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Author(s) :Lígia Pizzatto and Richard Shine
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# YOU ARE WHAT YOU EAT: PARASITE TRANSFER IN CANNIBALISTIC CANE TOADS

LÍGIA PIZZATTO[1] AND RICHARD SHINE

*School of Biological Sciences A08, The University of Sydney, New South Wales 2006, Australia*

ABSTRACT: Pathogen transfer may be an important but poorly understood cost of cannibalism. Does the consumption of smaller conspecifics by Cane Toads (*Rhinella marina*) result in transfer of viability-reducing parasites such as nematode lungworms (*Rhabdias pseudosphaerocephala*)? Our experimental trials confirm that cannibalistic toads can indeed become infected, and our results are probably the first evidence of macroparasite transmission via intraspecific predation in amphibians. Our results also show that parasites acquired via cannibalism are viable, develop into fertile adults, and reduce the locomotor performance of the hosts. How cannibalism contributes to nematode transmission and spread in natural populations is not known, but we propose a scenario in which this interaction would be likely to increase the lungworm prevalence, intensity, or persistence.

*Key words:* *Bufo marinus*; Bufonidae; Disease; Host–parasite; Intraspecific predation; Pathogen; *Rhinella marina*

CANNIBALISM—the ingestion of bodies or body parts of conspecific organisms—has been studied by anthropologists since the 15th century (see Lindenbaum, 2004), and by zoologists since early last century (Glaser, 1905). This type of predatory interaction can affect ecological dynamics at population (Moksnes, 2004; Wissinger et al., 2010) and community (Rudolf, 2007*a*,*b*) levels. The advantages of cannibalism are obvious: Conspecifics provide additional nutrition to the predator, enhancing individual survivorship (Lindenbaum, 2004) and development (Claessen et al., 2002), and enabling population persistence during food scarcity (Getto et al., 2005; Wise, 2006). Cannibalism also can reduce intraspecific competition (Polis, 1981; Wise, 2006).

Cannibalism is common in natural conditions, but there is considerable variation in the frequency of this behavior within and among species. Given the enormous advantages of this predatory interaction, why is it not even more widespread? In an evolutionary context, the answer to this question presumably lies in costs of cannibalism. For example, a predator that eliminates conspecifics can reduce its own inclusive fitness if the victim is a close relative (Hamilton, 1964), and can face increased risk of injury if it attacks an individual with similar fighting abilities as itself (Dawkins, 1976). However, such risks often may be low because cannibals typically target smaller or more vulnerable conspecifics (Polis, 1981), and some species recognize and avoid eating kin (Pfenning, 1997). The risk of acquiring parasites and diseases may be an important cost of cannibalism (Pfenning et al., 1998). Pathogen transfer is more likely from eating conspecifics than from eating heterospecifics, probably because of the host specificity of many pathogens (see Pfenning, 2000; Pfenning et al., 1998). Controversially, the idea that disease transmission acts as a selective force on the evolution of cannibalism is not supported by evidence from ecological models (Bolker et al., 2008; Rudolf and Antonovics, 2007; see also Rudolf, 2010, for factors driving the evolution of cannibalism). In salamanders, only very high levels of infection by an iridovirus can cause a strong decrease of cannibal allele frequencies (Bolker et al., 2008). In addition, because both cannibalism and disease dynamics are density dependent, they tend to reduce population density and, thus, exclude each other from the population (Bolker et al., 2008). However, that disease transmission is unlikely to select against cannibalism does not mean that it cannot increase its costs.

The controversy about the importance of disease transmission as a cost of cannibalism and the way it affects natural populations evinces the need for further studies in a

[1] CORRESPONDENCE: e-mail, ligia.oceanica@gmail.com

different range of host–parasite systems. To date, the frequency and consequences of parasite transfer through cannibalism have received little scientific attention (Rudolf and Antonovics, 2007). For example, the only studies on pathogen transmission by cannibalism in amphibians involve micropathogens (ranavirus, Gray et al., 2009; Pearman et al., 2004; and bacteria, Pfenning, 2000; Pfenning et al., 1998).

We studied the role of cannibalism by Cane Toads (*Rhinella marina,* formerly *Bufo marinus*; new nomenclature following Pramuk et al., 2008) on the transmission of the lungworm *Rhabdias pseudosphaerocephala* to address the questions: (1) Can the lungworms be transferred by cannibalism? (2) If so, then do parasites taken up this way influence the cannibal's viability?

## Materials and Methods

### *Study Species*

Cane Toads are large anurans native to South and Central America, but have been widely translocated for biocontrol of agricultural pests (Kraus, 2009; Lever, 2001). Toads were brought to Australia in 1935, and have since spread over more than a million square kilometers and inflicted severe ecological damage by fatally poisoning native predators (Shine, 2010). In Australia, some, but not all, populations of Cane Toads contain the nematode lungworm *R. pseudosphaerocephala* (Phillips et al., 2010), which is an American taxon apparently brought to Australia with the toads (Dubey and Shine, 2008). The lungworm life cycle is generalized from other species in the genus. The infective larvae (L3) penetrate the host skin and migrate to the lungs, where they develop into hermaphroditic adults. The adults lay the eggs in the lungs, and from there the eggs pass to the intestines and hatch into L1 larvae (Anderson, 2000; Baker, 1979; Langford and Janovy, 2009). These larvae are usually first seen in Cane Toad feces after 30 d postexposure (L. Pizzatto, personal observation), which is similar to the pattern seen in *Rhabdias americanus* in *Bufo americanus* (Anderson, 2000; Langford and Janovy, 2000). After being defecated, L1 larvae develop into L3 larvae or free-living dioecious adults that produce L3 larvae (Anderson, 2000; Baker, 1979; Langford and Janovy, 2000).

Because the parasite is apparently toad specific (Dubey and Shine, 2008; Pizzatto et al., 2010) and can reduce the survival and growth rates of metamorph Cane Toads, it may provide a means for toad biocontrol (Kelehear et al., 2009). Potentially, the probability of persistence of the lungworm in Cane Toad populations may be affected by the occurrence of cannibalism: When dry conditions prevent metamorphosing toads from dispersing away from the edges of natal ponds, large metamorphs predate heavily on smaller conspecifics (which comprise >60% of the diet of the larger animals; Pizzatto and Shine, 2008). Hence, any effects of cannibalism-mediated parasite transfer are of particular interest in this system.

### *Methods*

Sixteen small toads (mean 15.17 g ± 6.33 g SD) were collected in Middle Point Village (12.6°S, 131.30°E; datum = WGS84), 60 km east of Darwin in the wet–dry tropics of the Northern Territory. Because toads in this area are already exposed to the lungworm, the animals were initially treated with the antiparasite drug ivomectin (Ivomec®, 0.002 mg/g of toad mass, injected subcutaneously) and kept in well-ventilated plastic boxes (50 × 50 × 40 cm; up to 4 individuals per box). Each box had a pool of nonchlorinated water and each toad was fed two lab cockroaches every third day. Boxes were cleaned daily, and a second dose of ivomectin was administrated 15 d after the first dose to guarantee complete removal. In prior studies on this host–parasite system, we confirmed that this deworming approach was effective by regularly checking the feces and dissecting preinfected and treated individuals. In the current experiments, we used a microscope to inspect feces found in the boxes in order to confirm the absence of lungworm larvae prior to the experimental infections. Although treating the toads for previous (natural) infections was logistically demanding, we opted for using field-collected animals as cannibals because raising toads from eggs to the size at which they become cannibalistic would take several months and intensive husbandry and quarantine care.

Thirty days after the second dose of ivomectin, each toad was weighed, placed on a vinyl floor (average room temperature 28.01°C ± 1.31 SD), and stimulated to hop by touching the posterior part of its body with a long stick. We scored the time each animal took to hop 200 times, after which most animals appeared exhausted and became increasingly reluctant to move. All toads were then housed in well-ventilated individual boxes (33 × 21 × 11 cm). Each box contained a pool of nonchlorinated water and a preboiled soil substrate, and was kept over a plastic tray containing iodine scrub (Betadine® Surgical Scrub; 7.5% weight/volume povidone, which is an iodine solution) to prevent ingress of nematode larvae. All toads were fed every third day exclusively on live conspecific metamorphs (comprising 6%–8% of the cannibal's body mass). Eight of the toads were fed noninfected live metamorphs and the other eight were fed infected metamorphs.

Metamorphs to be used as food for the cannibals were raised in captivity from eggs under uncontaminated conditions; see Pizzatto et al., 2010), kept in groups of about 30 individuals in plastic boxes (33 × 21 × 11 cm), and fed every third day on ad libitum termites. To obtain infected metamorphs, we added about 3000 infective larvae of *R. pseudosphaerocephala* to the soil of the treatment boxes. Exposure to this high dose of larvae, which is a level often present in a single fecal pellet from an infected adult toad (L. Pizzatto, personal observation), ensured that metamorphs would contain at least some larvae at the time they were fed to the cannibals. These larvae were cultured from adult worms collected from the lungs of adult toads from Middle Point, and prepared as described by Kelehear et al. (2009). The infected metamorphs were fed to the larger toads less than 9 d after this infection treatment, before the lungworms could mature and begin producing eggs. Thus, any parasites transferred to the larger toads were the same lungworms as had infected the small toads, not the progeny of those animals (the larvae require at least 10 d postinfection to reach the lungs of metamorphs and begin developing into adults; L. Pizzatto, unpublished data; see also Anderson, 2000, for other toad lungworm species). To ensure further that any parasite transfer was direct (via cannibalism) rather than via free-living infective larvae, boxes were checked daily and changed if any fecal material was present. After 42 d, all toads were weighed, and their hopping performance was scored as in the start of the experiment. We then euthanized the toads by immersion in 4 g/L buffered tricaine methanesulfonate (MS222; Sigma, USA), and checked their lungs for the presence of Lungworms.

We used repeated-measures analyses of variance to compare control vs. treatment groups for (1) the times taken to perform 200 hops, and (2) body sizes at the beginning and end of the experiments. Preliminary analyses showed that including the total mass of prey consumed by each toad during the experiment did not affect the results of these analyses. We used logistic regression to verify that the proportion of toads containing mature lungworms differed between experimental and control groups at the end of the experiment.

## Results

Of eight toads that were fed on infected metamorphs, five (62.5%) had adult worms in the lungs (1–50, median = 9 worms) at the end of the experiment. None of the controls had any worms, which confirmed that the treatment affected lungworm uptake (logistic regression on presence/absence $c^2 = 5.73$, 1 df, $P < 0.02$; ANOVA on numbers of worms $F_{1,11} = 5.26$, $P < 0.05$). Control toads took less time to hop 200 times when compared to the treatment group (interaction time * treatment $F_{1,8} = 11.05$, $P < 0.015$; see Fig. 1), but growth rates did not differ significantly between the two groups (interaction time * treatment $F_{1,9} = 0.008$, $P = 0.93$).

## Discussion

In Cane Toads, cannibalism can confer a cost in terms of macroparasite transfer. To our knowledge, this is the first example of macroparasite transmission via intraspecific predation in amphibians. Our data show that the infective larvae of *R. pseudosphaerocephala*, initially taken into the cannibal's body inside a smaller metamorph toad, can move from the

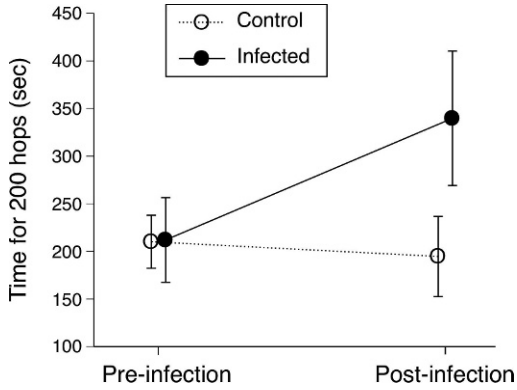


FIG. 1.—The effect of cannibalism-mediated parasite transmission on the locomotor performance of Cane Toads (*Rhinella marina*). The graph shows mean values and standard errors for the times taken to complete 200 hops by these toads before and after they were allowed to consume either parasite-infected or noninfected conspecifics.

host's alimentary tract to the lungs. Larvae ingested in this way appear to be fully viable: Inside the cannibal, the worms were able to mature in the lungs and affect the locomotor performance of their new host. Similar scenarios occur in other amphibians and their microparasites. For example, mass die-offs of Tiger Salamanders (*Ambystoma tigrinum*) were associated with bacterial infections, and cannibal morphs were overrepresented among the dead larvae (Pfenning et al., 1991, 1998). Spadefoot Toads fed on conspecifics containing high pathogen loads grew more slowly than did those fed similarly infected noncongeneric tadpoles (Pfenning, 2000).

Cannibalism can play an important role in the spread and persistence of pathogens within and among host populations. In humans, cannibalism has been linked to the origins of tapeworm (*Taenia* sp.) infections (Baer, 1940; Hoberg et al., 2000), and the spread of Kuru, a neurodegenerative prion disease associated with the consumption of corpses by the Fore people of Papua New Guinea (Anderson, 2008). Among nonhuman animals, infections by the widespread nematode *Trichinella* spp. are maintained by cannibalism and scavenging behavior among wild carnivores, facilitating transmission of the worm to domestic animals and humans (Pozio, 2000). Cannibalism of autotomized tails may be the main transmission mode of sarcosporidians among the insular lizards *Gallotia stehlini* (Matuschka and Bannert, 1987, 1989).

Despite the potential for cannibalism to act as a pathway for transmission of diseases, it is rarely a major factor in this regard (see Rudolf and Antonovics, 2007). If cannibalism is a one-to-one relationship, the number of infected individuals does not increase as a result of the pathogen transfer (the cannibal becomes infected, but the infected victim dies), and thus the disease cannot spread (Rudolf and Antonovics, 2007). Disease spread is more likely to occur via group cannibalism or scavenging, in which many individuals share an infected victim (Rudolf and Antonovics, 2007). Cannibalism in Cane Toads does not exhibit these features, and hence would not be expected to increase the spread of the parasite under the model of Rudolf and Antonovics (2007).

However, other features of Cane Toad cannibalism change this outcome. The primary cannibalistic cohort includes the largest metamorphs, which feed on their smaller conspecifics around the edges of natal ponds during the long tropical dry season (Pizzatto and Shine, 2008), when high desiccation rates prevent the smaller animals from dispersing (Child et al., 2008). Infection with *R. pseudosphaerocephala* reduces the locomotor abilities of metamorph Cane Toads (Kelehear et al., 2009), suggesting that infected animals (small, slow) are disproportionately likely to be victims of the larger cannibals. Those cannibals obtain most of their nutrition from smaller conspecifics over a prolonged period, increasing the cumulative probability of a cannibalistic toad consuming an infected metamorph. Lastly, a young toad's body size strongly affects its vulnerability to mortality sources such as desiccation (Child et al., 2008), predation (Ward-Fear et al., 2010), and lungworm infection (Kelehear et al., 2009). Because survival rates of metamorph toads are very low and size dependent (Alford et al., 1995), the (large) cannibals may be the only animals that survive long enough to recruit into the adult population. Under these conditions, cannibalism-mediated pathogen transfer could elevate the proportion of infected toads through time by selective mortality of small infected individuals that

are very unlikely to survive. The mechanism differs from that envisaged by Rudolf and Antonovics (2007), instead with cannibalism in this system transferring lungworms from a "doomed" host to a "safe" host. The end result is that cannibalism-mediated pathogen transfer could increase prevalence, intensity, and parasite persistence in a population. We have limited unpublished field data that offer some support to our idea. From 16 cannibal-sized toads collected in the absence of small metamorphs (and thus probably not involved in cannibalism, at least recently), parasitism intensity was nine worms per toad, as in our experiment, but prevalence was only 18.7% (opposed to 62% in the experiment).

In conclusion, we demonstrated that a macroparasite of Cane Toads can be transmitted via cannibalism and remains viable in the cannibal host. Further ecological modeling (including size-dependent mortality of infected individuals) and additional field studies (including sampling toads involved in cannibalistic interactions) are needed to test our hypothesis about how cannibalism could affect disease dynamics at the population level.

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