizes according to convention [32, 79] as $\log_e(X_{+P}/X_{-P})$, where X_{+P} and X_{-P} are the mean biomass densities, averaged over the final six weeks of the experiment, of resource X in the presence and absence of fish, respectively. Habitat pairs (i.e., X_{+P} and X_{-P}) were assigned based on spatial proximity in the field, although randomly assign- ing habitat pairs did not affect the qualitative results presented below. We used *ANOVA* to determine whether producer and prey effect sizes were different in high- versus low-colonization rate ponds. Changing the number of post-fish addition sampling dates included in the cascade strength analyses did not change the qualitative results presented below (see Appendix B for cascade results when only week twelve is included). To determine how biomass densities depended on time, fish presence and colonization rate, we performed linear mixed effects (LME) model analyses on the producer and prey time series post-fish addition (Table 1). For each community property, Y (i.e., producer and prey biomass densities), we fit a model of the form $Y = B_1(\text{time}) + B_2(\text{fish}) + B_3(\text{distance}) + B_4(\text{time} \times \text{fish}) + B_5(\text{time} \times \text{distance}) + B_6(\text{fish} \times \text{distance}) + B_7(\text{time} \times \text{fish}$

$\times$ distance) + G + E, where B_1-B_7 are regression coefficients, G is a random effect of pond identity (to account for the fact that repeated measurements were made on each pond), and E is a vector of errors. To improve normality of residuals, producer biomass densities were fourth-root transformed and prey biomass densities were fifth-root transformed prior to analyses. We also examined whether local resource conditions at the time of fish additions were different in habitats experiencing different colonization rates. To do this, we compared prey and producer biomass densities in low- and high-colonization rate ponds immediately before fish additions (the week four censuses) using *ANOVA*. LME models were fitted using the *nlme* package in the statistical programming environment, R [71].

2.3.4 Mathematical Model

Our model is a coarse-grained representation of our experimental study system and represents a straightforward alteration of traditional cascade models [62, 64]. A simple food chain model with the addition of colonization and adaptive for- aging strategies, in which the omnivore dynamically apportions

foraging effort toward either producers or prey based on their availability and profitability, can be represented by:

$$\frac{dR}{dt} = I_R + rR(1 - \frac{R}{K}) - a_N RN - q_R a_P RP \tag{2.1}$$

$$\frac{dN}{dt} = e_R a_N RN - mN - q_R a_P PN \tag{2.2}$$

$$\frac{dq_X}{dt} = v q_X \left(\frac{\partial \beta}{\partial q_X} - \sum_{k \epsilon S} q_k \frac{\partial \beta}{\partial q_k} \right) \tag{2.3}$$

where R is the producer biomass density, N is the prey biomass density, and P is the omnivore biomass density, which we make constant to mirror experimental conditions, and q_R and q_N are the preferences of the omnivore for producers and prey, respectively. The parameter v is a rate constant that sets the timescale on which changes in foraging effort occur, β is the omnivore's per capita biomass production rate, $\beta = f_R q_R a_P R + f_N q_N a_P N$, and the sum is taken over all S species in the omnivore's diet. Producers and prey colonize the system at rates, I_R and I_N, respectively. Other parameters are defined in Appendix B, and

correspond to a biomass interpretation of standard food chain models with Type I functional responses.

We incorporated adaptive foraging strategies using a pair of dynamical variables, denoted generally as q_X, which are based on replicator equations (e.g., [48]) and represent the proportion of total foraging effort an omnivore will apportion toward one of its resource species, X [2, 48]. In this context, the replicator equation implies a food choice rule that attempts to maximize biomass production: the omnivore increases its foraging effort toward resource X (i.e., q_X) if a unit change in q_X causes the omnivore's biomass production to exceed that derived from cur- rent foraging effort. We re-visit some of the assumptions behind the model, and present the results of simulations with Type II functional responses in Appendix B.

To study the effects of colonization rate on producer and prey effect sizes over a broad range of potential conditions, we simulated the model using 10^5 randomly generated parameter sets. We generated 100 values of colonization rates, I_X, evenly spaced on the interval $1 \times 10^{-4} \leq I_X \leq 5 \times 10^{-1}$ and simulated the model 1000 times for each value of I_X, drawing all other parameter

values (except P and a_P, which we make constant to mirror experimental conditions) independently from uniform distributions. To compare modeling results with our experimental results, equilibrium biomass' were summarized similarly to our data as log response ratios, where X_{+P} is the equilibrium biomass of resource X simulated with P > 0 and X_{-P} is the equilibrium biomass of X simulated with the same set of parameter values but with $P = 0$. Parameter distributions were based around values from the omnivory food chain model of McCann [58] and ranges were chosen because they produced interior equilibria with positive values for R, N, q_N, and q_R when matching parameter sets were used in models with omnivores ($P > 0$) and without omnivores ($P = 0$), and because they yielded realistic cascade strength values − for instance, effect sizes less than -3 or greater than 3 are rarely observed in nature [79]. This approach yields model results from thousands of unique "communities," which allowed us to explore the generality of our findings without making specific assumptions about variation in community composition or particular species' characteristics in our mesocosm experiment.

During each simulation, the model was run to equilibrium; simulations that did not generate fixed interior equilibria within 106 time steps were excluded from our analyses. To explore the importance of adaptive omnivory in generating empirically observed patterns, we also simulated fixed omnivory by performing the same procedure described above, except that we held q_R constant and $q_N = 1 - q_R$. Numerical simulations were accomplished using the *deSolve* and *rootSolve* libraries in R [71]. For additional details on parameter ranges used and other modeling specifics, see Appendix B.

2.4 Results

2.4.1 Effects of Colonization Rate on Cascades in Mesocosms

Cascade strengths in field mesocosms depended on colonization rate (Fig. 2.1a). Fish facilitated producer biomass densities, inducing a classic cascade, in communities experiencing high colonization rates (Fig. 1a; *ANOVA*; $F_{1,14} = 23.51$, $P = 0.0003$). When colonization rate was low however, fish depressed producer biomass densities. Likewise, fish effects on prey depended on

colonization rate (Fig. 1a; $ANOVA$; $F_{1,14} = 10.18$, $P = 0.007$): higher colonization rates led to an increasingly strong depression of prey biomass by omnivores. In short, differences in colonization rates in our study system generated consistent spatial variation in fish effects on both producers and prey; as colonization rate increased, fish effects shifted from a relatively weak depression of prey and depression of producers, to a relatively strong depression of prey and facilitation of producers. Analyses of biomass dynamics confirmed that fish had qualitatively different effects on producers in high- (positive effect of fish, Table 2.1) compared to low- (negative fish × distance interaction, Table 2.1) colonization rate ponds (compare Figs. 2.2a and 2.2b). We did not detect an interaction between time and fish presence for producers in ponds experiencing either colonization rate.

Fish had a negative effect on prey in both high- and low-colonization rate ponds (compare Figs. 2.2c and 2.2d; negative effect of fish, Table 2.1). The effect of fish on prey biomass densities was weaker in low colonization rate ponds however (positive distance × fish inter- action, Table 2.1), consistent with the results of the cascade analysis. Producer and prey biomass densities increased through time in all mesocosms (Table 2.1). We also detected a negative

interaction between time and fish presence for prey, indicating that prey biomass increased more slowly in the presence of fish in these mesocosms (Table 2.1).

Analyses of biomass dynamics confirmed that fish had qualitatively different effects on producers in high- (positive effect of fish, Table 2.1) compared to low- (negative fish × distance interaction, Table 2.1) colonization rate ponds (compare Figs. 2.2a and 2.2b). We did not detect an interaction between time and fish presence for producers in ponds experiencing either colonization rate. Fish had a negative effect on prey in both high- and low-colonization rate ponds (compare Figs. 2.2c and 2.2d; negative effect of fish, Table 2.1). The effect of fish on prey biomass densities was weaker in low colonization rate ponds however (positive distance × fish interaction, Table 2.1), consistent with the results of the cascade analysis. Producer and prey biomass densities increased through time in all mesocosms (Table 2.1). We also detected a negative interaction between time and fish presence for prey, indicating that prey biomass increased more slowly in the presence of fish in these mesocosms (Table 2.1).

We compared prey and producer biomass densities from the week four census (immediately preceding fish additions) in all ponds to show that local resource conditions at the time of fish arrivals depended on colonization rate. At

this time (Fig. 2.2, vertical dashed lines), our colonization rate treatment had generated differences in producer and prey biomass densities, indicating that fish experienced different resource conditions upon entering their respective webs, and that this depended on colonization rate. Specifically, fish added to high colonization rate communities entered webs with higher prey (Figs. 2.2c and 2.2d, vertical dashed lines; $ANOVA$; $F_{1,30} = 51.56$, $P < 0.001$) and lower producer (Figs. 2.2a and 2.2b, vertical dashed lines; $ANOVA$; $F_{1,30} = 31.91$, $P < 0.001$) availabilities, whereas fish added to low colonization rate communities experienced lower prey and higher producer availabilities. These differences were present not only at the time of fish additions, but could be detected throughout the experiment by analyzing the dynamics of fishless ponds. Reducing colonization rate (i.e., increasing distance from the source lake) had a positive effect on producers and a negative effect on prey (Fig. 2.2; Table 2.1) in these ponds, again indicating that resource conditions shifted from relatively low producer and relatively high prey, to high producer and low prey biomass densities as colonization rate decreased.

2.4.2 Effects of Colonization Rate on Cascades in Mesocosms

Cascade strengths in field mesocosms depended on colonization rate (Fig. 2.1a). Fish facilitated producer biomass densities, inducing a classic cascade, in communities experiencing high colonization rates (Fig. 2.1a; *ANOVA*; $F_{1,14} = 23.51$, $P = 0.0003$). When colonization rate was low however, fish depressed producer biomass densities. Likewise, fish effects on prey depended on colonization rate (Fig. 2.1a; *ANOVA*; $F_{1,14} = 10.18$, $P = 0.007$): higher colonization rates led to an increasingly strong depression of prey biomass by omnivores. In short, differences in colonization rates in our study system generated consistent spatial variation in fish effects on both producers and prey; as colonization rate increased, fish effects shifted from a relatively weak depression of prey and depression of producers, to a relatively strong depression of prey and facilitation of producers.

Analyses of biomass dynamics confirmed that fish had qualitatively different effects on producers in high- (positive effect of fish, Table 2.1) compared to low- (negative fish $\times$ distance interaction, Table 2.1) colonization rate ponds

(compare Figs. 2.2a and 2.2b). We did not detect an interaction between time and fish presence for producers in ponds experiencing either colonization rate. Fish had a negative effect on prey in both high- and low-colonization rate ponds (compare Figs. 2.2c and 2.2d; negative effect of fish, Table 2.1). The effect of fish on prey biomass densities was weaker in low colonization rate ponds however (positive distance $\times$ fish interaction, Table 2.1), consistent with the results of the cascade analysis. Producer and prey biomass densities increased through time in all mesocosms (Table 2.1). We also detected a negative interaction between time and fish presence for prey, indicating that prey biomass increased more slowly in the presence of fish in these mesocosms (Table 2.1).

We compared prey and producer biomass densities from the week four census (immediately preceding fish additions) in all ponds to show that local resource conditions at the time of fish arrivals depended on colonization rate. At this time (Fig. 2.2, vertical dashed lines), our colonization rate treatment had generated differences in producer and prey biomass densities, indicating that fish experienced different resource conditions upon entering their respective webs, and that this depended on colonization rate. Specifically, fish added to high colonization rate communities entered webs with higher prey (Figs. 2.2c and 2.2d,

vertical dashed lines; $ANOVA$; $F_{1,30} = 51.56$, $P < 0.001$) and lower producer (Figs. 2.2a and 2.2b, vertical dashed lines; $ANOVA$; $F_{1,30} = 31.91$, $P < 0.001$) availabilities, whereas fish added to low colonization rate communities experienced lower prey and higher producer availabilities. These differences were present not only at the time of fish additions, but could be detected throughout the experiment by analysing the dynamics of fishless ponds. Reducing colonization rate (i.e., increasing distance from the source lake) had a positive effect on producers and a negative effect on prey (Fig. 2.2; Table 2.1) in these ponds, again indicating that resource conditions shifted from relatively low producer and relatively high prey, to high producer and low prey biomass densities as colonization rate decreased.

2.4.3 Mathematical Models and Diet Information Reveal Mechanism

Our modeling analyses suggest that when omnivores forage adaptively, variation in prey colonization rate, I_N, generates a dramatic spatial gradient of omnivore effects on both producers and prey highly consistent with results of the

mesocosm experiment (Fig. 2.1a). This, however, does not occur when foraging preferences q_R and q_N are fixed (Fig. 2.1b), suggesting that a switch in preference towards producers in low colonization ponds is required for the spatial patterns we observed. Observed changes occurred through the strong influence of prey colonization rate, I_N, on omnivore foraging strategies (i.e., q_X; eq. 1). As I_N increases, prey become increasingly biomassive and the omnivore shifts its foraging effort increasingly away from the nutritionally inferior producers [34]. The topology of the system becomes more like a linear food chain, with increasingly strong omnivore-prey interactions and increasingly weak omnivore-producer interactions, as prey colonization rate increases.

In contrast, producer colonization rate, I_R, has little impact on either producer or prey effect sizes (Fig. B.1; Appendix B). This is due to the high sensitivity of q_N and q_R to the influx of the more profitable resource, which we assume are the prey. Thus, although our experiment's distance treatment potentially altered both prey and producer colonization rates, our model analyses suggest that changes in prey colonization rate, I_N, generated the observed shift in cascade strength, and that this holds across a range of assumed relationships

between distance from the source pool, I_N and I_R. The relative effects of I_N and I_R also hold across a range of values in the other parameters; the average impact of each model parameter on cascade strength is discussed in Appendix B and shown in Fig. B.1.

Differences in the gut contents of fish from ponds at different distances sup- ported the hypothesis that spatially mediated shifts in fish foraging strategies altered the effects of fish on lower trophic levels in our study system. The guts of fish taken from high colonization rate ponds contained a higher fraction of prey than those taken from low colonization rate ponds (Fig. 2.3a; *ANOVA*; $F_{1,7} = 17.53$, $P = 0.0041$), suggesting that fish foraging strategies depended on habitat colonization rate. Data from laboratory feeding trials confirmed that *G. affinis* is an adaptive forager. If fish foraged in a manner consistent with a fixed omnivory model, then we would have expected the ratio of prey to producer biomass consumed by individual fish to increase linearly as a function of the prey to producer biomass ratio in the environment. But, this was not the case. Instead, this relationship displayed non-linearities (Fig. 2.3b; significant quadratic term; $P < 0.001$), again suggesting that producers and prey

do not comprise a fixed proportion of the total diet and that the fish forage adaptively depending on producer and prey availabilities in the environment.

2.5 Discussion

Trophic cascades may be influenced by many factors that have so far received little empirical attention [45]. Here, we demonstrate that cascade strength is strongly influenced by a feedback between organismal movement at the landscape scale, resource conditions at the community scale and foraging behavior at the scale of the individual predator. Moreover, our study provides new empirical evidence that spatial variation in the strength of omnivory that likely exists within many landscapes ([49]; Fig. 2.3) has important consequences for emergent patterns in community structure and system dynamics.

Although we use experimental pond mesocosms to demonstrate the effects of colonization-foraging feedbacks on the emergence of cascades, we expect many of the qualitative features observed in this study apply to other ecological systems. One requisite feature for these feedbacks to emerge is an omnivorous top predator that is capable of exhibiting rapid behavioral responses to changes in resource conditions. Food web data show that omnivory is pervasive, with most

predators feeding from more than one trophic level to some degree [3, 86]. One caveat is that, although omnivory is ubiquitous in food webs, this does not necessarily imply that omnivores consume both animal prey and primary producers (e.g., intraguild predators and hyperparasitoids are omnivores that do not necessarily consume producers), as in our system. However, the general result – that variation in the exogenous supplies of resources in a consumer's diet can influence its foraging strategies and, therefore, how top-down control manifests in food webs – is one that likely extends to many organisms facing diet choices regardless of resource types they exploit [5, 48]. For instance, adaptive carnivores can prompt changes in the composition of lower trophic levels, with implications for key system properties like nutrient flux rates [74] and food web persistence [48].

Behavioral changes in individuals' foraging strategies have now been observed in taxa as diverse as rodents [34], birds [50], large mammals [72], arthropods and fish [48], many of which are high level consumers in their respective webs. The empirical patterns we observed emerge in models over a wide parameter range when we included adaptive behavior (Fig. 2.1a), but did not emerge when trophic preferences were fixed (Fig. 2.1b). This result highlights

the importance of how diet choice is represented in theoretical studies; for instance, adaptive versus fixed models of omnivory [2, 56]. That adaptive and fixed diet choice models yield qualitatively different dynamics is well known for communities of several interacting species in closed systems [1]. These disparate outcomes may be especially important for the study of spatially assembled habitats where resource dynamics can be strongly influenced by habitat connectivity and exogenous processes.

The behavioral mechanism we have identified altered community structures in response to spatial heterogeneity in colonization rates. Yet, it is possible that spatial variation in other factors could influence the outcomes we observed. Mesocosms in our study were placed in an open field with very little variation in other factors that could have influenced community structure, such as shading and allochthonous nutrient inputs. Additionally, our mesocosms were the same size and experienced identical initial conditions. However, variation in habitat size and initial conditions do influence natural systems; pond size in particular varies naturally across the SJMR landscape. At least one experiment has demonstrated that predators have stronger effects on prey in small habitats compared to large ones [76, 77], although the opposite can be true depending on

the particular relationships between habitat size and colonization rate for species in a given food web [45]. Predicting the effects of habitat size on cascade strengths in our system would require a detailed understanding of the effects of pond diameter and depth on colonization success and biomass distributions for species in basal, mid and top trophic levels. Uncovering the interactions between habitat distance, size and the adaptive foraging strategies of consumers therein is an important enterprise for future research.

Some species in our system (e.g., plankton) are capable of forming egg banks in dry sediment that individuals emerge from when ponds are inundated. This generates a lagged impulse of individuals following rewetting, separate from colonization processes, at both producer and prey trophic levels. Some zooplankton eggs for instance can remain diapaused for decades, and emergence rates display complex relationships with numerous abiotic factors like dissolved oxygen, pH and the recurrence interval of flooding events [38]. Because our observed outcomes are driven in large part by the presence of spatial variation in local resource conditions, the potential for egg banks to mask the patterns we observed would be limited to scenarios in which emergence forces homogenous resource conditions across a landscape. Insofar as egg banks do not erase spatial

variation in local resource conditions, we expect that the behavioral mechanism we have identified would still influence cascades.

An alternative potential explanation for the observed spatial variation in cascade strength is that it was driven by isolation's effect on species richness rather than biomass densities among trophic levels. In our field experiment, we observed that ponds experiencing low colonization rates had nearly half the number of prey species that ponds experiencing high colonization rates contained, on average (Appendix B). However, current cascade theory would predict cascades to be stronger (i.e., larger in magnitude) in low colonization rate ponds, all else constant, because of this reduced prey species richness [7, 32, 80]. Instead, we saw the strongest cascades in nearby, high richness ponds, and trophic depression of producers by fish in isolated, low richness ponds. Changes in species composition could have also caused the observed spatial variation in cascade strength. It is possible that certain prey species in this system are less edible to *Gambusia*, and that these species preferentially colonized isolated ponds, which prompted the observed behavioral shift. However, *G. affinis* is a well-documented generalist [70]; we detected confamilials of most encountered prey taxa in the guts of sacrificed fish, suggesting a low sensitivity to taxonomic composition

(Appendix A). Included in the gut contents were other predator species (e.g., Libellulid dragonflies), which tended to be rare and are generalist carnivores [88], making them unlikely candidates for depressing prey and producer biomasses in low colonization rate ponds.

Finally, our modeling analysis suggests that the patterns we observed hold despite a wide range of "community compositions," which is reflected in the broad range of parameter space over which the empirical patterns are maintained in the model. Thus, it is unlikely that these patterns were driven by differences in prey community composition *per se*. Moreover, we did not detect an effect of habitat colonization rate on the probability that a fish had to be replaced due to mortality or somatic growth (Appendix B); we did not find evidence that changes in aspects of omnivores other than foraging strategies occurred in response to manipulating colonization rates. Instead, our modeling, laboratory, and field results point to the strength of cascades being controlled by prey colonization rate, local producer and prey dynamics and omnivore foraging strategies.

Prior work has demonstrated that spatial isolation can modify cascades through effects on the composition and body masses of the predator guild [13] and predator extinction frequencies [16]. Our results suggest that even when

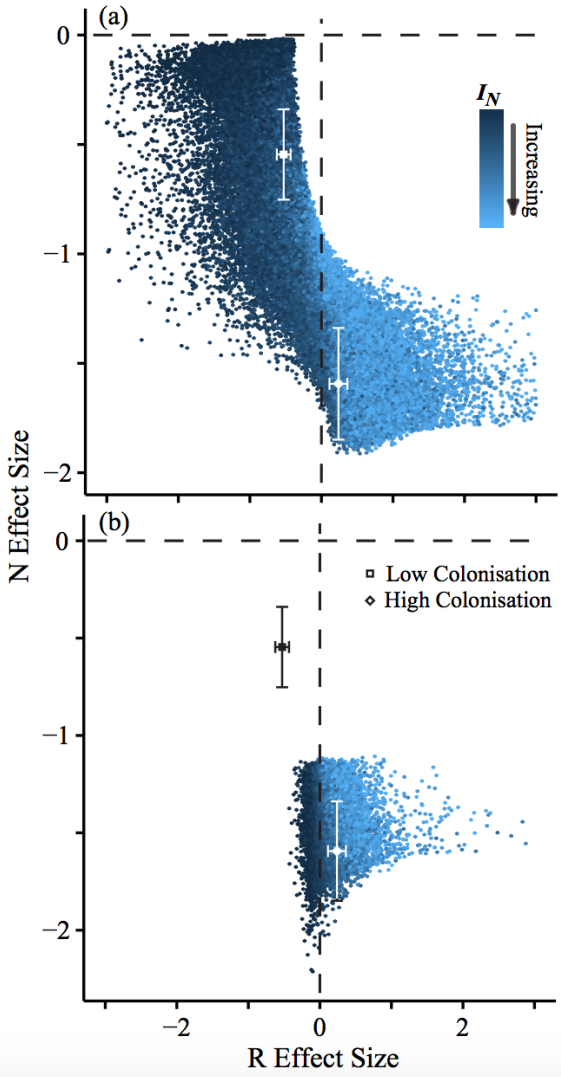
predator populations remain intact or vary on much slower timescales, changes in local foraging behavior driven by exogenous resource supplies can have equally dramatic effects. As a consequence, a deeper quantitative understanding of how food web interaction strengths are influenced by features of the spatial environment will be required to predict ecosystem responses to changing environmental conditions.

Table 2.1: Results of statistical analyses

Property	**Treatment**	**Sign**	**F(df)**	**P − value**
R	Time	+	19.14(3,84)	<0.001
	Distance	+	10.24(1,28)	0.0034
	Fish	+	2.1(1,28)	0.044
	Time×Distance	-	5.16(1,92)	0.042
	Time×Fish	n.a.	5.16(1,92)	0.23
	Distance×Fish	-	24.99(1,28)	<0.001
	Time×Distance×Fish	n.a.	0.344(3,84)	0.793
N	Time	+	35.72(3,84)	<0.001
	Distance	-	31.12(1,28)	<0.001
	Fish	-	31.79(1,28)	<0.001
	Time×Distance	-	31.79(1,28)	0.0003
	Time×Fish	-	2.92(3,84)	0.039
	Distance×Fish	+	9.68(1,28)	0.0043
	Time×Distance×Fish	n.a.	1.98(3,84)	0.123

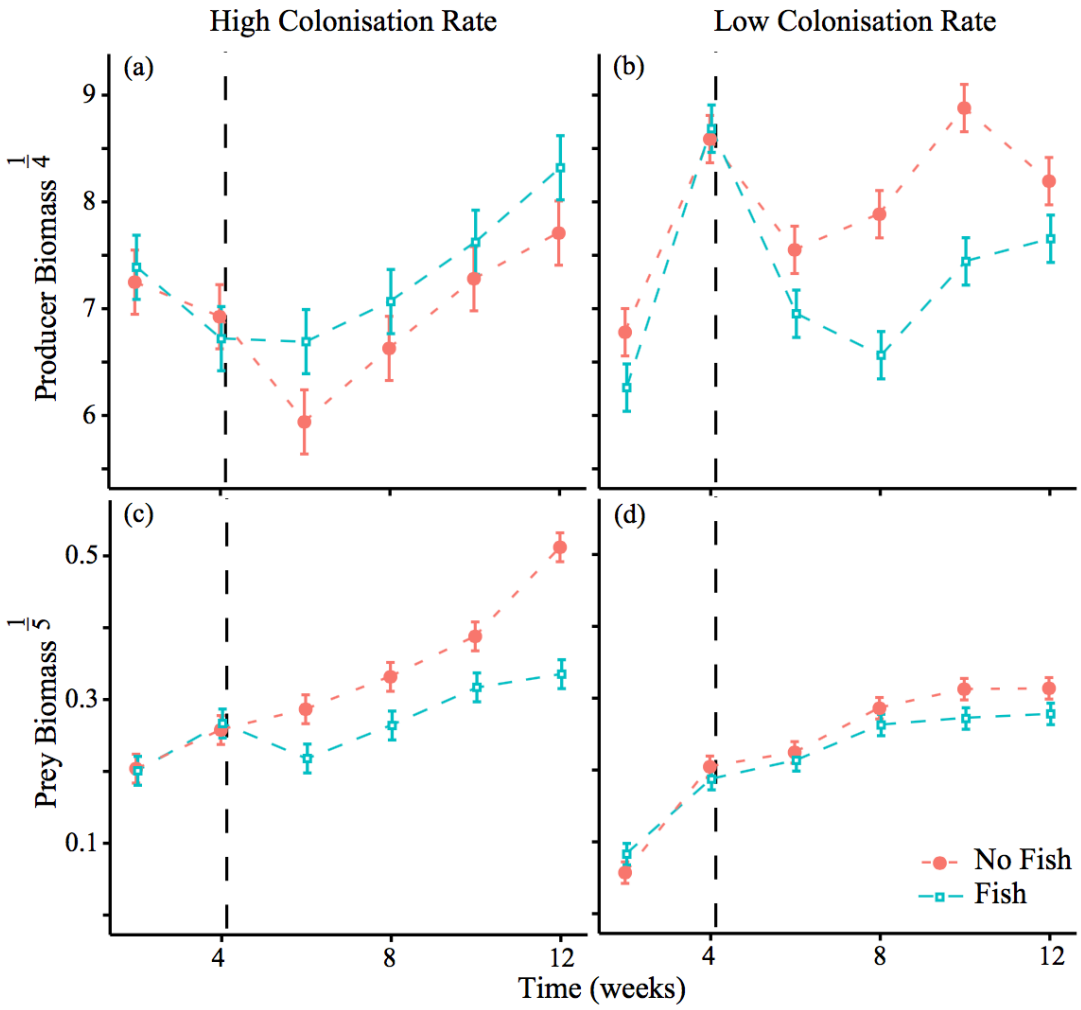
Results from analysis of community properties. “Property” column indicates the community property to which statistics refer (either producer or prey biomass densities). “Sign” column indicates whether the treatment variable resulted in an increase (+) or decrease (-) in the value of the community property.

Figure 2.1: Effects of colonization rate on trophic cascades



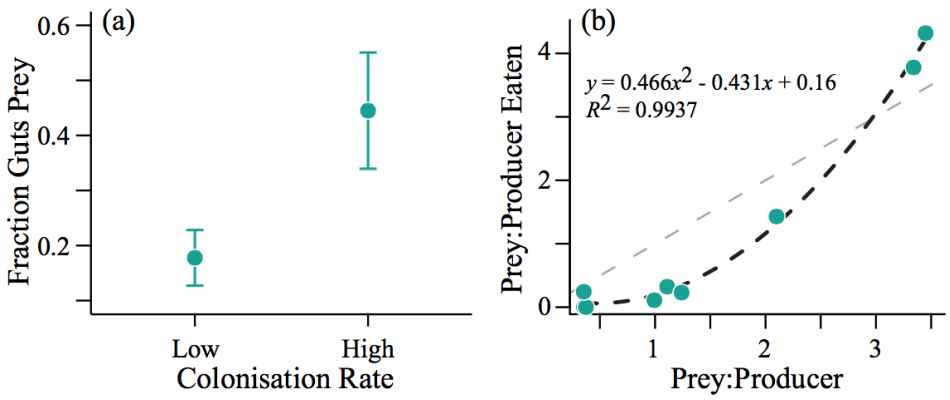
Effects of colonization rate on cascade strength (± SEM) from experimental ponds (squares = low colonization rate ponds; diamonds = high colonization rate) superimposed over results of models (colored points). Producer and prey effect sizes are on the x- and y-axes, respectively. (a) 10^5 simulations of the adaptive foraging model. (b) 10^5 simulations of the fixed foraging model with q_R = 0.2. Points represent stable internal equilibria resulting from individual simulations and are shaded according to the associated value of I_N, with lighter colors corresponding to higher colonization rates. Experimental points are shaded only for contrast against the background.

Figure 2.2: The dynamics of experimental ponds



Effects of fish and colonization rate on producer and prey biomass dynamics. Fish- and fishless ponds are indicated by blue squares and red circles, respectively. Dynamics of mean producer fluorescence (as a proxy for biomass) are shown for (a) high- and (b) low-colonization rate ponds (± SEM). Dynamics of mean prey biomass densities (mg mL-1 ± SEM) are shown for (c) high- and (d) low-colonization rate ponds.

Figure 2.3: Results of laboratory feeding experiments



Adaptivity in fish foraging in response to local resource conditions. (a) The fraction of fish guts occupied by prey (± 2 SEM) was higher in fish sacrificed from high colonization rate ponds. (b) Results of laboratory feeding trials show *G. affinis* exhibits a non-linear relationship between the ratio of prey:producers in the diet and the prey:producer biomass ratio in the environment. This non-linearity is consistent with predictions from adaptive foraging models.

Chapter 3

Network Size and Topology Regulate Trophic Cascades in Size-Structured Food Webs

3.1 Abstract

Variation in food web size and topological complexity is widespread in nature and probably influences trophic cascades, but experiments designed to test this hypothesis do not exist. Here I use simulations of empirically parameterized food web models to study the effects of these basic web features on the emergence of trophic cascades. I generate web topologies across a complexity gradient using a niche model, parameterize species interactions using allometric scaling rules and

study the effects of invading each web with a new predator on producer community biomass. I find that the probability of cascade emergence and cascade magnitudes decrease with increasing web size and increase with complexity, measured as the average degree of the network. This appears to hold because of the higher potential for producer compensation in large webs, which masks cascades at the producer community scale. Producers in more complex webs are more likely to share a suite of consumers however, and as a result show less compensation or more uniform responses to predator invasions. My results suggest that the search for a general theory of cascades in complex networks should focus on identifying features of network architectures that promote intraguild compensatory dynamics.

3.2 Introduction

The size and structure of ecological networks, where nodes represent species and edges represent trophic links, are highly variable in nature [23, 24]. Conceptual theories predict that these basic ecosystem features ought to regulate trophic cascades [68, 80]. But, understanding the relationships between the architecture of food webs and the processes that regulate cascades suffers from

insufficient data [80]; experiments designed to test these assertions do not exist [19]. Even basic theoretical questions about the effects of food web size and topological complexity on the emergence of cascades remain unanswered. This is in large part because cascade theory draws heavily from models that make simplifying assumptions like very few species in discrete trophic levels, non-saturating consumers [8] and unrealistic combinations of parameter values [90, 94]. Simple food chain models are tractable, can describe the gross dynamics of some systems (e.g., [27]) and provide analytical insight into ecological phenomena [15], but food chain models have so far failed to yield a general predictive theory of cascades [41]. The oft-ignored complexities of real ecosystems in modeling studies are a strong candidate for explaining this mismatch.

Network size may influence cascades through effects on biomass compensation or compensatory dynamics [33, 80]. This occurs for instance when, in large webs with many basal species, the biomass of one or a small number of producer populations changes while other populations change proportionately in the opposite direction; compensation has the potential to weaken or mask cascades. Shifts in the temporal correlation of fish biomass dynamics, that are consistent with community-level compensation, have been observed with

increasing species diversity in the northwest Atlantic [33] and with increasing functional diversity in a Nova Scotian marine system [78], which hint that compensation may more readily emerge in larger webs since both species and functional diversity increase with food web size. This implies that community-scale cascades may appear weaker or be less likely to emerge, because of this higher potential for compensation, in larger food webs [33, 80].

Observations that cascades appear to be strongest in freshwater lakes have been interpreted as evidence for the functional simplicity of lake webs [83] and the high generality of their constituent species. Experiments and observations indeed suggest that lake plankton are undifferentiated by planktivores [80], so phytoplankton and zooplankton can be sensibly aggregated into unified guilds and reasonably approximated by food chain models. This line of evidence and reasoning therefore would predict that cascades will be stronger or more likely to occur in highly connected (i.e., more complex) webs. Network complexity could also affect cascades through effects on compensation, but I am not aware of any studies that demonstrate relationships between features of network architecture and the strength of compensation in food webs.

Conversely, using techniques from information theory MacArthur [54] reasoned that the effects of changes in one population in a web on others shrinks as the number of links increases, and a small number of studies indeed evidence more diffuse predator effects in complex webs (e.g., [57]). These theories therefore predict that cascades will be weaker or less likely to occur in highly connected food webs. Therefore, based on prior theories two conceivable relationships between trophic cascades and complexity exist: cascades may be weaker or less likely to occur, or the opposite, as web complexity increases. The latter may be expected if high connectivity in a food web (i.e., webs with species that share many resources and consumers, on average) muffles compensation among intraguild populations whereas the former might hold if high complexity effectively diffuses predator effects and precludes their spread to the producer trophic level. Data provide mixed support for either possibility [7, 33, 32, 57], and issues of taxonomic aggregation in the literature make inferring compensation from observational studies difficult.

Here I use a simulation framework to study the effects of these basic food web features − size and topological complexity − on the emergence of trophic cascades in more biologically realistic ecological networks. I generate web

topologies using a niche model [91] and parameterize an energy flow model between species using empirical allometric scaling rules [8, 9, 94]; the model uses data on mass-metabolism, respiration and consumption rate relationships to constrain the strength of consumer-resource interactions [8, 9, 19, 94]. Across ranges of network size and complexity I study the effects of invading each web with a new top generalist predator on biomass in the producer level. I find that the probability of cascade emergence and cascade magnitudes decrease with web size but increase with complexity. This appears to hold, consistent with prior supposition, because large webs have a higher potential for compensation among producers that masks cascades. Conversely, producers in highly connected webs are more likely to share the same suite of consumers and therefore show weaker compensation, even when webs are large. My results highlight the pressing need for more network-based trophic cascade research and, critically, identify compensatory dynamics as an important driver of cascade emergence.

3.3 Methods

The niche model [91] successfully predicts the general structure of complex food webs [92] with only two free parameters: species richness, S and directed

connectance, C, defined as $\mathrm{L/S}^2$ where L is the number of trophic links. It can also be used as a generative model to create realistic food web topologies based on simple rules for assigning links. This procedure was used to generate 900 food web topologies (see below). Biomass dynamics were then simulated on these networks according to an allometric model.

3.3.1 The Niche Model

This model is discussed in detail by Williams & Martinez [91, 92] but briefly, a one-dimensional niche axis is assumed and each of S species are randomly assigned a 'niche value,' n_i, on this axis from the interval [0, 1]. Species i consumes all species within a range on the axis, r_i, that is placed by uniformly drawing the center from $[r_i/2,\ n_i]$. The range r_i is computed using a beta function to randomly draw values from [0, 1] with an expected value $2C$ and then multiplying that number by n_i. The model is constrained so that the species with the smallest $\mathrm{n_i}$ has $r_i = 0$; all webs contain at least one basal species. This approach was used to generate 900 food web simulations in a fully factorial design: initial species richnesses of $S = 10$, 20 and 30 were crossed with $C = 0.1$,

0.15 and 0.2 as input parameters (3 richnesses $\times$ 3 connectances $\times$ 100 iterations = 900 webs total). These values of C were chosen because they encompass the bulk of empirical connectance values [23]. Occasionally, model webs were generated that contained disconnected nodes or consisted of k disconnected subgraphs. When this occurred, the web was discarded and replaced by another niche model web consisting of a single connected component.

3.3.2 The Allometric Model

The flow of energy along the links in the food web was simulated with a multi-species allometric model that relates body mass to production, metabolic and consumption rates [8, 9, 90, 94]. This approach adds empirical realism to models by using body mass scaling to restrict possible parameter sets to biologically plausible combinations [90]. The equations for the rate of change of biomass of each species are

$$\frac{dB_i}{dt'} = B_i G_i(B) - \sum_{j=consumers} \frac{x_j y_{ji} B_j F_{ji}(B)}{e_{ji}} \tag{3.1}$$

for primary producers and

$$\frac{dB_i}{dt'} = -x_i B_i + x_i B_i \sum_{j=resources} y_{ij} F_{ij}(B) - \sum_{j=consumers} \frac{x_j y_{ji} B_j F_{ji}(B)}{e_{ji}} \qquad (3.2)$$

for consumers. All primary producers were assumed to have the same body mass, and time was scaled with the growth rate of primary producers [8, 90]. Here B_i is the biomass of species i and t' is re-scaled time. The metabolic parameter x_i is the mass specific metabolic rate of species i relative to the time scale of the system and the non-dimensional constant y_{ij} is the maximum ingestion rate (biomass per unit time) of resource i by consumer j relative to the metabolic rate of i (biomass per unit time). The function $G_i(B)$ is the normalized growth rate of primary producer population i, which follows the logistic growth equation, $1 - B_i/K_i$ where K_i is carrying capacity of basal species i. I assume implicit competition among producers occurs via a system-wide carrying capacity K and that n_{basal} basal species compete for this K according to $K_i = K/n_{basal}$. The assimilation efficiency e_{ji} is the fraction of the biomass of resource i lost due to consumption by consumer j that is metabolized [90]. The initial B_i of each species was uniformly selected from [0.1, 1].

The metabolic parameters are given by

$$x_i = \frac{a_{Ti}}{a_{rk}} \frac{M_k}{M_i}^{0.25} \tag{3.3}$$

$$y_{ij} = \frac{a_{ji}}{a_{Ti}}. \tag{3.4}$$

Here, M_i is the mass of an individual of species i and M_k is the mass of primary producers used for normalizing the time scale. The constants a_T, a_r and a_j (mass$^{0.25}$ $\times$ time^{-1}) are determined from empirical data on the allometry of metabolism, production, and maximum consumption respectively [8, 9, 90, 94]. I assume that all species are invertebrates, and so $a_r = 1$, $a_T = 0.314$ and $y_{ij} = 8$ (see [8] for the derivation of these values). The assimilation efficiency $e_{ij} = 0.45$ for consumption of producers and $e_{ij} = 0.85$ for consumption of consumers [94]. In order to reduce the size of the parameter space being explored, all species in a web are assumed to have a constant consumer-resource body size ratio Z so the mass of species i is $M_i = M_k Z^{T-1}$ where T is the trophic level of species i [8].

Trophic level was computed as the short-weight trophic level [92], which is a function of the shortest path between species i and the base of the web and the

mean trophic positions of all of i's resources. I report simulations in which $Z = 10^1$. Alternate values of Z had only modest effects on trophic cascades. Results of simulations with $Z = 10^2$, 10^3 and 10^4, which encompass most of the range of empirically observed values of Z [4], are shown in Appendix C and did not qualitatively differ from the results of simulations with $Z = 10^1$.

The function $F_{ji}(B)$ is the normalized multi-species functional response for consumer j and resource i, developed by Yodzis & Innes [94] and extended by others [8, 90, 92]. Adding predator interference [18] to a multispecies non-linear functional response [63] gives a functional response of the form

$$F_{ji} = \frac{\omega_{ji} B_i^{1+q}}{B_0^{1+q} + d_i B_j B_0^{1+q} + \sum_k \omega_{jk} B_k^{1+q}}. \tag{3.5}$$

Here d_i is a positive constant that sets the amount of predator interference [18] in the system and the sum in the denominator is over all k resources of consumer j. The parameter ω_{ji} is the relative preference of consumer j for resource i [56]. As in previous studies [8, 57, 56, 92] I assume $\omega_{ji} = 1/n_{rj}$, where n_{rj} is the number of resources of species j. The parameter B_0 is the half saturation density. I report

results for systems in which predator interference occurs but is weak ($d_i = 0.15$) and $B_0 = 0.5$. To stabilize simulations and avoid the collapse of food webs following predator invasions, I set the shape parameter $q = 0.2$. This gives a very slight relaxation of consumption at low resource biomasses, which is both biologically realistic [34] and probably too subtle to observe empirically (R.J. Williams *pers. comm.*).

3.3.3 Analyses

Simulations were run for 5000 model time steps at which point a top generalist predator invaded the food web. I assume that the predator is an indiscriminate secondary carnivore with body mass $M_P = 10^9$ that consumes any species in the web with $T > 2$; the invader consumes all predators and omnivores but not herbivores that are already present. This can be interpreted as the analogue to an experiment in which the same large-bodied generalist predator is added to many different food webs with known topologies. Following the predator invasion, each system was run for a further 5000 time steps. Cascade magnitudes were measured as the absolute value of the log response ratio $|\log_{10} B_{final}/B_{init}|$,

where B_{final} and B_{init} are producer community biomasses (summed over basal species) averaged over time steps 9000−10000 and 4000−5000 respectively. I define a cascade as a prompted change in producer biomass by at least a factor of 3, $|\log_{10} B_{final}/B_{init}| > \log_{10} 3$. The qualitative results presented below are unchanged by the choice of cutoff used to determine if a cascade occurred. Simulations were performed using the *deSolve* and *cheddar* libraries in R [71].

To analyze simulation results, I created sets of generalized linear models (GLMs) with binomial distributions and logit link functions. I used these models to study the effects of network size (S) and complexity (L/S) and their interaction on the probability of cascade occurrence following predator invasions. I define complexity as the network's average degree, L/S where webs with more links per species are said to be more complex. It is important to note that the frequentist statistical hypothesis tests employed here were not used to assess statistical significance (i.e., p-values) since power and p-values are determined by the number of simulations one chooses to run. Instead, I follow the suggestion of White et al. [89] and use the statistical models described below as a framework for partitioning variance and effect sizes in these multifactorial simulations and comparing effect sizes among factors. Starting with the model including the full

set of interaction coefficients, I generated a suite of candidate models of all possible combinations of coefficients from the full model and computed Akaike's Information Criterion (AIC) for each candidate. I selected the model with the lowest AIC as the best-fitting model. This process was automated using the *MuMIn* library in R [71].

To determine how the magnitude of cascades depended on network size and complexity I used *ANOVA* and the same model selection procedure with cascade magnitude $|\log_{10} B_{final}/B_{init}|$ as the response variable and the same set of predictor variables as described above. Overall, I am interested in (1) the probability that a cascade emerged and (2) the magnitude of predator effects and how these depend on S and L/S. I report these effects below using the notation $\beta_{\text{predictor}}$ (e.g., $\beta_{\text{L/S}}$ is the network complexity effect, which reflects the per unit impact of scaled L/S on the logit-transformed probability that a cascade occurred, in the case of GLMs). Only those coefficients that are included in best-fit models are reported below.

3.4 Results

Predator invasions had relatively small effects on total producer community biomass in most webs (Fig. 3.1), with only 22% of producer communities changing by at least a factor of three. Of the webs that exhibited cascades, 53% were small (initial $S = 10$); 29% occurred in medium webs (initial $S = 20$); 18% occurred in large webs (initial $S = 30$). The magnitude of the strongest cascade also decreased with increasing web size: 1.514, 1.511 and 1.29 for $S = 10$, 20 and 30 respectively. Food webs exhibited a variety of dynamics including fixed-point equilibria (Fig. 3.2a) and periodic or chaotic fluctuations (Fig. 3.2b) both before and after predator invasions. I did not note any systematic signatures in dynamics preceding cascades; cascades occurred when invasions prompted transitions from stable to unstable dynamics (Fig. 3.2c) and vice versa (Fig. 3.2d), or when they did not change at all (Figs. 3.2a and 3.2b).

Results of the best-fitting GLM analyses reveal that network size and complexity have opposite effects on the probability of cascade emergence. The model demonstrates that cascades are less likely to emerge in large food webs (Fig. 3.3; β_S = -1.32). On the other hand, the model shows that cascades are more likely to emerge in food webs with high connectivity (Fig. 3.3; $\beta_{L/S}$ =

0.86). The average magnitude of predator effects, measured as $|\log_{10} B_{final}/B_{init}|$, followed a qualitatively similar trend to those of cascade probabilities.

Cascade magnitudes decreased with network size (Fig. 3.4; β_S = -0.18) and increased with average degree (Fig. 3.4; $\beta_{L/S} = 0.11$) in these simulations. In both cases, the effect of network size was greater than the effect of average degree, L/S. Of the webs that did not exhibit cascades, more than 90% contained at least one producer population that exhibited a population-level cascade, $|\log_{10} B_{ifinal}/B_{iinit}| > \log_{10} 3$, where B_{ifinal} and B_{iinit} are the final and initial biomasses of basal species i. This indicates that the absence of community-level effects could be explained by compensatory changes in the biomasses of one or several species in opposite directions. To quantitatively assess this possibility, I computed variance ratios VR of biomass dynamics following predator invasions in all webs. This metric of compensation is based on the ratio of the variance in total community biomass B_C and the variances of individual producer populations B_i [37],

$$VR = \frac{varB_C}{\sum_{i=1}^{n} varB_i}. \tag{3.6}$$

In this case, a VR that is less than 1 is consistent with intraguild producer compensation since the sum of population variances are greater than variance in aggregate biomass; a VR greater than 1 is consistent with weaker compensation or uniform responses to invasions [37]. Fig. 3.5 shows the results of routine Gaussian kernel density estimation for the computed distributions of VR, separated into webs that did and did not exhibit cascades. The higher probability of compensation (i.e., VR is less than 1) is visible for webs that did not exhibit cascades, indicating that the absence of community-level effects could be explained in large part by biomass compensation at the producer population scale in these webs.

3.5 Discussion

The results of my simulations suggest that cascades are more likely to emerge in smaller and more connected food webs, a result in agreement with some prior interpretations of trophic cascades [69, 83]. Counter to ecological folk-wisdom however, this did not hold because predator effects were more diffuse in certain types of webs (e.g., [65, 68]). In other words, the absence of a cascade did not

coincide with predator effects that failed to transmit down the web to producers. Rather, apparent competition (due to shared herbivores) and implicit exploitative competition (due to a system-wide K) among producers lead to the common situation in which one or a small number of species increased in biomass, while others decreased proportionately, effectively masking the cascade at the guild scale. That this occurred in more than 90% of simulations suggests that the search for a general theory of cascades in complex networks should focus on identifying features of network architectures that promote compensatory dynamics among species. I know of no such studies in the literature.

In a meta-analysis of 114 cascade experiments, Borer et al. [7] did not detect an effect of species richness on the magnitude of cascades. The results presented here suggest that the effects of richness on cascades are conflated with variation in the network's average degree. It would be interesting to revisit the analyses of Borer et al. with this feature as an additional predictor variable, but unfortunately such data do not exist. A test of this prediction would require an enormous experimental effort involving predator addition experiments in many replicate food webs, whose first principles (i.e., species' abundances, population body mass distributions) and web topologies must be measured at the very least

immediately before and long after predator manipulations. Compiling this sort of quantitative food web time series data has only been undertaken once in the literature to my knowledge (see [28]).

Broadly, these results imply that trophic cascades in networks generated by the empirically motivated niche model are the exception rather than the rule. Although these models represent a step toward more ecological realism, they do lack several features of real ecological systems that could potentially affect cascades. For instance, I assume that body mass scales with trophic level. While this is true for the overwhelming majority of free-living species [9], parasites that occupy top trophic levels but are smaller than their hosts are notable exceptions [42], and researchers are only now beginning to explore the nuanced consequences of parasitism in food webs [14]. On the issue of generating realistic topologies, the niche model used here appears to capture concomitant predation on parasites, but not parasite-host linkages, well [47]. The extent to which parasites affect cascades is not generally known and is limited by questions on how to include them in food web models in the first place [14]. Addressing this limitation and others, including but not limited to extending models to account for the influences of adaptive behavior and spatial processes (e.g., dispersal of individuals

across a spatial network of interconnected habitats) on food web dynamics will be important enterprises for future research [27].

Changes in the temporal variability or autocorrelation of species biomasses have been proposed as potential early-warning indicators of pending ecosystem regime shifts. It is hypothesized that as complex systems approach a tipping point (e.g., [11]) their dynamics display a phenomenon called critical slowing down [87]: increased temporal autocorrelation and variance. Cascades have been proposed as a type of regime shift in ecosystems [11]. But, the observation that the types of dynamics exhibited by the present simulations were so variable suggests that couching cascades in terms of early-warning signals requires additional study. I find no evidence that any specific dynamical signature preceded cascades (Fig. 3.2). Invasions by the top predator were accompanied by shifts from periodic fluctuations to stable fixed points and vice versa, or no change in dynamics at all. Sometimes these were accompanied by excited transient fluctuations with large amplitudes and other times not, monotonically approaching the new system attractor instead. Whether cascades are analogous to regime shifts in ecosystems remains an open question, although the present

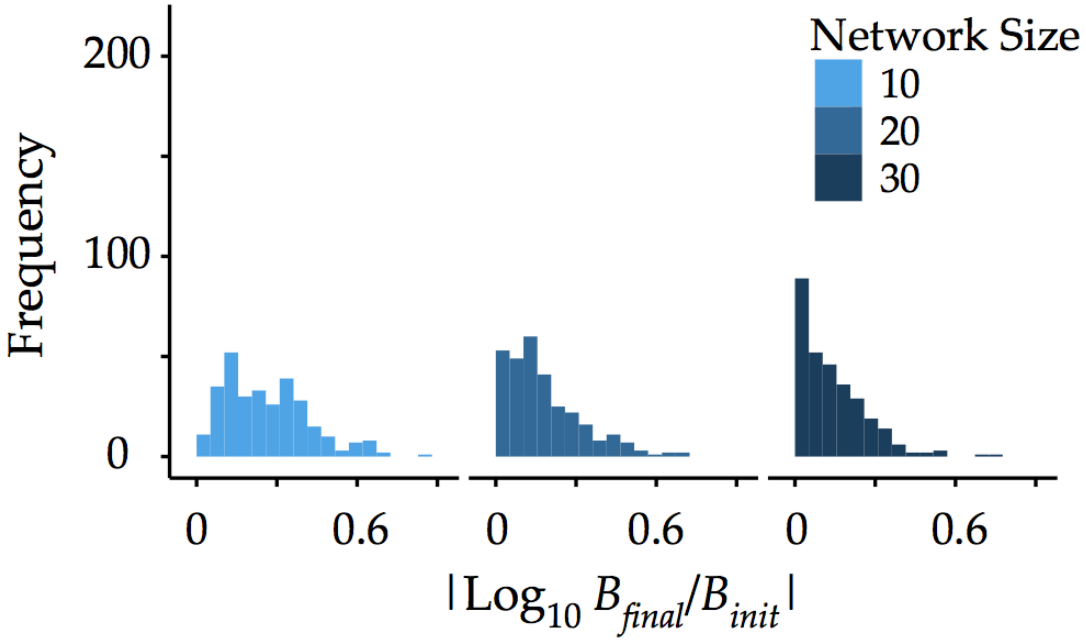
results suggest that dynamical signatures in population time series do not precede the induction of cascades.

These results are also consistent with suppositions that higher connectivity in trophic networks promotes cascades [83]. One potential explanation is that highly connected webs contain functionally similar species (i.e., species that share many resources and consumers) and as a result may be more reasonably coarse-grained into lower-dimensional systems that resemble food chains. New ways of detecting and comparing coarse structure in networks are emerging in the network theory literature (e.g., backbone detection and alignment algorithms, coarse equivalence, mixed membership stochastic block models; [61]). A promising avenue for future research will be to determine whether webs that exhibit cascades display network structures that coarsely resemble food chains. Different coarse-graining algorithms can lead to different outcomes however, and there is no consensus on which if any are best for reducing the dimensionality of ecological networks.

Identifying the abiotic and biotic features of ecosystems that regulate trophic cascades is a fundamental issue in ecology [68] and a practical problem for the management of pests and invasive species [75]. While the present study

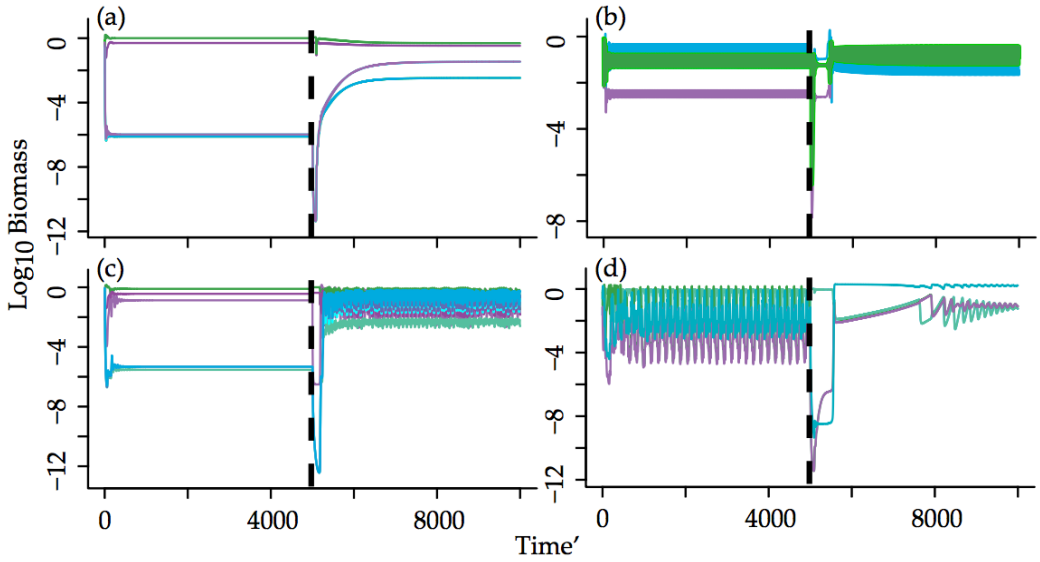
identifies features of model food web architecture that influence cascades, the potential for compensation [33, 37], complex indirect interactions and feedbacks [93] in real world networks together with insufficient data [80] combine to make the development of a predictive cascade theory of food webs a difficult problem.

Figure 3.1: Frequency distributions of cascade magnitudes



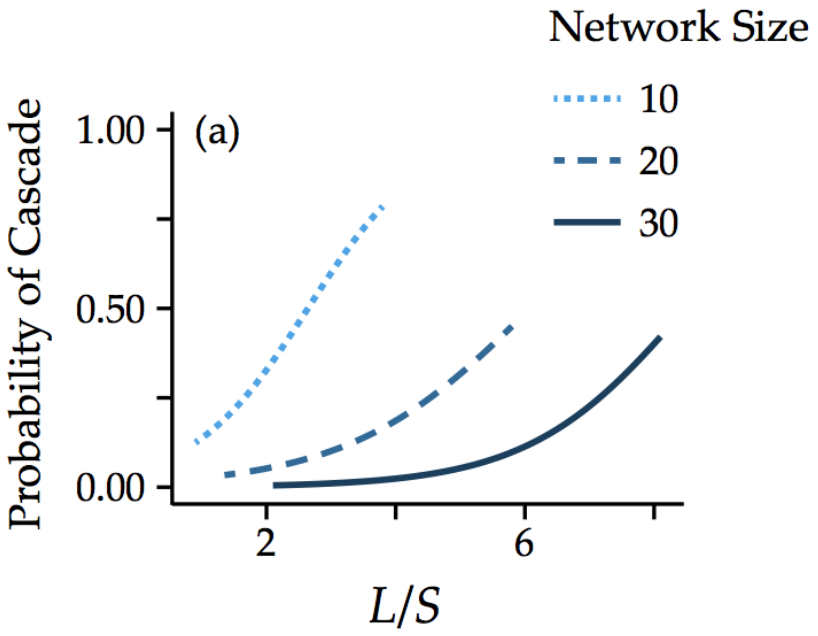
Frequency distributions of predator effects resulting from 900 food web simulations with $Z = 10^1$. Bars are colored according to initial network size, S. Results of all simulations are shown in Appendix C.

Figure 3.2: Predator invasions prompt different types of dynamical behavior



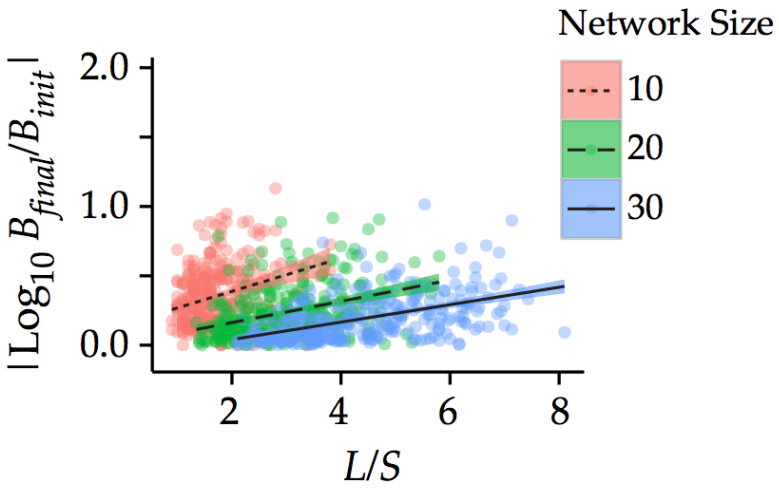
Dynamics of four representative food web simulations. Biomasses are $\log_{10}$ transformed and time is normalized by the growth rate of primary producers. Each line represents the dynamics of a single producer population. Vertical dashed lines are placed at the time of predator invasion. Transitions from stable to unstable (and vice versa) dynamics are visible.

Figure 3.3: Estimated probabilities of cascade emergences



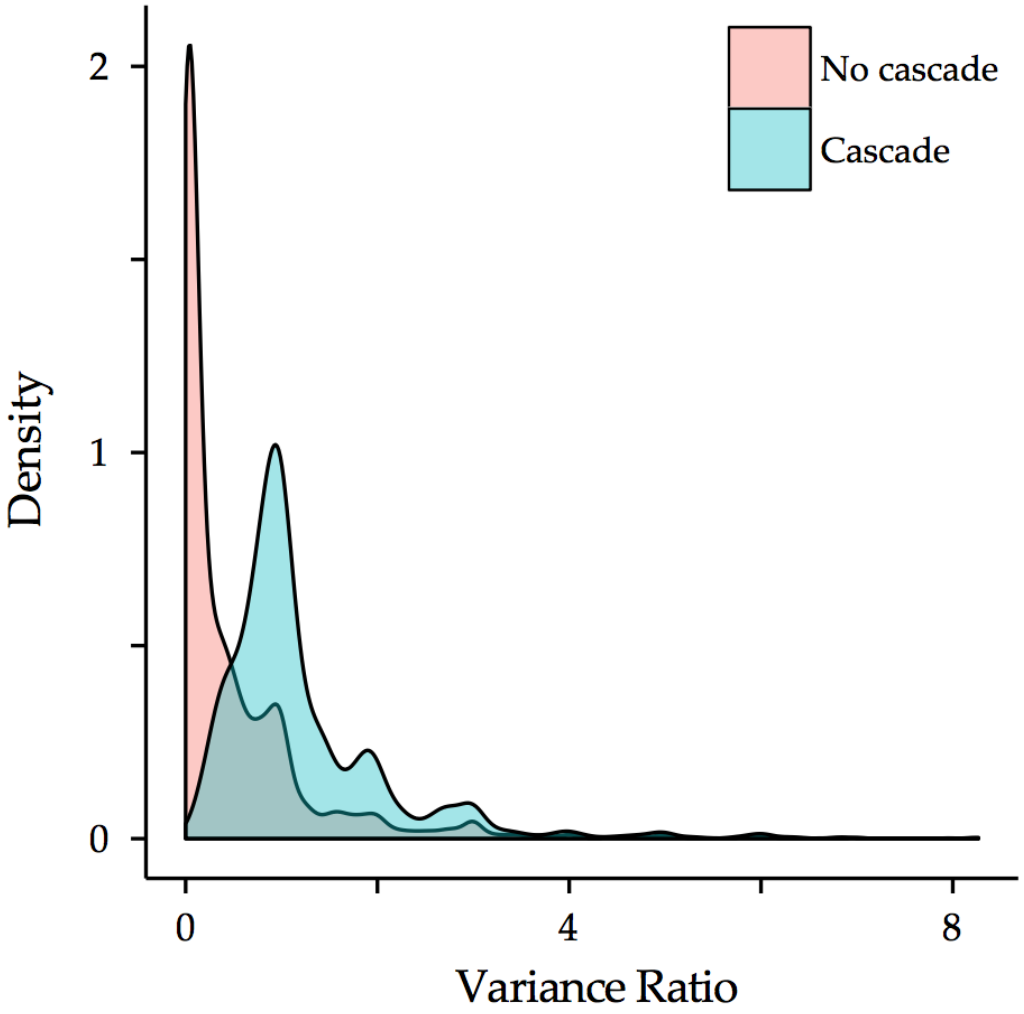
Probabilities of cascade occurrences were affected by network size and complexity. Lines are estimated probabilities from GLMs. Lines are colored based on initial network size, S. As high values of L/S relative to S are not observed in nature [23], the corresponding predictions are truncated to reflect a biologically plausible range only.

Figure 3.4: Cascade magnitudes depend on network size and complexity



The magnitude of predator effects depended on food web size and complexity in 900 web simulations. Best-fit lines from the model selection procedure with 95% confidence intervals are shown.

Figure 3.5: Compensation explains hidden cascades



Density plot showing the probability of compensation (i.e., Variance Ratio tends to be less than 1) across all simulations was higher when cascades did not occur. Density plots were generated using a Gaussian kernel estimation algorithm, and bandwidth was selected according to Silverman's rule-of-thumb [81].

Chapter 4

Strong Trophic Cascades in Enriched Omnivory Systems

4.1 Abstract

Understanding bottom-up enrichment effects on the outcomes of food web interactions, particularly trophic cascades, is a fundamental issue in ecology and a practical problem for ecosystem restoration and agricultural pest management. Theory predicts that enrichment will enhance cascades (i.e., stronger producer facilitation) in food chains. However, many if not most predators in webs are omnivores and enrichment has disparate effects on food chains and omnivory systems. A theory of enrichment effects on omnivory cascades is therefore a sorely

needed extension of trophic cascade models. Conceptual theories predict omnivory precludes the potential for cascades because of direct consumption of producers by top predators. Here I present results of analytical models across an enrichment gradient and show that, counter to intuition, omnivores that follow a fixed foraging strategy cause stronger cascades relative to chains when enrichment is high, despite the fact that they directly consume producers. I take a dynamical systems approach to demonstrate that resource competition between prey and omnivores forces higher prey suppression and producer facilitation at high enrichments. Finally I show that when omnivores forage adaptively enrichment prompts a rapid shift toward a pure herbivory strategy, which always weakens cascades relative to chains, yielding patterns more consistent with prior conceptual theories and experiments.

4.2 Introduction

Trophic cascades occur when predators reduce prey populations, indirectly affecting lower trophic levels. However, there is continued uncertainty about the mechanisms causing variation in the strength of observed cascades; theoretical predictions have received only mixed empirical support (e.g., [7, 32, 41, 79, 83]).

Statistical analyses of empirical data have hinted that other factors, like the prevalence of omnivory (i.e., feeding from more than one trophic level) in food webs [3, 86] causes the disaccord between trophic cascade theory and data, but experiments designed to test this hypotheses are scant [10, 29, 68, 80]. Meanwhile, worldwide losses of top predators stemming from human activities continue to cascade down food webs, affecting key system properties like biomass distributions across trophic levels [25].

Human activities have also affected bottom-up regulation of ecosystems by inducing changes in habitat enrichment, for instance through nutrient inputs to ecosystems, agricultural runoff, eutrophication or land use [31, 41, 73]. Top-down and bottom-up processes jointly regulate all ecosystems, and interactions between them in food webs can generate non-intuitive patterns in emergent community structure (e.g., [20, 21]); a predictive theory of trophic cascades will require their joint consideration [41].

An emerging consensus from experiments in freshwater systems, where anthropogenic changes to enrichment are an important management concern [12], is that enrichment enhances top-down trophic cascades in food chains with carnivorous predators [65]. Simple models support these data [73]. But, complex

feedbacks [56] and counter-intuitive enrichment-biomass relationships observed in some omnivory systems [60] question the extensibility of predictions from food chain models and experiments to ecosystems with omnivores [20, 21]. Extensions of cascade theory borne by models that incorporate variation in omnivory and enrichment are thus an important undertaking for the development of a predictive cascade theory. This endeavor will also inform the future efficacy of applied management strategies such as integrated pest management in agricultural systems, where enrichment is manipulated to maximize agricultural yield and natural enemies of pest herbivores are frequently omnivorous [17].

Conceptual theories hypothesize that omnivory ought to weaken cascades relative to analogous food chains because of direct consumption of producers by omnivores [65, 68], and a small number of experiments have supported this assertion [10, 29, 30]. Here I present results of analytical models that demonstrate this is not always the case. Namely, moderate omnivory in high enrichment habitats causes stronger cascades relative to food chains. This counterintuitive result occurs because prey biomass in omnivory systems rapidly decreases with enrichment despite a simultaneous increase in their resource. The added jeopardy of competing with one's predator augments the depression of prey by omnivores

through an additional competitive pathway. I take a dynamical systems approach to parse effects along this pathway and demonstrate that the net facilitation effect of omnivores on producer biomass production increases in magnitude with enrichment, and that this occurs at a faster rate than in analogous food chains. In contention with the analytical intuition I develop here however, prior experiments have uniformly pointed to omnivory weakening cascades [10, 27, 29, 65]. Insofar as these studies did not all occur in low enrichment habitats, additional explanatory mechanisms are needed for this inconsistency. I extend the present models to incorporate adaptive foraging behavior by the omnivore, and show that cascade strengths in these models are bounded by those in analogous food chains and thus stronger cascades in adaptive omnivory systems cannot emerge. Adaptive or flexible foraging is ubiquitous in real food webs [1, 48] and provides at least one feasible explanation for the apparent mismatch between the theoretical predictions developed here and data.

4.3 Model Motivations

I model trophic cascades in food chains with three species arranged in a linear sequence; producers are consumed by prey that are in turn consumed by

predators. Drawing an additional linkage between the predator and the producer yields the analogous omnivory system. I focus on equilibrium outcomes in order to pro- vide insights into how differences in predator foraging strategies (i.e., carnivory, fixed omnivory or adaptive omnivory) influence long-term community structure as measured by the trophic cascade.

Analyses of the three-node omnivory motif in closed systems indicate that the equilibrium biomasses of all species are particularly sensitive to variations in habitat enrichment and omnivory strength (parameters K and q in eqs. 4.1 and 4.4 below; reviewed extensively by McCann & Hastings [56] and Diehl & Feissel [20, 21]). For this reason, I concentrate my analyses on cascades in systems that vary in these features. Namely, I am interesting in understanding how enrichment and omnivory strength influence the relative magnitude of cascades in omnivory and food chain systems.

4.3.1 Food Chains and Fixed Omnivory Models

The models that govern population dynamics, which correspond to a biomass interpretation of Lotka-Volterra models with type I consumer functional responses are summarized in Table 4.1 and described by eqs. 4.1−4.6.

$$\frac{dR}{dt} = \rho R(1 - \frac{R}{K}) - \alpha NR \tag{4.1}$$

$$\frac{dN}{dt} = e\alpha NR - mN - \beta PN \tag{4.2}$$

$$\frac{dP}{dt} = g\beta PN - \mu P \tag{4.3}$$

represents the food chain and the analogous omnivory system can be represented as

$$\frac{dR}{dt} = \rho R(1 - \frac{R}{K}) - \alpha NR - q\beta PR \tag{4.4}$$

$$\frac{dN}{dt} = e\alpha NR - mN - (1-q)\beta PN \tag{4.5}$$

$$\frac{dP}{dt} = fq\beta PR + g(1-q)\beta PN - \mu P. \tag{4.6}$$

The state variables R, N and P are the biomasses of producers, prey and predators (or omnivores) respectively and K is enrichment, also called carrying capacity. I concentrate on how changes in K influence relative cascade strength – the strength of the cascade in the omnivory system relative to the strength of the cascade in the analogous food chain. I do this by deriving a comparative metric of trophic cascades, herein called R_{comp}.

As in prior studies [7, 32, 79] I define cascade strength as the log response ratio of equilibrium producer biomasses, $\log_e R^{*+}/R^{*-}$; for R^{*+} and R^{*-}, plus and minus signs denote the presence and absence of predators respectively, and the asterisk indicates the closed form solution is evaluated at equilibrium (solutions shown in Table 4.1). I differentiate between equilibrium producer biomasses in food chains and omnivory systems using R^{*+} and R^{*+} respectively. In its expanded form, R_{comp} is equal to the log response ratio of the omnivory system minus the log response ratio of the analogous food chain,

$$R_{comp} = log\frac{R_o^{*+}}{R^{*-}} - log\frac{R_c^{*+}}{R^{*-}}. \tag{4.7}$$

Note that the denominators on the right-hand side are equal (the R^{*-} solution) because the topologies of the predator-free systems are isomorphic for these motifs (i.e., they are both consumer-resource Lotka-Volterra systems and have the well known closed form solution, $m/\alpha e$). Using logarithm rules and taking advantage of this isomorphism, the right hand side of eq. 4.7 can be re-written as

$$R_{comp} = log \frac{R_o^{*+}}{R^{*-}} \frac{R^{*-}}{R_c^{*+}}. \tag{4.8}$$

Taking the product,

$$R_{comp} = log \frac{R_o^{*+}}{R_c^{*-}}. \tag{4.9}$$

Substituting the corresponding closed form solutions from Table 4.1 into eq. 4.9 and simplifying yields the R_{comp} metric used to compare cascades:

$$R_{comp} = log \frac{g\rho(-\alpha\mu + g\beta(mq + \rho - q\rho))}{((f - eg)Kq\alpha + g(q-1)\rho)(\alpha\mu - g\beta\rho)} \tag{4.10}$$

R_{comp} will be positive if cascades are stronger in omnivory systems and negative if cascades are stronger in food chains, for a given parameter set. I repeat this procedure to derive the prey analogue of R_{comp}, N_{comp}, by substituting the closed form solutions of equilibrium prey biomasses from eqs. 4.1-4.6 into eq. 4.9, yielding

$$N_{comp} = log \frac{-fgKq\beta + eKq\alpha\mu - fKq\beta\rho + fKq^2\beta\rho + \mu\rho - q\mu\rho}{(q-1)\beta(fKq\alpha - egKq\alpha + g(q-1)\rho)}. \tag{4.11}$$

By deriving R_{comp} and N_{comp}, I avoid costly simulation approaches and facilitate the exploration of parameter space while minimizing computational time.

4.3.2 Quantifying Net Interspecific Interactions

Trophic cascades are caused by both direct and indirect interactions in food webs [65, 68, 79]. To study changes in the strength of indirect interactions between predators and producers, I first compute the 'interaction matrix,' $\mathbf{A}[a_{ij}]$ where elements a_{ij} represent the effect of species X_j on the per capita biomass production rate of species X_i,

$$a_{ij} = \partial \frac{1}{X_i} \frac{dX_i}{dt} / \partial X_j. \tag{4.12}$$

In the absence of a direct interaction $a_{ij} = 0$; the interaction matrix denotes only the per capita level direct effects of species on one another in a system. Yodzis [93] derived the 'inverse interaction' matrix, $\mathbf{S}[s_{ij}] = -\mathbf{A}^{-1}$, and showed that its elements denote the net effects of an infinitesimal press input of X_j on X_i through

all possible direct and indirect pathways (see also Higashi & Nakajima [44] for an alternate derivation of this matrix). I concentrate on changes in the magnitude of s_{RP} , which denotes the net effect of predators (or omnivores) on producer *per capita* biomass production rates, across the enrichment gradient. Since omnivores consume producers and prey, s_{RP} can theoretically take positive or negative values, with a positive value indicating that the direct consumption of R by P is overridden by facilitation of R by P through the consumption of N.

There is no general consensus on how to measure indirect interaction strengths in model food webs [6, 51]. Here I follow the suggestions of Laska and Wootton [51] and Berlow et al. [6] and focus on per capita effects in the interaction matrix, as opposed to the Jacobian approach that describes changes in population-level biomass production rates among species (e.g., [55]). A focus on per capita effects facilitates the direct comparison of systems, since results are independent of population biomasses [6]. It is important to note however that theoretical cascades, which are analogous to the outcomes of species extirpations and the inverse matrix approach described above, which is analogous to a press experiment in which community structure remains intact but biomasses are changed, do describe different but related processes [51]. I use the inverse

interaction matrix approach to contrast the effects of a press addition of predators with that of omnivores on producer facilitation across the enrichment gradient, which may help develop intuition as to why differences, if any, between cascades across the foraging strategies studied here emerged. A logical hypothesis is that stronger cascades correspond to larger positive s_{RP} values (i.e., larger net facilitation of R by P).

4.3.3 Adaptive Omnivory

I incorporated adaptive foraging strategies into models by substituting q and (1 – q) in eqs. 4.4—4.6 with a pair of dynamical variables, q_R and q_N, which are based on replicator equations (e.g., [48]) and represent the proportion of total foraging effort an omnivore will apportion toward one of its resource species, X [2]. The replicator attempts to maximize biomass production by increasing its foraging effort toward resource X (i.e., q_X) if a unit change in q_X causes its biomass production to exceed that derived from current foraging effort [27]. The variable q_X is represented as

$$\frac{dq_X}{dt} = vq_X \left(\frac{\partial \gamma}{\partial q_X} - \sum_{k \epsilon S} q_k \frac{\partial \gamma}{\partial q_k} \right), \tag{4.13}$$

where v is a rate constant that sets the timescale upon which changes in foraging effort occur, γ is the omnivore's per capita biomass production rate, $\gamma = fq_R\beta R + gq_N\beta N$, and the sum is taken over all S species in the omnivore's diet. All other parameters are identical to those in Table 4.1. As it is difficult if not impossible to obtain a closed form solution of systems containing the replicator equation [40], I use simulations to draw comparisons between the fixed and adaptive foraging models. To do this over a wide range of potential conditions, I generated 50 values of K evenly spaced on the interval [2, 6.5] and simulated the model 100 times for each value of K, drawing all other parameter values independently from uniform distributions (Table 4.2). Simulations were performed using the *rootSolve* library in R [71]. For the expanded form and additional details on the replicator equation, see Appendix B and Fahimipour and Anderson [27].

4.4 Results and Discussion

4.4.1 Analyses of Food Chain and Fixed Omnivory Cascades

Fig. 4.1a depicts the relative strength of cascades for omnivory and food chain systems, R_{comp}, across a gradient of enrichment K and omnivory strength q. There are two interesting points. First, increasing enrichment causes a transition from weaker to stronger cascades in omnivory systems, relative to the food chain.

Parameter value ranges used to simulate adaptive foraging models. Parameters are defined in Table 4.1. Ranges were chosen because they produced stable internal equilibria for models with and without predators.

Second, increasing omnivory strength q enhances this effect. These results are counter-intuitive − under certain conditions, greater consumption of a producer by an omnivore leads to a stronger facilitation effect on that same producer. Fig. 4.1b shows that, consistent with prior theory [20, 60], a greater inhibition of prey by omnivores (via contemporaneous consumption and competition) occurs under high K and moderate q, which is at least partially responsible for the observed

shift. In other words, omnivory enhances trophic cascades when enrichment is high, in part because resource competition causes higher prey mortality than in food chains where competition does not occur.

Results of inverse interaction matrices show that the net effect of a press input of omnivores on producers is positive (i.e., $s_{RP} > 0$) despite the presence of a direct negative effect, and largest in the region of parameter space characterized by positive values of R_{comp} (mean $s_{RP} = 55.46$ when $R_{comp} > 0$ and mean $s_{RP} = 42.3$ when $R_{comp} < 0$; Fig. 4.2). All together, this suggests that stronger omnivory cascades occur under high K and intermediate q because these conditions cause stronger prey inhibition (Fig. 4.1b) and stronger producer facilitation (Fig. 4.2), relative to food chains.

Other parameters also influence this relationship, and can reverse this pattern such that increasing K leads to increasingly weak cascades in omnivory systems relative to food chains. This can be illustrated by computing the partial derivative of R_{comp} with respect to K,

$$\partial R_{comp}/\partial K = \frac{-g(f-eg)q\alpha\rho(-\alpha\mu + g\beta(mq+\rho-q\rho))}{((f-eg)Kq\alpha + g(q-1)\rho)^2(\alpha\mu - g\beta\rho)}, \qquad (4.14)$$

which will be positive for a given parameter set if R_{comp} will increase with a unit change in K as in Fig. 4.1. Figure 4.3 shows that the qualitative results presented above depend strongly on the prey's conversion efficiency, e. Increasing e leads to a shift from positive to negative values of $\partial R_{comp}/\partial K$, indicating that increasing enrichment causes stronger omnivory cascades when e is low, but weaker omnivory cascades when e is high (Fig. 4.4). This result should be interpreted with caution however, since the relatively large values of e that drive the sign change are greater than typical empirical estimates of conversion efficiencies (e.g., [19]), and may be unrealistic.

4.4.2 Simulations of Food Chains and Adaptive Omnivory Cascades

Adaptive omnivory foraging simulations over a wide range of parameter values show that increasing enrichment in this system always causes weaker cascades relative to food chains (i.e., $R_{comp} < 0$; Fig. 4.4a). One feature of the

replicator equation is that changes in foraging effort are lagged [1]. This feature adds biological realism to adaptive models, since it takes time to detect changes in resource conditions or employ a new behavior [1, 34]. The consequence of this lag for cascades in these systems however, is that any facilitation of producer biomass by the predator will then be followed by a lagged increase in foraging effort toward producers (i.e., optimal diet selection; [34]), suggesting that suboptimal fixed foraging strategies are needed to prompt stronger omnivory cascades. Data indicate that both suboptimal and quasi-optimal foraging strategies are common in nature and are observed across a wide variety of taxa [1, 34, 48, 50, 72].

I am aware of three experiments that test the effects of trophic omnivory on the strength of trophic cascades. Bruno and O'Connor [10] demonstrated that the addition of omnivorous pinfish to marine amphipod and isopod herbivore communities weakened cascades compared to communities with four other predators (crabs, shrimp, killifish and blennies). Similar results were observed by Flecker [30] in Venezuelan streams, and by Fahimipour and Anderson [27] herein, who observed cascade reversals in the presence of omnivorous fish predators in ephemeral ponds. These studies, in addition to prior conceptual theories [65, 68]

consistently suggest that omnivory ought to weaken cascades relative to food chains. Yet, analyses of fixed foraging models presented here reveal that this is not necessarily the case (Fig. 4.1). However by including adaptive foraging, I generate patterns across a wide range of potential conditions that are more consistent with prior expectations of omnivory-cascade relationships. Broadly, these results emphasize that knowledge of a predator's foraging strategies (e.g., fixed versus adaptive foraging) will be needed to generate predictions for the relative strength of cascades induced by distinct predators. Insofar as these studies did not all occur in low enrichment habitats, adaptive or flexible omnivory provides at least one feasible explanation for the mismatch between prior theories and the results of models analyzed here.

4.5 Conclusions

Observations from recent well-resolved food web data [3, 86, 92] now reveal that many if not most predators in nature are omnivorous to some degree. As a result, extensions of trophic cascade theories, including theories on the responses of cascades to increasing enrichment, toward omnivory systems will be needed to continue advancement toward a general predictive theory of indirect effects in

food webs [65]. The result that omnivory can enhance cascades under certain conditions obtained here is surprising in light of the near-unanimous consensus that omnivory in food webs ought to weaken cascades [10, 30, 65, 68, 80] due to direct consumption of producers by omnivores. I show that the opposite can occur because of the added mortality assumed by prey in omnivory systems, who must also compete with their predators for resources. Moreover, I demonstrate that across a wide range of conditions, the strength of adaptive omnivory cascades are always bounded by cascade strength in the analogous food chain − a result more consistent with prior supposition and data.

A growing number and diversity of factors that affect trophic cascades continue to emerge [65], which have improved our understanding of the ecological mechanisms that dampen or enhance indirect effects in food webs. Cascades are also becoming another signature of growing anthropogenic impacts on natural and agricultural ecosystems [11, 65]. Examples from agroecosystems [17, 75] and imperiled habitats that are the current object of management or restoration efforts [26] suggest that the future of cascade theories will progress to inform applied management problems and efforts to curb human alteration of ecosystems [65]. The present comparative analyses together with the documented ubiquity of

omnivory in nature suggest that omnivores contain promise for applications of cascade theory to ecological problems.

Table 4.1: Summary of analytical models

Parameter	Definition
ρ	Producer biomass production rate
K	Producer carrying capacity, or enrichment
α	Prey search rate
β	Net predator search rate
e	Conversion efficiency for prey consuming producers
f	Conversion efficiency for predators consuming producers
g	Conversion efficiency for predators consuming prey
m	Prey mortality rate
μ	Predator mortality rate
q	Omnivore's preference for producers
State Variable	**Closed Form Solution**
R_c	$R_c^{*+} = K - \frac{\alpha K \mu}{\beta \rho g}$
N_c	$N_c^{*+} = \frac{\mu}{g\beta}$
P_c	$P_c^{*+} = -\frac{eK\alpha^2\mu + gm\beta\rho - egK\alpha\beta\rho}{g\beta^2\rho}$
R_o	$R_o^{*+} = -\frac{gKmq\beta - K\alpha\mu + gK\beta\rho - gKq\beta\rho}{\beta(fKq\alpha - egKq\alpha - g\rho + gq\rho)}$
N_o	$N_o^{*+} = \frac{-fKmq^2\beta + eKq\alpha\mu - fKq\beta\rho + fKq^2\beta\rho + \mu\rho - q\mu\rho}{(q-1)\beta(fKq\alpha - egKq\alpha - g\rho + gq\rho}$
P_o	$P_o^{*+} = \frac{fKqm\alpha\beta - eK\alpha^2\mu - gm\beta\rho + gmq\beta\rho + egK\alpha\beta\rho - egKq\alpha\beta\rho}{(q-1)\beta^2(fKq\alpha - egKq\alpha - g\rho + gq\rho)}$

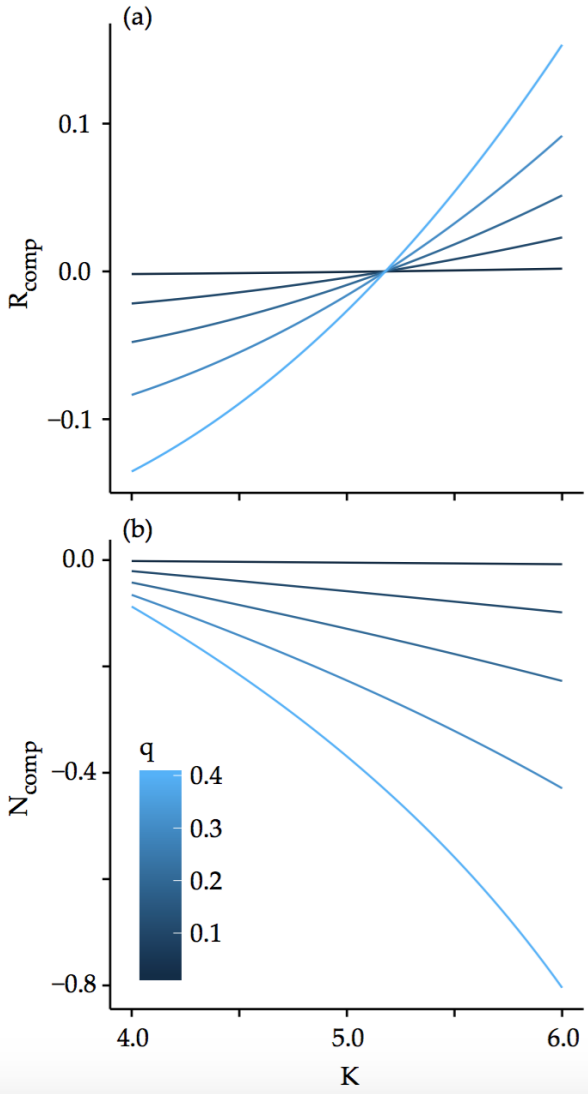
Summary of the analytical models described by eqs 4.1−4.6. The subscripts denote food chains, 'c' and omnivory systems, 'o.' Closed form solutions are provided for 3-species coexistence equilibria.

Table 4.2: Parameter ranges used for adaptive foraging simulations

Parameter	**Range**
ρ	[0.5, 1]
K	[2, 6.5]
α	[0.1, 0.15]
β	[0.15, 0.2]
e	[0.2, 0.4]
f	[0.08, 0.1]
g	[0.1, 0.2]
m	[0.01, 0.015]
μ	[0.038, 0.04]

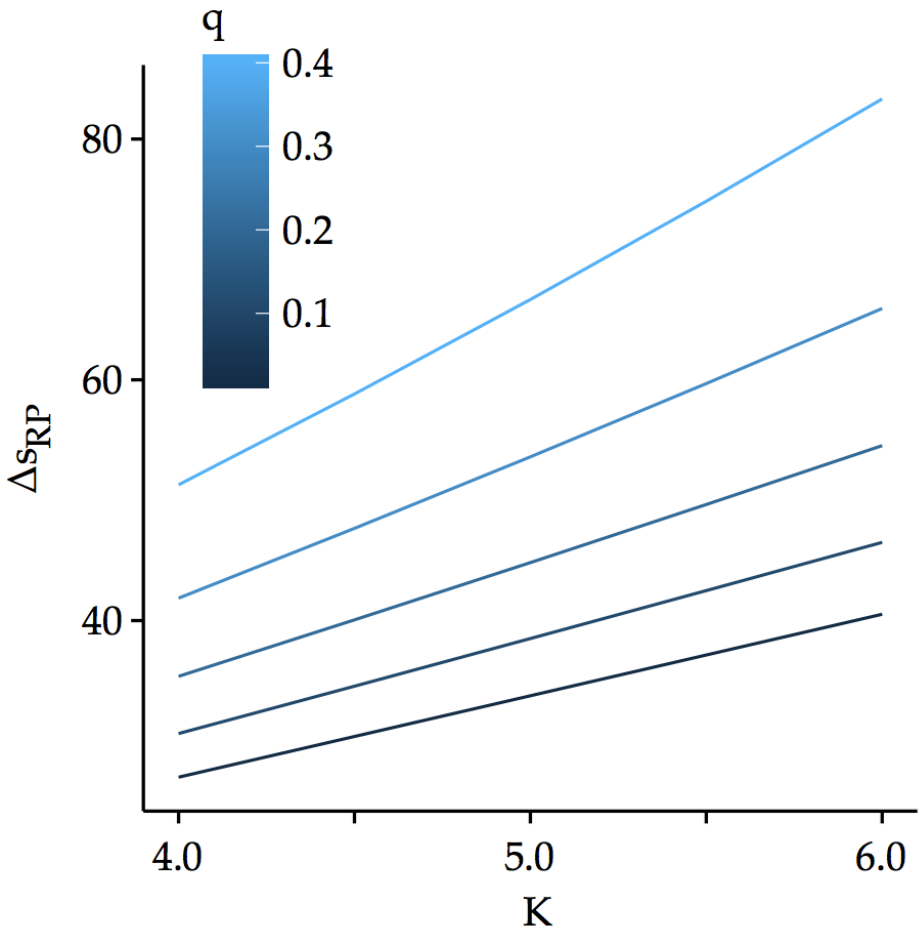
Parameter value ranges used to simulate adaptive foraging models. Parameters are defined in Table 4.1. Ranges were chosen because they produced stable internal equilibria for models with and without predators.

Figure 4.1: Comparative analyses of cascades in omnivory and food chain systems



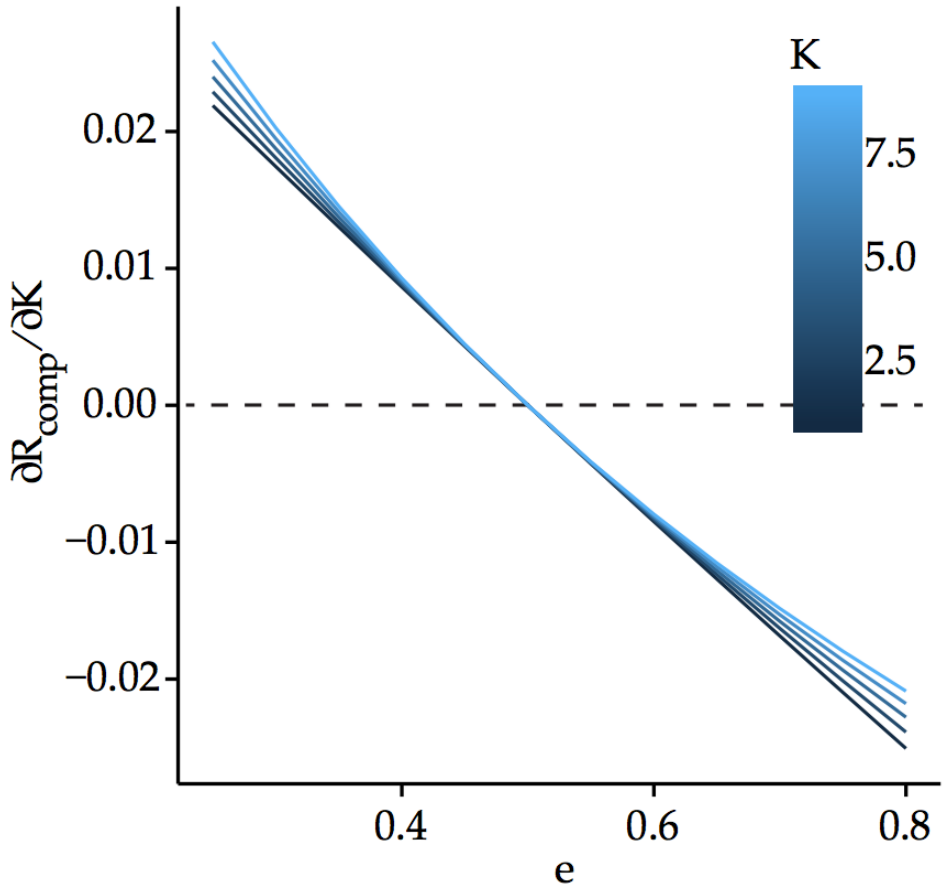
Results of R_{comp} analyses reveal that (a) increasing omnivory strength q weakens cascades relative to chains when enrichment is low, but enhances cascades relative to chains when enrichment is high. Lines are colored based on the associate values of q. Panel (b) shows that prey effect sizes become increasingly negative (i.e., prey biomass is increasingly depressed) as enrichment increases, relative to food chains.

Figure 4.2: Effects of press perturbations on producer facillitation



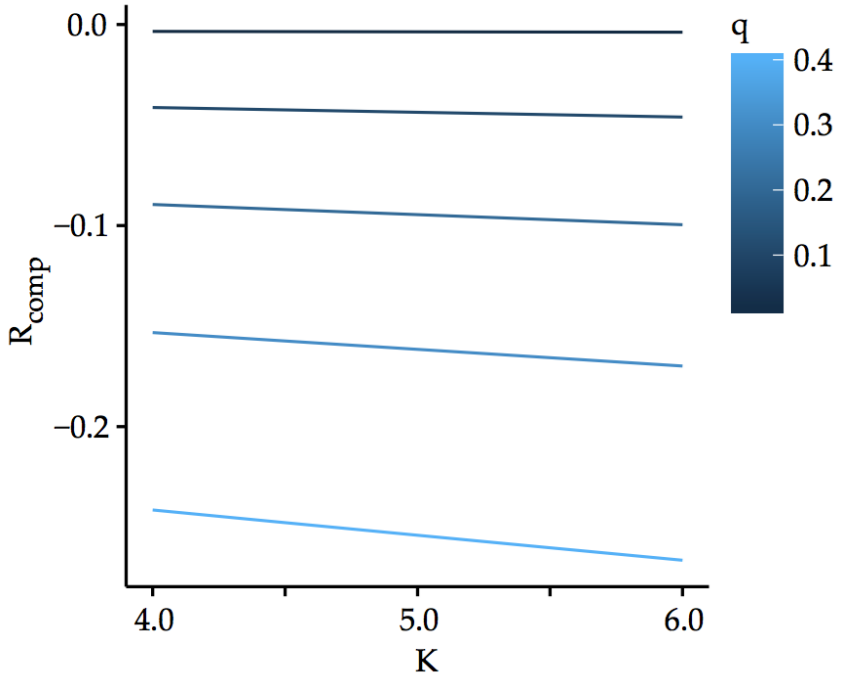
Results of inverse interaction matrix analysis. The y-axis s_{RP} represents the difference in facilitation effects between omnivores and predators. s_{RP} values greater than 0 indicate that the indirect facilitation of producers is stronger in omnivory systems, for a given parameter set. Increasingly steep slopes with increasing omnivory strength q are visible.

Figure 4.3: Prey conversion efficiencies alter enrichment effects



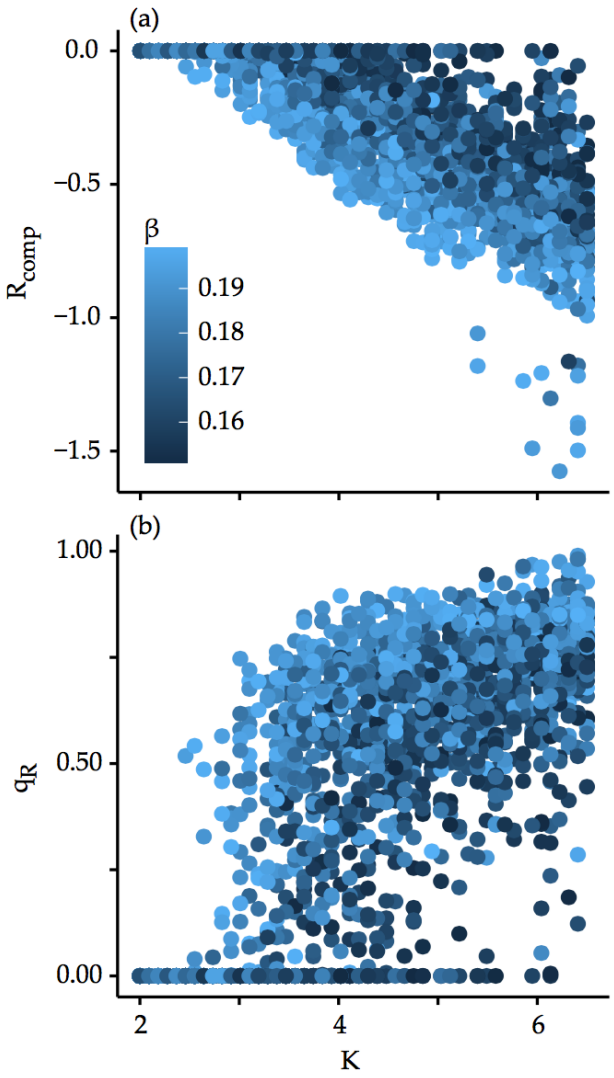
The partial derivative of R_{comp} with respect to K shows how the qualitative results presented in Fig. 4.1 also depend on prey conversion efficiency, e. The sign change from positive to negative values of the partial indicate that increasing prey conversion efficiencies leads to a shift from the patterns observed in Fig. 4.1, to a situation in which increasing enrichment and omnivory strength uniformly dampens cascades relative to food chains. An example of this shift is shown in Fig 4.4.

Figure 4.4: High prey conversion efficiencies reverse enrichment patterns



Results of R_{comp} analyses under high prey conversion efficiencies yield a case in which the patterns from Fig. 4.1 are reversed. Lines are colored based on the associated values of q.

Figure 4.5: Results of adaptive foraging simulations



Results of 5000 adaptive foraging simulations under a wide variety of potential conditions (see Table 4.2 for parameter value ranges) show that cascades in adaptive systems are bounded by those in analogous food chains. (a) Effects of enrichment on R_{comp} in this system. Note the horizon at $R_{comp} = 0$. Panel (b) shows the associated changes in foraging effort toward producers, q_R, rapidly approaches a pure herbivory strategy (i.e., $q_R = 1$) as enrichment increases, which explains the relatively weak cascades in adaptive systems. Points are colored according to values of β, which show increasing predator search rate modulates this effect.

Chapter 5

Conclusions

5.1 Conclusions

Trophic cascades are regulated by many biotic and abiotic factors. Identifying these factors is a fundamental issue in ecology [68] and a practical problem for conservation efforts [85], ecosystem restoration [65] and the management of pests and invasive species [75], yet most have received little theoretical or empirical attention [45, 80]. In this dissertation, I have investigated how spatial variation in animal behavior and habitat colonization rates, the network size and topology of the associated food web and feedbacks between bottom-up forcing and predator foraging strategies affect emergent community structure and trophic cascades. The studies outlined here add to the library of evidence that demonstrates predators' regulation of ecosystems, which is critical for biodiversity maintenance

[45, 85], will continue to suffer from anthropogenic stressors such as habitat fragmentation. Human activities continue to extirpate top predators at an alarming rate [25, 65, 85]. While the present work identifies several features of ecosystems that influence cascades, complex indirect interactions [2, 11, 20, 26, 57], feedbacks [93], and the myriad processes that influence cascades on different spatial and temporal scales in real ecological systems, together with insufficient data [80] continue to impede efforts to predict consequences of these losses.

The models and experiments reported here share a common theme in that they link processes that operate on different scales. In chapter two I demonstrated that animal movement at the landscape scale and foraging behavior at the scale of the individual predator controlled the strength and direction of cascades in experimental pond habitats [27]. With my third chapter I employed empirically parameterized food web models to demonstrate the strong regulating influence of network architecture on the magnitude of cascades and the probability of their emergence. Finally in chapter four, I used a comparative analytical approach to link bottom-up forcing to variation in predator foraging strategies and the strength of cascades. The challenge of understanding why trophic cascades vary in complex ecological systems has been severely hampered

by a lack of observational data and experiments designed to test theoretical predictions. My hope is that these results will encourage researchers to measure cascades in other complex ecological systems.

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Appendix A

List of Taxa Encountered in Irvine, CA

Table A.1: Taxa encountered during field experiment

Order	Family	Genus	Species	Order	Family	Genus	Species
Diptera	Ephydridae	Scatella	stagnalis*	Ploima	Lepadelladae	Lepadella	triptera
Coleoptera	Dytiscidae	Laccophilus	atristernalus*	Ploima	Brachionidae	Brachionus	sp b*
Diptera	Culicidae	Culex	tarsalis*	Ostracoda	Fam	Gen	sp a*
Diptera	Ephydridae	Ephydra	cinerea*	Cladocera	Daphniidae	Daphnia	sp a*
Diptera	Chironomidae	Gen	sp a*	Ploima	Lecanidae	Lecane	stokes*
Coleoptera	Dytiscidae	Colymbetes	sp a*	Ploima	Lecanidae	Lecane	inermis*
Coleoptera	Dytiscidae	Liodessus	hobbsi*	Ploima	Euchlanidae	Euchlanis	sp a
Coleoptera	Dytiscidae	Liodessus	sp a*	Ploima	Brachionidae	Anuraeopsis	fissa*
Anura	Fam	Gen	sp a	Ploima	Lecanidae	Monostyla	sp a*
Odonata	Libellulidae	Sympetrum	pallipes*	Ostracoda	Fam	Gen	sp b*
Diptera	Chironomidae	Gen	sp a*	Ploima	Lecanidae	Monostyla	cornuta*
Coleoptera	Dytiscidae	Hydroporus	sp a*	Ploima	Brachionidae	Lepadella	rhomboids*
Diptera	Tipulidae	Polymeda	cana	Ploima	Brachionidae	Notholca	sp a*
Odonata	Libellulidae	Miathyria	marcella*	Ploima	Lecanidae	Lecane	ludwigi*
Hemiptera	Corixidae	Sigara	sp a*	Ploima	Brachionidae	Notholca	squamula*
Ploima	Lecanidae	Monostyla	compies*	Ploima	Brachionidae	Notholca	sp b*
Ploima	Lecanidae	Monostyla	bulla*	Ploima	Brachionidae	Lepadella	ovalis*
Ploima	Brachionidae	Notholca	acuminata*				
Ploima	Euchlanidae	Euchlanis	alata				
Ploima	Lecanidae	Lecane	mira*				
Ploima	Lecanidae	Monostyla	lunaris*				
Ploima	Brachionidae	Brachionus	sp a*				
Ploima	Lecanidae	Lecane	luna*				
Ploima	Euchlanidae	Euchlanis	parva				

Prey taxa were identified to the highest possible resolution – species or genus in most cases. We use generic terms (e.g., fam, gen, sp) to indicate morphospecies – taxa that were not identifiable to full resolution, but that were clearly differentiable from other species that were present. Asterisks denote taxa with confamilials that were encountered at least once in the guts of fish sacrificed from the field experiment.

Appendix B

Supplementary Methods and Analyses

B.1 Model Details and Extensions

Model parameters not defined in the main text are as follows: r is the producer biomass production rate, K is producer carrying capacity, a_N is prey search rate, m is prey mortality rate, e_R is the conversion efficiency for a prey consuming a producer, f_R and f_N are the conversion efficiencies for an omnivore consuming a producer or prey and a_P is the omnivore's search rate. The full formulae for q_R and q_N for models with type I functional responses are:

$$\frac{dq_R}{dt} = vq_R(a_P f_R R - (q_R a_P f_R R + q_N a_P f_N N)) \tag{B.1}$$

$$\frac{dq_N}{dt} = vq_N(a_P f_N N - (q_R a_P f_R R + q_N a_P f_N N)) \tag{B.2}$$

Table B.1: Parameter value distributions

Parameter	Range	Value
r	(0.5, 1.2)	n.a.
K	(.5, 2)	n.a.
a_N	(0.3, 0.6)	n.a.
m	(0.2, 0.4)	n.a.
e_R	(0.05, 0.1)	n.a.
f_R	n.a.	$e_R - U(1 \times 10^{-4}, 5 \times 10^{-1})$
f_N	n.a.	$f_R - U(1 \times 10^{-2}, 2 \times 10^{-1})$
I_N	$(1 \times 10^{-4}, 5 \times 10^{-1})$	n.a.
I_R	$(1 \times 10^{-4}, 7.5 \times 10^{-1})$	n.a.
q_{R0}	(0.3, 0.7)	n.a.

To perform simulations, we drew parameters from the following uniform distributions based around parameters in the omnivory model of McCann [51] and shown in Table B.1. The parameter q_{R0} represents the initial conditions for predator foraging strategies. Note that we always assume $e_R > f_R$ and $f_N > f_R$. In other words, we assume prey convert a larger proportion of consumed producers into new biomass than omnivores and that prey are always more profitable to omnivores than producers. For simulations, we held P and a_P constant to mimic experimental conditions (i.e., we held fish body masses and densities constant throughout the field experiment) at $P = 1$ and $a_P = 1$. Fig. B.

1 shows the dependence of producer and prey effect sizes on each of the model parameters. High sensitivity of effect sizes to I_N , and their low sensitivity to I_R, are visible. Parameters v, r, e_R and a_N have relatively little effect on effect sizes.

A model with type II functional responses yields the same qualitative patterns as models with type I functional responses (Fig. B.2). This alternate model is given by:

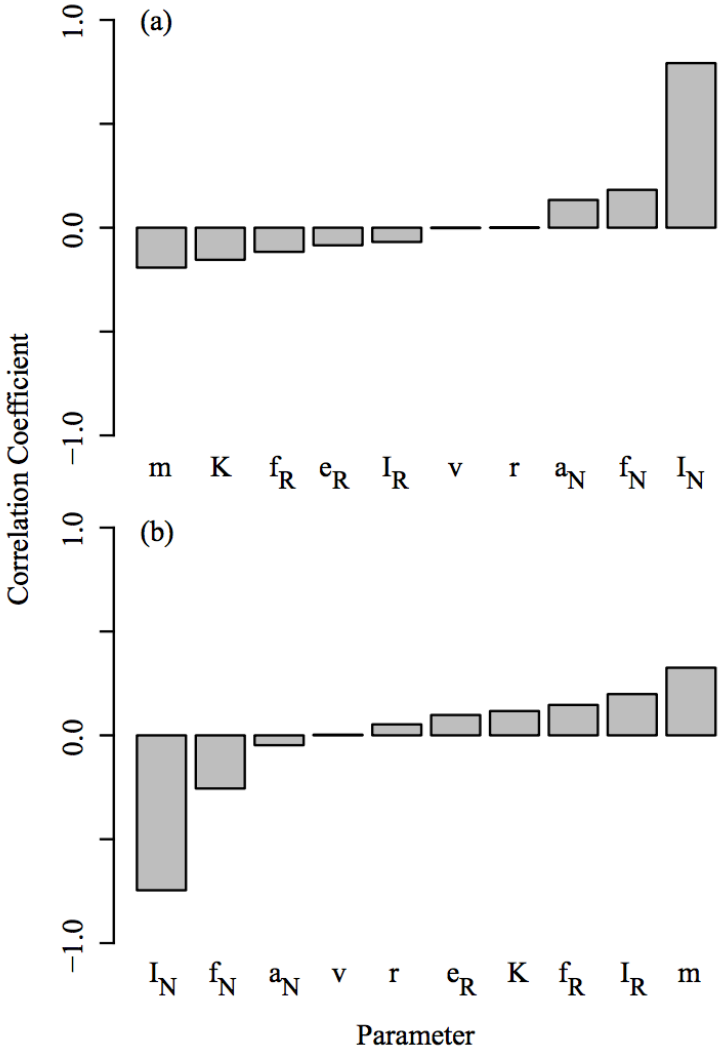
$$\frac{dR}{dt} = I_R + rR(1 - \frac{R}{K}) - \frac{a_N RN}{1 + h_R a_N R} - \frac{q_R a_P RP}{1 + j_R q_R a_P R + j_N q_N a_P N} \tag{B.3}$$

$$\frac{dN}{dt} = \frac{e_R a_N RN}{1 + h_R a_N R} - mN - \frac{q_R a_P PN}{1 + j_R q_R a_P R + j_N q_N a_P N} \tag{B.4}$$

$$\frac{dq_X}{dt} = vq_X \left(\frac{\partial \beta}{\partial q_X} - \sum_{k \epsilon S} q_k \frac{\partial \beta}{\partial q_k} \right) \tag{B.5}$$

All parameters are the same as above, except for the addition of $h_R \sim$ U(0, 3), prey handling time; $j_R \sim$ U(0, 4), omnivore handling time for producers; $j_N \sim$ U(0, 5) omnivore handling time for prey. To analyze this model, we performed the same procedure as described in the main text.

Figure B.1: Parameter impacts



Correlation coefficients describing the relationships between (a) producer and (b) prey effect sizes and parameters are shown for 10^5 randomly generated food chains with type I functional responses.

B.2 Supplementary Analyses of Field Experiment

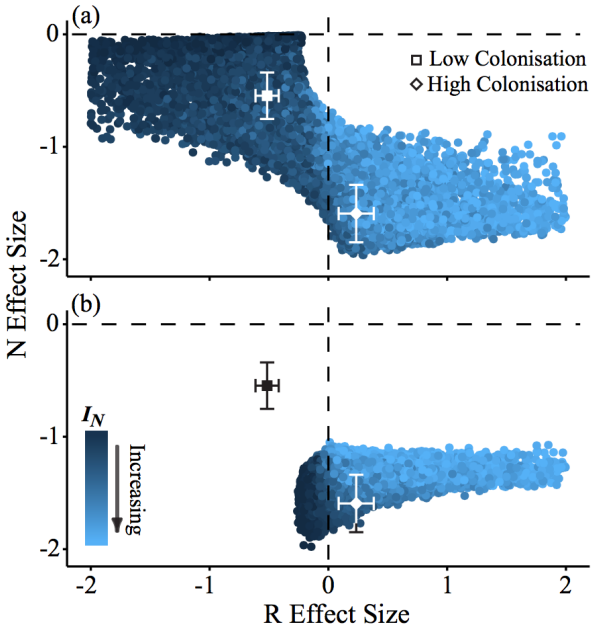
Changing the number of post-fish addition sampling dates included in the cascade strength analyses did not change the qualitative results presented in the main text. For instance, the results of analyzing only week 12 (the final time step) are qualitatively identical to those presented in the main text (*ANOVA*; prey effect size; $F_{1,14} = 9.06$, $P = 0.009$; producer effect size; $F_{1,14} = 5.38$, $P = 0.035$).

We also fit a model identical to that described in the main text to the dynamics of species richness in our ponds, to explore alternate explanations for our observed patterns (see main text). We observed that distance had a negative effect on prey species richness (LME; $F_{1,14} = 45.7254$, $P < 0.0001$), indicating that low colonization rate ponds contained fewer prey species. Low colonization rate ponds contained 5.9 species, on average, compared to 11.9 observed in high colonization rate ponds.

Over the course of the field experiment, we observed four fish deaths in high- and three fish deaths in low-colonization rate ponds. We performed logistic regression, and observed a non-significant effect of colonization rate on the

probability of a fish replacements due to mortality using the R [62] function *glm* with binomial distributions and logit link functions (non-significant distance effect, P = 0.623). During pond surveys, we also replaced any fish that displayed excessive somatic growth, which we define as exceeding 35 mm in length. Over the course of our experiment, this occurred for only two fish during the week 10 censuses – one in a high colonization rate pond, and one in a low colonization rate pond.

Figure B.2: Results of simulations with Type II responses

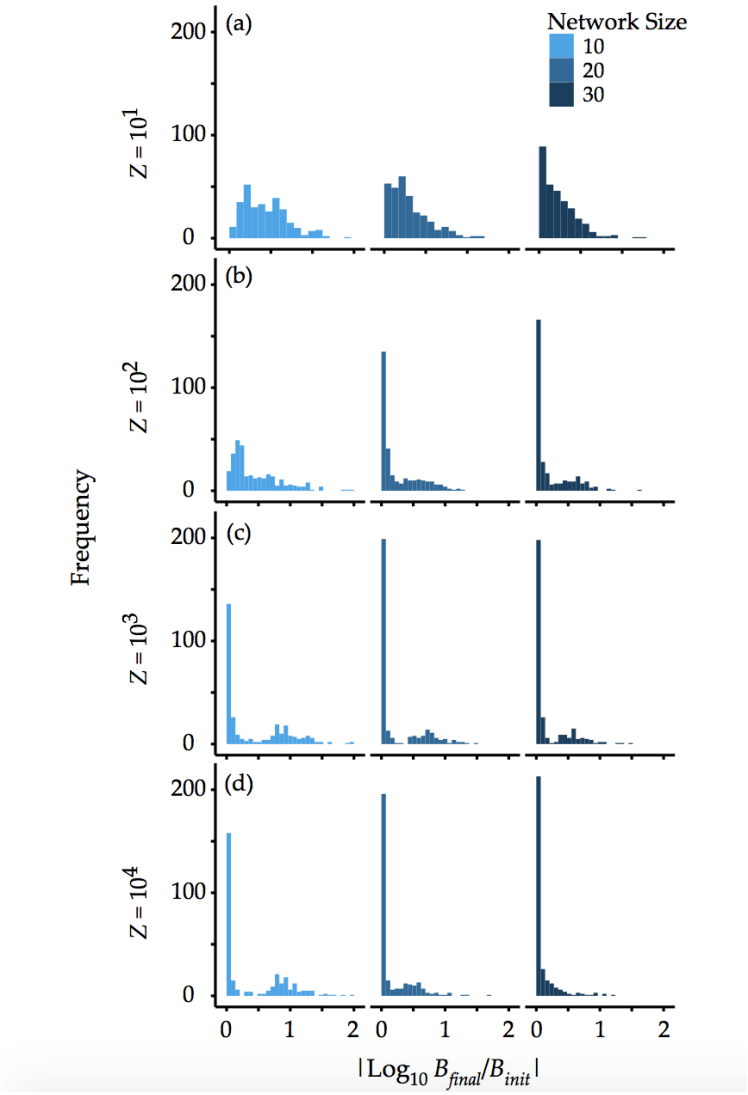


Results of 10^4 model simulations with type II functional responses, with experimental data (squares and diamonds) superimposed for comparison. Analyses of models with type II functional responses yield the same qualitative patterns presented in the main text for type I functional response models (see Fig. 2.1). Here, $P = 0.8$ and $aP = 0.4$.

Appendix C

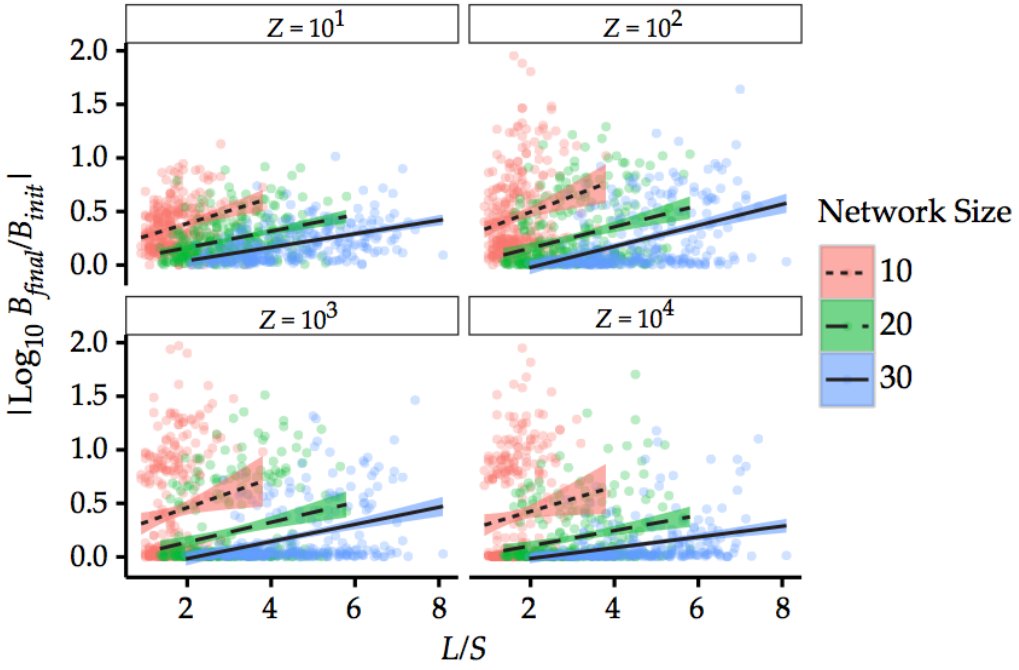
Additional Food Web Simulations

Figure C.1: Cascade magnitudes across all simulations



Results of cascade magnitudes from all simulations described in Chapter 3. System-wide $K = 10$.

Figure C.2: Cascade magnitudes as a function of covariates



Results of cascade magnitudes for all combinations of S, L/S and Z.