

OPPOSITE SHIFTS IN SIZE AT METAMORPHOSIS IN RESPONSE TO LARVAL AND METAMORPH PREDATORS

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Abstract. Predation risk can cause organisms to alter the timing of life history switch points. Theory suggests that increased risk in an early life stage should select for switching earlier and smaller, while increased risk in the subsequent stage should select for switching later and larger. This framework has frequently been applied to metamorphosis in amphibians, with mixed results. Few studies examining the effect of larval predation risk on metamorphosis have observed the predicted pattern, and no studies, to our knowledge, have examined the effect of increased risk during and after metamorphosis on the timing of this switch point. Here we examine the effect of larval and post-metamorphic predation risk on metamorphosis in the red-eyed treefrog, *Agalychnis callidryas*. We raised tadpoles in the presence or absence of cues from caged water bugs fed larvae and cues from spiders fed emerging metamorphs. Water bugs are effective larval predators, while spiders are poor larval predators but prey on metamorphs. Furthermore, since spiders forage on the water surface it is possible that tadpoles could assess future risk from this predator. Predators induced opposite shifts in life history. Tadpoles emerged smaller and less developed in response to water bugs, but later and larger in response to spiders. Interestingly, predator effects on larval duration were not independent; tadpoles delayed emerging in response to spiders, but only in the absence of water bugs.

Key words: complex life cycles; habitat shifts; induced defenses; leaf-breeding treefrogs; life history; metamorphosis; multiple predator effects; ontogenetic shift; phenotypic plasticity; predation risk; Smithsonian Tropical Research Institute; trait-mediated indirect effects.

INTRODUCTION

Many species from diverse taxa have evolved complex life histories in which the organism undergoes a series of transitions between successive stages with different selective pressures. Switch points between life stages, such as hatching and metamorphosis, are typically irreversible and may be accompanied by rapid morphological transformations, shifts in habitat, and changes in ecology. They can thus be thought of as ontogenetic niche shifts and are focal points for selection by two potentially variable environments. Variable environmental conditions in either or both stages can select for plasticity in switch point timing. As growth opportunities and predation risk vary between stages, the ability to time irreversible life history switch points in a way that enhances fitness is an important adaptation.

A number of theoretical studies have examined how individuals should shift the size and time of life history switch points in response to variation in growth and risk across life stages (recently reviewed in Benard [2004]). Werner and Gilliam (1984) and Werner (1986)

proposed that an organism should optimize the size and age of switching based upon size-specific mortality (μ) and growth (g) rates in larval and post-metamorphic habitats. Fitness is maximized when organisms switch stages in order to minimize the ratio of mortality rate over growth rate across life stages. Within this framework, increased mortality in an early stage results in metamorphosis at a smaller size and earlier age, while increased mortality in a later stage results in larger and later metamorphosis. While the “minimize μ/g ” rule is elegant in its simplicity and intuitively appealing, it assumes that reproduction is continuous and indefinite. As a result, it predicts a single size at metamorphosis for a given set of μ/g . Ludwig and Rowe (1990) and Rowe and Ludwig (1991) investigated the consequences of this assumption using dynamic optimization models with seasonal time constraints (e.g., reproduction, pond drying). These models better match the empirical evidence for many species, in that they predict an optimal size and age at switching that changes through the season. However, all else being equal their model makes similar predictions about the effect of stage-specific mortality on the timing of life history shifts.

Empirical studies testing for predator/pathogen-induced plasticity in the timing of life history switch points have focused on both hatching and metamorphosis. Predator-induced plasticity in the timing of hatching has been demonstrated in diverse taxa (e.g., salamanders, Sih and Moore 1993; frogs, Warkentin

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1995, Chivers et al. 2001, Laurila et al. 2002, Schalk et al. 2002, Johnson et al. 2003, Vonesh 2005; spiders, Li 2002; fish, Wedekind 2002) and is generally consistent with the predictions of the minimize μ/g framework (but see Laurila et al. 2002, Johnson et al. 2003). In response to increased embryonic risk, organisms frequently hatch earlier at a smaller size (e.g., Warkentin 1995, Johnson et al. 2003, Vonesh 2005). Hatching early is an effective strategy for escaping embryonic predation, but comes at the cost of reduced performance in subsequent stages (Warkentin 1999, Buckley et al. 2005, Vonesh 2005). In response to increased larval risk, organisms may delay hatching to hatch at larger sizes, reducing their exposure to larval predators and potentially increasing their survival in encounters with size-specific larval predators (Sih and Moore 1993, Moore et al. 1996). Nearly all of these studies focus on hatching plasticity in response to risk in a single stage. While a few studies have examined responses to both embryonic and larval predation risk (e.g., Laurila et al. 2002, Johnson et al. 2003), these studies do not test for interactions among stage-specific predators. To our knowledge, no studies have examined whether plastic responses to predators of different life stages are independent.

Theory has been less successful at predicting how predation risk affects the timing of metamorphosis in insects and amphibians. While most of the models above predict that larvae should metamorphose both smaller and earlier in response to increased larval stage risk, this prediction has been supported in only two of 40 experiments reviewed by Benard (2004). Two studies found that larvae metamorphosed earlier but at the same size (Laurila et al. 1998, Chivers et al. 1999), and 10 studies found larvae emerged smaller but at the same time or later. Interestingly, six studies observed the opposite of the response predicted by theory; metamorphosis occurred later and at larger sizes in the presence of larval predators. In the remaining studies, risk had no effect on metamorphosis. Benard (2004) discusses three mechanisms that may explain this mismatch between theoretical and empirical studies. In some experimental venues, predators may have indirect positive effects on prey growth resulting in larger rather than smaller size at metamorphosis. This effect is mediated through increased resource availability due to reduced foraging in the presence of predators (e.g., Peacor 2002). Another hypothesis is that larvae emerge later and larger because later in larval ontogeny they attain a size refuge from aquatic predators. This explanation is based on evidence that predation risk often declines with larval size. Larvae may reduce foraging early in ontogeny when they are vulnerable to predators and then increase foraging later in ontogeny after reaching a size refuge. In order to make up for reduced initial growth, organisms may remain larvae longer and emerge later and at larger sizes in the presence of predators than larvae in predator free environments (Van

Buskirk and Schmidt 2000). Finally, predator-induced changes in behavior or morphology can alter larval size-specific risk and growth, and this could potentially result in later and larger optimal timing of metamorphosis in the presence of predators.

While numerous studies have examined metamorphic shifts in response to larval predation risk, no studies, to our knowledge, have examined the effect of increased risk during and after metamorphosis on the timing of this switch point. There are at least two factors that have contributed to this gap in empirical studies of predator-induced switch point plasticity. First, post-metamorphic mortality risk is often more difficult to manipulate and quantify than larval mortality risk. Second, in order to exhibit adaptive plasticity in response to post-metamorphic risk, larvae must be able to estimate this future risk. Since larvae have little direct opportunity to sample post-larval habitats, it is not clear how they could respond to fine-scale (i.e., within generation) variation in post-metamorphic risk (Sih and Moore 1993).

While many species undergo ontogenetic niche shifts between life history stages, leaf-breeding treefrogs are particularly suitable for studies of switch point plasticity. Eggs, larvae, and frogs occur in different habitats, allowing the stage-specific selective pressures that shape plasticity in both hatching and metamorphosis to be isolated and quantified. Here we examine predator-induced plasticity in life history switch points in the red-eyed treefrog, *Agalychnis callidryas*. This species is widespread and locally common in low elevation forests from Yucatan through Panama (Duellman 1970). Eggs are attached to vegetation over ponds and swamps where they are vulnerable to arboreal and aerial predators. Hatched larvae drop into the water where they face aquatic predators. Hatching plasticity in response to embryonic risk is well documented in this species. Early hatching is an effective strategy for escaping from several species of egg-eating snakes (Warkentin 1995), wasps (Warkentin 2000), and pathogenic fungi (Warkentin et al. 2001). In this study, we test effects of larval and post-metamorphic predation risk on the timing of the next life history switch point, metamorphosis. We raised larvae in the presence or absence of cues from aquatic predators, *Belostoma* sp. giant water bugs, and predators of emerging metamorphs, *Thaumasia* sp. spiders. Water bugs and aquatic spiders are common predators of pond breeding amphibians in both temperate and tropical systems (e.g., water bugs, Brodie and Formanowicz 1983, McIntyre et al. 2004; spiders, Formanowicz et al. 1981, Hayes 1983, Donnelly and Guyer 1994), and both are abundant at our *A. callidryas* study ponds. Water bugs prey upon all size classes of *A. callidryas* larvae and prefer larger larvae in choice tests (J. R. Vonesh and K. M. Warkentin, unpublished data). Spiders are poor larval predators but are effective predators of emerging metamorphs (J. R. Vonesh and K. M. Warkentin, unpub-

lished data). Spider predation rates on frogs often decrease with increasing prey size (Formanowicz et al. 1981, Vonesh 2005). Furthermore, since these spiders forage on the water surface it is possible tadpoles could assess future risk from this predator. Given this pattern of stage- and size-specific risk to these predators, based on the μ/g framework we would expect that *A. callidryas* larvae should metamorphose earlier at smaller sizes in the presence of water bugs and later at larger sizes in the presence of spiders.

MATERIALS AND METHODS

To test for predator-induced plasticity in *A. callidryas*' behavior and life history we conducted an experiment in which larvae were exposed to cues from caged giant water bugs (*Belostoma* sp.) fed tadpoles and caged aquatic spiders (*Thaumasia* sp.) fed emerging metamorphs. This experiment was conducted in an ambient conditions laboratory at the Smithsonian Tropical Research Institute in Gamboa, Panama between July and October of 2004. We used a completely randomized 2×2 factorial design with four treatments: (1) no predator, (2) water bug alone, (3) spider alone, and (4) water bug and spider. Each treatment was replicated 10 times in an array of 60-L plastic tubs (40 cm deep \times 44 cm diameter) filled to a depth of 35 cm with aged tap water. Predator cages (40 cm deep \times 10 cm diameter) were constructed from fiberglass window screen (mesh diameter 1.2 mm) and suspended in the water on one side (determined randomly) of the tub. These enclosures allowed predators to access the surface and entire water column. Tub covers were covered with fine nylon mesh secured with elastic to prevent frogs from escaping and colonization by nonexperimental organisms.

Predators were collected from Ocelot and Quarry Ponds prior to the start of the experiment. Water bugs are effective predators of all sizes of *A. callidryas* larvae. In short-term predation trials, adult water bugs consumed an average of 5.1 ± 2.5 larvae \cdot d $^{-1}$ \cdot predator $^{-1}$ (mean \pm SD, $n = 18$ trials) across six size classes of *A. callidryas* larvae (range in larval total length [TL], 10.85–49.27 mm). While vulnerability to this predator appears to be hump-shaped with respect to larval size (J. R. Vonesh and K. M. Warkentin, unpublished data), *A. callidryas* larvae never attain a size refuge from this predator. Water bugs consumed 3.0 ± 0 larvae \cdot d $^{-1}$ \cdot predator $^{-1}$ ($n = 3$) of even the largest larval size class (TL = 44.77 ± 2.14 mm, mean \pm SD). In contrast, spiders are poor predators of *A. callidryas* larvae. Adult *Thaumasia* sp. spiders consumed only 0.5 ± 0.4 larvae \cdot d $^{-1}$ \cdot predator $^{-1}$ ($n = 20$) across larval sizes (range in larval TL, 9.8–44.2 mm). However, spiders were more effective predators of emerging metamorphs, consuming an average of 1.8 ± 0.5 metamorphs \cdot d $^{-1}$ \cdot predator $^{-1}$ ($n = 12$) in similar predation trials (J. R. Vonesh and K. M. Warkentin, unpublished data).

Agalychnis callidryas larvae used in the experiment were reared from 25 clutches collected from Ocelot Pond on 26 July. After hatching, larvae were pooled into a single 60-L tub and fed powdered alfalfa ad libitum until the start of the experiment. On 18 August, larvae and predators were randomly assigned to treatments, digitally photographed, and added to experimental tubs. Fifteen larvae were added to each replicate. A caged water bug, caged spider, or both were added to the predator treatments. Predators were in individual cages, with both cages on the same side in the multiple predator treatment. An empty cage served as a cage control in the no predator treatment. Predator and larval sizes were measured from digital photographs using the open source image analysis software ImageJ (available online;² total length [mean \pm SD]: water bugs, 39.92 ± 1.78 mm; spiders, 18.67 ± 2.08 mm; larvae, 21.83 ± 0.76 mm). There were no differences in initial larval or predator size among treatments (larvae, $F_{3,36} = 0.576$, $P = 0.634$; water bugs, $F_{1,18} = 0.062$, $P = 0.807$; spiders, $F_{1,18} = 0.138$, $P = 0.714$).

Predators and larvae were fed at 2–3 d intervals over the duration of the experiment (63 d). At each feeding, water bugs were fed an *A. callidryas* larva (Gosner stage 24–36; Gosner 1960) and spiders were fed an emerging metamorph (Gosner stage 41–44). Larvae were maintained on alfalfa powder ad libitum. To prevent fouling, excess food was removed weekly and water was replaced about every 14 d. Molting or dead predators and spiders with egg cases were replaced with similar sized individuals (four water bugs and 16 spiders replaced). Behavioral responses to treatments were quantified on two dates (31 August and 7 September) between 09:00 and 12:00 by a single observer. We recorded instantaneous counts of (1) the vertical location of the predator(s) and (2) each larvae, (3) the number of larvae in the half of the tub away from the predator cage, and (4) the number of larvae actively swimming (excludes larvae maintaining their position mid-water or slowly drifting). We averaged responses from these two dates and used these means as our response variables in subsequent analyses. On 8 September, larvae were removed, photographed, and measured in order to estimate larval growth rates. At this time we removed a random subsample of five larvae from each replicate for morphometric analyses (data not reported here). The remaining 10 larvae were returned and followed through to metamorphosis. As larvae neared metamorphosis, tubs were checked daily for emerging metamorphs. Metamorphs were removed from the experiment when they climbed out of the water. Immediately upon removal, we measured tail length (nearest 0.1 mm using dial calipers), then held them until complete tail absorption (Gosner stage 46; 2–3 d) and then measured snout–vent length (nearest 0.1 mm using dial

² (<http://rsb.info.nih.gov/ij/>)

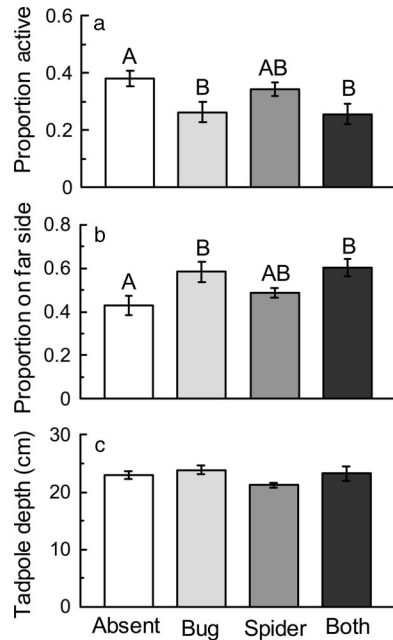


FIG. 1. Behavioral responses of *Agalychnis callidryas* tadpoles to larval and metamorph predator cues. (a) Proportion of tadpoles active, (b) proportion of tadpoles on the side of the container opposite the predator cages, and (c) average larval depth in the water column (height above the bottom). Bars show treatment means \pm SE. Bars with different letters were statistically different based on Fisher's LSD post hoc tests at $\alpha = 0.05$.

calipers) and mass (nearest 0.001 g using an electronic balance).

We tested for treatment effects on *A. callidryas*' behavior and life history using a two-factor multivariate analysis of variance (MANOVA) with water bug and spider as factors. Behavioral response variables included: (1) mean larval depth, (2) proportion of larvae on the side away from the predator cage, and (3) proportion of larvae active. Life history response variables included: (1) proportion surviving, (2) mean larval growth rate, (3) mean duration of larval period, (4) mean tail length at emergence from the water, and (5 and 6) mean mass and snout-vent length at tail absorption. Assumptions of normality and homoscedasticity of errors were assessed via Shapiro-Wilks and Bart-

lett tests, respectively. Proportional response variables were arcsine square-root transformed. Univariate ANOVA followed by Fisher's LSD post hoc test, where appropriate, were used to test for differences in individual response variables among treatments.

RESULTS

Giant water bugs and spiders foraged at different depths. Water bugs were only observed below the water surface (depth 26.3 ± 3.9 cm, mean and 95% CI, surface at 35 cm) while spiders foraged at or above the surface (36.8 ± 0.6 cm). The presence of the other predator had no effect on focal predator depth (water bug, $F_{2,17} = 1.167$, $P = 0.335$; spider, $F_{2,17} = 0.0001$, $P = 1.0$).

Agalychnis callidryas larvae altered their behavior in the presence of water bugs (MANOVA, Wilks' $\lambda_{3,34} = 6.02$, $P = 0.002$) but not in the presence of spiders (Wilks' $\lambda_{3,34} = 1.63$, $P = 0.201$). There was no significant interaction between bug and spiders on behavior (Wilks' $\lambda_{3,34} = 0.407$, $P = 0.749$). In the presence of water bugs, larvae decreased activity proportionally by $\sim 30\%$ (ANOVA, $F_{1,36} = 11.58$, $P = 0.002$; Fig. 1a), increased avoidance of the predator side of the container proportionally by $\sim 35\%$ ($F_{1,36} = 11.58$, $P = 0.002$; Fig. 1b), and tended to swim nearer the surface ($F_{1,36} = 3.034$, $P = 0.09$; Fig. 1c).

Both the presence of water bugs (MANOVA, Wilks' $\lambda_{6,31} = 0.503$, $P = 0.001$) and spiders (Wilks' $\lambda_{6,31} = 0.673$, $P = 0.043$) altered *A. callidryas*' life history. The overall water bug-by-spider interaction term in our MANOVA was not statistically significant (Wilks' $\lambda_{6,31} = 0.798$, $P = 0.283$). However, we included the interaction term in subsequent analysis of individual response variables because it was part of our initial design. Neither caged predator altered larval growth rates or survival (Table 1). Both growth rates and proportion surviving to emergence were high in all treatments (growth, mm TL/d; no predator, 1.23 ± 0.035 ; water bug, 1.20 ± 0.023 ; spider, 1.25 ± 0.036 ; both, 1.22 ± 0.045 ; survival, no predator, 0.88 ± 0.025 ; water bug, 0.86 ± 0.037 ; spider, 0.89 ± 0.038 ; both, 0.94 ± 0.022 ; mean \pm SE). Both water bugs and spiders had significant main effects on metamorph mass and snout-vent length (Table 1). In the presence of water bugs, metamorphs were 5% lighter (0.038 g, Fig. 2b) and 1.5%

TABLE 1. Results of ANOVA on the effects of water bugs and spiders on larval *Agalychnis callidryas* life history.

Life history variable	Water bug effect		Spider effect		Water bug \times Spider	
	F	P	F	P	F	P
Larval growth rate	0.586	0.449	0.344	0.561	0.008	0.929
Larval survival	0.759	0.390	2.425	0.128	0.505	0.482
Time to emergence	3.074	0.088	7.161	0.011	7.161	0.011
Metamorph mass	13.92	0.001	4.833	0.034	0.322	0.574
Metamorph SVL	21.91	<0.001	6.385	0.016	0.757	0.390
Tail length at emergence	11.89	0.001	0.342	0.562	0.044	0.836

Note: For all ANOVAs, $df = 1, 36$.

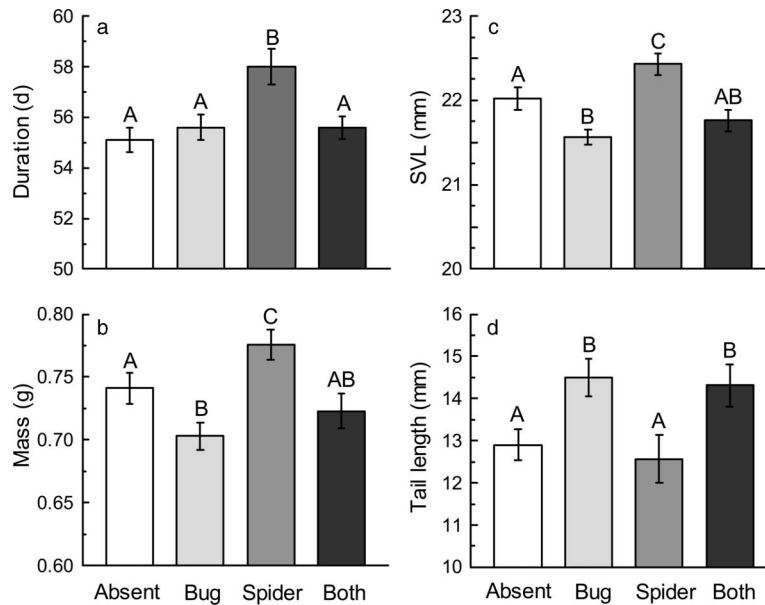


FIG. 2. Life history responses of *A. callidryas* tadpoles to larval and metamorph predator cues. Panels show treatment means \pm SE for (a) larval duration (days to emergence), (b) mass at metamorphosis (Gosner 46), (c) snout-to-vent length at metamorphosis (Gosner 46), and (d) tail length at emergence.

shorter (0.33 mm SVL, Fig. 2c), while in the presence of spiders, metamorphs were 5% heavier (0.035 g, Fig. 2b) and 2% longer (0.41 mm SVL, Fig. 2c) than those from the no predator treatment (Table 1). In addition, metamorphs emerging from the water bug treatment had significantly more tail remaining to be absorbed (12%, 1.5 mm, Fig. 2d) than those from the no predator treatment. There were no significant interactions between predators on these response variables (Table 1). The effects of predators on larval duration were more complicated. Water bugs appeared to have no effect on larval duration (Fig. 2a), while spiders caused metamorphs to delay emergence by 5.3% (2.9 d, Fig. 2a). Interestingly, predator effects on larval duration were not independent. Larvae emerged early in response to spiders only in the absence of water bugs (Table 1; Fig. 2a).

DISCUSSION

Our results show that *A. callidryas* larvae altered their behavior and shifted metamorphosis in response to predation risk in both current and future life stages and that these responses were not always independent. Larvae reduced activity, and emerged smaller and less developed (i.e., more tail remaining) in response to cues from larval predators. Larvae did not alter their behavior in response to post-metamorphic predators, suggesting larvae did not perceive these predators as an immediate threat. However, larvae did delay emergence and emerged larger in response to cues from post-metamorphic predators. Since predator treatments did not affect larval growth rate and larvae are unlikely to have the opportunity to sample growth rates in the

post-metamorphic arboreal habitats, these responses are likely driven by perceived differences in stage-specific predation risk. While delayed hatching in response to larval predators has been shown in other amphibians (Sih and Moore 1993, Moore et al. 1996, Schalk et al. 2002), to the best of our knowledge, this is the first example of delayed metamorphosis in response to post-metamorphic predation risk. Furthermore, while a few studies have examined life history switch point plasticity in response to different stage-specific predators (e.g., effects of egg and larval predators on hatching; Laurila et al. 2002, Johnson et al. 2003), ours is also the first study to test whether prey respond independently to risk in current and future life stages. Our results show that larvae delay emergence in response to post-metamorphic risk only in the absence of the larval predator.

We found no trade-off in growth associated with reduced activity in the presence of larval predators. We can think of two possible explanations for this. First, we maintained our tadpoles on ad libitum food rations. In a superabundant resource environment, it may be possible to maintain high growth rates at low activity levels (reviewed in Peacor and Werner [2004]). Second, *A. callidryas* are primarily water column filter feeders. Previous studies of predator-induced metamorphic plasticity in amphibians have usually focused on taxa that graze periphyton. Feeding, activity, and predation risk may be less closely related for filter feeders compared with periphyton grazing larvae. Grazers must move from patch to patch as resources are depleted, whereas filter feeders can likely maintain feeding rate while suspended nearly motionless or drifting in the

water column. This is likely a more common result than is generally appreciated; most studies that have looked for an effect of predator cues on growth rate prior to metamorphosis have failed to find an effect (Benard 2004).

Our results with respect to size at metamorphosis are consistent with the minimize μ/g framework. When larval risk is greater than post-metamorphic risk, individuals are expected to forgo growth in order to reduce exposure to predation and switch at smaller sizes. However there is likely a trade-off for switching smaller, as smaller size at metamorphosis has been shown to have negative fitness consequences in other amphibians (e.g., Altwegg and Reyer 2003, Vonesh 2005). When larval risk is lower than post-metamorphic risk, individuals are expected to switch at larger sizes. Switching later at a larger size may have a number of positive consequences for fitness (e.g., earlier time to sexual maturity, larger size at maturity) and is likely an adaptive response to spider predation, as vulnerability to spider predation decreases with increasing size in juvenile frogs (Formanowicz et al. 1981, Vonesh 2005). The cost associated with delaying metamorphosis, aside from larval predation risk, may be associated with time constraints on larval habitats (e.g., pond drying) and/or reproduction. The effects of predators on the timing of metamorphosis are less consistent with the minimize μ/g framework. We found no evidence of earlier switching in the presence of increased larval risk (i.e., water bugs). We did observe delayed metamorphosis in response to increased post-metamorphic risk (i.e., spiders). However, this response was only observed in the absence of water bugs.

Benard (2004) highlighted three alternative mechanisms to explain the lack of consistency between theory and empirical results in past studies that manipulated larval risk: indirect effects, predator size-refugia, and behavioral and/or morphological plasticity. However, none of these hypotheses seems to explain our results. First, indirect effects mediated through resources are unlikely to be important in our study, as food rations were not limiting and larval growth rates were consistent across treatments. Prey size refugia are not likely to be important in our study, as water bugs prey on all size classes of *A. callidryas* larvae. Finally, for predator-induced behavioral or morphological plasticity to shift the optimal strategy to later metamorphosis, predator-induced plasticity would have to decrease the overall mortality rate to below the mortality rate in the absence of the predator (Benard 2004). Most empirical studies (not surprisingly) find the opposite: higher mortality in the presence of predators. Thus, in general, predator-induced plasticity is predicted to give rise to later metamorphosis under few ecologically realistic scenarios. Instead, we suggest that at the high growth and development rates observed in our ad libitum resource conditions, there may have been physiological/developmental constraints preventing even earlier

emergence in response to water bugs. We plan to examine this question in future experiments, which manipulate resources as well as stage-specific risk. The result that larvae delayed emergence in response to spiders only in the absence of water bugs may be a short-term response to risk at metamorphic climax in which larvae respond to the more effective and immediate predation risk.

While our results show predator-induced plasticity in metamorphosis, the magnitude of these effects was not large. Larvae metamorphosed 5% lighter, 2% shorter, and with 12% longer tails in response to giant water bugs and 5% later, 5% heavier, and 2% larger in response to spiders compared to the no predator treatment. Although differences of this magnitude may have important consequences for individual fitness, it is worth pointing out that these differences are relatively small compared with differences due to, for example, variation in habitat quality among ponds. In experiments in which we raised *A. callidryas* larvae at specific densities in field enclosures, larvae from the lowest quality site (in terms of growth) emerged 63% later and 24% lighter than those from the highest quality site (J. R. Vonesh and K. M. Warkentin, *unpublished data*). Tadpoles in this study had larval durations similar to those reared at low densities in high quality ponds in the field experiment, but had 51% greater mass at metamorphosis. This indicates that the larvae in our experiment, which were fed ad libitum, experienced growth rates higher than those typically experienced in the field.

Life history switch points, such as hatching and metamorphosis, are ecologically pivotal events involving rapid changes in habitat, resource use, and/or morphology that dramatically alter species interactions. They are also foci of natural selection, because switching decisions are irreversible and small changes in switching strategies can have large effects on survival. Phenotypic plasticity of switch points is well documented, and the existence of trade-offs that make this plasticity adaptive is known for many systems. However, both the individual-level trade-offs that select for plasticity and the ecological consequences of plastic responses for populations depend on their environmental context. Understanding the selective pressures that shape plasticity requires information about the strength and form of trade-offs as well as the distribution of environmental variation. Future work will focus on evaluating the consequences of variation in multiple selective pressures across sequential variable environments. *Agalychnis callidryas* is the only species, to our knowledge, demonstrated to alter switch points in response to cues from predators of three succeeding life stages. Previous studies have shown that *A. callidryas* embryos hatch earlier in response to embryonic predation, and here we show this species also shifts metamorphosis in response to larval and post-metamorphic predators. These findings make *A. calli-*

dryas an ideal species for studies examining how variation across embryonic, larval, and post-metamorphic environments shape individual life history and how plasticity in individual life history contributes to demography.

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