

# To Grow or to Reproduce? The Role of Life-History Plasticity in Food Web Dynamics

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**ABSTRACT:** The size of an individual is a key feature influencing and determined by a species' life history and ecology. Here, I consider how life-history plasticity within a single species can influence the outcome of food web interactions along a productivity gradient. An individual can either reproduce early but remain susceptible to predators throughout its life (strategy 1) or delay reproduction and grow to a predator-invulnerable size refuge (strategy 2). At low productivity, strategy 1 is favored because the probability of growing to a size refuge is low compared to the probability of being eaten. Here, the system is consumer controlled, and predators have large effects on the food web. At high productivity, strategy 2 is favored because high food availability increases the probability of prey attaining size refuge before being eaten. Consequently, the system becomes less consumer controlled, and predators have weaker effects on food web dynamics. At intermediate productivity, either strategy 1 or strategy 2 can be favored, depending on the initial conditions of the system. Field and laboratory experiments with a common freshwater snail *Helisoma trivolis* and its insect predator *Belostoma flumineum* support both the key assumptions and predictions of the models.

*Keywords:* life-history plasticity, size refugia, food webs, snails, alternative equilibria.

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Predators and resources are two of the most important factors that limit the distribution and abundance of species in nature. However, the degree of influence of each factor varies considerably between and among systems and with environmental conditions (e.g., Hairston et al. 1960; Fretwell 1977; Oksanen et al. 1981; Power 1992; Wootton and Power 1993; Osenberg and Mittelbach 1996; Polis and Strong 1996; Brett and Goldman 1997; Leibold et al. 1997). Out of necessary simplicity, most models of food web

interactions assume that all individuals of a species are identical, and unchanging through time (but see, e.g., DeAngelis and Gross 1992 for more complex, individually based models). Nevertheless, individuals within a species are not identical and often change in a number of aspects throughout their lives.

In a companion article (Chase 1999, in this issue), I consider the food web dynamics of a prey species that changes in its susceptibility to predators through ontogeny. A number of species are highly susceptible to predators as juveniles but can grow too large to be effectively killed and consumed as adults. Such size refugia can have important effects on the outcomes of food web interactions, ranging from strong to weak consumer control, as well as the possibility of alternative stable equilibria depending on environmental and initial conditions (Chase 1999). This model assumed that individual life histories were fixed; species could not adjust their life history with environmental conditions. However, it is well known both theoretically and empirically that many species can adjust their life histories in response to food and mortality factors (i.e., predation) by allocating resources differently to somatic growth, reproduction, and/or defense (e.g., Law 1979; Michaud 1979; Crowl and Covich 1990; Resnick et al. 1990; Charlesworth 1994; Abrams and Rowe 1996). This and other sources of phenotypic plasticity have held a prominent role in evolutionary ecology (e.g., Futuyma and Moreno 1988; Stearns 1989; Scheiner 1993; Via et al. 1995; DeWitt et al. 1998), but explicit connections between such phenotypic plasticity and the broader context of ecological interactions and species abundance have rarely been bridged.

One such exploration into the influence of plasticity on ecological interactions has focused on adaptive behavioral traits (e.g., Abrams 1984, 1991, 1993, 1996, 1999; Gleeson and Wilson 1986; Werner and Anholt 1993; Oksanen and Lundberg 1995; Fryxell and Lundberg 1997; Grover and Holt 1998; Schmitz 1998). Results from these studies show how adaptive behavior of both prey and predators can influence food web dynamics, and in many cases, adaptive behaviors can profoundly change the outcomes of food

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web interactions relative to systems in which prey behavior is stereotyped.

In this article, I explore how adaptive life-history plasticity in relation to food acquisition and the ability of prey to grow to a size refuge can influence food webs. Life-history theory predicts that, given constraints, individuals should act in a way that maximizes their lifetime fitness (Roff 1992; Stearns 1992). Food resources play an obvious role in the parameters that affect an individual's life history (Werner and Gilliam 1984; Kozłowski 1992; Roff 1992; Stearns 1992; Charlesworth 1994). In addition, if predators are an important mortality agent, they can also profoundly influence a prey's life history (Law 1979; Michaud 1979; Werner and Gilliam 1984; Crowl and Covich 1990; Resnick et al. 1990; Charlesworth 1994; Abrams and Rowe 1996). The nature by which predation influences prey life history should depend on environmental conditions and food availability. In addition, the effects of predation on prey life histories may also influence the broader context of community level interactions within food webs, though this connection has received comparatively little attention.

I present a modification of a set of mechanistic food web models on the basis of those in the companion article (Chase 1999) by allowing prey individuals to have a simple life-history plasticity decision. In that article, I model the dynamics of two species that were stereotyped into different life-history strategies, one that is always susceptible to predators throughout its life, and the other that can grow too large to be eaten after passing through a susceptible juvenile stage. In this article, I assume that one prey species can be plastic for either of the two life-history strategies but cannot switch strategies past a critical period. Prey species can either follow strategy 1 and grow to maturity quickly and reproduce, but at the cost of somatic growth, so that they are susceptible to predators throughout their lives, or follow strategy 2 and delay reproduction in deference to growth until a size refuge from predation is reached and then reproduce. I derive the combinations of food availability and predation pressure under which each strategy is favored by using modified food web models.

The method of using food web interaction models for examining the optimization of different life-history strategies is a new approach with considerable promise. I show that at low productivity, strategy 1 dominates and the system is consumer controlled; at high productivity, strategy 2 dominates and the system becomes more weakly consumer controlled, and at intermediate productivity, either strategy (but not a mixture of strategies) can be favored depending on initial predator and prey densities. Furthermore, the domains of attraction of these alternative equilibria are determined by individual life-history optimization. Next, I present results from experiments with a

common freshwater snail, *Helisoma trivolis*, showing its ability to have a plastic response in growth and size at reproduction to food levels in the presence of the predatory insect, *Belostoma flumineum*. Finally, I present results from a mesocosm experiment that varies predators and initial conditions. I show that plastic life-history decisions of the snail resulting from different initial conditions greatly influenced snail population size structure and abundance, as well as food web and ecosystem processes.

### Food Web Models with Prey Life-History Plasticity

To derive the optimal life-history strategy of individuals that are plastic for certain traits, I use models identical to those used to explore food web structure. Food web models attempt to predict the abundance, interactions, and coexistence of two or more species. Given the assumption that life-history plasticity allows an individual of a species to choose one life-history strategy or the other, I directly substitute alternate life-history strategies for species in food web models to make an explicit connection between life history and food webs. To that end, I use the same methodology as food web models to explore the dynamics and interactions among species with life-history plasticity. The models I use are on the basis of the formulation by Holt et al. (1994; see also Leibold 1996), but models with other forms (e.g., Lotka-Volterra-based food chain models; Oksanen et al. 1981) give similar predictions. For simplicity, I use several conventional assumptions: food is limiting for both predators and prey; consumers do not interfere with one another; consumers remove prey at a constant rate (a Type I functional response; a saturating Type II response does not alter the qualitative predictions of the model, but graphical representation becomes impossible); and the system is closed so that the amount of nutrients is fixed and they are either in free form or tied up in consumer biomass. However, relaxing some of these assumptions has little influence on the overall qualitative dynamics of the system (Leibold 1996; Grover and Holt 1998).

#### *Prey Strategy 1: Reproduce Early*

The first prey strategy (strategy 1), in which prey ( $N$ ) consume a resource ( $R$ ) and are always susceptible to predators ( $P$ ), can be modeled in the context of a specific food chain. The dynamics are identical to those given by Holt et al. (1994):

$$\begin{aligned}
 dR/dt &= \frac{c'N}{b'} + \frac{cP}{b_p} + aNP\left(\frac{1}{b'} - \frac{b}{b_p}\right) - a'RN, \\
 dN/dt &= N(a'b'R - c' - aP), \\
 dP/dt &= P(abN - c),
 \end{aligned}
 \tag{1}$$

where  $c$  is the density-independent mortality rate for the species,  $a$  is the attack rate of the species on its resource,  $b$  is the conversion of resources consumed into new consumers through reproduction. Parameters without primes (e.g.,  $a$ ) refer to the predator's ( $P$ ) characteristics, while parameters with primes (e.g.,  $a'$ ) are for the prey ( $N$ ). Resource dynamics ( $R$ ) are given by the amount of nutrients recycled by dead consumers ( $c/b$ ) plus the incomplete assimilation of consumed prey minus those resources consumed (Holt et al. 1994).

The zero net growth isocline (ZNGI) for prey  $N$  is obtained by setting the dynamics of prey species ( $N$ ) to 0 (where the system is in equilibrium, neither increasing or decreasing), and solving for  $P$ :

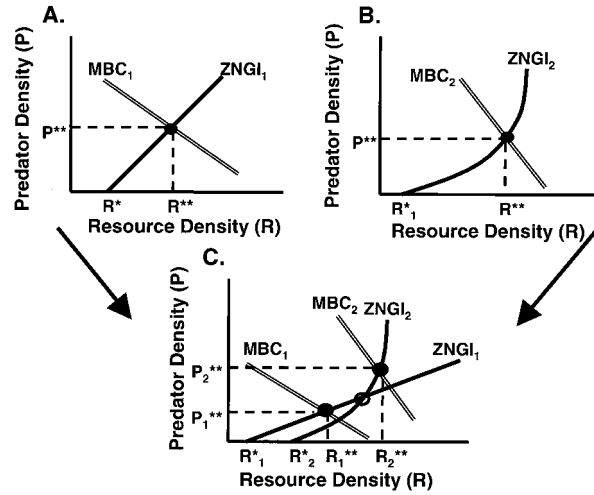
$$P = \frac{a'b'}{a}R - \frac{c'}{a}.
 \tag{2}$$

This ZNGI allows examination of the fate of prey individuals with variation of predator ( $P$ ) and resource ( $R$ ) abundance as in figure 1A. The  $x$ -intercept of this line, termed the species  $R^*$ , is the abundance of  $R$  that  $N$  maintains in the absence of  $P$  (see, e.g., Tilman 1982). In this case,  $R^*$  is solved by setting  $P = 0$  and solving for  $R$ , which gives  $R^* = c'/a'b'$ . The slope of the line denotes the effect of adding predators on the ability of  $N$  to have zero net growth (fig. 1A). The shallower the slope of its ZNGI, the more affected  $N$  is by  $P$ ; the slope of the ZNGI is given by  $a'b'/a$  (see Holt et al. 1994; Leibold 1996).

To examine a dynamic three-species food chain in two-dimensional graphs, Holt et al. (1994) described an important technique by using a mass balance constraint. Because the system is closed, resources are either free form or tied up in prey or predator biomass. Because the parameters  $b'$  and  $b_p$  represent the conversion of resources ( $R$ ) into  $N$  and  $P$ , respectively, the inverse of these  $N/b'$  and  $P/b_p$  represents the amount of resources tied up in the populations of  $N$  and  $P$ , respectively. Thus, for graphical simplicity, rather than presenting  $R$  as  $dR/dt$ ,  $R$  can instead be denoted in terms of nutrient mass balance within the closed system, giving

$$R = S - \frac{N}{b'} - \frac{P}{b_p},
 \tag{3}$$

where  $S$  is the environmentally determined supply rate of



**Figure 1:** A, Zero net growth isocline (ZNGI) and mass balance constraint (MBC) for life-history strategy 1. The point where the ZNGI and the MBC intersect is the equilibrium of resources ( $R^{**}$ ) and predators ( $P^{**}$ ).  $R^*$  is the abundance of resources that the herbivore can maintain in the absence of predators. B, ZNGI and MBC for life-history strategy 2, which delays reproduction and grows to size refugia. Letters and equilibria are as in A. C, ZNGIs and MBCs for both strategies overlaid on each other. Three equilibria are possible. The equilibria indicated by an open circle are locally unstable, whereas the two filled circles indicate locally (but not globally) stable alternative equilibria.  $R_1^{**}$  and  $P_1^{**}$  represent the equilibrium abundance of resources and predators, respectively, at the equilibria dominated by strategy 1, whereas  $R_2^{**}$  and  $P_2^{**}$  represent the equilibrium abundance of resources and predators, respectively, at the equilibria dominated by strategy 2.

nutrients. Substituting the equilibrium density of prey ( $N^*$ ), solved by setting  $dP/dt = 0$  and solving for  $N(N^* = c/ab)$ , and then solving equation (3) for  $P$  gives

$$P = b_p \left( S - R - \frac{cb'}{ab} \right).
 \tag{4}$$

Equation (4) is the mass balance constraint (MBC) for this system, and has a slope of  $-b_p$  and an intercept determined by the species-specific parameters, as well as the resource supply in the system. The MBC is plotted on the same  $P$ - and  $R$ -axes as the ZNGI, constraining the total biomass of the system to this line. The equilibrium point for this food chain is then determined by the intersection of the ZNGI and the MBC (fig. 1A).

#### Prey Strategy 2: Grow to a Size Refugia, then Reproduce

The above set of equations assumes that individual prey are identical in their susceptibility and reproductive output throughout their lives. However, as suggested earlier, this

may be too simplistic to describe a number of natural situations where individuals change significantly in a number of features throughout their lives. For mathematical tractability, I consider a two life-stage model to capture the main dynamics of a system in which juveniles ( $N_j$ ) are susceptible to predators but do not reproduce, while adults ( $N_A$ ) are immune from predation due to a size refuge, and reproduce. Both life stages consume the same resource ( $R$ ). Juveniles convert their resource acquisition into growth to the adult stage ( $g_j'$ ), while adults convert their resources into reproduction to the juvenile stage ( $b_A'$ ) and maintenance ( $c_A'$ ). The dynamic equations for such a system are

$$\begin{aligned} dR/dt &= \frac{c_A N_A}{b_A'} + \frac{cP}{b_p} + aN_jP \left( \frac{1}{g_j} - \frac{g_j}{b_p} \right) \\ &\quad - a_j'RN_j - a_A'RN_A, \\ dN_j/dt &= a_A'b_A'RN_A - g_j'a_j'RN_j \\ &\quad - c_j'N_j - aN_jP, \\ dN_A/dt &= g_j'a_j'RN_j - c_A'N_A, \\ dP/dt &= abN_jP - c, \end{aligned} \quad (5)$$

where the remaining parameters  $a$ 's,  $b$ 's, and  $c$ 's are as shown earlier (see also Chase 1999). The ZNGI for this system is solved by first determining the equilibrium densities of  $N_j$  and  $N_A$ . The equilibrium density of juveniles ( $N_j^*$ ) is given by setting the dynamics of predators ( $dP/dt$ ) equal to 0, and solving for  $N_j$ , which gives

$$N_j^* = c/ab. \quad (6)$$

Next, the equilibrium density of adults is determined by setting  $dN_A/dt = 0$ , substituting  $N_j^*$  (eq. [6]) for  $N_j$ , and solving for  $N_A$ , which gives

$$N_A^* = \frac{g_j'a_j'bcR}{ac_A'}. \quad (7)$$

Combining the dynamics of prey into a single equation ( $dN_j + dN_A$ ), and setting them both equal to 0, substituting  $N_j^*$  (eq. [6]) and  $N_A^*$  (eq. [7]) for  $N_j$  and  $N_A$ , respectively, and solving for  $P$ , thus yields the ZNGI for the entire prey population on  $P$ - and  $R$ -axes:

$$P = g_j'a_j' \frac{R}{c_A'} \left( \frac{a_A'b_A'R - c_A' - c_j'}{a} \right). \quad (8)$$

Equation (8) is thus the ZNGI for a species that has a two-stage life history characterized by a predator-vulner-

able juvenile stage and an invulnerable adult stage (fig. 1B). If, as above, resources are assumed to be in mass balance in a closed system, the equation for resources is

$$R = S - \frac{N_j}{b_A'} - \frac{N_A}{g_j'} - \frac{P}{b_p}. \quad (9)$$

Inserting  $N_j^*$  (eq. [6]) and  $N_A^*$  (eq. [7]) into equation (9) and solving for  $P$  thus gives the MBC for this system

$$P = b_p \left( \frac{-bcc_A' - a_jbb_A'cR - ab_A'c_A'R + ab_A'c_A'S}{ab_A'c_A'} \right) \quad (10)$$

(see fig. 1B).

#### *What Conditions Favor Each Strategy?*

The two above sets of equations describe the dynamics of two different life-history strategies. If individuals are plastic in their abilities to grow and to reproduce, an individual can either reproduce early and remain susceptible to predators throughout its lifetime (strategy 1) or grow to a size refugia and then begin reproduction (strategy 2). If the death rate of adults exceeds the growth rate of juveniles to adulthood (and size refugia; determined by the function including juvenile growth rate,  $g_j'$ ), the system becomes more consumer controlled, and life-history strategy 1 is optimal. If, however, the number of juveniles that reach adulthood exceeds the death and maintenance of adults ( $c_A'$ ), the system becomes less consumer controlled, and the strategy of growing to a size refugia (strategy 2) is more beneficial.

To determine under which conditions one strategy or the other (or both) should be favored, the ZNGIs and MBCs for each life-history strategy can be overlaid and examined together as in figure 1C. Equilibria for a strategy are determined by the point where the ZNGI intersects the MBC, but the stability of the equilibria is slightly more complicated. The population of each strategy is favored and increases in abundance to the right of its ZNGI. Where the ZNGIs cross within the MBC, as in figure 1C, there are three potential equilibria, each describing different optimal life-history strategies. The middle equilibrium, where both life-history strategies coexist, is unstable both locally and globally. This is because any slight perturbation from that point will tend toward dominance by one of the other two strategies. Thus, mixed life-history strategies should not occur with these dynamics (if, however, plasticity is in terms of some other defense that does not vary with ontogeny, more than one strategy can coexist in a single environment). The two outside equilibria, where either

strategy 1 or strategy 2 dominates, are locally stable in that once the equilibrium is reached, any slight perturbations will tend back toward the equilibrium. However, these equilibria are not globally stable because perturbations beyond a certain point will drive the system to the alternative equilibrium. Thus, depending on the initial conditions of predator and prey availability (see also Chase 1999), either strategy can become fixed and uninvadible by the other strategy.

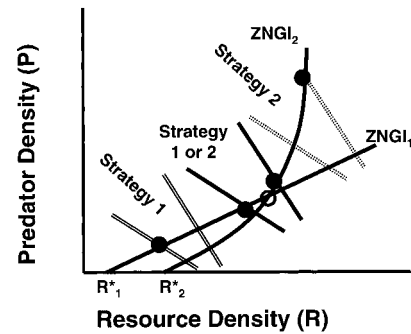
The two alternative stable equilibria, determined by the two life-history strategies, however, have very different outcomes on the dynamics of the food web. Strategy 1 is more strongly consumer limited. Strategy 2 is relatively less consumer limited. Strategy 2 will be favored when initial prey : predator ratios are relatively high. Dominance by strategy 2 leads to an overall higher biomass of prey and tends toward weaker food web interactions. Interestingly, however, strategy 1 is locally stable with low initial prey : predator ratios. Thus, optimal life-history decisions of individuals within a species can lead to conditions (i.e., dominance by strategy 1) that are very different from those that the species could attain by maximizing the overall abundance of the species (i.e., dominance by strategy 2).

Figure 1C only shows one specific set of ZNGIs and MBCs, where the two strategies' ZNGIs cross in between the two MBCs. This is necessary for the alternative stable equilibria discussed above to be feasible. ZNGIs are explicit properties of the species, but MBCs are functions of both species attributes as well as environmental conditions (notably environmental productivity). Increasing the environmental productivity of a system moves the relative positions of the MBC lines to the upper right part of the graphs.

Figure 2 shows three sets of MBCs, representing three qualitatively different levels of environmentally determined resource supply rates (increasing  $S$ ). The first set of MBCs represents low productivity. Here, strategy 1 is always favored. At low productivity, probability of growing to a size refugia relative to the predation risk is low because of low food availability. Thus, the optimal strategy for all individuals is to reproduce as soon as possible, at the cost of achieving size refugia. At intermediate productivity, either strategy can be favored depending on initial conditions (as discussed above). At high productivity, strategy 2 is always favored. Here, food is abundant, so that prey grow and achieve size refugia quickly.

*Domains of Attraction for the Alternative Equilibria*

To examine how individual fitness optimization favors one strategy or the other under the conditions that favor alternative equilibria (fig. 1C), I examined individual fitness of each strategy throughout the parameter space. For a



**Figure 2:** Effects of varying resource supply ( $S$ ) on the dominance of the different life-history strategies. ZNGIs for each strategy are as in figure 1. As MBCs are partially determined by resource supply, varying  $S$  varies the intercepts of each species' respective MBC. The double-lined MBCs with the lowest intercepts represent a low-productivity system. Strategy 1 alone represents the sole equilibrium. Solid MBCs with intermediate intercepts represent an intermediate-productivity system. Here, the equilibrium conditions are as in figure 1, and the two closed circles represent alternative stable equilibria where either strategy dominates depending on initial conditions. Dashed MBCs with the highest intercepts represent a high-productivity system. Here, the sole equilibrium is where strategy 2 dominates.

strategy to be favored, the average per capita fitness of individuals must be maximized relative to other strategies. Here, I take a novel approach and suggest that the per capita fitness of the different strategies are actually given by the function for prey dynamics ( $N$ ) relative to resources ( $R$ ) and predators ( $P$ ) in the above food web equations. From this, a simple way in which to convert the population dynamics of a strategy into the expected fitness of that strategy is to set the function describing per capita population growth ( $1/N \times dN/dt$ ) equal to the expected average fitness of that strategy ( $w$ ). Therefore, for individuals that reproduce early, and thus are unable to achieve size refugia (strategy 1), solving for the per capita prey ( $N$ ) dynamics in equation (1) gives the average per capita fitness function

$$w_1 = a'b'R - aP. \tag{11}$$

Similarly, solving for the per capita dynamics of both juveniles ( $N_j$ ) and adults ( $N_A$ ) for the strategy where individuals forgo early reproduction to grow to size refugia (strategy 2) in equation (5), the average per capita fitness function ( $w_2$ ) is given by

$$w_2 = (a'_j g'_j R - c'_j - a_j P)(a'_A b'_A - c'_A), \tag{12}$$

where  $a_j$  is the attack rate on juvenile prey. Setting  $w_1 = w_2$  gives the range of conditions where the average individual fitnesses of the alternative life-history strategies are

equal. To examine the line of equal fitnesses of each strategy on the same predator ( $P$ ) and resource ( $R$ ) axes as the ZNGIs and MBCs, setting  $w_1 = w_2$  (eq. [11] = eq. [12]) and solving for  $P$  gives

$$P = -\left(\frac{-c'Ra'b' - b'_A a'_A c'_A + b'_A Ra'_A a'_1 c'_A g'_1}{-a - a_1 b'_A a'_A c'_A}\right). \quad (13)$$

The line described by this equation shows the combinations of predator ( $P$ ) and resource ( $R$ ) abundance where the two life-history strategies have equal expected fitness. I term this line the “isofitness” line of the system. All combinations of  $P$  and  $R$  values above the isofitness line (i.e.,  $P$  greater than the value of eq. [13]) favor strategy 2, which delays reproduction and favors growth to achieve a refuge. Alternatively, all parameter values below the isofitness line (i.e.,  $P$  less than the value of eq. [13]) favor strategy 1, the strategy that reproduces early and does not achieve refugia. Figure 3 shows a typical isofitness line for this system on the  $P$ - and  $R$ -axes. The isofitness line follows a positive sloped trajectory with increasing  $R$ . As resource supply ( $R$ ) increases, the abundance of predators ( $P$ ) needed to cause individuals to select strategy 2 increases. This is because individual fitness is determined by the probability of being eaten by a predator relative to food availability that determines per capita reproductive output.

Figure 4 shows the isofitness line overlaid on the ZNGIs and MBCs of strategy 2 at intermediate resource supply (the ZNGIs cross between the MBCs). The position of the alternative equilibria discussed above can be considered with the isofitness line. In all cases, the isofitness line intersects at the same point as the two strategies’ ZNGIs (i.e., the unstable equilibrium point). At the intersection of the ZNGIs, both strategies are equally favored. The shaded

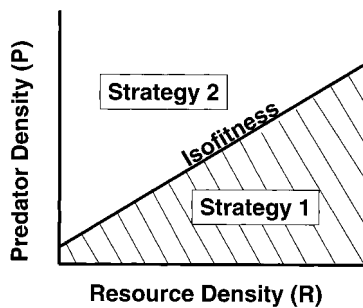


Figure 3: Range of predator ( $P$ ) and resource ( $R$ ) conditions that favor each life-history strategy. Isofitness line denotes the range of conditions in which each strategy has equal expected fitness (as determined by eq. [13] in text). Area below the line is the range where strategy 1 is favored. Area above the line is the range where strategy 2 is favored.

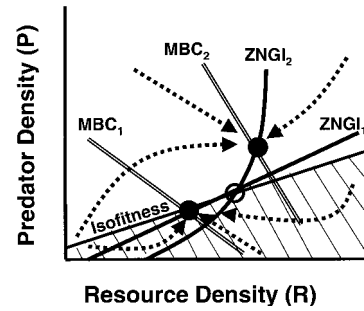


Figure 4: Isofitness line from figure 3 overlaid on the food web graphs from figure 1C. Ranges above and below the isofitness line represent domains of attraction that drive the system to either the upper or lower alternative stable equilibria depending on initial conditions. Dotted arrows qualitatively depict the direction the system will tend toward when started at different points along the phase space.

area below the isofitness line denotes the domain of attraction that carries the system to the lower stable equilibrium point dominated by strategy 1. The clear area above the isofitness line denotes the domain of attraction that carries the system to the upper stable equilibrium point dominated by the strategy 2.

From this analysis, it is interesting to note that individual fitness maximization causes the entire ecosystem to be structured in ways that might be very different than if individuals were not able to shift their life-history strategies. First is the fact that the system dominated by a relatively lower number of small reproductive individuals is locally stable under certain conditions, even though a higher biomass of large individuals in a size refuge is also locally stable under the same environmental conditions (i.e., fitness maximization does not equal biomass maximization). Second, the achievement of these alternative stable equilibria is determined by individual fitness maximization as mediated by the isofitness line. The system can be initially closer to one equilibrium than the other, but it is the conditions that an individual experiences relative to the isofitness line that will determine which equilibrium the system will achieve (fig. 4).

#### Conclusions and Some Limitations of the Models

The specific modeling scenario I have chosen, on the basis of that of Holt et al. (1994), does include several implicit assumptions that may limit a literal (i.e., quantitative) translation of the models with the reality of several empiric systems. Three of the assumptions that are most likely to influence the predictions of the models I have discussed here are the following: first, nutrients are assumed to be limiting so that the system can be assumed to be closed

in mass balance. This assumption will not hold if the limiting resource is energy. However, the qualitative predictions of this model will be identical if energy is assumed to be supplied externally and a slightly different modeling approach is used (Leibold 1996; Chase 1999). Second, predators have a Type I functional response, whereas a Type II response might be more realistic for many predators. The qualitative predictions of this model are robust to this assumption because the large invulnerable stage dampens the effects of the functional response. However, if the Type II response is very (perhaps unrealistically) strong, the quantitative predictions of the models can be altered. Third, I make the very simplistic assumption that prey can be divided into only two categories, whereas in reality, a more continuous function of size structure, reproduction, and susceptibility to predators is certainly more realistic. Analytically, however, this becomes a daunting task and, with it, the model will lose a great deal of generality. In the companion article (Chase 1999), I explored the rules for dominance of species with these types of dynamics. Here, the assumption of only two life stages was not likely to significantly affect the predictions of the model. However, in the present article, life-history decisions may be much more sensitive to this prediction. I present some discussion of this and associated assumptions and possible extensions of this model.

In the models discussed earlier, I have added a complexity to the typical food web models; specifically, that of the possibility for species to be plastic in a simple life-history decision that might importantly influence their lifetime fitness as well as the overall food web interactions. Evolutionary ecologists have frequently explored how environmental conditions such as food availability or mortality factors influence the plasticity of behavior and life-history responses of individuals. However, the inverse of this problem, exploring the implications of this plastic response to the food web context in which the species are imbedded has been explored with behavioral plasticity but not heretofore with a life-history problem.

I have modeled this complexity from a food web perspective, where such detail of the biology of species is rarely considered. However, obviously, while this is a complexity from a food web perspective, it is still a gross oversimplification of the processes of life-history optimization, growth, and reproduction. I have made several highly simplified (and in some cases suspect) assumptions about the processes of life-history plasticity. First and foremost, while I have explored how life-history plasticity responds to food and mortality factors, I have ignored how behavioral plasticity can modify these responses. It is well known that a species' behavioral plasticity can modify its interactions with food and mortality and have an important effect on the dynamics of the food web (e.g., Abrams 1984, 1991,

1993, 1996, 1999; Gleeson and Wilson 1986; Werner and Anholt 1993; Oksanen and Lundberg 1995; Fryxell and Lundberg 1997; Schmitz 1998). While it is likely that such behavioral plasticity can influence the dynamics I have discussed here, behavioral plasticity occurs on a much faster timescale (i.e., nearly instantaneous) and is highly reversible, whereas life-history plasticity takes place over a longer timescale and is not reversible. Thus, it can be reasonably approximated that behavioral plasticity can be abstracted in the context of a life-history plasticity model and would not likely influence the qualitative dynamics of the model.

As discussed earlier, perhaps one of the most unrealistic assumptions of the model is that the species is assumed to have only two stages, a susceptible juvenile and a reproductive adult. With this model structure, I implicitly assume that all individuals within a category have equal probabilities of growth or reproduction. This assumption cannot be strictly true. As it stands, the model assumes that any individual juvenile has an equal probability of growing to adulthood (dependent of food availability), whereas any individual adult has an equal probability of reproduction. The former assumption is particularly egregious. Instead, in a more biologically realistic model individual juveniles must pass through several developmental constraints before they can grow to the invulnerable adult stage. Such a stage-structured model would require a considerable amount of parameterization and not be particularly general. However, it is important to explore how the qualitative nature of the model might change with this added complexity (and realism). I performed a number of numerical simulations where various lags were introduced in the time it took from an individual's birth to the time it could become an invulnerable adult. While these lags had very slight effects on the relative positions of the ZNGIs and MBCs, the qualitative results were uniformly consistent with the much simpler two-stage model.

As a final consideration, I briefly discuss the limitations of using an implicitly fixed evolutionary model. The model I have used implicitly assumes that the phenotypic plasticity is a fixed character or trait of the species in question, and the species will express one or the other form of the plasticity depending on environmental conditions. That is, the model considers plasticity within an ecological context and is not able to deal with any influences of evolutionary selection that may constrain the model. For example, if there is a cost to maintaining the ability to be plastic, a model that allows selection would predict the loss of plasticity in low or high productivity environments, where only one strategy is always favored. Nevertheless, the type of model formulation presented here, where the expected fitness of different strategies are overlaid on an environmental template, may be able to provide important insights

into the ranges of conditions under which different strategies would be favored and selected for.

To conclude this theoretical section, it has not been my intention to identify all of the contingencies and to model correctly all of the complexities of a life-history problem, but rather to show how a simple depiction of this problem can be imbedded within the broader food web context to make qualitatively unique predictions. I have made use of an established procedure for examining food web interactions and modified them to examine how a form of life-history plasticity, specifically the ability to delay reproduction to achieve size refuge from predators, should influence and be influenced by food web interactions. The specific predictions this model makes are not novel, in and of themselves. This is because traditional life-history models explicitly account for age or stage-specific reproductive and mortality probabilities, and make predictions about which life-history strategies are optimal under which conditions (e.g., Law 1979; Michaud 1979; Charlesworth 1994; Abrams and Rowe 1996). Thus, given similar assumptions, these models make identical predictions to mine about which life-history strategies should be favored under which conditions of resource availability (which directly relates to reproductive probabilities) and predators (which directly relates to mortality probabilities). However, in the modeling framework I have presented here, it is possible to make an explicit link between individual fitness and optimization characteristics that are of interest to evolutionary biologists and the interactions within food webs that are of interest to ecologists. This link is not straightforward in the more traditional analyses. Furthermore, the framework I have presented provides explicit theoretical evidence showing how the lower-level processes of phenotypic plasticity can strongly influence the larger-scale patterns of ecological abundance and interactions.

In the following sections, I present results from an experimental system that, in part, motivated this theoretical study. First, I explore whether the system in question, a common, widespread, freshwater snail, conforms to the assumptions of life-history plasticity and vulnerability to predators. Next, I explore the predictions of the model, manipulating predators and initial conditions in experimentally controlled mesocosms. I show that both the assumptions and predictions of the modeling framework appear to be supported in this simplified aquatic food web.

#### Experimental Study System: Snails in Pond Food Webs

To explore the assumptions and predictions of this model, I used an experimental system in which both life-history plasticity and variable food web interactions have been documented. Freshwater snails (Mollusca: Gastropoda) are common and important consumers in temperate lakes and

ponds but are also prey to a number of predators (e.g., Lodge et al. 1987; Osenberg 1989; Martin et al. 1992; Bronmark et al. 1997; Chase 1998b). In addition, snails are known to be highly variable and plastic in a number of behavioral and life-history traits in response to both food availability and predation pressure (e.g., Osenberg 1989; Crowl and Covich 1990; Covich et al. 1994; Turner 1996; DeWitt 1998). I explored the model assumptions and predictions by using a wide ranging, abundant, and important herbivore, the pulmonate (lung-breathing) snail *Helisoma trivolis* (hereafter referred to as *Helisoma*), which was embedded in a food web consisting of a common snail predator, the insect *Belostoma flumineum* (Hemiptera: Belostomatidae; hereafter referred to as *Belostoma*) and several species of plants and algae that were potential sources of food for the snails. Experiments were performed and organisms were collected near the Kellogg Biological Station (Michigan State University) in southwestern Michigan (Barry County).

*Helisoma* is a relatively large, iteroparous snail that occurs primarily in small fishless ponds in the eastern and midwestern United States. It can be an important consumer of benthic primary productivity, and can be consumed by a variety of predators, including the abundant snail specialist *Belostoma* (Chase 1998b). Like other species of snails (e.g., Osenberg and Mittelbach 1989; Crowl and Covich 1990; DeWitt 1998), however, after growing through a susceptible juvenile phase, *Helisoma* can grow too large to be eaten by all of the predatory species that co-occur in these ponds. Such size refugia have important influences not only for this species' survival but for the dynamics of the entire food web (Chase 1998b).

#### *Tests of Model Assumptions: Life-History Plasticity in Response to Predators and Food*

To test the key assumptions of this model, that variation among individuals is plastic in response to predators and food, I examined the ability of *Helisoma* to respond in growth and size at first reproduction to predation and food. Crowl and Covich (1990) showed that a different snail species (*Physella virgata*) was able to respond to the presence of a chemical cue (i.e., smell) of predators eating conspecific snails by shifting their life history to grow larger and reproduce later in life (see also DeWitt 1998). However, these authors did not manipulate food levels, a potentially important factor that is predicted to covary with predation to influence life-history traits.

#### *Methods*

In 6-L plastic tubs placed outside under natural environmental conditions, I manipulated three levels each of

food and perceived predation risk for snails in a  $3 \times 3$  experimental design. Each treatment was replicated with six tubs. Young snails (1–2 wk old) were collected from egg pods that were laid in buckets containing a single adult snail. This was so that all snails used in these experiments were either full or half sibs (paternity within multiple clutches from a single snail can be mixed) to minimize genetic variation among experimental animals. Five snails were placed into each tub filled with ambient well water and an inoculation of algal species (snail resources) from local ponds. Resource and predation treatments were assigned randomly to tubs. To manipulate resource levels, nutrients (nitrogen [N] and phosphorus [P]) were added at three levels: first, ambient well water ( $1 \times$ ;  $200 \mu\text{g/L N}$ :  $40 \mu\text{g/L P}$ ),  $3 \times$  ambient nutrients ( $600 \mu\text{g/L N}$ :  $120 \mu\text{g/L P}$ ), and  $6 \times$  ambient nutrients ( $1,200 \mu\text{g/L N}$ :  $360 \mu\text{g/L P}$ ). This range of nutrients spanned the range of natural variation in pond productivity levels and stimulated algal growth accordingly (Chase 1998b). Three levels of predation risk were used: no risk, low risk, and high risk. Predation risk was manipulated by suspending 5-cm<sup>3</sup> permeable mesh (0.05-mm mesh) cages within each tub. No organisms were put into the no risk treatments. In the low risk and high risk treatments, one adult predatory *Belostoma* was placed into each cage. In these snails, risk appears to be conferred through chemical cues born in the water from consumed conspecifics and not from predator presence per se (as the insect alone gives no noticeable water borne cues; J. M. Chase, unpublished data). In treatments designated as low risk, predators were fed on average 0.25 small (4–7 mm shell length) *Helisoma* snails per day (i.e., one snail every fourth day), while in high risk treatments, predators were fed two snails per day.

Treatments were maintained for 4 mo to examine the size snails attained at first reproduction and their final size at the end of the experiment. I examined tubs every 3–7 d to explore for egg production. When one individual in a tub began reproduction (as indicated by the presence of yellow egg pods), the snails were measured, and the average shell length was recorded. At the termination of the experiment, snails were measured again to determine whether size at first reproduction and final size among snails were related. Finally, at the end of the experiment, snails from each tub were removed from their treatments, and all surviving individuals from each tub were placed in a 15-L bucket with one adult *Belostoma* to determine how susceptible snails from different treatments were to predators. After 48 h, any uneaten snails were assumed to be too large to be consumed by predators, because vulnerable snails are typically eaten within 1–2 h.

#### Results: Snail Size at First Reproduction

Results from this experiment showed that the size at first reproduction was strongly influenced by both resources (ANOVA,  $df = 2$ ,  $MS = 43.63$ ,  $F = 36.02$ ,  $P > .001$ ), predation risk (ANOVA,  $df = 2$ ,  $MS = 68.57$ ,  $F = 56.62$ ,  $P > .001$ ) and their interaction (ANOVA,  $df = 4$ ,  $MS = 9.74$ ,  $F = 8.04$ ,  $P > .001$ ; fig. 5). In particular, in the absence of predation risk, snails grew to a common, relatively small, size at first reproduction regardless of resource level (Tukey's HSD,  $P > .1$ ). In the low predation risk treatment, snail size at first reproduction was not different from the no risk treatments at the two lower food levels (Tukey's HSD,  $P > .1$ ), but snails grew to larger sizes before reproducing at the highest food level (Tukey's HSD,  $P < .05$ ). At the highest predation risk, size at first reproduction did not differ from the other predation risk treatments at the lowest food levels (Tukey's HSD,  $P > .1$ ), but individuals grew to much larger sizes before reproducing at both intermediate and high food levels (Tukey's HSD,  $P < .05$ ). In addition, size at first reproduction was strongly correlated with final size (regression;  $y = 1.23x + 0.11$ ,  $R^2 = 0.89$ ,  $P < .001$ ). Furthermore, both the slope ( $t$ -test;  $P > .13$ ) and intercept ( $t$ -test;  $P > .41$ ) were not significantly different from 1 and 0, respectively, suggesting size at first reproduction is a good indicator of final size.

#### Results: Snail Size-Specific Predation Risk

The proportion of individuals susceptible to predators in feeding trials (arcsine square-root transformed) varied with resources (ANOVA,  $df = 2$ ,  $MS = 2.52$ ,  $F = 24.56$ ,  $P < .001$ ), predation risk (ANOVA,  $df = 2$ ,  $MS = 4.84$ ,  $F = 74.36$ ,  $P < .001$ ) and their interaction (ANOVA,  $df = 4$ ,  $MS = 0.88$ ,  $F = 8.57$ ,  $P < .001$ ; fig. 6) in a manner directly inverse to the snail size at first reproduction. This

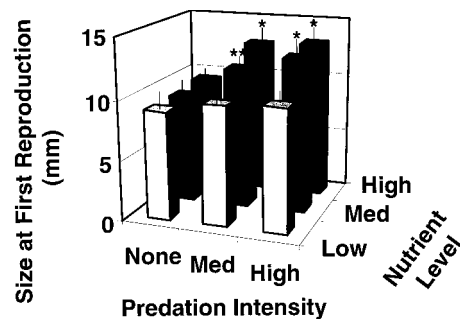
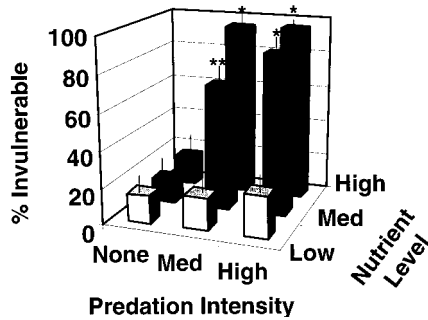


Figure 5: *Helisoma* mean size at first reproduction from the treatments' varying food levels and predation risk. Asterisks denote treatments that are significantly different from treatments with no asterisks (Tukey's HSD,  $P < .05$ ).



**Figure 6:** Percentage of *Helisoma* invulnerable to predators at the termination of the experiment varying food levels and predation risk. Asterisks denote treatments that are significantly different from treatments with no asterisks (Tukey's HSD,  $P < .05$ ).

shows that those individuals that delayed reproduction to grow larger were indeed considerably less vulnerable than those that grew to smaller sizes.

Thus, the key assumptions of the model presented above appear to be upheld. First, prey individuals of the common snail species *Helisoma* are highly plastic in their ability to grow and to reproduce; second, predation pressure and resource abundance are two primary factors that influence this plasticity; and third, this plasticity in size determines the prey's susceptibility to predators.

#### *Tests of Model Predictions: Influences of Life-History Plasticity on Food Webs*

To examine the predictions of the model presented above, I examined the dynamics and life-history characteristics of *Helisoma* in the context of a simplified food web. The model predicts that individuals should maximize their lifetime fitness by selecting one of the two basic life-history strategies: reproduce early but do not achieve size refugia (strategy 1) or defer reproduction to achieve size refugia from predation (strategy 2). The above experiments showed that *Helisoma* can achieve either of these strategies in a plastic way that depends on the interaction between food availability and predation pressure. Furthermore, the model predicts that these alternative life-history strategies can have profound effects on the realized food web structure of the system at intermediate levels of primary productivity. Specifically, when low initial prey : predator ratios favor individuals to exhibit life-history strategy 1, predators should have large and cascading effects on the food web. However, when high initial prey : predator ratios favor individuals to exhibit life-history strategy 2, predators should have weaker overall effects on the food web.

#### *Methods*

I performed an experiment in large controlled mesocosms (380-L cattle tanks) placed in array near the Kellogg Biological Station's experimental pond facility. These mesocosms provide an ideal media within which to examine food web interactions among these pond organisms in that they are easily controlled, yet adequately simulate many aspects of natural pond conditions (see e.g., Leibold and Wilbur 1992; Wilbur 1997; Chase 1998b). Because the model predicts life-history plasticity to have its most profound effects on food web structure at intermediate productivity, tanks were filled with well water, and nutrients (nitrogen [N] and phosphorus [P]) were added to approximate conditions of intermediate productivity observed in natural ponds (3,000  $\mu\text{g/L N}$  : 200  $\mu\text{g/L P}$ ; Chase 1998b). Natural densities of algae, macrophytes, duckweed, and zooplankton were collected from a variety of local ponds and inoculated into the mesocosms. Snails and predators were collected from natural ponds and stocked into the tanks within 1 wk after collection. Initially low or high snail abundance (10 or 40 *Helisoma*/tank) and the presence or absence of predators (0 or 10 second-instar *Belostoma* nymphs) was manipulated in a  $2 \times 2$  factorial design. Each treatment was replicated with four mesocosms, for a total of 16 mesocosms used in this experiment. Treatments were assigned to mesocosms randomly.

All of the species used in this simplified food web freely reproduced and completed their life cycles within the mesocosm environment. The experiment ran for a total of 180 d, allowing for a minimum of three generations of both insect predators and snail prey. Both predators and prey had significant numeric responses to the treatments and appeared to stabilize over the latter half of the experiment. Thus, this experiment was intended to explore the long-term community-level effects of life-history plasticity by allowing several generations of both snail and predator numerical responses so that they might achieve some sort of stable configurations in the different treatments. To quantify the transient dynamics and potential stability of these mesocosm food webs, snails and predators were censused every 15 d over the course of the experiment.

After the final census, all snails and predators were collected, counted, measured, and compared with species-specific length/dry weight conversions to estimate biomass. Plants (algae, duckweed, and macrophytes) were collected, dried, and weighed for biomass estimates.

At the termination of the experiment, snails were collected from their respective treatments and used in assays to determine the reproductive state and predator susceptibility of snails of different sizes from different treatments. Snails were divided into three categories, small (1–5 mm),

medium (6–10 mm), and large (>10 mm), to facilitate comparisons. Snails from each size category from each mesocosm (if there were any) were placed individually into small (1 L) containers at the termination of the experiment for up to 1 wk to examine if they were reproductive (as determined by egg production; these snails can store sperm for a very long time, and so it was not necessary to do assays with pairs of snails). Next, these same snails were placed with an adult *Belostoma* in feeding trials as above to examine the predator susceptibility of snails from the different treatments.

### Results

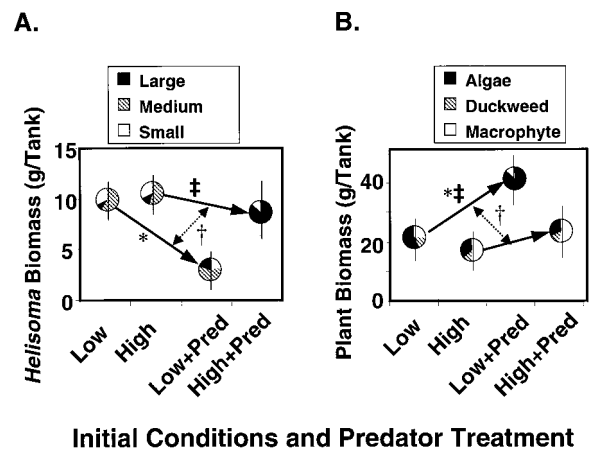
From the biweekly censuses, I estimated population growth and dynamics of the snail populations within each predator treatment. *Helisoma* populations in all treatments increased and then appeared to level off for the last half of the experimental time period. To quantify whether populations were stable by the end of the experiment, I averaged the change in abundance for each treatment from one census period to the next over the last 60 d of the experiment. For all treatments, the average change in abundance of each species was not significantly different from 0 (*t*-tests,  $P > .1$ ), indicating that the treatments appeared to be relatively stable. Furthermore, populations started at different initial snail densities without predators converged on similar numbers. Thus, the results of this experiment, and any effects due to initial treatment conditions, are more likely due to population level responses (i.e., alternative stable states) rather than differences in the initial starting conditions. I only present results from the final, exhaustive, censuses.

From the reproductive and vulnerability experiments performed on snails after the termination of the experiment, snails in the small size category (1–5 mm) were never reproductive (0% reproductive), regardless of treatment, and were always vulnerable to predators (100% of small snails consumed by predators). Snails in the medium size category (6–10 mm) were again generally susceptible to predators (84%  $\pm$  9% consumed). However, importantly, and in accordance with model predictions, these medium-size snails differed in their reproductive states depending on predator treatment (all percentages arcsine transformed; ANOVA,  $df = 1$ ,  $MS = 0.43$ ,  $F = 24.38$ ,  $P < .001$ ), initial condition treatment (ANOVA,  $df = 1$ ,  $MS = 0.54$ ,  $F = 31.21$ ,  $P < .001$ ), and their interaction (ANOVA,  $df = 1$ ,  $MS = 0.69$ ,  $F = 39.74$ ,  $P < .001$ ). Medium snails without predators in both the low and high initial density treatments were generally reproductive (73%  $\pm$  8% and 76%  $\pm$  6% reproductive, respectively). However, the presence of predators significantly influenced the effect of initial conditions on the reproductive state of medium snails

(Tukey's HSD,  $P < .05$ ). Medium snails in the low initial density treatment with predators were typically reproductive (83%  $\pm$  11% reproductive), whereas those in the high initial density treatment with predators were generally nonreproductive (14%  $\pm$  6% reproductive). Finally, large snails were almost always reproductive (91%  $\pm$  6% reproductive) regardless of treatment and were not susceptible to predators in feeding trials (0% consumed).

Predators reproduced frequently through the experiment, having up to three generations per mesocosm on average. There were no significant differences in predator abundance between any of the treatments (ANOVA,  $df = 1$ ,  $MS = 435.13$ ,  $F = 1.29$ ,  $P > .3$ ).

Figure 7A presents the results of the snail size structure and biomass from the experimental treatments at the termination of the experiment. Both snail biomass and size structure were influenced by predator presence and the interaction between predation and initial conditions (fig. 7A; table 1). However, biomass and size structure were not influenced by initial conditions alone, indicating that final differences in initial conditions treatments were likely due to the interaction with the presence of predators, and



**Figure 7:** A, Size structure and overall biomass of *Helisoma* from the mesocosm experiment manipulating predation and initial conditions. B, Abundance and composition of plants from the mesocosm experiment. Treatments are the following: *low* = low initial snail densities; *high* = high initial snail densities; *low + pred* = low initial snail densities plus predators; *high + pred* = high initial snail densities plus predators. Solid lines with arrows lead from each initial density treatment (low or high) without predators to its respective initial density treatment with predators. Asterisks next to a solid arrow indicate significant differences in the total biomass of herbivores or plants between treatments (ANOVA followed by Tukey's HSD;  $P < .05$ ). Double crosses next to a solid arrow indicate significant differences in the species composition (i.e., the relative proportion of each species) of *Helisoma* size classes (small, medium, or large) or plant groupings (algae, duckweeds, or macrophytes). Crosses next to a dotted line with double arrows indicate significant initial conditions by predator interactions (ANOVA;  $P < .05$ ).

**Table 1:** ANOVA results for several response variables from the mesocosm experiment

Dependent variable and source of variation	df	MS	F	P
Snail minimum reproductive size:				
Pred	1	14.06	5.870	.03*
Initial	1	39.06	16.30	.002*
Pred × initial	1	27.56	11.50	.005*
Snail susceptibility to predators:				
Pred	1	.89	21.34	.001*
Initial	1	.59	14.07	.003*
Pred × initial	1	1.09	25.93	<.001*
Snail biomass:				
Pred	1	107.74	18.28	.001*
Initial	1	132.71	22.52	<.001*
Pred × initial	1	119.24	20.24	.001*
Proportion small snails (1–5 mm):				
Pred	1	.00	.160	.69
Initial	1	.04	11.98	.01*
Pred × initial	1	.03	9.53	.01*
Proportion medium snails (5–10 mm):				
Pred	1	.38	30.03	<.001*
Initial	1	.48	37.98	<.001*
Pred × initial	1	.47	37.93	<.001*
Proportion large snails (>10 mm):				
Pred	1	.43	97.57	<.001*
Initial	1	.81	182.15	<.001*
Pred × initial	1	.78	176.00	<.001*
Plant biomass:				
Pred	1	967.21	24.51	<.001*
Initial	1	1,095.61	27.76	<.001*
Pred × initial	1	1,197.16	30.33	<.001*
Proportion algae:				
Pred	1	.14	8.88	.01*
Initial	1	.10	6.20	.03*
Pred × initial	1	.08	4.98	.04*
Proportion duckweeds:				
Pred	1	.01	5.85	.03*
Initial	1	.00	1.08	.32
Pred × initial	1	.00	.10	.76
Proportion macrophytes:				
Pred	1	.01	2.88	.12
Initial	1	.03	6.03	.03*
Pred × initial	1	.01	2.35	.15

Note: Factors include the presence and absence of predators (pred), the initial snail densities (initial), and their interaction (pred × initial). An asterisk indicates statistical significance.

not some other influence such as differences in time to reach equilibria. This result is expected if the communities had enough time for numeric responses to lead to some stable point, as the results from the biweekly censuses suggest. That is, it appears that there was enough time for the populations to respond beyond the initial transient differences that resulted from starting the populations at different densities. Regardless of initial conditions, snails

in the treatments without predators were dominated by a relatively high biomass of medium-sized individuals (Tukey's HSD,  $P > .1$ ). However, with predators, both snail biomass and size structure were strongly influenced by initial conditions (fig. 7A; table 1). At low initial snail densities, snails were dominated by small and medium-sized individuals, and were strongly reduced by predators. (Tukey's HSD,  $P < .05$ ) At high initial snail densities, large,

invulnerable individuals dominated the structure of the snail population, and predators did not influence overall snail biomass relative to the treatments without predators (fig. 7A). These results are in direct accordance with the predictions from the modeling framework presented above.

In addition to snail biomass and size structure varying with predators and initial conditions, so did the composition and abundance of other members of the food web (i.e., plants) in these mesocosms. Plants varied in both composition (algae, duckweed, and macrophytes) and abundance between predator and initial snail density treatments (table 1; fig. 7B). Plant biomass and composition was not influenced by initial snail densities without predators (Tukey's HSD,  $P > .1$ ). However, both plant abundance and composition varied considerably in the low and high initial snail densities with predators. Plant biomass was higher with predators (Tukey's HSD,  $P < .05$ ); this indicates that predators caused a trophic cascade in this treatment. Conversely, in the high initial snail treatment, plant biomass and composition was not influenced by predator presence or absence (Tukey's HSD,  $P > .1$ ); that is, no trophic cascade occurred in this treatment.

Remember that because both predators and prey went through several generations and had plenty of time for both life-history and numeric responses to differing food and predators, these food web results are not likely to be transient effects. Instead, the results presented here provide explicit experimental evidence on how life-history plasticity and optimal life-history decisions can influence both the abundance and size structure of prey species after several generations of predators and prey, as well as producing cascading, long-term effects on other members of the food web.

### Discussion

The domains of life-history, behavioral, and evolutionary ecology are rarely connected to the larger-scale dynamics of food webs and communities within which they are embedded because of the inherent complexities associated with doing so. However, some attempts at combining optimization with food web interactions have shown interesting theoretical results that are not borne out of food web models that do not consider such lower-scale processes (Abrams 1984, 1991, 1993, 1996, 1999; Gleeson and Wilson 1986; Oksanen and Lundberg 1995; Fryxell and Lundberg 1997; Grover and Holt 1998). In addition, some experimental studies have shown how behavioral optimization of predators and prey can influence food web dynamics (e.g., Power 1984; Turner and Mittelbach 1990; Chase 1998a; Schmitz 1998).

In this article, I examined the food web interactions of

a species with a simple life-history plasticity decision: reproduce early, at the cost of somatic growth, or forgo early reproduction and achieve a size refuge from predation, and then reproduce. I showed that allowing such plasticity in life-history decisions of a species can have interesting and important theoretical outcomes on the dynamics of the food web. At low productivity, strategy 1 is favored, but the system is consumer controlled. At high productivity, strategy 2 is favored, and consumer control is weakened. At intermediate productivity, strategy 1 or strategy 2, but not a mixture, can be favored depending on parameters and initial conditions. Here, individual optimization through isofitness analysis determines the domains of attraction and outcomes of food web interactions.

The predictions of the modeling framework I developed here can also be borne out of more traditional models of life-history optimization of age and size at first reproduction under varying mortality levels (e.g., Law 1979; Michaud 1979; Stearns 1992; Charlesworth 1994; Abrams and Rowe 1996) given similar assumptions. Traditional life-history modeling involves demographic analyses of stage or age specific parameters related to survival and reproductive output. However, none of these previous models have explored the influence of abundance of resources, prey, and predators on the final outcomes of life-history optimization, such as in the models I presented above. Furthermore, the theory I developed here, on the basis of food web models, provides an explicit link between life-history plasticity and the broader context of ecological interactions.

I tested some of the key assumptions and predictions of this modeling framework by using a common, important consumer in small pond food webs. The pulmonate snail *Helisoma* was shown to be highly plastic in its size at first reproduction, and in its ability to grow too large to be consumed by its common predator *Belostoma*. This plasticity resulted from variation in both resource abundance and predation risk. Furthermore, I showed that this life-history plasticity, resulting from variation in initial conditions, had a strong influence on both snail abundance and size structure, as well as on other members of the food web (i.e., plants) in experimental mesocosms.

In the modeling, I considered the two life-history strategies in relation to body size alone. However, body size per se is not necessary to achieve the sorts of dynamics discussed here; any trait that also varies with ontogeny and confers defense to a species can be similarly examined. Examples of such defenses include both mechanical (e.g., spines and thorns) and chemical defenses in both plants and animals. Any defensive trait that leads to a highly susceptible juvenile period, and a relatively invulnerable adult period, should lead to the results similar to those above.

Body size plays a key role, as both cause and effect, in a species' life history and ecology. Food availability and predators are two important factors that influence species' life histories and distribution and abundance. However, the link between life history and food web theory has rarely been made. The size of an individual determines its reproductive ability as well as its susceptibility to predators. If a prey individual has a high likelihood of being eaten, it should reproduce as soon as possible. Several types of prey become more susceptible to predators as they grow (e.g., fish feeding on zooplankton: Vanni 1987; Stibor 1992; intertidal limpets eaten by birds: Wootton 1997). If prey are more susceptible to predators with size, they should reproduce early and often. Such selective predation of larger, reproductive individuals does not substantially alter the dynamics of food web interactions from how they would be in the absence of such life-history plasticity. If, however, prey have the potential to grow too large to be effectively consumed, many conditions will lead to prey delaying reproduction in deference to growth (e.g., Law 1979; Michaud 1979; Charlesworth 1994; Abrams and Rowe 1996). As shown earlier, this form of life-history plasticity can have interesting and important effects on the outcomes of food web interactions.

Typical food web models assume individuals are unvarying throughout their lives. However, it is well known from evolutionary ecology that attributes of prey behavior and life history can vary considerably as functions of fitness optimization. Several authors have shown that prey across a wide range of taxa can grow larger and vary their morphology as a plastic response to reduce their risk in the presence of predators (e.g., Resnick 1983; Lively 1986*a*, 1986*b*; Crowl and Covich 1990; Resnick et al. 1990; Bronmark and Minor 1992; Spitze 1992; Wellborn 1994; Spitze and Sadler 1995; Belk 1998; DeWitt 1998). However, in all of these studies, the degree to which such life-history plasticity varied with environmental conditions, and how it influenced the dynamics of food web interactions, were not examined.

In this study, I have attempted to make an explicit link between the disparate research programs of life-history decisions, phenotypic plasticity, and food web ecology. By viewing life-history optimization within the broader community-level context, significant insights can be made at both levels. Further explorations into the link between lower level optimization of individuals with higher level ecological interactions in communities and ecosystems, both theoretical and experimental, should continue to provide interesting insights into the variation of food web interactions and community-level phenomena that are so commonly observed in nature.

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