

Antipredator behavior of chytridiomycosis-infected northern leopard frog (*Rana pipiens*) tadpoles

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Abstract: We investigated the effects of *Batrachochytrium dendrobatidis* Longcore, Pessier & Nichols, a pathogen implicated in global amphibian population declines, on antipredator behavior of northern leopard frog (*Rana pipiens* Schreber, 1782) tadpoles in response to visual and chemical cues of a fish predator, bluegill sunfish (*Lepomis macrochirus* Rafinesque, 1819). We placed infected and uninfected tadpoles in containers partitioned with a transparent divider and measured tadpole activity and distance from the center. Infected tadpoles had significantly lower activity levels across all treatments. When exposed to only visual cues, uninfected tadpoles positioned themselves farther from the center divider (and thus the predator) than infected animals. All tadpoles were at similar distances from the center when exposed to chemical cues only, likely because chemical cues alone do not provide spatial information on the location of predators. Infected tadpoles were significantly farther from the center divider than uninfected ones when exposed to visual and chemical cues together, suggesting that, although the mechanism is unknown, both cues are necessary to stimulate predator avoidance behavior for infected animals. In a second experiment, infected tadpoles experienced lower mortality than uninfected ones in the lethal presence of fish. Thus, effects of infection on behavioral antipredator responses are complex, but lower host susceptibility to predation, low activity, and greater distance from predators when both chemical and visual predator cues are present likely benefits *B. dendrobatidis*, which relies on host survival for transmission.

Résumé : Nous avons étudié les effets de *Batrachochytrium dendrobatidis* Longcore, Pessier & Nichols, un pathogène impliqué dans le déclin des populations d'amphibiens à l'échelle du globe, sur le comportement antiprédateur des têtards de la grenouille léopard (*Rana pipiens* Schreber, 1782) en réaction à des signaux visuels et chimiques d'un poisson prédateur, le crapet harlequin (*Lepomis macrochirus* Rafinesque, 1819). Nous avons placé des têtards infectés et sains dans des récipients comportant une paroi de division transparente et nous avons mesuré l'activité des têtards et leur distance du centre. Dans toutes les situations, les têtards infectés ont des niveaux d'activité plus faibles. Lors d'expositions aux seuls signaux visuels, les têtards sains se tiennent plus loin de la paroi de division centrale (et ainsi du prédateur) que les animaux infectés. Lors d'expositions aux seuls signaux chimiques, tous les têtards se tiennent à des distances semblables de la paroi médiane, probablement parce que les signaux chimiques seuls ne fournissent pas de renseignements spatiaux sur l'emplacement des prédateurs. Lors d'une exposition simultanée aux signaux visuels et chimiques, les têtards infectés se tiennent plus loin de la paroi médiane que les têtards sains, ce qui laisse croire que, par un mécanisme encore inconnu, les deux types de signaux sont requis pour déclencher un comportement d'évitement des prédateurs chez les animaux infectés. Dans une seconde expérience, les têtards infectés ont subi une mortalité réduite par rapport aux têtards sains en présence de poissons qui représentent un danger de mort. Ainsi, les effets de l'infection sur les réactions comportementales contre les prédateurs sont complexes, mais la vulnérabilité réduite à la prédation, l'activité faible et la distance plus grande des prédateurs en présence simultanée de signaux chimiques et visuels sont vraisemblablement bénéfiques à *B. dendrobatidis* qui dépend de la survie de son hôte pour sa transmission.

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Introduction

Ecologists increasingly acknowledge a role for pathogens in population dynamics (Real 1996) and in maintaining diverse communities and ecosystems (McCallum and Dobson 1995). Diseases can play an important role in affecting host fitness directly by reducing survival and reproductive rates

in host species (Daszak et al. 1999, 2003; Carey 2000). Diseases can also indirectly affect host fitness by altering behaviors so that susceptibility or exposure to pathogens is altered (Kiesecker and Blaustein 1999; Kiesecker and Skelly 2001; Parris and Cornelius 2004; Parris et al. 2004).

Pathogens are expected to be locally adapted to their hosts, as they generally evolve more rapidly than their hosts owing to shorter generation times and higher mutation rates (Ebert 1994, 1999; Kaltz and Shykoff 1998). As a result, behavioral modifications of the host are generally thought to benefit the parasite or pathogen, often in terms of transmission. For example, snails (*Potamopyrgus antipodarum* (Gray, 1843)) infected with the trematode parasite *Microphallus* Ward, 1901 spend more time on top of rocks than do uninfected snails, which increases their probability of consumption by the definitive host (Levri 1999).

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Infection also may induce host behaviors that can be adaptive for the host. For example, a typical ectothermic response to pathogens is for infected hosts to prefer warmer microhabitats, presumably to reduce pathogen proliferation within the host (i.e., behavioral fever; Kluger et al. 1975). Bullfrog (*Rana catesbeiana* Shaw, 1802) tadpoles infected with *Aeromonas* bacteria prefer warmer aquatic temperatures (Lefcort and Eiger 1993), which slow bacterial growth. However, because warm conditions usually are in shallow water with high aquatic predator densities (Arnold and Wassersug 1978), predation risk may be enhanced for infected tadpoles. Stereotypical responses of infected tadpoles therefore may facilitate pathogen transmission if predators serve as reservoirs. However, high predation may be disadvantageous for pathogens whose transmission depends on host survival (Dobson 1988; Parris et al. 2004).

Appropriate antipredator behavior is important for survival of larval amphibians because predation is a primary selective force in aquatic communities (Wellborn et al. 1996). Consequently, tadpoles of many taxa spatially avoid predators or reduce activity to reduce detection and predation risk (Lima and Dill 1990; Skelly and Werner 1990; Skelly 1994). Pathogens that affect host behavior may alter adaptive antipredator responses (Thompson 1990; Lefcort and Blaustein 1995; Thiemann and Wassersug 2000; Parris et al. 2004), potentially affecting host conspicuousness and risk of predation (Giles 1983; Dolinsky et al. 1985; Quinn et al. 1987).

Infection caused by the pathogenic fungus *Batrachochytrium dendrobatidis* Longcore, Pessier & Nichols (Chytridiomycota, Chytridales; Longcore et al. 1999) has been implicated in global amphibian declines (e.g., Berger et al. 1998; Bosch et al. 2001; Bradley et al. 2002; Collins et al. 2003; Daszak et al. 2003; Lips et al. 2003, 2004; Muths et al. 2003), although effects on individual species vary. *Batrachochytrium dendrobatidis* infects keratinized tissues in the skin of metamorphosed animals and is commonly spread through colonization by free-swimming zoospores (Longcore et al. 1999; Pessier et al. 1999; Nichols et al. 2001; Piotrowski et al. 2004). Mortality caused by chytridiomycosis is noted mainly among metamorphosed animals in the field (Berger et al. 1998; Lips 1999; Green and Kagarise Sherman 2001; Bell et al. 2004), but infected larvae may exhibit disfigurement of their keratinized mouthparts (Fellers et al. 2001; Rachowicz and Vredenburg 2004) and may demonstrate reduced growth and development (Parris 2004; Parris and Beaudoin 2004; Parris and Cornelius 2004). *Batrachochytrium dendrobatidis* is characterized as an emerging infectious disease (Morehouse et al. 2003; Daszak et al. 2003; Weldon et al. 2004), and it is particularly important to study behavioral responses to emerging pathogens that are often spread rapidly among host populations.

Chytridiomycosis has been observed in natural amphibian populations throughout the USA, and it has been implicated in population declines of the northern leopard frog, *Rana pipiens* Schreber, 1782 (e.g., Carey et al. 1999; Morell 1999; Muths et al. 2003). We tested the impact of *B. dendrobatidis* infection on *R. pipiens* tadpole antipredatory behavior and susceptibility to predation. We exposed tadpoles to the lethal and nonlethal presence of bluegill sunfish (*Lepomis macrochirus* Rafinesque, 1819) in laboratory containers and tested whether infection alters tadpole antipredator behavior or af-

fects risk of predation. The distributions of *R. pipiens* and *L. macrochirus* overlap throughout much of the northern USA, and thus predation from *L. macrochirus* is likely an important selective force for *R. pipiens* tadpoles in natural populations (Smith 1983; Woodward 1983; Bodie et al. 2000).

Materials and methods

Pathogen effects on behavioral responses to predators

We used 40 uninfected and 40 chytridiomycosis-infected *R. pipiens* tadpoles to test for differences in tadpole behavior in the nonlethal presence of *L. macrochirus*. Behavior was monitored in plastic tubs (34 cm × 19 cm × 11 cm) with a clear Plexiglas® divider (6 mm thick) firmly attached in the center, filled to a depth of 7 cm (1.6 L per side). Containers were marked with lines on the bottom every 5 cm to estimate the distance of a tadpole from the center.

Tadpoles were derived from laboratory breedings between four *R. pipiens* pairs. Following oviposition in the laboratory (11 February 2004), tadpoles from the four egg clutches were kept separate in 37 L aquaria to test for parentage effects on resultant offspring performance. On 16 February, after reaching the free-swimming stage (stages 25–27; Gosner 1960), we haphazardly selected 80 tadpoles (20 from each clutch) and added them to individual 1.0 L plastic containers containing 400 mL of water. Tadpoles were fed ground rabbit chow ad libitum and reared on a 12 h light : 12 h dark photoperiod. We maintained all containers at room temperature (22 ± 3 °C).

Tadpoles were acclimated for 7 days before being infected. We administered *B. dendrobatidis* infections by exposing 40 randomly selected tadpoles to water baths containing infectious concentrations of fungal zoospores (7000 zoospores/mL; Davidson et al. 2003; Parris and Baud 2004). Our design therefore simulated transmission by water, the likely mode of *B. dendrobatidis* transmission in natural populations (Nichols et al. 2001; Johnson and Speare 2003; Piotrowski et al. 2004). We cultured *B. dendrobatidis* in the laboratory on tryptone – gelatin hydrolysate – lactose (TGhL) agar in 9 cm petri dishes according to standard protocol (Longcore et al. 1999; Davidson et al. 2003). After 6 days of growth on fresh medium, we harvested zoospores that emerged 30 min after adding sterile water to fungal cultures. An additional group of 40 *R. pipiens* tadpoles exposed to a fungal-free TGhL agar wash served as controls. After a 7 day exposure period, we replaced water in all containers with uncontaminated dechlorinated tap water. Parris and Baud (2004) confirmed this exposure protocol as an effective method for infecting tadpoles.

Our design tested for differences in responses of uninfected versus infected tadpoles to the nonlethal presence of visual and (or) chemical fish predator cues. On 28 February, we collected adult *L. macrochirus* from natural populations at the Edward J. Meeman Biological Field Station (Shelby County, Tennessee; 35°22'N, 90°1'W). Fish were returned to the laboratory and reared in 37 L aquaria and fed *R. pipiens* tadpoles ad libitum. Uninfected and infected tadpoles were exposed to one of four predator cue treatments: (1) no predator cues; (2) visual predator cues only; (3) chemical cues only; (4) visual and chemical cues. All trials were conducted 1–2 March by randomly selecting a tad-

pole and placing it in one of the four treatments at random. Tadpoles in the visual cue treatment were exposed to the visual presence of two *L. macrochirus* on the opposite side of the center divider. The chemical cue treatment consisted of tadpoles placed in one side of the plastic tub in which two *L. macrochirus* had resided for the previous 24 h; fish were removed immediately prior to starting the treatment. Simultaneous presence of visual and chemical cues was accomplished by placing a tadpole in the end of the tub containing water in which *L. macrochirus* had resided for 24 h, while relocating fish to the opposite side of the tub immediately prior to the trial. Control treatments had neither visual nor chemical predator cues present. Each tadpole was exposed to only one predator cue treatment. Fish were haphazardly selected and had a body length of 48 ± 8 mm (mean \pm 1 SE) across all treatments. Each of the 8 treatment combinations was replicated 10 times. All experimental animals were cared for in accordance with the principles and guidelines of the Canadian Council on Animal Care.

Tadpoles were allowed to acclimate to their respective tubs for 20 s, after which we recorded tadpole location relative to the center divider and activity level (swimming any time during the interval) at 20 s intervals for 20 min. Mean distance from the center divider was used to estimate tadpole distance, and proportion of time periods spent active during the 20 min observation period was calculated as an estimate of activity level.

Histological examinations were used to confirm *B. dendrobatidis* infection in tadpole mouthparts following the experiment. We randomly selected 10 *B. dendrobatidis* exposed and 10 unexposed tadpoles, euthanized them with MS-222, and fixed them in 10% formalin. A subsample of 10 tadpoles likely would yield robust data on infectivity, because previous work documented our exposure protocol to be 100% effective at infecting tadpoles (Parris and Baud 2004). Tadpole mouthparts were embedded in paraffin, sectioned at 6 μ m, and stained with hematoxylin and eosin. We used serial sectioning and processed approximately 200 sagittal sections per mouthpart (sensu Rachowicz and Vredenburg 2004).

Pathogen effects on susceptibility to predation

We tested for differences in susceptibility of uninfected versus *B. dendrobatidis* infected *R. pipiens* tadpoles to predation by *L. macrochirus*. One hundred and sixty additional *R. pipiens* tadpoles were obtained from laboratory breedings (see above) and exposed to *B. dendrobatidis* for 7 days. We exposed 80 tadpoles (16 groups of 5) to 400 mL of *B. dendrobatidis* inoculated water in 1.0 L containers. Sixteen additional groups of 5 tadpoles exposed to a TGhL agar wash served as controls (see above). Treatments consisted of uninfected or infected *R. pipiens* tadpoles reared with *L. macrochirus* in 15 L of water (37 L aquaria). The two treatments were each replicated 8 times in 16 aquaria. All tadpoles were between Gosner (1960) stages 25–27 prior to testing. We reared tadpoles in both treatments at an initial density of 10 animals/aquarium (0.67 animals/L), which is within the range of natural densities of *R. pipiens* tadpoles (Morin 1983; Werner and Glennemeier 1999). After tadpoles were acclimated for 24 h, we measured and haphazardly added a single fish to each predator aquarium (*L. macrochirus* body

length = 59 ± 6 mm); fish size did not differ between treatments. The response variable estimated was predation, which was measured as the disappearance of or injury to tadpoles. We observed animals three times daily and used one minus the proportion of tadpoles remaining at the end of 3 days as our estimate of predation. We chose a 3 day experimental period because by day 3 at least one tadpole was eaten in all aquaria and, in some aquaria, all tadpoles were consumed.

Tadpoles from unexposed and pathogen-exposed conditions did not differ statistically in body size for both experiments (total body length unexposed = 15 ± 3 mm; exposed = 15 ± 4 mm; mean \pm 1 SE), and all were within the gape of fish predators. At the end of both experiments, we thoroughly disinfected all equipment by adding bleach (6% sodium hypochlorite) to yield a 10% solution, which kills *B. dendrobatidis* (Johnson and Speare 2003). All equipment and water were disinfected in similar fashion throughout the experiments, ensuring no contamination by *B. dendrobatidis*.

Response variables and statistical analyses

We used multivariate analysis of variance (MANOVA) to test for main and interaction effects of the independent factors clutch, infection status, and predator cue on the dependent variables tadpole activity level and distance. We then used Bonferroni-adjusted (significance level of 0.025 for two response variables) univariate analyses of variance (ANOVA) contrasts on each response variable to test for significant contributors to the multivariate effects (PROC GLM; SAS Institute Inc. 1990). We tested for correlations between behavioral responses prior to conducting separate ANOVAs. There was no significant correlation between the two larval responses ($r = 0.1381$, $P = 0.2341$), thereby justifying the use of separate ANOVAs for activity level and distance. Preliminary ANOVAs indicated that trial date had no significant effect on either activity level ($F_{[1,64]} = 0.01$, $P = 0.9689$) or distance ($F_{[1,64]} = 0.27$, $P = 0.6076$); this factor was removed from subsequent analyses. We angularly transformed mean activity level data, and log-transformed mean tadpole distance from the center divider, prior to analysis to meet parametric assumptions (Sokal and Rohlf 1995). We also performed two-tailed Student's *t* tests to assess significant differences between infection status means within each predator cue treatment. Survival data in the lethal presence of predators were analyzed using *G* statistics for log-likelihood ratio goodness-of-fit tests on untransformed data (PROC FREQ; SAS Institute Inc. 1990) because of significant departures from normality and heteroscedasticity of error variances between uninfected and infected treatments.

Results

Histology with serial sectioning detected *B. dendrobatidis* infection in all sampled pathogen-exposed tadpoles. Histological examinations indicated that infected oral discs displayed hyperkeratosis and pigment loss (Fellers et al. 2001; Rachowicz and Vredenburg 2004), while no controls were infected.

MANOVA indicated significant main effects of clutch and infection status, and a significant predator cue \times infection status interaction effect on combined larval responses, but no significant predator cue main effect (Table 1). *Batracho-*

Table 1. Summary of MANOVA and univariate ANOVAs of proportion of time spent active and distance from center divider (distance) for uninfected and *Batrachochytrium dendrobatidis* infected northern leopard frog (*Rana pipiens*) tadpoles reared in the absence or presence of visual and (or) chemical cues from predatory bluegill sunfish (*Lepomis macrochirus*) in laboratory containers.

ANOVA: activity level and distance										
Source	df	Wilks' λ	F	P	Response	Source	df	Mean squares	F	P
Clutch	6, 128	0.7590	3.16	0.0064	Activity level	Clutch	3	0.0648	3.06	0.0342
Predator cue	3, 30	0.9671	0.36	0.9047		Predator cue	3	0.0099	0.47	0.7056
Infection status	3, 30	0.8544	5.46	0.0065		Infection status	1	0.1922	9.08	0.0037
Predator cue \times infection status	6, 60	0.8019	2.50	0.0256		Predator \times infection	3	0.0183	0.87	0.4629
					Distance	Error	65	0.0212		
						Clutch	3	0.1514	2.69	0.0533
						Predator cue	3	0.0102	0.18	0.9086
						Infection status	1	0.2175	3.87	0.0535
						Predator \times infection	3	0.2222	3.95	0.0119
					Error	65	0.0562			

Note: Tadpoles were derived from four clutches. Significance levels for univariate tests were interpreted at 0.025 (Bonferroni-corrected for two response variables).

chytrium dendrobatidis exposure significantly reduced tadpole activity level, but this response was not significantly affected by predator cues (Table 1, Fig. 1A). Although the predator cue \times infection status interaction was not significant, activity levels were similar for uninfected and infected larvae in the presence of visual or chemical predator cues. However, uninfected larvae had significantly higher activity than infected ones in the absence of cues ($t_{\alpha=0.05(2), 17} = 2.51$, $P = 0.0290$) and in the presence of visual and chemical predator cues simultaneously ($t_{\alpha=0.05(2), 17} = 2.13$, $P = 0.0489$; Fig. 1A). Activity level significantly differed among tadpoles from different egg clutches (Table 1).

Tadpole distance was significantly affected by the interaction between predator cue and infection status, although predator cue and infection status main effects were not significant (Table 1). Infected tadpoles were observed closer to the center divider than uninfected ones in the presence of visual predator cues only ($t_{\alpha=0.05(2), 16} = 4.42$, $P = 0.0004$), but were found farther away from the center divider than uninfected animals when both visual and chemical predator cues were present simultaneously ($t_{\alpha=0.05(2), 14} = 2.22$, $P = 0.0438$; Fig. 1B). Tadpoles from different clutches were marginally different in distance (Table 1).

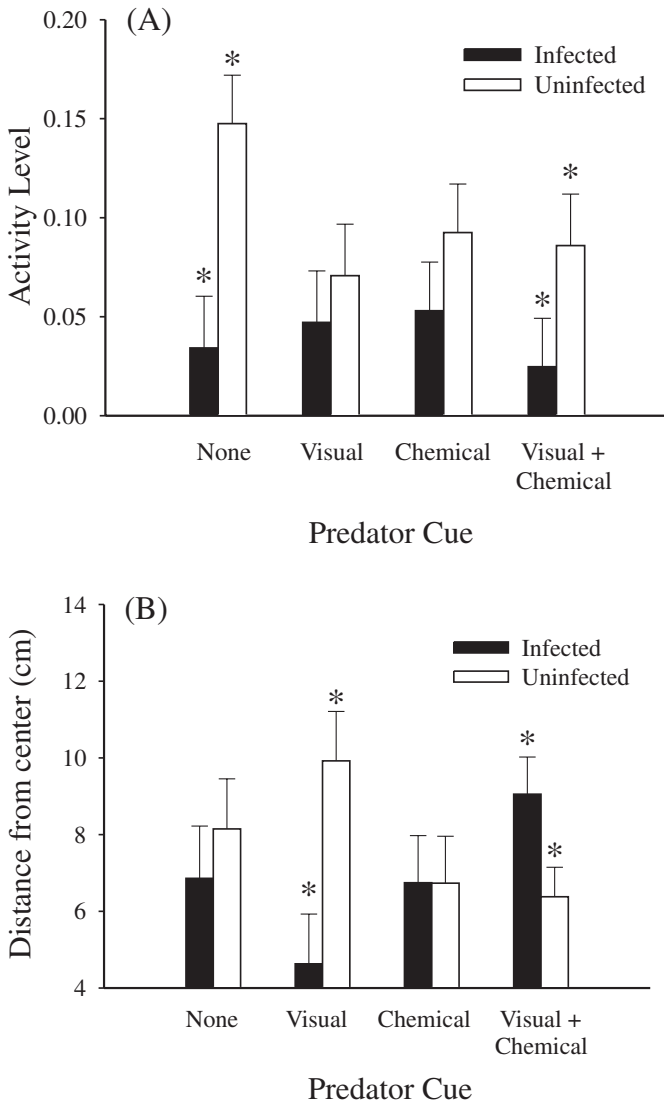
In the lethal presence of *L. macrochirus*, significantly more uninfected than infected tadpoles were consumed (proportion of uninfected tadpoles surviving = 0.313 ± 0.057 , infected = 0.525 ± 0.057 ; $G_1 = 7.486$, $P = 0.0064$, $n = 160$).

Discussion

Behavioral changes are important in modifying ecological interactions such as predation, but studies of how disease can modulate susceptibility to predators are rare (Giles 1983; Parris et al. 2004). Our experiments suggest that infection by *B. dendrobatidis* induces behavioral responses that likely minimize predation risk for *R. pipiens* tadpoles in some environments. Animals infected with chytridiomycosis exhibited stereotypical responses to infection by decreasing activity levels. Infections often cause a physiological response to disease that can change host behavior (Thompson 1990); however, we did not test the physiological mechanism herewithin. Low activity and high survival with predators are responses expected by hosts to a pathogen that requires its host to remain alive. *Batrachochytrium dendrobatidis* spreads among hosts by releasing zoospores from infected hosts, and an environmental reservoir is currently unknown (Longcore et al. 1999; Pessier et al. 1999). Thus, maintaining amphibian host survival may be important for pathogen persistence and spread.

Uninfected tadpoles reduced their activity levels in the presence of predator cues. Low activity in predator environments may benefit tadpoles because their immobility reduces predation risk from many visually foraging aquatic predators, such as sunfish (e.g., Lawler 1989; Werner and Anholt 1993). However, reduced conspicuousness to predators comes at a cost, because low activity levels reduce foraging efficiency (Skelly 1994; Anholt et al. 1996), decrease competitive ability (Morin 1983), and prolong development (Skelly 1994); thereby, ultimately increasing time exposed to aquatic predators. Thus, activity patterns for uninfected tadpoles re-

Fig. 1. (A) Proportion of time spent active and (B) distance from center divider for uninfected and *Batrachochytrium dendrobatidis* infected northern leopard frog (*Rana pipiens*) tadpoles observed for 20 min trials in the absence of predator cues (none), or in the presence of visual, chemical, or combined visual and chemical cues from bluegill sunfish (*Lepomis macrochirus*) in laboratory containers. Asterisks indicate significant differences between infection status means within predator cue treatments. All values are treatment means + 1 SE.



flect a trade-off between avoiding predators and obtaining resources (Sih 1985; Nicieza 2000).

Much of the variation in activity between uninfected and infected tadpoles was driven by differences in the absence of predator cues, and in the presence of combined visual and chemical cues. In predator-free environments, uninfected tadpoles should be at a selective advantage relative to infected tadpoles because of higher activity and probable increased foraging efficiency. But by exhibiting significantly lower ac-

tivity under conditions of combined visual and chemical predator cues, infected larvae may avoid predation more efficiently than uninfected ones; a conclusion that is consistent with our predation susceptibility experiment. Parris (2004) demonstrated that *R. pipiens* tadpoles infected with *B. dendrobatidis* exhibit longer larval period lengths and smaller body masses at metamorphosis when reared in the absence of predators in artificial ponds, suggesting that *R. pipiens* tadpoles with reduced activity also may have slower development and growth, which often reduces fitness components in terrestrial life-history stages (e.g., Smith 1987; Parris 2001). It is also important to note that infected tadpoles maintained low activity levels across all treatments and did not facultatively adjust their activity in relation to predator cues. Thus, low activity, although potentially conveying resistance to predation, may be a by-product of infection (Giles 1983) rather than an adaptive host response to predator cues (Godin and Sproul 1988).

When exposed to visual predator cues only, infected tadpoles remained closer to the center divider than uninfected tadpoles. Infection therefore may increase susceptibility to predation because of impaired visual recognition capabilities. However, use of vision as a primary antipredator sensory modality is rare in larval amphibians that often inhabit benthic areas, which present challenging visual environments because of vegetation, plankton, or suspended particulate matter (Petranka et al. 1987; Stauffer and Semlitsch 1993; Kiesecker et al. 1996). When exposed to chemical cues only, infected and uninfected tadpoles did not differ in distance from the center divider; this was expected because chemical cues alone did not provide sufficient information on spatial location of predators in our laboratory containers. However, in the simultaneous presence of non-contact visual and chemical predator cues, infected tadpoles had a performance advantage by positioning themselves farther from the center divider than uninfected tadpoles. This interpretation is based on the assumption that proximity to predators increases conspicuousness and risk of predation (Lima and Dill 1990). Chemical cues are known to play important roles in predator recognition of amphibians (Petranka et al. 1987; Kiesecker et al. 1999), especially at night, in turbid water, or in complex structural aquatic environments. Thus, our experiment demonstrates that detection of combined visual and chemical cues elicits infection-mediated avoidance behaviors in *R. pipiens* tadpoles infected with chytridiomycosis. The requirement for combined chemical and visual stimuli to induce a response, as opposed to chemical cues alone, suggests that visual cues give tadpoles additional information about the location of the predator that is not present in chemical cues alone. However, the underlying mechanisms for these responses were not tested in this study. Future work could involve studies of neurological responses to infection to better understand behavioral differences between infected and uninfected tadpoles.

Low mortality in the lethal presence of fish predators, coupled with low activity levels and significantly greater distance from predators when exposed to nonlethal visual and chemical cues, suggests that infection by *B. dendrobatidis* does not increase *R. pipiens* tadpole mortality in predatory environments. In fact, uninfected tadpoles suffered 21% greater mortality than infected ones in the lethal presence of preda-

tors. Although not tested in our experiment, patterns of differential mortality could also be a result of fish avoiding infected tadpoles. It is unknown if fish can recognize and respond to diseased prey, or if *L. macrochirus* can be infected by *B. dendrobatidis*. However, previous experiments suggest that amphibians themselves avoid contact with and preying upon diseased conspecifics (Pfennig et al. 1998; Kiesecker et al. 1999).

Lower predation of infected relative to uninfected tadpoles is likely beneficial for pathogen transmission. *Batrachochytrium dendrobatidis* can remain infectious in water for up to 7 weeks (Johnson and Speare 2003), but it is a pathogen that is spread between amphibian hosts and predation is not required for transmission. If antipredator responses benefit the pathogen by keeping the host alive, then selection should favor the retention of efficient antipredator and avoidance responses in infected animals. Alternatively, reduced host activity and spatial avoidance of predators could be patterns actively induced by the pathogen, and not necessarily an adaptive host response. *Batrachochytrium dendrobatidis* may be lethal to infected metamorphs for some species of anurans (e.g., Berger et al. 1998), but infection does not directly increase mortality in tadpoles (Parris and Baud 2004; Parris and Cornelius 2004). Sublethally infected tadpoles thus could serve as an intraspecific pathogen reservoir (sensu Brunner et al. 2004) for *B. dendrobatidis* to infect newly metamorphosing froglets and adults in natural aquatic environments.

Because of the increasing evidence of *B. dendrobatidis* infection in natural populations (Daszak et al. 2003; Morehouse et al. 2003; Stuart et al. 2004), it is important to discern the mechanisms by which disease can affect amphibians. Tadpole behavioral responses to pathogens likely reflect both the physiological effects of infection and the selection pressures on host and pathogen. Infection-induced low host activity reduces conspicuousness to aquatic predators, but reduces foraging efficiency in the absence of predators. Efficient movement away from combined visual and chemical cues may preclude infected animals from suffering high mortality from predators in natural environments. Reduced predation and enhanced antipredator responses for infected relative to uninfected tadpoles likely benefits *B. dendrobatidis* because this pathogen may use the tadpole life-history stage as a pathogen reservoir and transmission vector. Our study suggests that pathogen infection may alter host behavior such that host survival — and consequently pathogen opportunities for transmission — is increased. Future work should focus on the underlying mechanisms to explain such host behaviors.

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