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An Examination of the Effects of Prey Density, Mortality, Nutrients, and Foraging Tradeoffs on a System With Inducible Defenses: An Empirical and Theoretical Approach

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AN EXAMINATION OF THE EFFECTS OF PREY DENSITY, MORTALITY, NUTRIENTS, AND FORAGING TRADEOFFS ON A SYSTEM WITH INDUCIBLE DEFENSES: AN EMPIRICAL AND THEORETICAL APPROACH

by

Benjamin C. Ralston Daniel

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ABSTRACT

An Examination of the Effects of Prey Density, Mortality, Nutrients, and Foraging Tradeoffs on a System with Inducible Defenses: an Empirical and Theoretical Approach

by

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Utah State University, 2024

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Inducible defenses are a form of phenotypic plasticity, where prey are able to express changes in morphological, behavioral, or life history traits in the face of an increased threat of predation. Different biotic and abiotic factors in the surrounding environment can influence this adaptive response to predation, which in turn can influence the strengths of the interactions between species in a community. At the top of the food chain predators are important in controlling prey density and increasing mortality of top predators can reduce the constraints on their prey leading to trophic cascades. However, adaptable prey responses to predation can lead to counterintuitive responses of the trophic levels to predator mortality. The death of predators also plays a key role in the cycling of nutrients in a system. Energy flows up the trophic levels of a food chain through consumption, but recycling of dead biomass and excretion allows for some of those resources to be reclaimed by the lowest trophic level. In this thesis, I analyze the responses and dynamic consequences of inducible defenses in a prey species, through a combination of experimental and theoretical techniques. First, I examined how prey density plays a role in modulating the prey population's induction of defenses at varying levels of

predation threat in a laboratory-based experiment using protists and flatworms. This experimental work showed that the reduction in magnitude of defense induced linked to increased prey density may be due more to a competitive reduction in size rather than a density-dependent reduction in the mechanism of defense induction. Second, I investigated how the interactive effects of inducible defenses and nutrient recycling affect how predators respond to increases in their own mortality using a theoretical model. In the same framework, I also examined how the cost of defense induction may interact with changes in the rate of nutrient recycling to alter community stability. I found that nutrient recycling led to an increased negative response of predators to their own mortality while also providing an observable increase to predator density due to bottom-up enrichment with no observable effect on system stability.

(72 pages)

PUBLIC ABSTRACT

An examination of the effects of prey density, mortality, nutrients, and foraging tradeoffs on a system with inducible defenses: an empirical and theoretical approach

Benjamin C. Ralston Daniel

To grasp the functioning and stability of ecosystems, it is important to understand species interactions. With many ecosystems becoming more imperiled from urbanization and anthropogenic influences it is important to understand ways in which species can adapt to rapid changes in their environment. Phenotypic plasticity is one such tool at nature's disposal to initiate rapid change, where species with the same genetic makeup can have different expressed traits depending on their environment. Inducible defenses are one such form of phenotypic plasticity in which prey can express different levels and forms of defense depending on the threat of predation present in their environment. In this thesis, I work to determine the mechanisms by which *P. aurelia* balance the costs and benefits of producing defenses through the manipulation of predator and prey densities to encourage a better understanding of this form of phenotypic plasticity. Using an experimental framework, I show that prey density leads to a reduction in base morphology but may be linked to increased defense induction in this protist. At the top of the food chain predators are important in controlling prey density and increasing mortality of predators can reduce the constraints on prey growth leading to a cascading effect through a food chain. However, adaptable prey responses to predation can lead to counterintuitive reactions of the trophic levels to predator mortality. Furthermore, the death of predators plays a key role in the cycling of nutrients in a system. Energy flows up the trophic levels of a food chain through consumption, but recycling of dead biomass and excretion allows for some of those resources to

be reclaimed by the lowest trophic level. In this thesis, I also investigate inducible defenses in a theoretical setting to better understand how adaptable traits may interact with nutrient recycling and foraging costs to influence responses to predator mortality and system stability. I found that nutrient recycling led to an increased negative response of predators to their own mortality while also providing an observable increase to predator density due to bottom-up. Overall, I further our understanding of inducible defenses in natural and theoretical settings.

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Chapter I: Introduction

Phenotypic plasticity, or the ability for organisms with the same genotype to produce distinct phenotypes when exposed to different environments, has been a topic of interest for decades now (Pigliucci 2005). It has gone from being considered a nuisance to those studying evolution to the center of intriguing questions about rapid adaptability and its potentially key role in responding to changing environments (Agrawal 2001). One of the key questions regarding phenotypic plasticity is what the costs of plasticity are, and how they help define the limits of the related responses (DeWitt et al. 1998).

Inducible defenses are phenotypically plastic responses to, most commonly, variable levels of predation (Harvell 1990, Tollrian and Harvell 1999). These defenses allow for prey to invest energy into defense–be it behavioral, morphological, or life-history based–only when there is a sufficient threat present (e.g. Parejko and Dodson 1991, Agrawal et al. 1999, Luquet and Tariel 2016, Reger et al. 2018). Through this flexibility in producing defenses, prey are balancing production and maintenance costs against the threat of predation. The balancing of the costs and benefits of defense induction leads to a change in the connectivity of the species in this community, where defenses reduce the direct effect of interactions between prey and predator and prey species on lower trophic levels. The increase in connections in a food web and the modulations of connectivity strength has generally been considered to have a stabilizing effect on communities (McCann et al. 1998, McCann 2000, Thébault and Loreau 2005, Vos et al. 2004), and there is theoretical evidence that supports this idea of community stabilization (Yamamichi et al. 2019). There are many ways that this cost-benefit tradeoff can be influenced by biotic and abiotic factors in an ecosystem. For instance, the availability of nutrients has the potential to change the expected response of prey to the same level of predation (McClure et al. in review). However, if we think about costs in the manner of needing energy to invest into these induced defenses, we can also see that conspecific density likely plays a role in the response to predation. Density-dependence of prey response to predators could occur either due to direct competition between a greater number of individuals reducing growth opportunities, or a lesser realized threat of predation due to a greater number of prey (e.g. safety in numbers). Reduced magnitude of defense induction at higher densities of conspecifics has

been demonstrated in several species (Tollrian et al. 2015). Density-dependent modulation of defense induction is hypothesized to stem from prey utilizing conspecific cues to perform risk assessment (Van Buskirk et al. 2011). However, there is little research on whether this density-dependent adjustment of defense induction is due to competition or prey-perceived threat of predation, and whether it affects aspects of defense induction other than the magnitude of the defense induced.

Alongside the consideration of costs to express the defense, are costs of the expressed defense on other attributes of an organism. As an example, *Paramecium aurelia*, a ciliate protist, increases its width and reduces its speed in response to predation pressure, a readily measurable form of induced defense (Hammill et al. 2010). The increased width observed in *P. aurelia* likely incurs some form of metabolic cost to generate and could be considered a cost to the phenotypic plasticity in this organism. However, we can also consider that a reduction in the speed of this organism likely reduces its foraging ability. The impacts on foraging ability as an additional cost of the induction of defense leads to the consideration of another set of species interactions, that of prey and their uptake of primary producers. The costs of defense are inextricably linked to both the ability for prey to garner energy and the benefits provided by the defense in the face of high predation risk. Therefore, changes to bottom-up and top-down effects within a community might also be influenced by the presence of an inducible defense.

Predators play a key role in any system by consuming prey from lower trophic levels and releasing nutrients back into the system through waste and decay of their biomass (e.g. Schindler and Eby 1997, Vanni, 2002). It follows that nutrient recycling and predator mortality rates would therefore play a key role in community dynamics, influencing the impact of bottom-up and top-down effects in the system. Nutrient recycling dictates the rate at which extra nutrients are returned for use by primary producers, and changes to predator mortality influence the strength of top-down control of lower trophic levels. It is also likely that predator mortality linked with nutrient recycling could lead to a reduction in top-down control and an increase in bottom-up productivity. Further, incorporating prey with an adaptable response to predation pressure could drive complex changes to the expected responses to top-down and bottom-up control. Indeed, the presence of an adaptable prey trait added to a predator-prey model has

been demonstrated to be capable of increasing predator equilibrium densities with increased predator mortality, termed a hydra effect (Abrams and Matsuda 2005, Abrams 2009). This concept has been argued to be potentially useful in the context of fisheries management, however, in the case of harvest, the nutrients at the top of the trophic level are being removed from the system entirely which may lead to different expected responses to predator mortality.

The work presented in this thesis aims to address gaps regarding the nature of the dilution effect and the theoretical implications of explicitly accounting for nutrient dynamics in systems with inducible defenses. In Chapter 2, we attempt to parse the effects of competition and density-dependent modulation of defense induction in the protist *Paramecium aurelia* in response to predation risk. A range of densities of *P. aurelia* were exposed to a gradient of predator chemical cues to observe how prey density modulates the induction of morphological defense by *P. aurelia* to the gape-limited predator *Stenostomum spp.* In Chapter 3, I used a theoretical model to investigate the interaction between nutrient recycling, foraging tradeoffs in defended morphs, and predator mortality. This involved designing a tri-trophic food chain model with explicit nutrient dynamics and an inducible defense and analyzing the system over a range of parameters both numerically and analytically. The details of the mathematical analysis can be found in the appendix of this thesis. Chapter 4 summarizes the impact of this work as well as recommendations for future research.

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Chapter II: Changes in rates of defense induction in response to conspecific and predator densities

Abstract

Inducible defenses allow prey to adaptively react to varying levels of predation threat. The main trigger of prey defense response is generally predator cues. However, for a given density of predators, prey can induce less when prey densities are larger, due to a reduction in individual predation threat with increased prey density, termed the dilution effect. Density dependence of defense induction has been observed by comparing high and low prey density treatments between high and low predator cue concentrations. *Paramecium aurelia* is a protist species known to induce morphological defenses which we expect to reduce induction rates at higher prey densities. To discern the underlying mechanism driving this density dependent reduction in defense, I measured induction levels in *P. aurelia* across gradients of prey and predator densities. Our results suggest that a competitive reduction in size is driving the reduction in the morphological defense produced by *P. aurelia* at high prey densities and that the "gap of induction", the total change in size to achieve the maximum level of induction, at a given prey density was larger at higher densities, indicating a potential attraction effect of prey density. I found similar density-dependent reductions in the magnitude of induction as seen in previous research. However, I also found a positive correlation between the gap of induction and prey density, indicating a potential combination of cost and adaptation to increased risk of predation at higher densities.

Introduction

The dynamics and species composition of ecosystems are controlled by complex interactions between biotic and abiotic factors. The biomass of the different trophic levels in an ecosystem can be thought to be controlled by both resources (bottom-up) and consumers (top-down) (Hunter and Price 1992, Power 1992). Previous research lent credit to the idea that top-down effects might be more important than bottom-up effects in determining trophic level

biomass due to the idea of trophic cascades (Paine 1980). However, it has become clearer that the direction of trophic control varies greatly with other abiotic factors (Rogers et al. 2020). Additionally, the strength of both bottom-up and top-down effects are mediated by the interactions between predators and prey. Predator-prey interactions have been studied in-depth in laboratory, field, and theoretical settings (Gause et al. 1936, Paine 1966, Rosenzweig 1971, Lubchenco and Menge 1978). The effects of predation have been shown to ripple down through food webs (top-down effects) but have also been shown to be dependent on other factors such as enrichment (bottom-up effects; Pace et al. 1999). In this study, we contribute to the growing description of how species interactions can be mediated, specifically through prey defenses and their regulation by bottom-up and top-down effects.

There are a wide range of prey responses to predation, many of which can be classified as some form of defense. There are two main forms of defense, constitutive and inducible (Garcia et al. 2021). Constitutive defenses are permanent adaptations to predation whereas induced defenses are phenotypically plastic traits that can be altered by prey in response to the threat of predation (Tollrian and Harvell 1999). Induced defenses are predicted to be selected over constitutive defenses when reliable cues for induction are available and predation is temporally or spatially variable (Harvell 1990), as the costs of expressing defenses are only incurred when a threat is present. Inducible defenses can be morphological such as trichome production on radish leaves (Agrawal et al. 1999) and neck teeth formation in some *Daphnia spp.* (Parejko and Dodson 1991), behavioral such as predator avoidance in snails (Luquet and Tariel 2016), and some can be plastic changes in life-history traits such as the modification of age and size at reproductive maturity in *Daphnia pulex* (Reger et al. 2018). However, there must also be a cost to the induction of defenses or there would be no fitness benefit associated with an uninduced state (Tollrian and Harvell 1999). This leads to a balancing of costs and benefits for prey: investing in defense induction to avoid mortality from predation but only when that investment does not hinder competitive ability to the point of exclusion. To determine this cost-benefit analysis many prey species use a measure of predation pressure, either visual or environmental cues of predator presence, in the environment to help estimate the benefits of defense induction (Schoeppner and Relyea 2009). This balancing act means that defenses are not always static,

which leads to flexible connections between species in the ecosystem.

The flexibility of these species connections can alter community stability by changing species-interaction strengths and subsequently altering community dynamics (McCann et al. 1998, McCann 2000, Verschoor et al. 2004, Thébault and Loreau 2005) Therefore, the mechanism by which defenses are induced in a system are important to understanding the stability of communities with adaptable prey responses to predation. For example, the connectivity of food webs can be altered from the top-down as prey react to higher levels of predation threat. However, it is important to note that predation threat is dependent on both predator and prey density.

In systems where predators eat one form of prey, the threat of predation on a single individual can go down as prey density increases (Bertram 1978). This is due to the dilution effect, wherein at higher prey densities the uptake of prey by predators can become saturated, and individual prey will be less at risk of predation through safety in numbers. Defense induction has also been shown to be modulated by the density of conspecifics through a reduction of predation risk, adding further complexity to the cost-benefit balancing act that is defense induction (Tollrian et al. 2015). Tollrian et al. 2015 also showed that *Daphnia* responds to conspecific cues in the environment meaning that some species of prey may be able to perform a form of risk assessment through monitoring of the ratio of conspecific and predator cues in the environment. This fits neatly into the idea of risk assessment, which was highlighted by Peacor (2003) as an important aspect of considering prey modulation of phenotype in response to predation. The idea of using information on predator density and prey density to inform plastic decisions regarding predation risk was also supported by a modulation of tadpole anti-predator behavior only to changes in the ratio of prey to predator cue, indicating a monitoring of per capita predation risk (Van Buskirk et al. 2011). So even in the case of increased predator density, the induction of defense can still be dependent on prey density, and, therefore, consideration of the lower trophic levels is also important.

There is also the consideration that intraspecific competition could lead to a reduction in defense induction, as with increasing density the costs of the induced defense could become an increasing hindrance to survival (e.g. Pettersson and Brönmark 1997, Fyda and Wiackowski

1998) There is also the potential that, given a phenotype is costly to express, under higher levels of competition it may be impossible for an individual to garner the prerequisite energy to produce said trait. Overall, this indicates that the mechanism of defense induction could be density-limited due to available resources, especially in the case of a morphological inducible defense which requires energetic investment rather than behavioral shifts.

In this article, I explore the mechanism driving density-dependent modulation of defense induction in the protist *Paramecium aurelia* which increases its width in response to predation pressure, a readily measurable form of induced defense (Hammill et al. 2010). They do so in response to predation by the gape-limited flatworm (*Stenostomum spp.*) as an increase in width increases predator handling time and a reduction in speed reduces the chances of encountering a predator. The increase in width is likely energetically costly for *P. aurelia* as seen in other ciliate protozoans (Fyda and Wiackowski 1998) while the reduction in speed may reduce the foraging ability of the ciliate. Both costs impact their competitive ability with non-induced morphs. Like other morphological, inducible defenses, this reduction in fitness should lead to a threshold of predation threat at which P. aurelia will induce (Hammill et al. 2008). Ciliates, like *P. aurelia*, are also one of the organisms in which prey density has been observed to affect the magnitude of defense induction (Tollrian et al. 2015). Previous studies also suggest that *P. aurelia* are aware of conspecific densities, altering their dispersal away from the same predation threat under different levels of conspecific density (Hammill et al. 2015). This ability to alter anti-predator responses in the face of predator cues but at varying conspecific densities is an extra layer of complexity we need to account for when investigating how inducible defenses scale up to affect population dynamics.

To explore how induction rates in *P. aurelia* are modulated by prey and predator density I measured the defense response of the protist *P. aurelia*, as the change in width, under a range of conspecific densities with different levels of predator cue present. My goal was to determine how prey density modulates the reaction norm of induction of *P. aurelia*. Specifically, is the density-dependence of the magnitude of induction separate from any density-dependent modulation of size seen in the absence of predators? My results indicated that the reduction in maximum defense induction seen in *P. aurelia* at high densities of conspecifics is related

to a density-driven reduction in size. We also found evidence for higher prey densities to be correlated with an increase in the "gap of induction", the total change in size to achieve the maximum level of induction.

Methods

Study Species

I used the freshwater ciliate *Paramecium aurelia* as the prey species due to its rapid generation time and easily quantified inducible defense. *Paramecium aurelia* can reproduce asexually, and isolated individuals can be used to form and maintain clonal populations. We used clonal lines of *P. aurelia* populations replicated from a single individual and maintained at Utah State University through monthly subsampling and relocation into autoclaved protist media. Our experiment used three clones that varied in their ability to induce morphological defenses: EV2, FD4, and P. Aur. The clonal line P. Aur. was raised from a commercially purchased population of *P. aurelia* (Carolina Biological Supply Company, Burlington, NC). Previous investigations have indicated that P. Aur induces defenses in the presence of chemical cues from predators (Hammill et al. 2023). FD4 is a clonal line stemming from one individual collected from the Logan River in Logan, Utah, USA, 41.74°N, 111.79°W, and has been observed to be consistently wider than other clones and shows minimal change in body width when exposed to predator chemical cues. The clonal line of EV2 are all individuals descendant from a single P. AUR individual that was part of a population housed with predators for 6 months where the whole community was subsampled and inoculated into new media every week. The continual subsampling applied selective pressure for EV2 to reproduce rapidly in the presence of predators to reach large population sizes before predators reach sufficient densities to drive the populations to extinction. This selection pressure to rapidly reproduce has resulted in EV2 being smaller than its P. AUR ancestral stock and less responsive to predator cues. *Stenostomum spp.* flatworms are a known predator of *P. aurelia* (Kratina et al. 2007). Populations of *Stenostomum spp.* were also maintained at Utah State University in protist media for the production of predator kairomones. Predator cues were produced by freezing *Stenostomum* at densities of 200 individuals per ml. This predator cue was thawed and used in induction experiments.

I conducted the experiments in media containing a bacterial community consisting of *Serratia fonticola* and *Bacillus subtilis* present in cultures on Carolina Protozoan Pellets (Carolina Biological Supply).

Study Design

The study included 9 different predator treatments and 7 different prey densities in a full 7x9 factorial design with 5 replicates for each of the three different clonal lines of *P. aurelia* described above. I performed the experiments in 24-well plates. I used the following prey density treatments: 1, 5, 10, 15, 20, 30, and 60 protists. I placed the protists into the same total volume of protist media (300 μg), predator kairomone solution (300 μg), and extra storage media. I used extra storage media to standardize the difference in volume, stemming from the volume needed to isolate 60 protists versus all of the lower prey density treatments. I used the following predator density treatments: 0, 1, 3, 5, 10, 15, 20, 40, and 60 flatworms (I calculated the actual volume of thawed kairomone using the 200 worms/mL ratio of the frozen predator cue solution). I assigned treatment combinations randomly over 63 wells across three 24-well plates for each replicate.

I used the same protist media across all experiments produced by dissolving 0.23 g of crushed protist pellet (Carolina Biological Supply Company, Burlington, NC) in 1 liter of Arrowhead Mountain Spring Mineral Water (USA) filtered through Melitta Super Premium Unbleached brown filters (Melitta Group, Germany). I poured the media into mason jars which I autoclaved for sterile storage. Populations of protists and *Stenostomum* were maintained using storage media at a concentration of 1 pellet to 2 liters of water.

After treatment application, I left the protists in their wells for 24 hours. I then stained the protists with 5% Lugol's solution and took photos of 3-4 randomly selected protists from each well. I then measured the length and width of the imaged protists using ImageJ (Schneider et al., 2012).

Statistical Methods

I examined the relationship between induction, prey density, and predator density using an exponential function:

$$f(x) = a(p)(1 - e^{-bx}) + c(p)$$

where (1)

$$a(p) = a + B_1 * p$$

$$c(p) = c + B_2 * p$$

$$f(x) = e^{(a+B_1*p)}(1 - e^{-e^b x}) + e^{(c+B_2*p)} \quad (2)$$

Where $f(x)$ is the measurement of interest of the protist defense, a represents the gap of induction as a linear function of prey density (p), b represents the rate of induction as predator density (x) increases, and c represents the baseline induction level as a linear function of prey density. Together a and c determine the maximum level of induction.

I further refined the model (eq. 2) such that parameters a and c are exponentially related to prey density, p. I exponentiated the relationships with prey density to ensure positive parameter values. All parameters were allowed to vary for each clone to form a hierarchical model. B_2 determines the relationship between maximum induction and the baseline morphology with prey density. For example, if B_1 is set to zero then the relationship between maximum induction and baseline morphology to prey density is only associated with the parameter c and the maximum induction is just the minimum size defined by c plus a. Any extra density-dependent effect on the magnitude of induction, likely non-competitive components of the dilution effect, would therefore be picked up by B_1. The separation of these effects can be clarified, if B_2 is set to zero. Then it is easier to see that the only relationship to prey density is one that affects only the magnitude of induction and no longer the baseline morphology because there is no relationship between c and prey density. Overall, this means that a relationship between prey density and B_2 changes the magnitude of induction alongside the baseline morphology in the

absence of predators. Alternatively, a prey density relationship to a, picked up by B_1, indicates some density-dependent modulation of the gap of induction, the distance from the baseline morphology and the maximum level of induction.

I examined the model using both the width-to-length ratio and the width as the response variable. I used width-to-length ratio data because it is a simple metric for the increase in width of protists, however, I also looked at trends in width independent of length to see if the observed patterns were truly driven by increases in width. We ran the models in a Bayesian hierarchical framework in JAGS through R using a combination of JAGS-related packages (Denwood 2016, Plummer 2003, Plummer 2023, Kellner 2024, R Core Team 2024, Su and Yajima 2024). I ran both models with three chains, one million iterations, a default burn-in rate of half of the iterations, and a thinning rate of 500.

The models were fit assuming log-normal errors. I used uninformative priors for each of the parameters and the respective variances as outlined in Table 1. I used priors with arbitrarily low and high values for the uniform distributions of variance to ensure that the model fit was not influenced by the definition of the priors. Convergence of the model was assessed through trace plot convergence, alongside parameter estimates having an $\hat{R}$ below 1.05 with effective sample sizes greater than 300.

Table 1: Table of the prior and hyper prior distributions of the exponential model.

Dispersion of error $\sim Unif(0.001, 10)$
Dispersion of parameter $\sim Unif(0.001, 5)$
Parameters $\sim N(hyperprior, \frac{1}{\text{dispersion}^2})$
Hyper priors $\sim N(0, \frac{1}{\text{dispersion}^2})$

Results

Width to Length Ratio Model

The model fit with the width-to-length ratio data converged for all parameters (Fig. 1). Both P. AUR and EV2 clones demonstrate a weakly positive relationship between prey density and the gap of induction. This is indicated by the median estimate of B_1 for the model fit for P.

AUR (0.018, 95% credible interval: -0.0009 - 0.036) and EV2 (0.017, 95% credible interval: -0.0023 - 0.032) both of which were positive (Fig. 1). Additionally, the probability of direction of the posterior distribution of B_1 for both clones was 97%, indicating the estimates for B_1 for both clones were likely different than zero. These probability of directions can be presented as two-sided p-values for the presence of effect to be $p = 0.06$ (Makowski et al. 2019). This positive relationship between a and prey density indicates an increase in the gap of induction (the gap from basal morphology to maximum induction) with increasing prey density. The posterior distribution of B_1 for FD4 was more centered on zero seen by its negative median estimate with a probability of direction of the distribution being positive at 48%, indicating a nearly 50-50 distribution around zero. The posterior distribution of B_1 for FD4 indicates a lack of a relationship between the gap of induction and prey density for this clone. The posterior distributions of B_2 are similar for all clones with 100% of the distributions being less than zero (Fig. 1). This indicates that all the clones have a negative relationship between the baseline morphology and some component of the magnitude of induction.

Looking at the model fit (Fig. 2), we can see the median estimates at zero predator density highlight the effects of the competition-driven reduction in size, seen in the reduction in the y-intercept at higher prey densities. We can see that for P. AUR and EV2 the gap of induction increases with prey density, as the size change from base morphology to maximal induction gets larger with prey denstiy. FD4 seems to have a more constant response to predation across prey densities reflecting its B_1 posterior distribution being heavily centered around zero. This effect can be seen in the raw data showing that there does seem to be a difference in the gap of induction with increasing prey density. The gap of induction, calculated as the percent difference between the mean width-to-length ratio at a predator density of 5 and a predator density of 60, at a prey density of 5 is 7.4% for P. Aur, 1.7% for FD4, and 4.7% for EV2 while at a prey density of 60 the gap of induction is 12.8% for P. Aur, 9.7% for FD4, and 8.2% for EV2. The raw data suggests this increase in the gap of induction may be present for all clones, which may disagree with my model fits due to the limited variance explained by the model (Fig. S3).

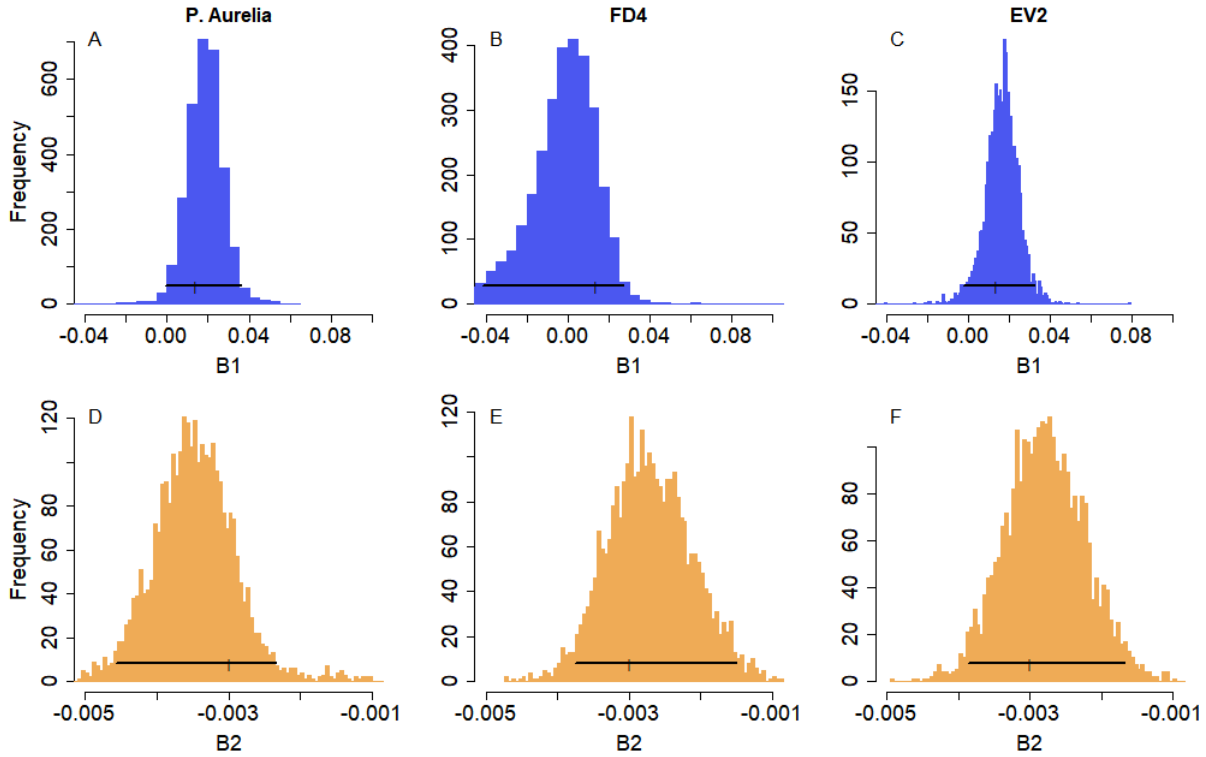

Figure 1: Shows the posterior distribution of B_1 (A-C) and B_2 (D-F) for the model fit with width-to-length ratio data collected from the clones P. AUR, FD4, and EV2. The horizontal black lines represent the 95% credible intervals of each posterior distribution and the black tick marks are the medians of the posterior distributions. Listed here are the medians and 95% credible intervals for the B_1 posterior distributions: P. AUR = 0.018 (-0.00093 - 0.036), FD4 = -0.0008 (-0.042 - 0.027), and EV2 = 0.017 (-0.0023 - 0.032). Listed here are the medians and 95% credible intervals for the B_2 posterior distributions: P. AUR = -0.0035 (-0.0046 - -0.0023), FD4 = -0.0027 (-0.0038 - -0.0015), and EV2 = -0.0028 (-0.0039 - -0.0017)

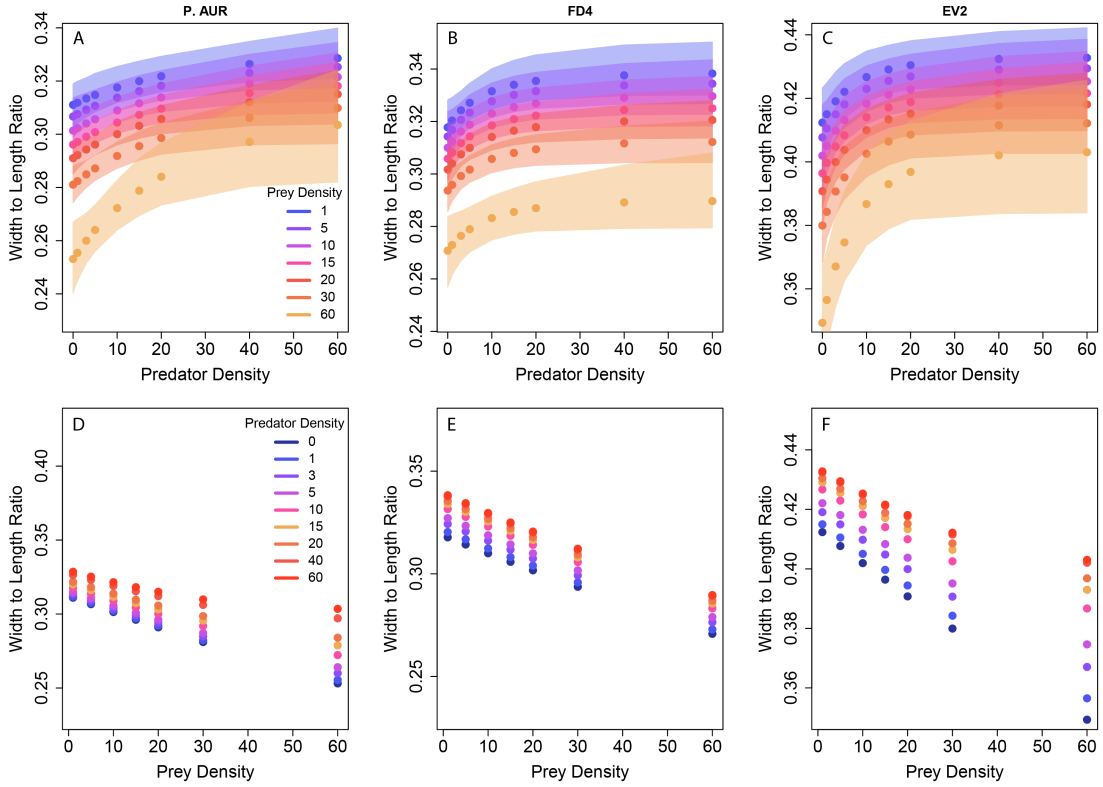

Figure 2: A-C show the median model estimate of width-to-length ratio at a given predator density colored by prey density. The polygons are the 95% intervals. D-F show the median estimates of width-to-length ratio at a given prey density colored by predator density. Column 1- P. AUR Column 2- FD4 and Column 3- EV2.

Width Model

The model fit with the width data converged for all parameters (Fig. S2). The posterior distribution of B_1 for each clone is more centered around zero as seen by the smaller yet still positive median values for P. AUR and EV2 (Fig. 3). The probability of direction for P. AUR's B_1 posterior distribution is 95% while the probability for EV2's posterior distribution of B_1 is now 78%. This indicates an even weaker relationship to the gap of induction with raw width. Again, all clones had similar B_2 posterior distributions, indicating a negative relationship between prey density and basal morphology and magnitude of induction (Fig. 3).

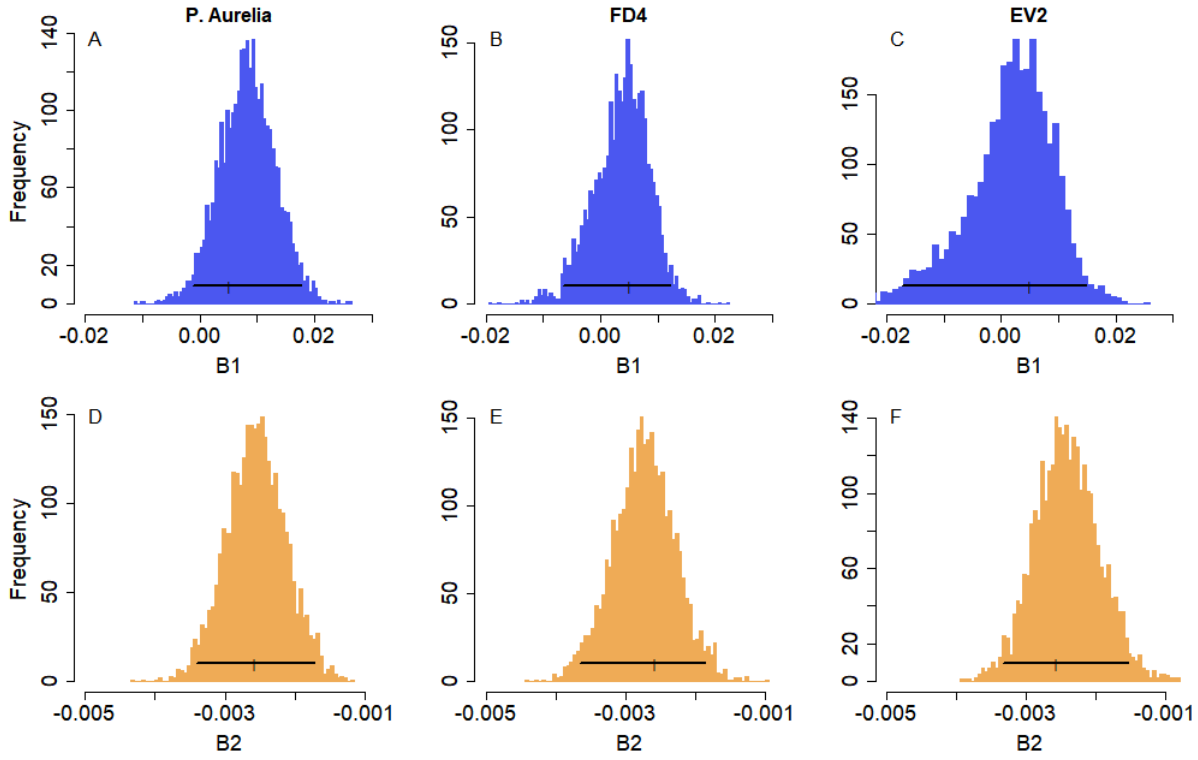

Figure 3: Shows the posterior distribution of B_1 (A-C) and B_2 (D-F) for the model fit with width data collected from the clones P. AUR, FD4, and EV2. The horizontal black lines represent the 95% credible intervals of each posterior distribution and the black tick marks are the medians of the posterior distributions. Listed here are the medians and 95% credible intervals for the B_1 posterior distributions: P. AUR = 0.0083 (-0.0012 - 0.018), FD4 = 0.0041 (-0.0066 - 0.012), and EV2 = 0.0023 (-0.017 - 0.015). Listed here are the medians and 95% credible intervals for the B_2 posterior distributions: P. AUR = -0.0026 (-0.0034 - -0.0017), FD4 = -0.0027 (-0.0037 - -0.0019), and EV2 = -0.0024 (-0.0033 - -0.0015).

Increasing prey density results in reduced prey widths across all predator densities, and prey density has little impact on the gap of induction for all three clones (Figure 4).

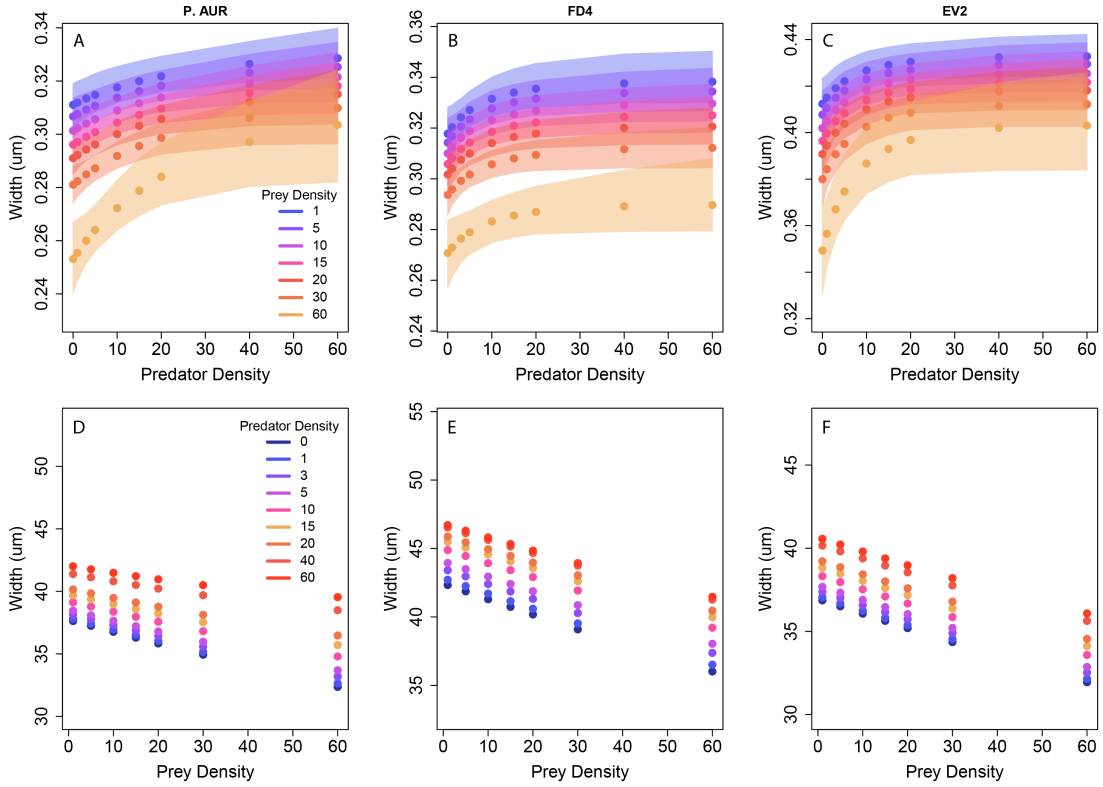

Figure 4: A-C show the median model estimate of width at a given predator density colored by prey density. The polygons are the 95% intervals. D-F show the median estimates of width at a given prey density colored by predator density. Column 1- P. AUR Column 2- FD4 and Column 3- EV2.

Discussion

My goal was to try and interpret the density-dependent mechanisms driving defense induction in the protist *Paramecium aurelia*. My model suggests similar trends in a reduction in the magnitude of defense induction at higher levels of prey density, similar to the density-dependence of induction observed by Tollrian et al. (2015). This is highlighted by the negative relationship, B_2, between prey density and the parameter adjusting the basal morphology and part of the maximum induction of the induction curve, c. My model also showed a weak positive relationship between prey density and the gap of induction, suggesting that these protists may induce more at higher prey densities. This observation is also supported by the raw data indicating that the percent difference in width-to-length ratio of this gap of induction does appear to get larger with prey density. The positive relationship between prey density and the gap of induction could be linked to attractive effects of prey density on predators, wherein prey density increases the risk of predation (Bertram 1978). Overall, I observed similar patterns to previous research on density-dependence of defense induction and noted that the gap of induction of *P. aurelia* indicates a potential attractive effect of prey density on predation threat.

The reduction in the magnitude of defense seen in this experiment was also shown to be linked to the basal morphology of the protists. Like previous studies on density-dependent modulation of defense induction, I saw that defense induction was dependent on both predator and prey densities (Wiackowski and Staronska 1999, Van Buskirk et al. 2011, Tollrian et al. 2015). These previous studies indicated that some of the species were able to respond to conspecific cues to accurately estimate the threat of predation and balance the costs of defense induction. My model tries to parse apart two portions of the density-dependent response to predation with a relationship to just the magnitude of defense and a relationship to the magnitude of induction as well as the basal morphology of the protist, and this is where our findings differ from the literature. I saw that the reduction in the magnitude of defense observed in *P. aurelia* was also linked to reductions in the base morphology of the protist, indicating that the reduction in defense could be linked to a competitive reduction in size. This was expected as all prey densities were given the same volume and concentration of growth media, and it is known that competition can drive organisms to have smaller body sizes because they require fewer

nutrients for maintenance, making them less susceptible to starvation and reducing the impact of competition (Peters 1986). This also leads to a reduction in growth which has also been seen as a cost to the induction of defense seen in ciliate protists (Fyda and Wiackowski 1998). However, there is also the possibility of space-limited competition, or competition for some other unseen resource such as oxygen in driving the reduction in size seen in this experiment. My findings also offer a potential method for identifying the dilution effect and attraction effect from the induction curve in prey with inducible defenses.

The weak positive relationship of prey density to the gap of induction highlights an interesting aspect of analyzing the curve of induction. My model shows that it is possible to parse apart the density-dependent aspects of defense induction that are dependent and independent of base morphology. In this experiment, I saw that there is a reduction of defense correlated with reduced size in the absence of predation threat. However, I also saw a weak positive trend between the gap of induction and conspecific density. Even if this effect is weak it still indicates an aspect of the curve of induction that could be linked to important effects of density-dependent predation that have been pointed out in the literature. The positive relationship I observed could be linked to an attractive effect of prey density on predators, wherein increased prey density can lead to an increased risk of predation. The correlation between prey density and increased defense could indicate that *P. aurelia* evolved alongside *Stenostomum spp.* with an attractive effect of prey density on flatworm predation rate, leading to a greater investment in defense at higher prey densities. This highlights the complicated ways in which conspecific density plays a role in the plastic modulation of morphology and inducible defenses. At the same time, it highlights that the gap of induction assessed from an induction curve may be able to inform us about the relationship between prey density and predation threat if the effects of competition can be accounted for.

We also saw that the effect of predator density was less pronounced than we would have expected. However, the model also suggests that the predator density that drives defense induction in these protists is very low. This low level of predator density required to induce defenses may stem from the high risk of mortality that these predators present (Hammill et al. 2023). The high risk posed by predation means that there is a large benefit of defense induction even

at low predator densities and would explain the immediate response even to predator chemical cues. In future experiments, using a larger range of low-level predator cues may reveal a better understanding of the relationship between induction and predator density in this system. It could be that these protists simply switch this defense on and off in response to the presence of any predator and the defense then scales with increasing predation. We may expect this relationship if the costs of inducing defenses are relatively low compared to the risk of predation. There is also the consideration that perceived predation risk may scale with prey density as well, considering the positive relationship between the gap of induction and prey density seen in this experiment. Alongside more low-level predator density treatments, I would consider having the upper end of prey densities be near 40. This should allow holding the total volume constant across all treatments to be easier which was likely a large source of observation error in this experiment. I was still able to detect some interesting patterns, but the potential influence of observation error meant the model was only able to explain a small portion of the variance (Fig S3).

In conclusion, I saw a density-dependent reduction in the magnitude of defense induction. However, my model linked this more to a reduction in size due to assumed competition. Alongside this relationship, I also saw a weak positive correlation between prey density and the gap of induction, potentially indicating prey density is associated with an increased risk of predation for *P. aurelia*. In the future, I should look at smaller concentrations of predator cues to try and determine if the response to predation is presence-absence in this species or a more graded response with an inflection point that could also be associated with conspecific density. I would also like to observe the relationship between prey density and the gap of induction with no sources of nutrient competition to see if the density dependence does stem from conspecific cues alone. Overall, *P. aurelia* requires more study to fully understand the nature of how conspecific density changes its adaptive response to predation.

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Supplement

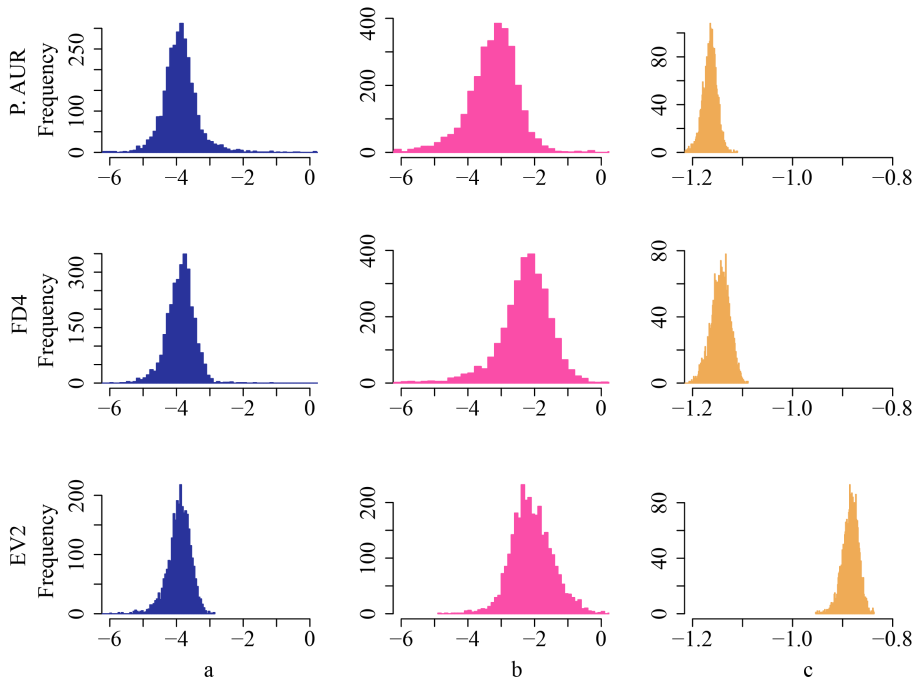

Figure 5: The posterior distributions of a, b, and c for the model fit with the width-to-length data. Row 1 are the posterior distributions for the clone P. AUR, row 2 are the posterior distributions for FD4, and row 3 are the posterior distributions for EV2.

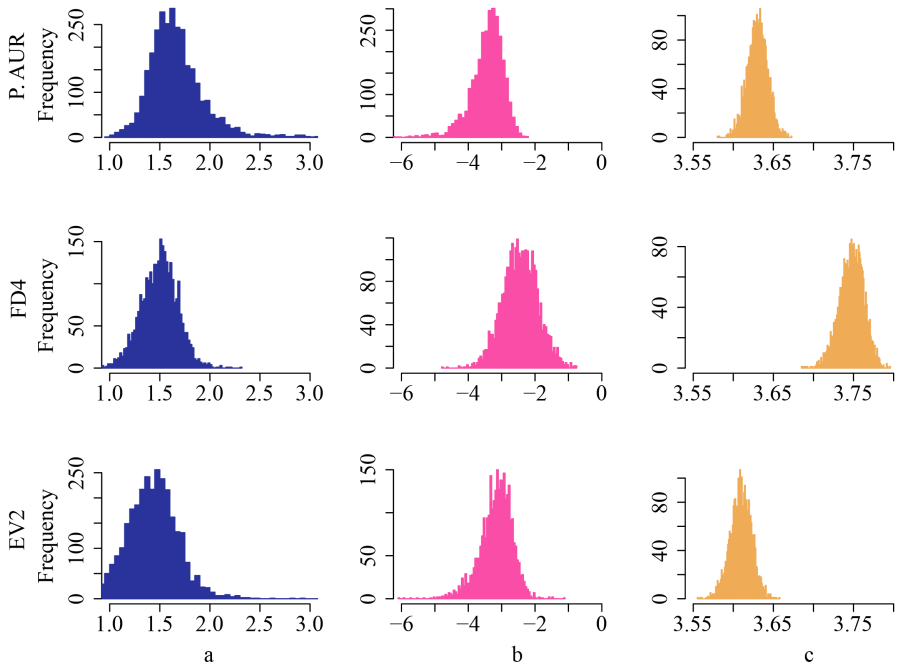

Figure 6: The posterior distributions of a, b, and c for the model fit with the width data. Row 1 are the posterior distributions for the clone P. AUR, row 2 are the posterior distributions for FD4, and row 3 are the posterior distributions for EV2.

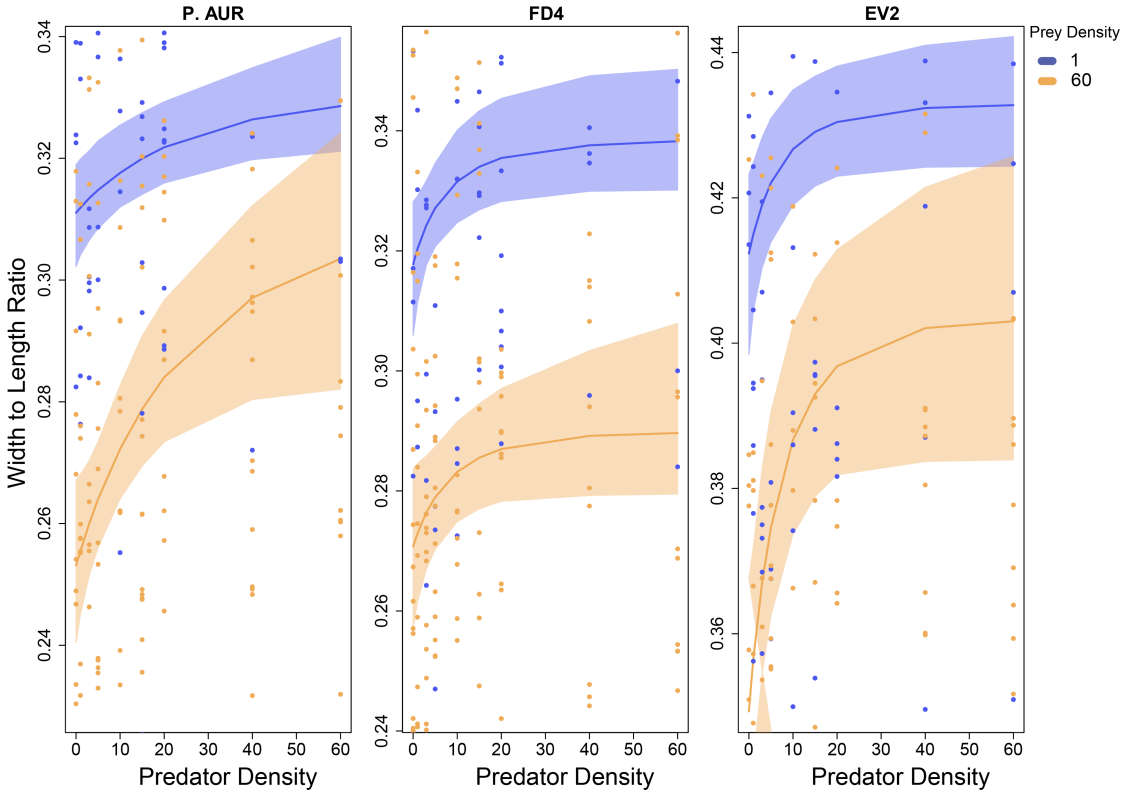

Figure 7: Shows the raw data alongside the model fits for the lowest and highest prey density treatments for the entire gradient of predator treatments. Highlights the spread of the raw data, represented by the points, alongside the median predicted curve, represented by the lines. The polygons are the 95% credible intervals.

Chapter III: Impacts of nutrient recycling and foraging trade-offs in a tri-trophic food chain model with inducible defenses

Abstract

Predators are key players in communities: they can suppress prey species through top-down control and can provide key sources of nutrition for humans accumulating biomass up the trophic ladder. Using a tri-trophic mathematical model, I explored how inducible prey defenses and nutrient recycling jointly affect responses to predator mortality and community stability. My work agrees with prior models' expectations for adaptive responses to predator mortality, however, it also highlights the potential for nutrient recycling to shift and intensify the responses. I also observed that the stabilizing effects of inducible defenses appears to be unaffected by nutrient recycling rates.

Introduction

The dynamics and species composition of ecosystems are controlled by complex interactions between biotic and abiotic factors. The biomass of the different trophic levels in an ecosystem can be thought to be controlled by both resources (bottom-up) and consumers (top-down) (Hunter & Price, 1992; Power 1992). The strength of both bottom-up and top-down effects are mediated by the interactions between predators and prey. The effects of predation have been shown to ripple down through food webs (top-down effects) but have also been shown to be dependent on other factors such as enrichment (bottom-up effects; Pace et al., 1999). Therefore, any modulation to the interactions between species is important in understanding the cascading nature of top-down and bottom-up effects.

Prey adaptation, specifically induced prey defenses, can alter top-down and bottom-up effects observed in communities. Induced defenses are phenotypically plastic traits that can be altered by prey in response to the threat of predation (Tollrian and Harvell, 1999). Inducible defenses can be morphological, behavioral, or changes to life-history traits (see Parejko and Dodson, 1991; Luquet and Tariel, 2016; Reger et al., 2018). Inducible defenses are consid-

ered to have a stabilizing effect on natural systems (Verschoor et al., 2004). However, the effects stemming from adaptable prey defenses can lead to harder-to-predict interactions between species. For instance, Abrams and Vos (2003) showed that adding both density dependence and an adaptive prey response led to multiple indeterminate effects on trophic level equilibrium density in response to increasing mortality at each level. Additionally, Abrams and Matsuda (2005) showed that in a predator-prey system with two prey species of differing susceptibility to predation, the associated adaptive change in prey population defenses through the fluxes in the abundance of the two prey allows for an increase in predator harvesting to drive an increase in predator equilibrium population, termed the "hydra effect". It seems important to consider what other aspects of a community might influence these responses to top-down responses to increased predator mortality

An important attribute of inducible defenses to consider in any model of inducible defenses is the cost of induction on the fitness of the defended morph, which is usually coded into the equations of the model to represent a decrease in the growth rate of defended individuals. Increasing the cost of induction will have a direct reduction on the population of induced prey. However, how this tradeoff interacts with other aspects of the system may be important.

Nutrient cycling is critically important to the function of natural systems, yet can be difficult to manipulate and measure. The recycling of nutrients can lead to variable enrichment in ecosystems and is an important aspect of terrestrial and aquatic ecosystems(e.g. Fenchel, 2008; Prescott et al., 1993; Vanni, 2002). Nutrient recycling also serves as an important link between terrestrial and aquatic ecosystems (e.g. Cummins et al., 1989; Rex and Petticrew, 2008). Theory on how nutrient recycling affects stability has shown that recycling has the potential to be both stabilizing and destabilizing to population dynamics (Bandyopadhyay et al., 2004; Lu, 2004; Ruan, 2001; Ruan and He, 1998). Alongside the general lack of knowledge, there has been little investigation into how nutrient recycling might change the balance of costs and benefits of the mechanisms of inducible defenses. When we consider that top predators in aquatic ecosystems provide an important ecosystem service in the form of nutrients recycled back into the system from their excretion, egestion, and eventual mortality (Schindler and Eby, 1997; Vanni, 2002), and adaptive prey responses to predation can lead to counterintuitive responses

to predator mortality (Abrams and Matsuda, 2005), including nutrient recycling in a model with an inducible defense could reveal important interactions between recycling, adaptable prey responses, and predator mortality. Adding nutrient recycling to a model of inducible defenses also adds density-dependent structure to bottom-up effects. All of these aspects add poorly understood interactions with inducible defenses.

To explore how inducible defenses and nutrient recycling jointly affect top-down and bottom-up effects, I analyze a tri-trophic model where the middle species (the prey) has an inducible defense. Using the model, I assess how top-down responses to increased predator mortality and community stability are affected by nutrient recycling and inducible defenses in the prey. I found that increased nutrient recycling rates pushed the community towards bistability, that the cost of induction tended to reduce the area of bistability generated by increased nutrient recycling, and faster induction rates also reduced the area of bistability.

Methods

Model Overview

I model a system with three trophic levels with inducible defenses in one trophic level and nutrient recycling (Fig. 8 and Table 2). The first trophic level represents primary producers that uptake nutrients from the system. The second trophic level represents the prey species which can induce a defense. I am assuming that there are only two morphs either defended or undefended and that defended morphs benefit from lower predation rates, but the defense comes at the cost of reduced foraging ability. The third trophic level represents the top predator of the system. My model also contains an explicitly accounted for growth-limiting nutrient. I will refer to this growth-limiting nutrient as nutrients from here on. The nutrients have a constant input from outside the system and a proportion of nutrients are recycled from the mortality of the prey and predator. This model structure closely represents experimental approaches to studying inducible defenses (e.g. Wiackowski and Staronska, 1999; Hammill et al., 2023, and others) which should leave room for future work, either in the form of testing model predictions or parameterizing portions of the model with biological data.

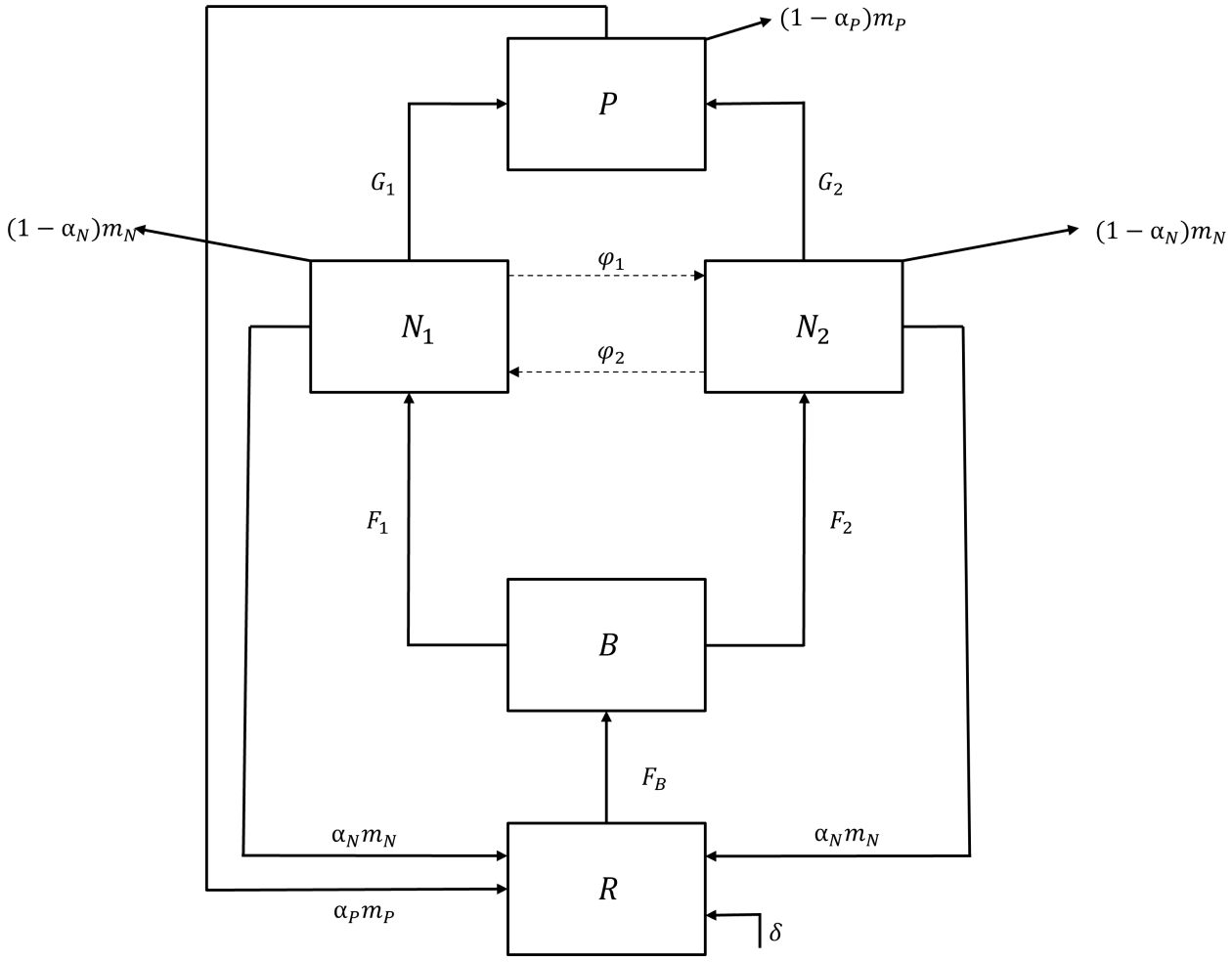

Figure 8: Diagram of Model: (1) Squares represent state variables, where R is the density of a limiting resource, B is the density of the primary producer, N_1 is the density of prey without induced defenses, N_2 is the density of prey with induced defenses, and P is the density of the predator. (2) Solid arrows denote the flow of matter: this includes matter transfer due to consumption and decomposition, and matter loss due to mortality and removal. (3) Dashed arrows denote trait change between the induced and uninduced populations. (4) Each arrow is labeled with its respective per capita rate of change.

Table 2: Definitions of model variables and parameters

Switching Function and Recycling Model	
Variables	
R	The density of a growth-limiting nutrient
B	The density of primary producers
N_i	The density of prey, where $i \in [1,2]$, and $i = 1$ refers to the uninduced morph and $i = 2$ refers to the induced morph
P	The density of the top predator
N	The density of total prey
γ	The proportion of total prey that are defended
Parameters	
δ	The constant inflow of limiting nutrient
m_i	Per capita mortality rates for species i
α_i	The proportion of nutrients recycled from species i
Functions	
F_B	Per capita uptake rate of limiting nutrients by primary producer
F_i	Prey per capita consumption rate of primary producers by prey type i, where $i \in [1,2]$, and $i = 1$ refers to the uninduced morph and $i = 2$ refers to the induced morph
φ_1	Per capita rate of induction
φ_2	Per capita rate of loss of induction
G_i	The functional response of predator on prey morph i, where $i \in [1,2]$, and $i = 1$ refers to the uninduced morph and $i = 2$ refers to the induced morph
Default Parameter Values	
$e_B = 2, e_{N1} = 0.8, e_{N2} = 0.8, e_P = 0.3, \delta = 10, m_N = 0.5, m_P = 0.4, \varepsilon = 2,$ $\rho = 0.2, \lambda = 1, c_0 = 0.3, c_1 = 0.05, h_0 = 0.24, h_N = 0.21, a_{N1} = 2, a_{N2} = 1, b = 0.2$	

My model is described by the following set of differential equations:

$$
\begin{aligned}
\frac{dR}{dt} &= \overbrace{\delta}^{\text{nutrient in-flow}} + \overbrace{\alpha_N m_N N_1 + \alpha_N m_N N_2 + \alpha_P m_P P}^{\text{recycled nutrients}} - \overbrace{F_B(R)B}^{\text{consumption}} \\
\frac{dB}{dt} &= \overbrace{e_B F_B(R)B}^{\text{growth}} - \overbrace{F_1(B)N_1 - F_2(B)N_2}^{\text{consumption}} \\
\frac{dN_1}{dt} &= \overbrace{e_{N_1} F_1(B)N_1}^{\text{growth}} - \overbrace{G_1(N_1,N_2)P}^{\text{consumption}} - \overbrace{m_n N_1}^{\text{mortality}} + \overbrace{\varepsilon[N_2\varphi_2(P) - N_1\varphi_1(P)]}^{\text{trait change}} \\
\frac{dN_2}{dt} &= e_{N_2} F_2(B)N_2 - G_2(N_2,N_1)P - m_n N_2 + \varepsilon[N_1\varphi_1(P) - N_2\varphi_2(P)] \\
\frac{dP}{dt} &= \overbrace{P e_P[G_1(N_1,N_2,P) + G_2(N_2,N_1,P)]}^{\text{growth}} - \overbrace{m_P P}^{\text{mortality}}
\end{aligned}
\tag{3}
$$

where,

$$
G_i(N_1,N_2) = \frac{(c_0 + c_i)N_i}{(1 + bP + \sum_{j=1}^{2} h_0(c_0 + C_j)N_j} \tag{4}
$$

$$
F_i(B) = \frac{a_{N_i} B}{1 + a_{N_i} h_N B} \tag{5}
$$

$$
\begin{aligned}
\varphi_1(P) &= 1 - \frac{1}{1 + \rho P^{\lambda}} \\
\varphi_2(P) &= \frac{1}{1 + \rho P^{\lambda}}
\end{aligned}
\tag{6}
$$

$\frac{dR}{dt}$ is the equation defining the rate of change in the density of the nutrients in the system. It is defined by inputs from a constant inflow of nutrients (δ) and a proportion (α_i) of nutrients recycled from the per capita death rate (m_i) of the prey and predator trophic levels and outputs from consumption by primary producers ($F_B(R)B$). The per capita uptake rate $F_B(R)$ is left undefined as it is later simplified out of the model.

$\frac{dB}{dt}$ is the equation defining the rate of change in the density of primary producers in the system. The growth of primary producers is defined by a conversion (e_B) of an uptake rate of nutrients ($F(R)$). The consumption of primary producers is defined by a similar uptake rate

by the prey morphs ($F_1(B)$ and $F_2(B)$ where, because I am assuming, some cost to fitness due to defense induction in the form of reduced growth rates for induced prey, $F_2(B) < F_1(B)$. For each prey morph, the per capita consumption rate is defined by Eq. (5) where a_{N_i} is the encounter rate and h_N is the handling time.

$\frac{dN_i}{dt}$ are the equations defining the rate of change in the density of the prey presenting a specific morph of defense, where N_1 refers to the uninduced morph and N_2 refers to the induced morph. These equations follow the same general structure of growth, consumption, mortality, and trait change. Growth is just the conversion (e_{N_i}) of the consumption of primary producers ($F_{N_i}(B)$) into reproductive output where, as defined above, $F_2(B) < F_1(B)$ due to the costs of defense induction. Consumption of prey is also differential based on defense induction where the consumption of defended prey is less than the consumption of undefended prey, ($G_2(N_2,N_1,P) < G_1(N_1,N_2,P)$). I assume the predator has a Beddington-DeAngelis functional response. Thus, for each morph, the per predator predation rate is Eq. (4) where $c_0 + c_i$ is the encounter rate for morph i, h is the handling time (assumed to be the same for both morphs), and b is the interference parameter. I also include a per capita mortality rate (m_N) that is the same between defended and undefended morphs. Lastly, I model the rates of induction and loss of induction using switching functions (Yamamichi et al., 2019), Eq. (6) where φ_i is the probability an individual switches to phenotype i, ε is the maximum rate of switching, ρ defines the density at which the rate is half of its maximum, and λ is a shape parameter where larger values cause a more step-like function.

For inducible defenses, there are three main choices of model concerning the form of trait change: switching function (SF) models, fitness gradient models, and optimal trait models (Yamamichi et al., 2019). I use an SF model because they can produce sigmoidal responses of defense induction to predator density, similar to the induction threshold response curves produced by morphological inducible defenses (Hammill et al., 2008; Yamamichi et al., 2019). Specifically, SF models have a set of equations of predator density which define the probability that a prey expressing one phenotype switches to another. SF models tend to stabilize predator-prey population dynamics (Yamamichi et al., 2019), and they have been used in previous studies (Abrams and Matsuda, 2005; Yamamichi et al., 2011).

To simplify the model analysis, we assumed nutrient dynamics are much faster than the changes in densities of the three species. This means that resource density is at a quasi-steady state defined by $\frac{dR}{dt} = 0$. Solving that equation for $BF_B(R)$ and substituting it back into $\frac{dB}{dt}$ yields a system with one reduced dimension. I then convert the model from a form that tracks the densities of each prey phenotype (N_i) to a form that tracks total prey density ($N = N_1 + N_2$) and the proportion of defended prey ($\gamma = N_2/N$). This transforms the discrete trait model Eq. (3) into a continuous trait model; the derivation is identical to that in Cortez (2011) and Cortez et al. (submitted). The advantage of this approach is that I reduce the dimensions of the model down to the biotic components which provides a framework for easier analysis. The formulation of the model into a continuous trait form also allows for easier analysis by separating the density and trait effects into separate equations.

The converted model is then represented by the equations:

$$\begin{aligned}
\frac{dB}{dt} &= e_B\Big(\overbrace{\delta}^{\text{influx of nutrients}} + \overbrace{\alpha_N m_N(1-\gamma)N + \alpha_N m_N \gamma N + \alpha_P m_P P}^{\text{recycled nutrients}}\Big) - \overbrace{F_1(B)(1-\gamma)N - F_2(B)\gamma N}^{\text{consumption}} \\
\frac{dN}{dt} &= \overbrace{e_{N_1}F_1(B)(1-\gamma)N + e_{N_2}F_2(B)\gamma N}^{\text{growth}} - \overbrace{G_1(N,P)P - G_2(N,P)P}^{\text{consumption}} - \overbrace{m_N N}^{\text{mortality}} \\
\frac{d\gamma}{dt} &= \overbrace{(1-\gamma)\varepsilon\varphi_1(P) - \gamma\varepsilon\varphi_2(P)}^{\text{phenotypic plasticity}} + \overbrace{\gamma(1-\gamma)[(e_{N_2}F_2(B) - G_2(N,P)P) - (e_{N_1}F_1(B) - G_1(N,P)P)]}^{\text{phenotypic sorting}} \\
\frac{dP}{dt} &= \overbrace{Pe_P[G_1(N,P) + G_2(N,P)]}^{\text{growth}} - \overbrace{m_P P}^{\text{mortality}}
\end{aligned} \tag{7}$$

where,

$$\begin{aligned}
F_i(B) &= \frac{a_{N_i}B}{1 + a_{N_i}h_N B} \\
G_1(N,P) &= \frac{(c_0 + c_1)(1-\gamma)N}{1 + bP + h_0(c_0 + c_1)(1-\gamma)N + h_0 c_0 \gamma N} \\
G_2(N,P) &= \frac{c_0 \gamma N}{1 + bP + h_0(c_0 + c_1)(1-\gamma)N + h_0 c_0 \gamma N}
\end{aligned} \tag{8}$$

The equations $\frac{dB}{dt}$ and $\frac{dP}{dt}$ can be interpreted the same as in the discrete trait model. The

equation $\frac{dN}{dt}$ can now be interpreted as the change in density of the combined prey population, both induced and uninduced morphs. The pieces $\frac{dN}{dt}$ are mainly the same just a combination of the two equations from the discrete trait model without the switching function. The first two terms of the trait equation describe the change in the proportion of defended individuals due to induction and the loss of induction or phenotypic plasticity. The latter two terms in the trait equation describe the effects of phenotypic sorting, i.e. changes due to differences in the reproduction and predation rates of the two phenotypes. The phenotypic sorting term isolates the individual fitness gradient, which represents changes in the proportion of defended individuals due to births and deaths of each prey morph.

Following Cortez (2011) and Cortez et al. (submitted), I assume the phenotypic sorting terms are negligibly small relative to the phenotypic plasticity terms. This is because I am focusing on systems where induction and loss of induction rates are fast relative to the generation time of the prey; mathematically, this corresponds to ε being relatively large and the phenotypic sorting term being relatively small. The benefit of this assumption becomes obvious in my analysis wherein partial differentiation of the trait change equation solves to zero in the absence of the phenotypic selection terms.

Jacobian

The Jacobian of a set of differential equations is the matrix of all of the system's first-order partial derivatives. The Jacobian for Eq. (7) has the form:

$$J = \begin{bmatrix} \frac{\partial}{\partial B}(\frac{dB}{dt}) & \frac{\partial}{\partial N}(\frac{dB}{dt}) & \frac{\partial}{\partial P}(\frac{dB}{dt}) & \frac{\partial}{\partial \gamma}(\frac{dB}{dt}) \\ \frac{\partial}{\partial B}(\frac{dN}{dt}) & \frac{\partial}{\partial N}(\frac{dN}{dt}) & \frac{\partial}{\partial P}(\frac{dN}{dt}) & \frac{\partial}{\partial \gamma}(\frac{dN}{dt}) \\ \frac{\partial}{\partial B}(\frac{dP}{dt}) & \frac{\partial}{\partial N}(\frac{dP}{dt}) & \frac{\partial}{\partial P}(\frac{dP}{dt}) & \frac{\partial}{\partial \gamma}(\frac{dP}{dt}) \\ \frac{\partial}{\partial B}(\frac{d\gamma}{dt}) & \frac{\partial}{\partial N}(\frac{d\gamma}{dt}) & \frac{\partial}{\partial P}(\frac{d\gamma}{dt}) & \frac{\partial}{\partial \gamma}(\frac{d\gamma}{dt}) \end{bmatrix} = \begin{bmatrix} - & - & + & + \\ + & + & - & \pm \\ 0 & + & - & - \\ 0 & 0 & + & - \end{bmatrix} \tag{9}$$

Each entry of the Jacobian defines how a change in one variable affects the dynamics of another variable. The solution of the signs of the Jacobian entries as seen on the right-hand side of Eq. (9) can be found in the appendix. These signs can be interpreted as the different influences of density on density, density on trait, trait on density, and trait on trait effects. The

interactions between species are summarized by J_{11}, J_{12}, J_{13}, J_{21}, J_{22}, J_{23}, J_{31}, J_{32}, and J_{33}. Changes in density also feed into changing the proportion of individuals expressing defenses defined by the trait equation and summarized by J_{41}, J_{42}, and J_{42}. J_{41} and J_{42} simplify to zero due to our assumption of phenotypic selection being negligible relative to the trait change of the inducible defense because without phenotypic selection the only variables informing induction are predator density (P) and the proportion of prey expressing defenses (γ). The effects of the change in the trait in the prey population affect both growth rates (defined by J_{14}, J_{24}, and J_{34}) and the dynamics of the trait itself (J_{44}). The structure of the Jacobian allows us to decompose the responses to predator mortality and equilibrium stability into effects of densities on density dynamics, densities on trait dynamics, trait on density dynamics, and trait on trait dynamics.

Analysis

The analysis of the model used to answer my first question focused on equilibrium stability and the responses of the trophic levels at equilibrium to increased mortality. The analysis included mathematical techniques to analyze the expected response of state variable density at a stable equilibrium to changes in top-predator mortality and numerical simulation to provide observations of the system response at equilibrium to varying predator mortality rates. All calculations were done in Maple (Maplesoft, 2023); the formulas are presented in the appendices.

For the analytical techniques, I evaluated the Jacobian of my system assuming the system was at a stable equilibrium. I used the expected signs of the Jacobian to evaluate the response of equilibrium densities of each state variable in response to increasing predator mortality. This was done using the partial derivative of the density of each state variable at equilibrium with respect to predator mortality (m_P) shown below for the predator (Eq. (10)). This partial derivative was also calculated for each of the other state variables (appendix S2). The expected responses to predator mortality in the absence of adaptation and nutrient recycling were determined for the system using each state variable's respective analog to Eq. (11) as this was the only part of the partial derivative with a potentially indeterminate sign. The determinants were then evaluated for the effects of trait change and nutrient recycling.

$$\frac{\partial P^*}{\partial m_P} = \underbrace{\frac{|J^{[3,3]}|}{|J|}}_{+} \overbrace{(-1)^6}^{+} \overbrace{(-\frac{\partial P}{\partial m_P})}^{+} \tag{10}$$

where,

$$J^{3,3} = \begin{vmatrix} \frac{\partial \dot{B}}{\partial B} & \frac{\partial \dot{B}}{\partial N} & \frac{\partial \dot{B}}{\partial \gamma} \\ \frac{\partial \dot{N}}{\partial B} & \frac{\partial \dot{N}}{\partial N} & \frac{\partial \dot{N}}{\partial \gamma} \\ 0 & 0 & \frac{\partial \dot{\gamma}}{\partial \gamma} \end{vmatrix} = \frac{\partial \dot{\gamma}}{\partial \gamma}(\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial N} - \frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial B}) \tag{11}$$

To investigate the effects of recycling and foraging rate on system stability, we used numerical analysis to investigate the type of stability of the system at a range of parameter values for predator mortality (m_P), recycling (α), and maximum uptake rate of the defended morph (a_{N2}). I used the Jacobian to compute stability via eigenvalues (Yodzis, 1981). I also investigated system stability under different rates of adaptation, ε, to further understand how the adaptability of the prey related to the parameters of interest. All calculations were performed in Maple (Maplesoft, 2023).

My analytical predictions hold for all areas of biologically relevant parameter space. To illustrate the dynamics, I use the default parameter values listed in Table 2 for figures with relevant changes described in the figure captions.

Results

Responses to Predator Mortality

My analysis resulted in the following equations of local sensitivity of the trophic level densities at equilibrium to predator mortality (appendix S2),

$$\frac{\partial B^*}{\partial m_P} = \overbrace{\frac{(-1)^4}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{+} \left(\overbrace{-\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial \gamma}}^{\pm} + \overbrace{\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{N}}{\partial N}\frac{\partial \dot{B}}{\partial \gamma}}^{+} + \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial P}}^{-} - \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial P}\frac{\partial \dot{N}}{\partial N}}^{-} \right) \tag{12}$$

$$\frac{\partial N^*}{\partial m_P} = \overbrace{\frac{(-1)^5}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} \left(\overbrace{-\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial \gamma}}^{\pm} + \overbrace{\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{B}}{\partial \gamma}\frac{\partial \dot{N}}{\partial B}}^{+} + \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial P}}^{-} - \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial P}\frac{\partial \dot{N}}{\partial B}}^{-} \right) \tag{13}$$

$$\frac{\partial P^*}{\partial m_P} = \overbrace{\frac{(-1)^6}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{+} \left(\overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial N}}^{+} - \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial B}}^{+} \right) \tag{14}$$

Eq. (12) is the change in the equilibrium density of primary producers with respect to changes in predator mortality. The first two terms are dependent on trait change sensitivity to predator density ($\frac{\partial \dot{\gamma}}{\partial P}$), while the last two terms both change with the effect of trait change with respect to the proportion of induced individuals ($\frac{\partial \dot{\gamma}}{\partial \gamma}$). The first term's sign is dependent on the cost or benefit of defended individuals to the whole prey population ($\frac{\partial \dot{N}}{\partial \gamma}$), while the magnitude can be modulated by recycling terms present in $\frac{\partial \dot{B}}{\partial N}$. Recycling also determines the magnitude of the last two terms (part of $\frac{\partial \dot{B}}{\partial N}$ and $\frac{\partial \dot{B}}{\partial P}$). Eq. (13) is similarly structured to Eq. (12) with respect to the trait-dependent effects, however, recycling is only present in the fourth term ($\frac{\partial \dot{B}}{\partial P}$). It is easier to see in Eq. (14) that the response to mortality is dependent on the balance between the intraspecific effects of the first term and the interspecific effects of the second term. The only trait-dependent portion in the equation scales both of these terms and is therefore unimportant in determining the response.

In the absence of adaptation (i.e., fixed levels of defense) and nutrient recycling, increased predator mortality causes densities to change in a way consistent with a trophic cascade: predator density decreases ($\frac{\partial P^*}{\partial m_P} < 0$), prey density increases ($\frac{\partial N^*}{\partial m_P} > 0$), and primary producer density increases ($\frac{\partial B^*}{\partial m_P} < 0$). This agrees with prior theoretical studies (Oksanen et al., 1981; McCann et al., 1998). The predator response is typically negative, but a positive response (i.e., a hydra effect) can occur if the intraspecific producer and prey interactions are much stronger than the interspecific interactions. Overall, this means that if the prey-producer dynamics are stable the system will exhibit classic trophic cascade responses (appendix S2.1).

When inducible defenses are present and recycling is absent, the response of the predator

density to mortality is still only dependent on the dynamics of the lower trophic levels. The responses for the lower trophic levels are the same as the above. However, in some cases, prey and primary producers can respond in the opposite direction.

$\frac{\partial B^*}{\partial m_P}$ can become positive if $\frac{\partial \dot{B}}{\partial P}$ is very large which is possible in a scenario with high levels of nutrient recycling. We can see that an increase in primary producer growth linked to the rate of recycling of dying predators links primary producer growth to predator mortality, and if the rate of recycling is great enough, then primary producers may increase their growth in response to increased predator mortality. The switch in response of primary producers to predator mortality in cases where $\frac{\partial \dot{\gamma}}{\partial P}$ is large and $\frac{\partial \dot{N}}{\partial \gamma} > 0$. The switch in response here is due to prey trait change being highly sensitive to predator density and an overall benefit to prey fitness due to a higher level of mean defense. This situation could arise when prey share benefits from defense such as chemical defenses which could benefit any individuals near the defense rather than just the defended individual. Lastly, the switch in response of primary producers occurs if $\frac{\partial \dot{\gamma}}{\partial P}$ is large and $\frac{\partial \dot{B}}{\partial \gamma}$ is very large. Therefore, the classic cascade response in primary producers can be altered if prey trait change is highly sensitive to predator density and there is a large cost to foraging from inducing defenses. The change in response in this scenario may seem counterintuitive at first, as increasing predator mortality would lead to more undefended prey individuals, which are more effective at consuming primary producers, however looking at $\frac{\partial \dot{B}}{\partial B}$ we can see that producer density negatively impacts producer growth rates so a situation with increased consumption of primary producers could benefit primary producer growth. The same is true for the switching of the response of $\frac{\partial N^*}{\partial m_P}$, meaning the non-trophic-cascade responses occur under the same scenarios for the lower trophic levels.

When nutrient recycling is present, it does not change the direction of the responses to increased predator mortality, but it does change the magnitude. First, it makes the predator's response more negative because increased recycling decreases the impact of prey density on primary producer growth rate ($\frac{\partial \dot{B}}{\partial N}$ is larger with increasing α_N) which overall increases the magnitude of the second term in Eq. (14). However, while the predator response to predator mortality is steeper when nutrient recycling is higher, predator density is higher because of a bottom-up effect (Fig. 9). Second, it reduces the sensitivity of prey to predator mortal-

ity through the increase in primary producer growth rate from recycled nutrients from dying predators. Third, it also reduces the sensitivity of primary producers to predator mortality. This occurs through the increase in the magnitude of the positive relationship between producer growth and predator mortality (increasing $\frac{\partial \dot{B}}{\partial P}$ with α_P) and the more positive relationship between prey density and producers driven by recycling dead prey ($\frac{\partial \dot{B}}{\partial N}$ becomes more positive with increasing α_N). $\frac{\partial \dot{B}}{\partial N}$ also decreases the magnitude of the response to predator mortality modulated by the costs of defense for prey ($\frac{\partial \dot{N}}{\partial \gamma}$) which can be positive or negative.

Table 3: Table of the effect on the expected response of equilibrium densities of the different trophic levels (B,N,P) to increasing predator mortality at different levels of model complexity.

	Response to increased predator mortality		
	Primary Producer (B)	Prey (N)	Predator (P)
Absence of adaptation and recycling	$-$	$+$	$\pm$
Effect of adding adaptation	$\pm$	$\pm$	0
Effect of nutrient recycling	$\pm$	$\pm$	$-$

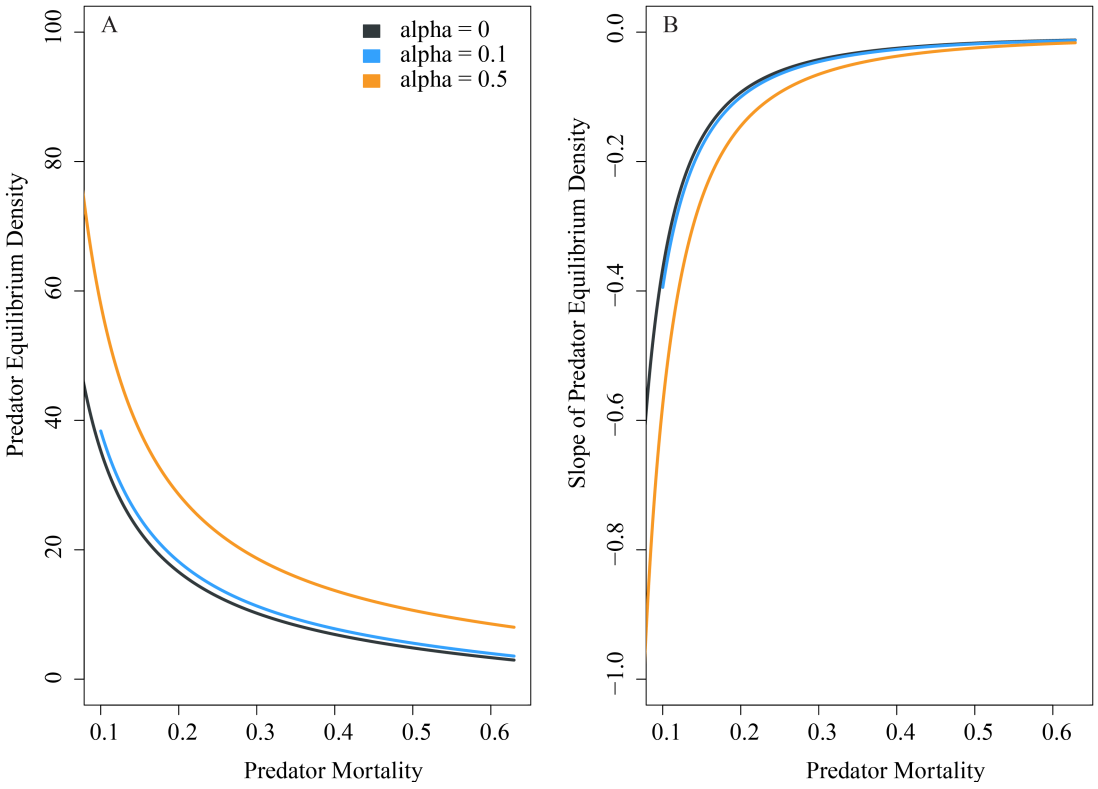

Figure 9: Shows (right) the response and (left) the sensitivity of predator density to predator mortality at different levels of nutrient recycling. (A) Shows the equilibrium density of the predator (P) over a gradient of predator mortality at different rates of nutrient recycling $\alpha = 0$, $\alpha = 0.1$, and $\alpha = 0.5$. (B) Shows the slope of the response of each scenario in panel A. Predator mortality (m_P) and recycling rate (α) deviate from default parameter values as defined by the x-axis and legend.

Effects of Nutrient Recycling, Foraging Costs, and Predator Mortality on Stability

I next explore how nutrient recycling, foraging costs of defense, and predator mortality affect the stability of the system. These results are numerical because stability analysis of the four-dimensional model was analytically intractable. Overall, I found that the type of stability present was unaffected by the level of nutrient recycling, foraging costs, or predator mortality.

Discussion

I explored the joint impacts of nutrient recycling and inducible defenses on top-down and bottom-up processes in a tri-trophic model with a prey species with an inducible defense. I found that trophic level sensitivity to predator mortality is altered by prey adaptability depending on trait change sensitivity to predator density and costs of defense, increased nutrient recycling increases predator sensitivity to its mortality, and the rate of nutrient recycling can induce bistability in a system. I also observed that the relationship between stability and recycling is altered by foraging costs to induce defense and the rate of adaptation. The implications of these results highlight important notes on previous research and avenues for future research.

The chain of species responses to bottom-up enrichment or top-down mortality in simple food chain models has generally been thought to follow the formula of trophic cascades. Therefore, many would expect that with increasing predator mortality there would be a loss of top-down pressure, leading to an increase in the intermediate trophic level and a subsequent decrease in the trophic level below that one. This pattern is presumed to cascade down to the primary producers of the system (Oksanen et al., 1981; McCann et al., 1998), and my model predicts the same pattern in the absence of adaptation. Adding in adaptation, I observed a set of indeterminate effects similar to those seen in Abrams and Matsuda (2005). The classic trophic cascade expectations were possible in my model, but I also found that all of the trophic levels could switch expected responses to predator mortality depending on trait change sensitivity to predator density and costs of defense induction. I also found that higher rates of nutrient recycling had the potential to switch the expected responses in the lower trophic levels, the cascade of bottom-up enrichment being linked to the death of the top predator had the ability to change the expected response to the top-down pressure of predator mortality when recycling

rates were high enough. However, predator sensitivity to its mortality only increased with increasing rates of recycling. The bottom-up cascade of enrichment was seen in the increased equilibrium densities of predators but the actual sensitivity of predators to mortality was also increased. Overall, it is clear that nutrient recycling is important to the expected responses to bottom-up and top-down effects in ecosystems.

Many studies predict that plasticity is stabilizing (Cortez, 2011; Yamamichi et al., 2019, 2011). My results agree with this conclusion. Adding nutrient dynamics to the system and a simple form of nutrient recycling did not lead to any adaptability-based alteration to the system stability. Overall, there was no noteworthy interaction between nutrient recycling and stability in my model.

Adaptive responses are present in many communities in nature (Bradshaw and Hardwick, 1989). In terms of ecosystem management, my findings indicate that it is important to consider the rate at which prey can respond to predation, foraging costs, and the rate at which nutrients cycle through a system. I observed that changing rates of nutrient recycling may lead to increased sensitivity of predators to their mortality. However, I did not see any changes to system stability linked to increasing recycling rates or foraging trade offs. In the future, it would be important to consider implementing other modes of recycling, such as through implementing direct time-lags into the rate of recycling or through large, pulsed mortality events similar to those seen in salmon habitat (Chen et al., 2011). There is potential that these temporally offset forms of recycling could lead to changing response to time-lagged shifts in response to predator mortality and leave room for non-stabilizing effects related to phenotypic plasticity.

Overall, I highlight that including explicit nutrient dynamics and recycling could be important in more accurately estimating the responses of species to top-predator mortality. My work highlights the need to understand how nutrient recycling can alter the expected responses of species interaction, especially in systems with phenotypic plasticity.

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Chapter IV: Conclusion

The goal of my research was to better understand the nature of density-dependence in defense induction and theoretically how inducible defenses might interact with other forms of shifting top-down and bottom-up effects. I found evidence in agreement with the density-dependent reduction in the magnitude of induced defenses in ciliate protists similar to Tollrian et al. (2015). However, my model suggests that this reduction in magnitude is linked to the reduction in size in the absence of predation threat, likely due to the metabolic strain of interspecific competition (Peters 1986). My results suggest that the prey in this system may have to overcome a larger "gap of induction" to try and express a similar level of defense as those in a less competitive environment. In the theoretical portion of my research, I found that adding a simple form of explicit nutrient dynamics added an interesting layer of interaction in the predicted effect of inducible defenses in a tri-trophic food chain model. I found that increasing recycling led to a more negative response of predator equilibrium density to predator mortality, but that it also led to an increased bottom-up enrichment that seemed to outpace the increased response of predators to their mortality. The effects on stability appeared to be non-existent and the expected stabilizing response of inducible defenses held true for my system with explicit nutrient dynamics. My research highlights a lack of understanding of the mechanisms of prey response to predation in systems with inducible defenses and a need for accounting for the non-intuitive feedback that may occur with the many facets added to species interaction through these inducible defenses.

Overall, inducible defenses could introduce counterintuitive responses to different top-down and bottom-up pressures, and these effects are not easily untangled from the plethora of other interactions between species within a community and even simplified abiotic factors in an ecosystem. I believe more data over a smaller gradient of predator cues could reveal more clearly the mechanisms of the density-dependent response to predation of *P. aurelia*. I would also suggest adding in more relevant vectors of nutrient recycling to my model, such as time-lagged recycling, large, pulsed mortality events (Chen et al. 2011), and predator effluent as a form of recycled nutrients (Allgeier et al. 2017). I also suggest merging the two forms of analysis from my thesis, with enough data on the predator-prey system from Chapter 2 many

of the parameters in the model of Chapter 3 could be parameterized giving better ideas of my model's predictions to different scenarios of recycling and foraging trade offs.

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Chapter 3 Appendix

S1 The Partial Derivatives and Expected Signs

Here, I determine the signs of the Jacobian entries for the continuous trait version of the model. Throughout, I assume all quantities are evaluated at $N^* > 0, P^* > 0, B^* > 0$ and $0 \leq \gamma \leq 1$. In addition, I assume the effects of phenotypic sorting are negligibly small.

Partials of $\dot{B}$

The entry J_{11} simplifies to,

$$\begin{aligned}\frac{\partial \dot{B}}{\partial B} &= \frac{\partial}{\partial B}\left(e_B(\delta + \alpha_N m_N(1-\gamma)N + \alpha_N m_N \gamma N + \alpha_P m_P P) - \frac{a_{N_1}B}{1+a_{N_i 1}h_N B}(1-\gamma)N - \frac{a_{N_2}B}{1+a_{N_2}h_N B}\gamma N\right) \\ &= 0 + \frac{(1+a_{N1}h_N B)(a_{N1}(1-\gamma)N) - (a_{N1}(1-\gamma)NB)(a_{N1}h_N)}{(1+a_{N1}h_N B)^2} - \frac{(1+a_{N2}h_N B)(a_{N2}\gamma N) - (a_{N2}\gamma NB)(a_{N2}h_N)}{(1+a_{N2}h_N B)^2} \\ &= \frac{a_{N1}N(-1+\gamma)}{(1+a_{N1}h_N B)^2} - \frac{a_{N2}N\gamma}{(1+a_{N2}h_N B)^2} < 0\end{aligned} \tag{S1}$$

where the first term is negative because $0 \leq \gamma \leq 1$.

The entry J_{12} simplifies to,

$$\begin{aligned}\frac{\partial \dot{B}}{\partial N} &= \frac{\partial}{\partial N}\left(e_B(\delta + \alpha_N m_N(1-\gamma)N + \alpha_N m_N \gamma N + \alpha_P m_P P) - \frac{a_{N_1}B}{1+a_{N_1}h_N B}(1-\gamma)N - \frac{a_{N_2}B}{1+a_{N_2}h_N B}\gamma N\right) \\ &= e_B\alpha_N m_N(1-\gamma) + e_B\alpha_N m_N\gamma N - \frac{a_{N_1}B(1-\gamma)}{1+a_{N_1}h_N B} - \frac{a_{N_2}B\gamma}{1+a_{N_2}h_N B} \\ &= e_B\alpha_N m_N - \frac{a_{N_1}B(1-\gamma)}{1+a_{N_1}h_N B} - \frac{a_{N_2}B\gamma}{1+a_{N_2}h_N B} < 0\end{aligned} \tag{S2}$$

Because we assume that nutrient recycling is relatively low (α_N small), this entry is negative.

The entry J_{13} simplifies to,

$$\begin{aligned}\frac{\partial \dot{B}}{\partial P} &= \frac{\partial}{\partial P}\left(e_B(\delta+\alpha_N m_N(1-\gamma)N+\alpha_N m_N \gamma N+\alpha_P m_P P)-\frac{a_{N_1}B}{1+a_{N_1}h_N B}(1-\gamma)N-\frac{a_{N_2}B}{1+a_{N_2}h_N B}\gamma N\right)\\ &= e_B\alpha_P m_P P > 0\end{aligned} \tag{S3}$$

The entry J_{14} simplifies to,

$$\begin{aligned}\frac{\partial \dot{B}}{\partial \gamma} &= \frac{\partial}{\partial \gamma}\left(e_B(\delta+\alpha_N m_N(1-\gamma)N+\alpha_N m_N \gamma N+\alpha_P m_P P)-\frac{a_{N_1}B}{1+a_{N_1}h_N B}(1-\gamma)N-\frac{a_{N_2}B}{1+a_{N_2}h_N B}\gamma N\right)\\ &= -e_B\alpha_N m_N N+e_B\alpha_N m_N N+\frac{a_{N_1}BN}{1+a_{N_1}h_N B}-\frac{a_{N_2}BN}{1+a_{N_2}h_N B}\\ &= \frac{a_{N_1}BN}{1+a_{N_1}h_N B}-\frac{a_{N_2}BN}{1+a_{N_2}h_N B} > 0\end{aligned} \tag{S4}$$

where the positive sign follows from our assumption that defended prey have lower consumption rates than undefended prey ($a_{N2} < a_{N1}$).

Partials of $\dot{N}$

The entry J_{21} simplifies to,

$$\begin{aligned}\frac{\partial \dot{N}}{\partial B} &= \frac{\partial}{\partial B}\left(e_{N_1}\frac{a_{N_1}B}{1+a_{N_1}h_N B}(1-\gamma)N+e_{N_2}\frac{a_{N_2}B}{1+a_{N_2}h_N B}\gamma N-\frac{NP((c_0+c_1)(1-\gamma)+c_0\gamma)}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N}-m_N N\right)\\ &= \frac{(1+a_{N1}h_N B)(e_{N1}(1-\gamma)Na_{N1})-(e_{N1}(1-\gamma)Na_{N1}B)(a_{N1}h_N)}{(1+a_{N1}h_N B)^2}+\frac{(1+a_{N2}h_N B)(e_{N2}\gamma Na_{N2})}{(1+a_{N2}h_N B)^2}\\ &= \frac{e_{N1}a_{N1}N(1-\gamma)}{(1+a_{N1}h_N B)^2}+\frac{(1+a_{N2}h_N B)(e_{N2}\gamma Na_{N2})}{(1+a_{N2}h_N B)^2} > 0\end{aligned} \tag{S5}$$

Noting that the $\frac{\partial \dot{N}}{\partial N}$ equation has a form $\dot{N} = Nf(N)$ and $f(N^*) = 0$, entry J_{22} reduces to,

$$\frac{\partial \dot{N}}{\partial N} = \frac{\partial}{\partial N}\left(e_{N_1}\frac{a_{N_1}B}{1+a_{N_1}h_NB}(1-\gamma)N + e_{N_2}\frac{a_{N_2}B}{1+a_{N_2}h_NB}\gamma N - \frac{NP((c_0+c_1)(1-\gamma)+c_0\gamma)}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N} - m_N N\right)$$
$$= \frac{P((c_0+c_1)(1-\gamma)+c_0\gamma)(h_0(c_0+c_1)(1-\gamma)+h_0)}{(1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N)^2} > 0 \tag{S6}$$

The entry J_{23} simplifies to,

$$\frac{\partial \dot{N}}{\partial P} = \frac{\partial}{\partial B}\left(e_{N_1}\frac{a_{N_1}B}{1+a_{N_1}h_NB}(1-\gamma)N + e_{N_2}\frac{a_{N_2}B}{1+a_{N_2}h_NB}\gamma N - \frac{NP((c_0+c_1)(1-\gamma)+c_0\gamma)}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N} - m_N N\right)$$
$$= -N((c_0+c_1)(1-\gamma)+c_0\gamma)\frac{1+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N}{(1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N)^2} < 0 \tag{S7}$$

The entry J_{24} simplifies to,

$$\frac{\partial \dot{N}}{\partial \gamma} = \frac{\partial}{\partial B}\left(e_{N_1}\frac{a_{N_1}B}{1+a_{N_1}h_NB}(1-\gamma)N + e_{N_2}\frac{a_{N_2}B}{1+a_{N_2}h_NB}\gamma N - \frac{NP((c_0+c_1)(1-\gamma)+c_0\gamma)}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N} - m_N N\right)$$
$$= \frac{-e_{N_1}a_{N_1}BN}{1+a_{N_1}h_NB} + \frac{e_{N_2}a_{N_2}BN}{1+a_{N_2}h_NB} - \frac{NP(-Pbc_1-c_1)}{(1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N)^2} \tag{S8}$$

The sign of entry J_{24} can be positive or negative because the sum of the first two terms is negative and the third term is positive. The entry is more likely to be positive when predation rates (c_1) and predator inference (b) are higher and more likely to be negative when there are large costs to defense (a_{N2} smaller).

Partials of $\dot{P}$

The entry J_{31} simplifies to,

$$\frac{\partial \dot{P}}{\partial B} = \frac{\partial}{\partial B}\left(P(e_P(\frac{(c_0+c_1)(1-\gamma)N}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N} + \frac{c_0\gamma N}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N}) - m_P)\right)$$
$$= 0 \tag{S9}$$

The entry J_{32} simplifies to,

$$\begin{aligned}\frac{\partial \dot{P}}{\partial N} &= \frac{\partial}{\partial N}\left(P(e_P(\frac{(c_0+c_1)(1-\gamma)N}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N}+\frac{c_0\gamma N}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N})-m_P)\right)\\ &= \frac{Pe_P(-c_1\gamma+c_0+c_1)(bP+1)}{(1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N)^2} > 0\end{aligned} \tag{S10}$$

Noting that the $\frac{\partial \dot{P}}{\partial P}$ equation has a form $\dot{P} = Pf(P)$ and $f(P^*) = 0$, entry J_{33} reduces to,

$$\begin{aligned}\frac{\partial \dot{P}}{\partial P} &= \frac{\partial}{\partial P}\left(P(e_P(\frac{(c_0+c_1)(1-\gamma)N}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N}+\frac{c_0\gamma N}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N})-m_P)\right)\\ &= -\frac{be_PPN((c_0+c_1)(1-\gamma)+c_0\gamma)}{(1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N)^2} < 0\end{aligned} \tag{S11}$$

The entry J_{34} simplifies to,

$$\begin{aligned}\frac{\partial \dot{P}}{\partial \gamma} &= \frac{\partial}{\partial \gamma}\left(P(e_P(\frac{(c_0+c_1)(1-\gamma)N}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N}+\frac{c_0\gamma N}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N})-m_P)\right)\\ &= Pe_PN(-\frac{c_1}{1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N}-\frac{N(-c_1\gamma+c_0+c_1)c_1h_0}{(1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N)^2})\\ &= \frac{-Pe_PNc_1(bP+1)}{(1+bP+h_0(c_0+c_1)(1-\gamma)N+h_0c_0\gamma N)^2} < 0\end{aligned} \tag{S12}$$

Partials of $\dot{\gamma}$

The entry J_{41} simplifies to,

$$\frac{\partial \dot{\gamma}}{\partial B} = \frac{\partial}{\partial B}\left((1-\gamma)\varepsilon\varphi_1(P) - \gamma\varepsilon\varphi_2(P)\right) = 0 \tag{S13}$$

The entry J_{42} simplifies to,

$$\frac{\partial \dot{\gamma}}{\partial N} = \frac{\partial}{\partial B}\left((1-\gamma)\varepsilon\varphi_1(P) - \gamma\varepsilon\varphi_2(P)\right) = 0 \tag{S14}$$

The entry J_{43} simplifies to,

$$\frac{\partial \dot{\gamma}}{\partial P} = \frac{\partial}{\partial P}\left((1-\gamma)\varepsilon\varphi_1(P) - \gamma\varepsilon\varphi_2(P)\right) = \frac{\varepsilon\rho P^{\lambda}\lambda((1-\gamma)+\gamma)}{P(1+\rho P^{\lambda})^2} = \frac{\varepsilon\rho P^{\lambda}\lambda}{P(1+\rho P^{\lambda})^2} > 0 \tag{S15}$$

The entry J_{44} simplifies to,

$$\frac{\partial \dot{\gamma}}{\partial \gamma} = \frac{\partial}{\partial \gamma}\left((1-\gamma)\varepsilon\varphi_1(P) - \gamma\varepsilon\varphi_2(P)\right) = -\varepsilon\left(1 - \frac{1}{1+\rho P^{\lambda}}\right) - \frac{\varepsilon}{1+\rho P^{\lambda}} = -\varepsilon < 0 \tag{S16}$$

S2 Expected response of equilibrium values to a change in a parameter

For a system of two differential equations where only one of the equations is dependent on a parameter of interest (m),

$$\begin{aligned} \frac{dx}{dt} &= xf(x,y) \\ \frac{dy}{dt} &= yg(x,y,m), \end{aligned} \tag{S17}$$

the equilibrium conditions are $0 = x^* f(x^*,y^*)$ and $0 = y^* g(x^*,y^*,m)$. The local sensitivities of the equilibrium densities to the parameter are found by taking the derivative of the two equations with respect to m,

$$\begin{aligned} 0 &= \frac{\partial f}{\partial x}\frac{\partial x^*}{\partial m} + \frac{\partial f}{\partial y}\frac{\partial y^*}{\partial m} \\ 0 &= \frac{\partial g}{\partial x}\frac{\partial x^*}{\partial m} + \frac{\partial g}{\partial y}\frac{\partial y^*}{\partial m} + \frac{\partial g}{\partial m} \end{aligned} \tag{S18}$$

Writing this system in matrix form yields,

$$\begin{bmatrix} 0 \\ -\frac{\partial g}{\partial m} \end{bmatrix} = \overbrace{\begin{bmatrix} \frac{\partial f}{\partial x} & \frac{\partial f}{\partial y} \\ \frac{\partial g}{\partial x} & \frac{\partial g}{\partial y} \end{bmatrix}}^{\text{Jacobian}} \begin{bmatrix} \frac{\partial x^*}{\partial m} \\ \frac{\partial y^*}{\partial m} \end{bmatrix}, \tag{S19}$$

which can be solved to to get

$$\begin{bmatrix} \frac{\partial x^*}{\partial m} \\ \frac{\partial y^*}{\partial m} \end{bmatrix} = J^{-1} \begin{bmatrix} 0 \\ -\frac{\partial g}{\partial m} \end{bmatrix} \tag{S20}$$

Applying Cramer's rule yields,

$$\begin{aligned}\frac{\partial x^*}{\partial m} &= \frac{|J^{[2,1]}|}{|J|}(-1)^{2+1}\left(-\frac{\partial g}{\partial m}\right)\\ \frac{\partial y^*}{\partial m} &= \frac{|J^{[2,2]}|}{|J|}(-1)^{2+2}\left(-\frac{\partial g}{\partial m}\right)\end{aligned} \tag{S21}$$

where $|J^{[i,j]}$ is the submatrix of J where row i and column j have been removed. We can determine the signs of each sensitivity because the Jacobian entries have known signs.

The above process can be generalized to models with any number of dimensions (Cortez and Abrams, 2016; Yodzis, 1988). For my model, the responses to increased predator mortality are

$$\frac{\partial x_i^*}{\partial m_P} = \frac{|J^{[3,i]}|}{|J|}(-1)^{3+i}(-\frac{\partial \dot{P}}{\partial m_P}) \tag{S22}$$

where x_i is B for $i = 1$, N for $i = 2$, P for $i = 3$, and γ for $i = 4$ and $J^{[3,i]}$ is the Jacobian with row 3 and column i removed. Note that $|J| > 0$ for my system because I am analyzing the responses of stable equilibria. This is because the determinant of the Jacobian is equal to the product of its eigenvalues, and any Jacobian of a four dimensional system evaluated at a stable equilibrium must have 4 eigenvalues with negative real parts. It follows, that the products of these eigenvalues will lead to the determinant of the Jacobian always being positive. Also note that $\frac{\partial P}{\partial m_P} < 0$ because an increase in predator mortality decreases the predator growth rate.

S2.1 Responses in the absence of adaptation and nutrient recycling

I start by showing the responses in systems without adaptation. To do this, I fix the proportion of defended individuals at a value γ^*. The Jacobian for the density dynamics is,

$$\hat{J} = \begin{bmatrix} \frac{\partial}{\partial B}(\frac{dB}{dt}) & \frac{\partial}{\partial N}(\frac{dB}{dt}) & \frac{\partial}{\partial P}(\frac{dB}{dt}) \\ \frac{\partial}{\partial B}(\frac{dN}{dt}) & \frac{\partial}{\partial N}(\frac{dN}{dt}) & \frac{\partial}{\partial P}(\frac{dN}{dt}) \\ \frac{\partial}{\partial B}(\frac{dP}{dt}) & \frac{\partial}{\partial N}(\frac{dP}{dt}) & \frac{\partial}{\partial P}(\frac{dP}{dt}) \end{bmatrix} = \begin{bmatrix} - & - & + \\ + & + & - \\ 0 & + & - \end{bmatrix} \tag{S23}$$

where the signs of all entries of $\hat{J}$ are the same as in the Jacobian for the full model (J). I determine the response to mortality in the absence of adaptation utilizing Eq. (S22) where $|\hat{J}|$

is negative. Computing the sensitivities yields,

$$\frac{\partial B^*}{\partial m_P} = \overbrace{\frac{(-1)^4}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} |\hat{J}^{[3,1]}| = \overbrace{\frac{-1^4}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} \left(\overbrace{\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial P}}^{+} - \overbrace{\frac{\partial \dot{B}}{\partial P}\frac{\partial \dot{N}}{\partial N}}^{+} \right) \tag{S24}$$

$$\frac{\partial N^*}{\partial m_P} = \overbrace{\frac{(-1)^5}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{+} |\hat{J}^{[3,2]}| = \overbrace{\frac{-1^5}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{+} \left(\overbrace{\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial P}}^{+} - \overbrace{\frac{\partial \dot{B}}{\partial P}\frac{\partial \dot{N}}{\partial B}}^{+} \right) \tag{S25}$$

$$\frac{\partial P^*}{\partial m_P} = \overbrace{\frac{(-1)^6}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} |\hat{J}^{[3,3]}| = \overbrace{\frac{-1^6}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} \left(\overbrace{\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial N}}^{-} - \overbrace{\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial B}}^{-} \right) \tag{S26}$$

If there is no nutrient recycling ($\frac{\partial \dot{B}}{\partial P} = 0$), then increased predator mortality causes decreases in the primary producer ($\frac{\partial B^*}{\partial m_P} < 0$), increases in the prey ($\frac{\partial N^*}{\partial m_P} > 0$), and increases or decreases in the predator ($\frac{\partial P^*}{\partial m_P}$ positive or negative). These responses match the classical predictions from trophic cascades (McCann et al., 1998; Oksanen et al., 1981). Note that the predator response is typically negative, but a positive response (i.e., a hydra effect) can occur if the intraspecific producer and prey interactions are much stronger than the interspecific interactions. In our model, hydra effects arise at a stable equilibrium only when predator interference (b) is sufficiently high.

S2.2 Predictions for B^* with inducible defenses

$$\begin{aligned} \frac{\partial B^*}{\partial m_P} &= \overbrace{\frac{(-1)^4}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{+} |\hat{J}^{[3,1]}| \\ &= \overbrace{\frac{(-1)^4}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{+} \left(\overbrace{-\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial \gamma}}^{\pm} + \overbrace{\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{N}}{\partial N}\frac{\partial \dot{B}}{\partial \gamma}}^{+} + \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial P}}^{-} - \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial P}\frac{\partial \dot{N}}{\partial N}}^{-} \right) \end{aligned} \tag{S27}$$

$\frac{\partial B^*}{\partial m_P}$ is expected to be negative in the absence of trait adaptation and recycling. However, it

can become positive in any of the following scenarios,

1. $\frac{\partial \dot{B}}{\partial P}$ is very large which is possible in a scenario with high levels of nutrient recycling.

2. $\frac{\partial \dot{\gamma}}{\partial P}$ is large and $\frac{\partial \dot{N}}{\partial \gamma} > 0$. This corresponds to a scenario where prey trait change is highly sensitive to predator density and when higher mean defense leads to increased fitness for all prey.

3. $\frac{\partial \dot{\gamma}}{\partial P}$ is large and $\frac{\partial \dot{B}}{\partial \gamma}$ is very large. This is a scenario where prey trait change is highly sensitive to predator density and there is a large cost to foraging from inducing defenses.

The magnitude of B^*'s sensitivity to m_P can be altered by increasing recycling.

1. As seen above, with increasing recycling $\frac{\partial \dot{B}}{\partial P}$ get larger which in turn reduces the magnitude of $\frac{\partial B^*}{\partial m_P}$ and can potentially switch its sign.

2. Increasing the rate of recycling decreases the magnitude of $\frac{\partial \dot{B}}{\partial N}$ which decreases the magnitude of the first term of the determinant whose sign is dependant on the sign of $\frac{\partial \dot{N}}{\partial \gamma}$. This also reduces the magnitude of the third term of the determinant which approaches zero reduces the magnitude of $\frac{\partial B^*}{\partial m_P}$.

S2.3 Predictions for N^* with inducible defenses

$$\begin{aligned}\frac{\partial N^*}{\partial m_P} &= \overbrace{\frac{(-1)^5}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} |\hat{J}^{[3,2]}| \\ &= \overbrace{\frac{(-1)^5}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} \left(\overbrace{-\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial \gamma}}^{\pm} + \overbrace{\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{B}}{\partial \gamma}\frac{\partial \dot{N}}{\partial B}}^{+} + \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial P}}^{-} - \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial P}\frac{\partial \dot{N}}{\partial B}}^{-} \right)\end{aligned} \tag{S28}$$

$\frac{\partial N^*}{\partial m_P}$ is expected to be positive in the absence of trait adaptation and recycling. However, it can become negative in any of the following scenarios,

1. $\frac{\partial \dot{B}}{\partial P}$ is very large which is possible in a scenario with high levels of nutrient recycling.

2. $\frac{\partial \dot{\gamma}}{\partial P}$ is large and $\frac{\partial \dot{N}}{\partial \gamma} > 0$. This corresponds to a scenario where prey trait change is highly sensitive to predator density and when higher mean defense leads to increased fitness for all prey.

3. $\frac{\partial \dot{\gamma}}{\partial P}$ is large and $\frac{\partial \dot{B}}{\partial \gamma}$ is very large. This is a scenario where prey trait change is highly sensitive to predator density and there is a specifically a large cost to foraging from inducing defenses.

The magnitude of N^*'s sensitivity to m_P can be altered by increasing recycling.

1. As seen above, with increasing recycling $\frac{\partial \dot{B}}{\partial P}$ get larger which in turn reduces the magnitude of $\frac{\partial N^*}{\partial m_P}$ and can potentially switch its sign.

S2.4 Predictions for P^* with inducible defenses

$$\begin{aligned}\frac{\partial P^*}{\partial m_P} &= \overbrace{\frac{(-1)^6}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{+}|\hat{J}^{[3,3]}| \\ &= \overbrace{\frac{(-1)^6}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{+}\left(\overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial N}}^{+} - \overbrace{\frac{\partial \dot{\gamma}}{\partial \gamma}\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial B}}^{+}\right)\end{aligned} \tag{S29}$$

As stated above $\frac{\partial P^*}{\partial m_P} < 0$ for cases when the prey-producer dynamics are stable and is positive otherwise.

Here induction has generally no effect on the response to predator mortality. Higher rates of nutrient recycling leads to larger values of $\frac{\partial \dot{B}}{\partial N}$ which increases the magnitude of $\frac{\partial P^*}{\partial m_P}$, meaning increasing recycling increases predator sensitivity to predator mortality so long as the prey-producer dynamics are stable.

S2.4.1 Hydra Effects

Hydra effects are only present in a system where the determinant of the sub matrix of the Jacobian without the predator terms is greater than zero (Cortez and Abrams, 2016).

$$\begin{vmatrix} \frac{\partial \dot{B}}{\partial B} & \frac{\partial \dot{B}}{\partial N} & \frac{\partial \dot{B}}{\partial \gamma} \\ \frac{\partial \dot{N}}{\partial B} & \frac{\partial \dot{N}}{\partial N} & \frac{\partial \dot{N}}{\partial \gamma} \\ \frac{\partial \dot{\gamma}}{\partial B} & \frac{\partial \dot{\gamma}}{\partial N} & \frac{\partial \dot{\gamma}}{\partial \gamma} \end{vmatrix} > 0 \tag{S30}$$

Which can be simplified to:

$$\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial N}\frac{\partial \dot{\gamma}}{\partial \gamma} - \frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial B}\frac{\partial \dot{\gamma}}{\partial \gamma} > 0 \tag{S31}$$

This means that hydra effects do not arise if $\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial N} - \frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial B} > 0$ which is the case when prey-producer dynamics are stable. However hydra effects can arise when $\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial N} - \frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial B} < 0$ when prey-producer dynamics are exhibiting limit cycles. This is the same condition for hydra effects when defense levels are fixed as seen in section S2.1. This means inducible defenses in my model do not increase or decrease the likelihood of a hydra effect occurring as none of the equations dependent on trait are involved in this determinant.

S2.5 Predictions for γ^* with inducible defenses

$$\begin{aligned} \frac{\partial \gamma^*}{\partial m_P} &= \overbrace{\frac{(-1)^7}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} |\hat{J}^{[3,4]}| \\ &= \overbrace{\frac{(-1)^7}{|\hat{J}|}(-\frac{\partial \dot{P}}{\partial m_P})}^{-} \left(\overbrace{\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{B}}{\partial B}\frac{\partial \dot{N}}{\partial N}}^{+} - \overbrace{\frac{\partial \dot{\gamma}}{\partial P}\frac{\partial \dot{B}}{\partial N}\frac{\partial \dot{N}}{\partial B}}^{+} \right) \end{aligned} \tag{S32}$$

Similar to the prediction for P^*, $\frac{\partial \gamma^*}{\partial m_P} > 0$ when prey-producer dynamics are stable and is negative otherwise.

S3 Appendix References

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