REVIEW

Getting out alive: how predators affect the decision to metamorphose

Rick A. Relyea

Abstract Metamorphosis has intrigued biologists for a long time as an extreme form of complex life cycles that are ubiquitous in animals. While investigated from a variety of perspectives, the ecological focus has been on identifying and understanding the ecological factors that affect an individual's decision on when, and at what size, to metamorphose. Predation is a major factor that affects metamorphic decisions and a recent review by Benard (Annu Rev Ecol Evol Syst 35:651–673, 2004)) documented how predator cues induce metamorphic changes relative to model predictions. Importantly, however, real predators affect larval prey via several mechanisms beyond simple induction. In this paper, I contrast the leading models of metamorphosis, provide an overview of the multiple ways that predators can directly and indirectly affect larval growth and development (via induction, thinning, and selection), and identify how each process should affect the time to and size at metamorphosis. With this mechanistic foundation established, I then turn to the well-studied model system of larval amphibians to synthesize studies on: (1) caged predators (which cause only induction), and (2) lethal predators (which cause induction, thinning, and selection). Among the caged-predator studies, the chemical cues emitted by predators rarely induce a smaller size at metamorphosis or a shorter time to metamorphosis, which is in direct contrast to theoretical predictions but in agreement with Benard's (Annu Rev Ecol Evol Syst 35:651–673, 2004) review based on a considerably smaller dataset. Among the lethal-predator studies, there is a diversity of outcomes depending upon the relative importance of induction versus thinning with the relative importance of the two processes appearing to change with larval density. Finally, I review the persistent effects of larval predators after metamorphosis including both phenotypic and fitness effects. At the end, I outline a number of future directions to allow researchers to continue gaining insight into how predators affect the metamorphic decisions of their prey.

Keywords Phenotypic plasticity · Environmental heterogeneity · Evolution · Life history

Introduction

The process of metamorphosis has long fascinated biologists from a range of disciplines (Gilbert and Frieden 1981; Kaltenbach 1996; Wassersug 1997; Hall and Wake 1999; Truman and Riddiford 2002). The ecological focus on metamorphosis, especially among insect and amphibian ecologists, has striven to understand how genetic and environmental factors combine to cause organisms to make adaptive decisions on both the time to metamorphosis and the size at metamorphosis (Rose 2005). Amphibian ecologists have had a particularly strong interest in metamorphosis because so many species of amphibians undergo dramatic changes between the larval and adult stages. As a result, amphibians have inspired numerous models of metamorphosis as well as a tremendous amount of empirical work (Wilbur and Collins 1973; Wilbur 1980; Werner 1986; Newman 1992; Benard 2004).

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A wide range of factors are important in affecting the size and time to metamorphosis including the intensity and timing of competition (Barnett and Richardson 2002; Resetarits et al. 2004), degradation of the larval environment (Denver et al. 1998), and the presence of predators (Werner 1986; Benard 2004). During the past decade, numerous studies have focused on the effects of predators on amphibian traits with the goal of obtaining a mechanistic understanding of metamorphic decisions. While a recent review examined how predator cues (i.e., caged predators) affect metamorphic outcomes (Benard 2004), it did not consider the impact of real predation which can produce substantially different outcomes. The objective of this review is to take a more comprehensive approach in order to understand how predators affect metamorphosis by including both predator cues and lethal predators. In doing so, I use the extensive data on amphibians as a model system, but the insights gained apply to a wide variety of metamorphosing organisms.

This review has five major sections. First, I compare and contrast existing models of metamorphosis. Second, I review the multiple processes in which predators affect prey organisms that are relevant to metamorphic decisions and how these processes relate to existing models. Third, I synthesize caged-predator studies to understand how predation cues alone induce changes in metamorphosis (using an expanded collection of studies from that of Benard 2004). Fourth, I synthesize lethal predator studies to understand how actual predation affects metamorphosis. Finally, I review how larval predator environments can have carry-over effects beyond metamorphosis.

Models of metamorphosis: when (and at what size) should animals metamorphose?

For organisms to evolve the ability to respond to environmental variation, there are a number of conditions that must be met (reviewed in Schlichting and Pigliucci 1998). These conditions are certainly met in most larval amphibians experiencing predation risk, including spatial and temporal variation in predator environments (Van Buskirk and Relyea 1998), reliable environmental cues (typically chemical cues emitted by predators; Kats and Dill 1998), and the presence of trade-offs between metamorphosing early and small versus metamorphosing late and large (Berven and Gill 1983; Smith 1987; Semlitsch et al. 1988). Given that the conditions for metamorphic plasticity are met and given the numerous empirical observations that confirm the phenomenon, we can turn to evolutionary models to determine *how* animals should adaptively alter their size and time to metamorphosis.

The first models of metamorphic plasticity focused on the importance of amphibian growth and differentiation in determining the time to metamorphose. The original model

(Wilbur and Collins 1973; made more formal recently by Day and Rowe 2002) proposed that there is a minimum and maximum size of metamorphosis that produces a "window" of metamorphosis within a particular size range. Within that size window, the authors proposed that current growth rate determined the time to (and therefore the size at) metamorphosis. If the current growth rate is good (relative to some long-term evolutionary average growth rate in the terrestrial stage), metamorphosis should be delayed to take advantage of the beneficial larval environment. If the current growth rate is poor, metamorphosis should be accelerated to minimize the time spent in the poor growth conditions of the larval environment. Thus, the emphasis of Wilbur and Collins was on larval growth. However, they were not oblivious to the importance of predation. In fact, they state, "if the body size is small and the growth rate is slow, metamorphosis will proceed. Natural selection has favored the initiation of metamorphosis rather than remaining in the pond and risking predation and the other dangers of the aquatic community" (Wilbur and Collins 1973, p. 1,311). Thus, it is clear that the authors recognized the importance of predation in favoring metamorphosis, but they implicitly assumed that predation risk was a constant.

The Wilbur and Collins' model inspired a number of subsequent models. For example, Smith-Gill and Berven (1979) came at the problem from a more physiological perspective and emphasized the primacy of differentiation rate of the individual rather than growth rate, believing that, "timing of metamorphic climax is dependent primarily upon differentiation rates, while body size at any particular stage of metamorphosis is a function of both growth and differentiation rates" (Smith-Gill and Berven 1979, p. 563). Thus, while growth rate and differentiation rate are frequently (but not perfectly) correlated, only differentiation rate reliably predicts time to metamorphosis. It is not evident from the Smith-Gill and Berven model that predation plays any role in the metamorphic decision.

Focusing on a different system that also experiences complex life cycles (centrarchid fish), Werner and Gilliam (Gilliam 1982; Werner and Gilliam 1984; Werner 1986) observed that predation risk could be quite variable among habitats and this variable risk could also play an important role in the decision to metamorphose. They proposed that, within the size window that permits metamorphosis, organisms with complex life cycles should minimize the ratio of mortality rate (μ) to growth rate (g) when comparing two habitat choices (e.g., aquatic versus terrestrial habitats or littoral versus pelagic habitats). Hence, when mortality rate (background mortality plus mortality from predators) is constant, the minimization of μ/g is conceptually equivalent to the Wilbur and Collins' prediction (Wilbur and Collins 1973); animals should make a decision that maximizes their growth rate. If growth is fast (again, relative to some long-

term evolutionary average growth rate in the terrestrial stage), metamorphosis should be delayed; if growth is slow, metamorphosis should be accelerated. However, when mortality rate is not constant, then prey must balance the benefits of growth in the aquatic environment against the risks of mortality in the aquatic environment. At a constant growth rate, higher risks of mortality should induce prey to metamorphose at a smaller size (which should also cause metamorphosis at an earlier time, although the model does not make explicit predictions about time to metamorphosis). As extensions of this model, others have demonstrated that time constraints can alter the predictions of optimal size and time of metamorphosis (Ludwig and Rowe 1990; Rowe and Ludwig 1991). If there is initial size variation and time constraints on reproduction, the optimal timing of metamorphosis can differ among individuals based on initial size. Still others have applied game theory to incorporate frequency-dependent predation and dilution effects (Bouskila et al. 1998).

In addition to these models of metamorphosis, there are a variety of related models that examine the optimal time to maturity for an organism (e.g., Law 1979; Michod 1979; Stearns and Koella 1986; Abrams and Rowe 1996). As pointed out by Abrams and Rowe (1996), these models are not asking the same question as models of metamorphosis because most animals must continue to grow and develop after metamorphosis before they achieve maturity. Nevertheless, some of these models can still be instructive. The model of Abrams and Rowe (1996) is particularly instructive because it examines the situation in which growth and development are either fixed or flexible and, importantly, their model not only incorporates growth versus predation risk trade-offs but also the indirect positive effects that predators can have on prey resources either by reducing prey number or by reducing prey foraging rates. It is clear from their model that a wide range of outcomes are possible. However, when both age and size at maturity are flexible, the direct effect of predators (i.e., the perceived risk of predation) is expected to decrease size at maturity, but the indirect positive effect of predators on prey resources (i.e., more food) favors an increase in size at maturity (age at maturity may increase or decrease with regard to both factors). Therefore, the combination of the direct and indirect effects on time to maturity depends upon the relative magnitude of the two effect sizes. As we shall see, recognizing the multiple effects that predators can have on their prey is critical to understanding how predators affect time and size at metamorphosis.

What effects do predators have on amphibians?

For nearly a century, biologists have explored how predators affect the survival and phenotypes of amphibians

(Adolph 1931; Bragg 1956; Turner 1962; Wilbur 1972; Morin 1983; Werner 1986; Relyea 2001a; Van Buskirk 2002). A number of early investigators estimated natural survival rates at <9% although, in all cases, it was not clear how much of the mortality was due to predation (Turner 1962; Herreid and Kinney 1966; Brockelman 1969; Calef 1973). Subsequent experiments have confirmed that predators alone can have a major negative impact on larval survival under both natural pond conditions (Wilbur 1972; Smith 1983; Werner and McPeek 1994; Smith and Van Buskirk 1995; Relyea 2002c) and under artificial pond conditions (i.e., mesocosms; Morin 1983; Van Buskirk 1988; Wilbur and Fauth 1990; Semlitsch 1993; Relyea 2002d). Given the great risk that predators pose, it is therefore not surprising that many amphibians have evolved the ability to adjust the amount of time that they spend in the larval stage when they find themselves in high and low risk environments.

When considering how predators affect amphibian metamorphosis, we must consider the assumptions underlying the models versus the reality of how prey respond to predators in nature. When we examine the models, we see that the only factors that induce changes in these traits are prey growth rate and, in some cases, a prey's assessment of its risk of predation. Moreover, the only traits assumed to be phenotypically plastic are the two life history traits (size and time to metamorphosis). However, predators have several additional effects on larvae that may play an important role in metamorphic decisions, including a reduction of competition by thinning the prey population, selection on phenotypic variation via the non-random killing of prey, and the induction of behavioral and morphological traits that come at the cost of reduced growth and development. Below I review what is known about each of these predatory processes.

The thinning effect of predators

While predation is a dead end for those prey that are eaten, it can be beneficial for those that survive because thinning the prey population can reduce competition and improve the growth of the surviving prey. Of course, the impact on the metamorphic decision depends upon whether the species in question possesses developmental plasticity and whether there are sensitive developmental windows during which the increased growth must occur. For instance, in many species, increased food resources that occur late in the larval period do not increase development rate to metamorphosis but do increase mass at metamorphosis (Leips and Travis 1994). To understand how thinning affects size and time to metamorphosis, we need to conduct thinning experiments that track larval growth and development from early ontogeny through metamorphosis. A few studies have examined the separate impact of thinning by removing

larvae by hand at the same rate as a predator thins. For example, thinning alone can cause a 10–33% increase in the mass of larval wood frogs (*Rana sylvatica*; Van Buskirk and Yurewicz 1998) and a 74% increase in the growth of gray tree frogs (*Hyla versicolor*; Relyea 2002d). Although both experiments were terminated prior to metamorphosis (to allow measurement of larval morphology), it is likely that these larval effects would lead to larger and faster developing metamorphs. Surprisingly, we currently lack direct estimates of the thinning effect of predators on size and time to metamorphosis.

The selection effect of predators

Recent studies have demonstrated that predators can cause selection on larval behavior and morphology, but relatively little research has been conducted on the role of selection by predators on amphibian metamorphosis. Among behavioral traits, both within and across species, more active species of larvae are killed more often by predators (Skelly 1994; Relyea 2001c). Among morphological traits, several investigators have found that larvae survive predation better when they are larger and when they possess relatively deep tails (Travis et al. 1985; Van Buskirk et al. 1997; Van Buskirk and Relyea 1998; Van Buskirk and Schmidt 2000). The question of interest is whether selection by predators on a larval population can affect the observed size and time to metamorphosis either directly by removing a non-random subset of the population or indirectly if the larval traits under predatory selection are correlated to metamorphic traits (Emerson et al. 1988; Relyea 2001b). This remains an important area for future investigations.

The induction effect of predators

In contrast to the paucity of data on the separate thinning and selection effects of predators on amphibian metamorphosis, we have a great deal of data on the effect of predator-induced fear on amphibian metamorphosis. This abundance of data has occurred because two decades ago investigators discovered that amphibians respond to chemical cues that are emitted by aquatic predators (Petranka et al. 1987) and are composed of both alarm cues and kairomones (Schoeppner and Relyea 2005). Operationally, this meant that researchers could simply put a predator in a cage and the cues could diffuse out of the cage. While the predator can scare the larvae, it is unable to kill any of the larvae (assuring that the prey density remains constant). In terms of the models of metamorphosis, this means that one can isolate the effect of predator induction without the confounding processes of predator thinning and selection.

The past decade has witnessed an explosion of studies documenting predator-induced changes in amphibian behavior and morphology. In general, many species of larvae forage less, use refuges more, and develop relatively deep tails and small bodies (Relyea and Werner 1999; Lardner 2000; Relyea 2001a; Lane and Mahony 2002; Van Buskirk 2002). These defenses appear to be adaptive because they make the larvae more resistant to predation (Van Buskirk et al. 1997; Van Buskirk and Relyea 1998; Van Buskirk and Schmidt 2000) but come at the cost of slower growth and development (Skelly 1992; Van Buskirk 2000; Relyea 2002a, b) because predator-induced larvae spend less time feeding, have relatively smaller scraping mouthparts, and have relatively shorter and likely less efficient intestines (Relyea and Auld 2004, 2005). Whereas individuals should metamorphose sooner with predators if only life history traits are plastic, the occurrence of costly plastic behavior and morphology may cause the individual to delay metamorphosis until sufficient growth has been achieved to enter the window of metamorphic size (although we may also have to consider the indirect positive effects on the resources of the larvae; Peacor 2002).

In summary, when we examine studies in which investigators examine the impact of predators on metamorphosis, we must consider the multiple processes that are occurring (induction, thinning, or selection) and the magnitude of their effects on metamorphosis. Below, I provide an extensive review of the studies that have examined each of these processes alone and combined together and consider (where possible) the impact of the simultaneous changes in behavior and morphology.

A review of caged-predator studies

In searching the literature, I found 41 studies that reared larval amphibians in the presence and absence of caged predators which is an expansion of the 24 studies previously considered by Benard (2004; in both reviews, multiple amphibian species within one publication were treated as separate studies). Importantly, these 41 studies have a number of inherent biases to consider when extrapolating these data to amphibians in general (Appendix 1). First, of the 44 families of amphibians, existing studies come from only eight families (six anuran families and two caudatan families) with the majority of studies (85%) coming from just three families of anurans: Bufonidae, Hylidae, and Ranidae. Second, the studies do not represent a broad range of geography: 49% come from Europe, 41% come from North America, and 10% come from Australia. We appear to have no studies of this type on Asian, South American, and African amphibians. Third, studies conducted so far have used a surprisingly low diversity of predator species: 44% have used dragonflies (almost entirely aeshnid dragonflies), 17% have used fish, 17% have used beetle larvae, 5% have used

salamanders, and the remaining 17% used a variety of other predators. Finally, the studies are biased by experimental venue: 54% have been conducted in the laboratory, 46% have been conducted in outdoor mesocosms, and, surprisingly, no studies have been conducted under natural conditions (e.g., enclosures placed into ponds).

When comparing the results of these 41 studies (Appendix 1), it is clear that investigators have obtained a variety of results. In a few cases, the conclusion from a study was equivocal because the presence or absence of caged predators was crossed with other environmental conditions (e.g., competition treatments, drying treatments) that made the response to predators context-specific (e.g., Laurila and Kujasalo 1999; Nicieza 2000; Altwegg 2002a; Barnett and Richardson 2002). However, one of the most strikingly consistent results was that in 95% of all studies the amphibians did not emerge earlier with caged predators; they emerged either at the same time (55%) or later (40%) than larvae reared in no-predator environments (a result also found by Benard 2004). Similarly, in 86% of all studies, larvae living with caged predators did not metamorphose at a smaller size; they metamorphosed at a size that was either the same (54%) or larger (32%) than larvae living in no-predator environments. Both of these results are in direct contrast to the prediction of some models (e.g., Werner 1986) that adding caged predators will increase a larva's perceived mortality risk and, as a result, induce amphibians to metamorphose earlier and at a smaller size.

Given this apparent contrast between theory and experiments, we need to more closely examine the diversity of metamorphic outcomes. We can begin by considering the two cases in which amphibians emerged earlier with caged predators. Laurila et al. (1998) found that *Bufo bufo* tadpoles exhibited no difference in growth between caged-dragonfly (*Aeshna juncea*) and no-predator environments, despite a predator-induced activity reduction early in the experiment. Thus, the response appears to be adaptive and in accord with models that incorporate predation risk (e.g., Werner 1986; Abrams and Rowe 1996). Kiesecker et al. (2002) found that *Rana aurora* tadpoles emerged earlier in response to caged newts. In this case, the tadpoles also emerged at a smaller size due to the shorter period of time spent as larvae. These studies demonstrate that caged predators can induce a shorter time to metamorphosis, but it is rare.

The lack of earlier metamorphosis in 95% of the studies begs the question, why do most amphibians raised with caged predators metamorphose at the same time or later than amphibians raised without caged predators? For amphibians that exhibit no change in their time to metamorphosis, we cannot rule out the possibility that these species simply lack developmental plasticity. For example, of the six species of hylids examined, only one of them exhibited

plastic development. This suggests that there may be a phylogenetic constraint in the hylid family (although our current sample is really too small to draw any firm phylogenetic conclusions). In a few cases, we know that the animals possess developmental plasticity but they only exhibit this plasticity under a particular subset of conditions (Laurila and Kujasalo 1999; Nicieza 2000; Barnett and Richardson 2002). For example, Laurila and Kujasalo (1999) found that caged predators induced *Rana temporaria* to have a longer time to metamorphosis when hydroperiod was constant but no change when the hydroperiod was shortened, suggesting that the tadpoles were balancing the simultaneous challenges of predation risk and desiccation. Thus, a lack of response can be due to either phylogeny or due to environmental context.

For those amphibians that are capable of exhibiting predator-induced developmental plasticity, why do so many exhibit a longer time to metamorphose? The most likely explanation is that these animals take longer to metamorphose because they are paying a cost for producing antipredator defenses throughout their larval period. As detailed above, a wide variety of larval amphibians exhibit predator-induced changes in behavior and morphology that together cause slower growth and development (Van Buskirk 2000; Relyea 2002a, b). For example, *Bombina bombina* and *Bombina variegata* both develop relatively deep tails with caged aeshnid predators and both metamorphose at a later time with caged aeshnid predators (Vorndran et al. 2002). Similar patterns can be seen in *Rana esculenta*, *Rana lessonae*, *Rana ridibunda*, *Rana sphenocephala*, *R. sylvatica*, and *Triturus alpestris* (Van Buskirk and Schmidt 2000; Babbit 2001; Van Buskirk and Saxer 2001; Altwegg 2002b; Relyea 2002c). These repeated patterns suggest that the predator-induced defenses that cause slower larval development in turn cause a longer time to metamorphosis.

The cost of predator-induced larval defenses can be expressed not only as slower development, but also as slower growth. As noted by Smith-Gill and Berven (1979), growth and development are often correlated, but they are not perfectly correlated, particularly late in the larval period (e.g., Leips and Travis 1994). When growth and development are highly correlated, amphibians living with caged predators should experience slower rates of growth and development and, as a result, they metamorphose later but at the same size as conspecifics reared in no-predator environments (because the slower daily growth rate is extended over a greater number of days). This pattern appears to be common (Wildy et al. 1999; Babbit 2001; Van Buskirk and Saxer 2001; Altwegg 2002a; Relyea 2002c).

When growth and development are not highly correlated, there are two possible outcomes. If development is negatively affected by the induction of larval defenses but growth rate is not, we should observe cases in which

amphibians reared with caged predators take longer to metamorphose and emerge at a larger size than larvae reared without caged predators. This is a common occurrence (Laurila et al. 1998; Laurila and Kujasalo 1999; Nicieza 2000; Van Buskirk and Schmidt 2000; Babbit 2001; Altwegg 2002a, b; Barnett and Richardson 2002; Vorndran et al. 2002). Alternatively, if growth rate is negatively affected by the induction of larval defenses but development is not, we should observe cases in which amphibians living in caged-predator environments metamorphose at the same time but experience a slower daily growth rate and, as a result, emerge at a smaller size. This pattern appears to be much less common, having been observed only twice (Skelly and Werner 1990; Lardner 2000). For example, Skelly and Werner (1990) found that predators induced no change in the time to metamorphosis of *Bufo americanus* but did induce a 41% reduction in prey foraging and a 28% smaller size at metamorphosis. The time to metamorphosis was unchanged compared to no-predator controls, but because of reduced foraging, the tadpoles ended up emerging at a smaller size. Because metamorphic outcomes depend upon predator effects on growth and development, we need to understand how predators affect both traits over ontogeny and determine when correlations between growth and development are strong versus weak. With this information available, we can make much better predictions about how predators will affect metamorphosis.

In trying to summarize the effects of caged predators on amphibian metamorphose, a number of patterns emerge. First, as also noted by Benard (2004) and confirmed herein with a much larger collection of studies, there is overwhelming evidence that predation cues from caged predators rarely causes a smaller size at metamorphosis (14% of studies) and even more rarely causes a shorter time to metamorphose (5% of studies). The most likely reason for this consistent rejection of the theoretical prediction is that predators do not only directly cause larvae to alter their life history traits, but also cause larvae to alter their behavioral and morphological traits and these trait changes can indirectly reduce growth and development (Tollrian and Harvell 1999). Moreover, these trait changes can vary over ontogeny (Relyea 2003c; Benard 2004) and the impacts of changing resource intake on growth versus development can be stage-specific (Leips and Travis 1994). The relative magnitudes of these growth and developmental costs largely determine the final size and time to metamorphosis. That is not to say that amphibians are incapable of reducing their time to metamorphosis; clearly this has been observed (Laurila et al. 1998). However, it is to say that while it may be advantageous to emerge earlier to avoid predation, the other demands on the larvae to defend themselves may override this pressure. Thus, immediate larval defense (with the associated costs) may take primacy over longer-term, optimal metamorphic decisions. This means that the models of metamorphosis that incorporate mortality rate and growth risk are not necessarily incorrect (e.g., Werner 1986), but our experimental tests of the models simply do not match the models' implicit assumptions that only life history traits are altered and that predation risk does not alter growth rate. The reality is that predator cues can have direct negative effects on prey growth by inducing costly behavioral and morphological defenses [of course, under high competition, predators could have indirect positive effects on prey growth by initially reducing foraging thereby stimulating an increase in resources (Abrams and Rowe 1996; Relyea and Werner 2000; Peacor 2002); however, such high competition is less likely to occur when lethal predators are present and killing prey]. If prey are within their window of size in which metamorphosis is possible, most models predict that this reduction in prey growth should make the larval habitat even less desirable and induce earlier metamorphosis. However, empirical studies suggest that by reducing growth, caged predators actually cause a temporal shift in the window of metamorphic size. Hence, both the timing of the metamorphic window *and* the metamorphic decisions within that window appear to be inducible by predators.

Future studies of caged-predator effects on size and time to metamorphosis could benefit from a number of considerations. The first priority is to document how larvae alter their defenses over ontogeny and repeatedly quantify growth and development throughout the larval period so that we can have a more mechanistic understanding of how predators alter the final size and time to metamorphosis (e.g., Skelly and Werner 1990; Van Buskirk and Schmidt 2000). The second priority emerges from the fact that our current estimates of predatory effects are dominated by the use of one small group of predators, the aeshnid dragonflies. If we hope to understand how predators generally affect metamorphic decisions, we need to examine a wider range of aquatic predators (Kurzava and Morin 1998; Nyström et al. 2001; Relyea 2001a, c, 2003a). We also suffer from a fixation on 2 × 2 × 2... (etc.) factorial experiments that only examine the presence and absence of a predator. It would be instructive to examine metamorphic responses across a range of densities of predators to determine how the metamorphic decision changes with increased predation risk. In short, we need to go beyond simply rearing amphibian larvae with and without predators, measuring their size and time to metamorphosis, and then inferring the mechanism underlying the metamorphic decision without the data to support the inference. Even though our current studies come from a restricted set of families, we need to understand the mechanistic underpinnings of metamorphosis in these families before we spread our efforts across additional species and families. A more

mechanistic approach will likely lead to new models of metamorphosis that incorporate the complexity of predator-induced defenses that the past two decades of empirical work have discovered.

A review of lethal-predator studies

In contrast to caged-predator studies, lethal-predator studies examine the combined impacts of induction, thinning, and selection on amphibian metamorphosis. In my search for lethal-predator studies, I excluded cases in which the predator had no significant effect on prey survival (e.g., Figiel and Semlitsch 1990; Beachy 1997) and those studies in which nearly all prey were killed (providing less reliable results based on only a few individuals). Using these criteria, I found 51 studies (Appendix 2). As with the caged-predator studies, lethal-predator studies have several biases. First, the studies come from only six amphibian families, with the majority of studies (87%) coming from just three families of anurans: Bufonidae, Hylidae, and Ranidae. Second, the geographic range is again quite restricted: North America (80%), Europe (16%), and Africa (4%). Third, the types of lethal predators used are very different than the caged-predator studies with 57% using salamanders (almost entirely newts, *Notophthalmus viridescens*), 25% using dragonflies, 6% using fish, and 6% using multiple predators. Interestingly, this predator bias may simply reflect the fact that most studies incorporating lethal predators have been conducted by researchers affiliated with the tremendously productive academic lineage of Henry Wilbur (e.g., Wilbur, Van Buskirk, Semlitsch, Parris, Morin, and Fauth) and these researchers frequently use predatory salamanders. Finally, the venues employed are quite different than in caged-predator studies: 78% have been in mesocosms, 14% have been in pens placed into ponds or streams, 8% have examined patterns in natural ponds, and 0% have been in the laboratory. We must keep these biases in mind when examining patterns of metamorphosis with lethal predators.

When examining the impact of lethal predators on the size and time to metamorphosis, we must first contemplate how to interpret the potential outcomes. From the above review of caged-predator studies, it is clear that induction rarely results in earlier time to metamorphosis. However, the thinning effect of lethal predators should reduce competition which should allow shorter times to metamorphosis and faster growth (providing that the thinning occurs early enough in the larval period). It is fair to say that we have so little information on the impact of selection that we cannot yet make any predictions of its relative impact on metamorphosis. Therefore, an important caveat in my interpretation of lethal predator studies below is that I limit myself to interpretations that consider only the costs of induction (e.g., reduced foraging) and the benefits of thinning; the final outcome, of course, will depend upon the relative magnitude of these processes. One way to help determine the relative magnitude of thinning and induction would be to examine the growth rate of the animals because thinning by predators typically causes faster growth whereas induction by predators typically causes slower growth [except under high competition (Peacor 2002), but high competition is less likely when lethal predators are killing off the competition]. While most authors do not analyze or report growth rate, it can be easily estimated (mass at metamorphosis/time to metamorphosis).

Past studies using lethal predators have produced a variety of outcomes on the size and time to metamorphosis (Appendix 2). In 32% of the studies, researchers have found that larvae raised with lethal predators metamorphosed earlier than conspecifics raised without lethal predators. This suggests that thinning played a dominant role in the time to metamorphosis in these studies. However, size at metamorphosis was nearly evenly split: half of the studies observed a smaller size at metamorphosis (six of the seven cases involved *B. americanus*) while the other half observed a larger size at metamorphosis. In the cases of emerging earlier and smaller, an examination of daily growth rates provides an interesting quandary. For example, *B. americanus* tadpoles that emerge earlier and smaller can have either slower daily growth rates (Wilbur and Fauth 1990) or faster daily growth rates (Van Buskirk 1988), suggesting that the decision to invest energy into more rapid growth versus more rapid development may be more complex than we realize. In cases where larvae emerged earlier and larger (e.g., *Hyla chrysocelis*, *Hyla gratiosa*, *Pseudacris triseriata*, *Rana sphenocephala*), these animals experienced a much higher daily growth rate with lethal predators (i.e., achieved greater mass in fewer days), suggesting that the thinning effect of predators was the dominant factor in causing this metamorphic decision.

In the remaining studies of lethal predators, 51% found equal times to metamorphosis while 17% found longer times to metamorphosis. When there are equal times to metamorphosis, one must conclude that these animals are either not capable of responding to lethal predators or that the delaying effects of induction and the accelerating effects of thinning equally offset each other. Within this group, one can find cases of either smaller, equal, or larger sizes at metamorphosis. Because time to metamorphosis is not different, these cases represents larvae that experienced slower, equal, and faster daily growth rates, respectively. This suggests that the primary mechanisms were induction, induction plus thinning, and thinning, respectively. Interestingly, such a diversity of outcomes was predicted by Abrams and Rowe (1996) in their model of optimal times to

maturity. The few cases of longer times to metamorphosis with lethal predators always had equal or larger size at metamorphosis. These cases suggest a primacy of induction effects which can slow down development. When this slower development is combined with a slower or equivalent growth rate, we produce metamorphs that are of equal or larger size.

The above studies demonstrate that induction and thinning can vary in their relative importance to the metamorphic outcome. However, it would be helpful if we could predict a priori the conditions under which each process would dominate. For the induction process, the magnitude of predator-induced defenses and the associated costs are greatest under low competition and lowest under high competition (reviewed in Relyea 2004). In contrast, the thinning process is expected to have minimal positive effects on prey growth when competition is low (i.e., food is not limiting) but large positive effects on prey growth when competition is high because the limited resources can be divided among fewer individuals. Thus, if we were to examine the impact of lethal predators across a range of prey densities, we should observe delayed metamorphosis at low prey density (a dominance of induction effects) but accelerated metamorphosis at high prey density (a dominance of thinning effects). Vonesh (2005) provides an excellent study of lethal predator effects across a range of prey density and the results are consistent with these predictions.

The existence of multiple mechanisms producing the same metamorphic outcome in lethal-predator studies underscores the importance of actually identifying the responsible mechanisms rather than conducting "input–output" experiments and inferring a mechanism. As in the case for comparing the results of caged predator studies against model predictions, it is critical that we recognize that when we add lethal predators we not only alter mortality risk, but we frequently alter growth rate through a number of indirect pathways. Therefore, we cannot use lethal-predator experiments to test model predictions under the assumption that mortality risk is the only factor that is changing. Future studies should quantify growth and development throughout larval ontogeny with lethal predators to obtain the needed parameters to predict metamorphic responses. We also need studies that examine the separate and combined effects of the three predator processes (induction, thinning, and selection) on metamorphic decisions. Recent studies of these processes on larval behavior, morphology, and growth have demonstrated that this is empirically possible (Van Buskirk and Yurewicz 1998; Relyea 2002d). As is the case for caged-predator studies, we need to conduct lethal-predator studies with a wider diversity of predators and a range of predator densities. Future work could also include a wider range of amphibian families, but this seems to be of lower priority for making conceptual progress.

The effects of larval predators after metamorphosis

In most studies of animal metamorphosis, investigations often end when metamorphosis is completed. However, as reviewed by Pechenik et al. (1998), environments experienced early in ontogeny can have dramatic impacts on traits later in ontogeny across a broad range of taxa. In amphibians, the lasting effects of size and time to metamorphosis have been of interest for some time. For example, several researchers have found that while time to metamorphosis occasionally correlates with post-metamorphic performance, it is size at metamorphosis that appears to have a much more important effect. A larger size at metamorphosis can persist for up to 2 years and size at metamorphosis is positively correlated with improved juvenile survival, larger size at reproduction, earlier times to reproduction, and improved mating success, and the production of more and larger eggs (Howard 1980; Berven 1981, 1982; Berven and Gill 1983; Smith 1987; Semlitsch et al 1988; Gerhardt 1994; Altwegg and Reyer 2003). Thus, the correlative evidence suggests that larval predator effects on size at metamorphosis may have long-lasting fitness effects.

Despite the potential long-term effects of larval predators, their impacts have rarely been examined. In experiments with caged predators, several researchers have found that living with caged predators as larvae has no effect on post-metamorphic survival or growth (Altwegg 2002b; Lane and Mahony 2002; Relyea and Hoverman 2003). There appear to be no studies of lethal-predator effects on post-metamorphic survival or growth. I was also unable to find any caged- or lethal-predator studies that have followed individuals to reproductive maturity to examine the impacts of larval predators on size and age at reproduction, long-term post-metamorphic survival, or egg production. Such studies are clearly long-term endeavors that would require a great deal of time, money, and energy.

In addition to post-metamorphic life history traits, we can also ask how other types of post-metamorphic traits are affected by experiencing predators in the larval environment. For example, Benard and Fordyce (2003) found that tadpoles living with the smell of crushed conspecifics showed no observable changes in phenotype as tadpoles, but did produce threefold higher concentrations of bufadienolide toxins after metamorphosis (the adaptive value of this response remains unclear). Morphological traits can also be affected by larval predator environments. For example, caged predators experienced during the larval stage can cause the development of relatively longer legs in wood frogs (*R. sylvatica*; Relyea 2001b), relatively shorter and more muscular legs in water frogs (*Rana ridibunda*; Van Buskirk and Saxer 2001), and no difference in gray tree frogs (*H. versicolor*; Relyea and Hoverman 2003). Thus, the few data suggest that we can obtain a variety of outcomes

on post-metamorphic morphology as a result of either species-specific responses to larval predators or perhaps due to differences in experimental venue. In those cases where larval predators cause leg length changes in post-metamorphic anurans, the changes were relatively small (<10%) and would not likely affect hopping ability (Zug 1972, 1986; Van Buskirk and Saxer 2001).

Synthesis and future directions

We have come a long way in identifying how predators affect metamorphic decisions. It is clear that when conducting and interpreting our experiments, we must carefully consider the multiple processes that are occurring during predation (induction, thinning, and selection) and recognize that amphibian larvae (and many other prey taxa) can change multiple traits (behavior, morphology, and life history). This means that metamorphic models which try to optimize life history traits, without taking into account the effects of thinning and induction will often arrive at predictions that do not match the empirical data. Models that incorporate integrated traits such as growth and total mortality risk can incorporate this diversity of predator effects (e.g., Werner 1986), provided that they recognize that predators alter not only mortality risk, but also alter growth and the timing of the metamorphic window through several pathways (e.g., Abrams and Rowe 1996). It might prove useful to also develop models in which each predator process (selection, induction, and thinning) is modeled as a separate parameter so that one could understand their interactive effects on metamorphic decisions.

The way in which we conduct our experiments could also be improved. Progress toward understanding plasticity in size and time to metamorphosis will not be made by conducting more experiments in which we simply raise prey in the presence and absence of caged or lethal predators and then infer a causal mechanism that fits the experimental endpoint. As we have seen, multiple mechanisms can produce the same phenomenon. If we want to know *why* animals arrive at a particular metamorphic outcome, we need to follow them over ontogeny, quantify their multiple relevant traits (behavior, morphology, growth, and development), and quantify resource levels over time (Benard 2004). For example, predator defense strategies can change dramatically during the larval period with a reliance on more costly behavioral defenses early in ontogeny and a reliance on less costly morphological defenses later in ontogeny (Relyea 2003c). As a result, caged predators can cause larvae to experience slower growth early in ontogeny but faster growth later in ontogeny (Relyea and Werner 2000; Van Buskirk and Schmidt 2000). In this case, the final mass at metamorphosis for animals reared with caged

predators could be less than, equal to, or greater than animals reared without predators, depending on the magnitude of the growth effects early versus late in ontogeny. In lethal-predator studies, we not only need to track animals over ontogeny and understand how they change their traits, we also need to understand the relative importance of thinning versus induction by conducting experiments that manipulate these two processes separately and together (e.g., Van Buskirk and Yurewicz 1998; Relyea 2002d). These additional experimental steps will require more time and energy, but the advances in our understanding will be worth the effort.

While empirical traditions are wonderful testaments to our past successes, they also limit our ability to broadly generalize our results. As we have seen, the vast majority of our experiments have been conducted on three anuran families (primarily those species inhabiting North America and Europe). On the positive side, such intense research effort on these three families opens up the possibility for a future phylogenetically based meta-analysis to determine which patterns are due to ecological conditions versus phylogenetic constraints. While such an analysis has yet to be undertaken, we likely have sufficient data to proceed along this avenue of research.

The predator taxa that we use represent a tiny fraction of the predators with which larval amphibians live. Based largely (but not entirely) on tradition, it is apparent that most investigators examining caged predator effects have chosen to work with one family of predators, the aeshnid dragonflies, while most investigators of lethal predator effects have chosen to work with a very different family, newts in the family Salamandridae. Thus, not only is our taxonomic reference for predators narrow, it may very well be that the two types of experiments (caged versus lethal predators) are not even comparable because they rarely use the same type of predator. Conducting numerous small experiments with different types of predators would be helpful, but separate experiments can suffer from differences in experimental conditions. To best understand the impact of different types of predators on amphibian metamorphosis, we need to conduct single, large experiments in which we can examine the impact of predation risk on metamorphosis by using different species of predators, different combinations of predators, and different densities of predators. We also need experiments that combine predator effects with other challenges to growth and development including competition, desiccation, and anthropogenic stressors (e.g., Laurila et al. 1998; Babbit 2001; Altwegg 2002a; Relyea 2003b, 2004). Such experiments, conducted with a mechanistic perspective (as described above), will rapidly advance our understanding of how amphibians (and probably many other taxa) make their metamorphic decisions.

Finally, there is a great need to determine whether all of the focus on the size and time to metamorphosis even matters to amphibians in their subsequent post-metamorphic life. While we have a number of excellent correlations suggesting that size is important, few investigators have addressed whether larval predator environments per se affect post-metamorphic survival, growth, morphology, and reproduction. Addressing some of these questions requires experiments that take much more time (additional months or years), making it doubtful that we will ever know as much about this part of the life cycle. However, at the very least, recent studies have shown that even relatively short-term post-metamorphic assessments can produce a number of surprising discoveries (Relyea 2001b; Van Buskirk and Saxer 2001; Benard and Fordyce 2003).

Conclusions

The copious literature available on larval amphibians and their predators offers the capacity to generate a great number of insights about metamorphosis that likely apply to a variety of metamorphosing organisms as well as those organisms that possess less dramatic forms of complex life cycles (Werner 1988). The vast majority of these organisms serve as prey to some predator and, as a result, possess inducible defenses that come with associated costs of growth and development (Werner 1988; Hall and Wake 1999; Truman and Riddiford 2002; Benard 2004). Therefore, in all of these taxa, one would expect a diversity of metamorphic decisions in response to predators which arise as the combined outcome of the three major predatory processes: induction, thinning, and selection. The conditions under which each of these processes might dominate are predictable and future models that incorporate these mechanisms will lead us to an improved understanding about how prey make this important life history decision.

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