The Mysterious Vanishing Frogs of North America

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I have a passion for all things slimy, wet, and creepy-crawly. Some of the best times of my life have been spent on my knees, digging in the dirt for earthworms, traipsing back from ponds with buckets of putrid swamp water teeming with tadpoles, or chasing fat little toads in knee-high grass. I love the outdoors and all of the ugly animals that inhabit it. I like to catch them, watch them, and – especially – photograph them. For the longest time, lizards have been the main focus of my photographic endeavors, but last summer, inspired by a book on frogs from the local library, I set out to document the lives of these often overlooked amphibians. I live in southern Florida near the Everglades, and I remember catching, mating, and raising dozens of frogs as a kid, so I didn't expect to have to lug my tripod and lenses very far to find a suitable subject. But much to my dismay, I didn't find a single frog in an entire afternoon of searching. In fact, in the year and half since my initial search, I've seen only four frogs.

I immediately assumed that pollution had decimated the populations of my favorite amphibian. I have no hard evidence, but I believe that this is a common belief shared by many fellow lay naturalists. This makes scientific sense; frogs spend most of their lives in water, have thin, easily permeable skin during all stages of their lives, and lay their gelatinous eggs in water. Pesticides and other pollutants accumulate in water, where they can easily diffuse into the thin skin of frogs. It all seems perfectly logical.

The catch is, this perfectly logical and widely believed answer is probably false. While there are never any absolutes in ecology, there is a large body of evidence that suggests that declining frog populations are in fact the result of a natural parasite, and that frogs are even evolving to fight the parasite. The facts and science behind declining frog populations will be discussed shortly, but almost as interesting as the facts themselves is how the story was reported in the media, and how the reporting influenced the nation's thinking on frogs.

The frenzy over deformed frogs began on a lazy summer day in August 1995. Cindy Reinitz, a teacher at a school in Henderson, Minnesota, was walking with her middle school students down a deserted, dusty back road on the way to a field trip at a local farm. Along the way, the kids began to chase frogs along the side of the road. When one of the kids showed her a frog with a missing leg, her first instinct was that one of her less than angelic students had pulled the leg off. However, a closer examination of the frog showed no fresh wound, and when she and her middle schoolers set out to capture more frogs, they noticed that a large number of the frogs were missing legs, had more than two legs, or were deformed in some other way (Souder, 1).

The frog story had all of the elements that make a newspaper reporter's ears perk up: children – to provide excellent visuals and add just the right amount of "cute" factor, a defenseless victim – the frogs, an ultimate evil – pollution, and a possible danger to everyone – the frogs could ostensibly be "canaries in a mineshaft" (idea from Reaser &

Johnson, 3). By 1997, an alarming number of newspaper articles had been written on the topic, enough that a prominent researcher, Stanley Sessions of Hartwick College, was prompted to comment, “I have never seen a scientific or biological phenomenon grow so fast with so few publications.” (Kaiser, 1). Later, in a 1998 letter to Science Magazine, Sessions noted that, “Approximately half of the recent reports of deformed amphibians in the United States and Canada are from a single study (my own) of one site in California, published in 1990” (Sessions letter to Science Magazine, 1).

However, baseless speculation is not the exclusive domain of the media. In September of 1997, the Minnesota Pollution Control Agency (MPCA) issued a press release describing unpublished research showing that African clawed frog embryos developed abnormally in local pond water, even though standard tests found the water safe to drink. Residents were naturally alarmed; many of them obtain their drinking water from these very ponds. Scientists quickly assailed these claims; how could results regarding the African clawed frog pertain to the distantly related Minnesota frog populations? More concretely, the scientists at the pollution control agency had overlooked a critical point: African clawed frog embryos won’t grow properly in ordinary water unless a specific salt is added. When the researchers added the salt, the embryos were completely normal (Kaiser, 4). While the MPCA’s actions were certainly justified, given the apparent public health problem, they highlight a key problem that applies to more or less every facet of the frog debate: it’s easy to become panicked when dealing with what appear to be significant public health problems, but it’s critical to remain clear-headed and to trust only scientific, validated evidence.

Complex problems have complex answers that are rarely apparent at first glance. Yet when the world heard about the Minnesota schoolchildren's discovery, the first instinct of most laypeople and even wildlife professionals was to sigh ruefully and blame pollution. But Mark's Theory of Complex Problems proved correct yet again; pollution is actually the least likely of the four competing theories that scientists have proposed to explain the dramatic decreases in frog populations. The foremost explanation is that a parasite called a Trematode is causing the deformities through a purely mechanical means (i.e. the physical presence of the parasite causes the deformities – not any toxins produced by the parasite). Sessions is the chief proponent of this theory, and has been researching the phenomena since 1987 (Sessions & Ruth, 1). The second most probable theory is that natural predation and the cannibalistic tendencies of the tadpoles result in various deformities (Sessions web page, "Predation Hypothesis"). Frogs, and most amphibians, have the extraordinary ability to regenerate parts of their bodies; it is entirely possible that the majority of the observed deformities are result of a leg being bitten off and not regenerating, or a small nip near a leg erroneously causing a new, third leg to grow. The third theory deals with UV-B radiation. It's well known that UV-B radiation is harmful, and that UV-B radiation has been observed to markedly decrease the yield of a batch of eggs (Blaustein, 1), but it is not clear that UV-B radiation is the root cause of the observed frog deformities (Sessions web page, "UV Hypothesis"). Pollution is the fourth most likely cause of the frog deformities. The agent most frequently proposed as the culprit is Retinoic acid; the common mosquito pesticide, Methoprene, is a retinoid mimic. Evidence regarding the specific type of deformations produced by Retinoic acid has all

but eliminated retinoids as a possible cause of frog deformities (Sessions, Franssen, and Horner, 3).

Trematodes are interesting parasites. They begin their lives as cilia-covered free-swimming larva, called miracidium, and incubate inside the bodies of pond snails. As these snails happily scour the muck at the bottom of North American ponds, the Trematodes are multiplying and maturing inside the snail's body. The Trematodes multiply and mature inside the snail's body, and eventually they emerge from the snail with a powerful tail that allows them greater mobility than juvenile miracidium. These Trematodes in the second stage of their lifecycle, called cercariae, infest young tadpoles and form cysts inside of their bodies, in which metacercariae develop. The metacercariae represent the final stage of Trematode development, and emerge from the frog's body when it is eaten by a predator (typically a bird). It is the bird that is the primary host for the Trematode. (University of Wisconsin BioLab, 1).

Stanley Sessions, a biologists at Hartwick College, is the foremost proponent of the Trematode theory. Between 1986 and 1987, he was involved in a routine survey of the endangered frog species A. marcrodactylum in Southern California. Several of the frogs were either found dead or died during the survey, and were subsequently examined microscopically (Sessions & Ruth, 2). Alarming numbers of metacercarial cysts were found in the tissue of the frogs. The cysts, oddly enough, were highly localized in the pelvis and hindlimbs of the frogs (Johnson, 1). Sessions suspected that the cysts were causing the observed limb deformities, but was unsure of the mechanism by which the

cysts caused these deformities. He hypothesized that the cysts were disrupting the tissue by their simple physical presence (i.e. they do not produce a toxin or other chemical that causes deformities). To test his hypothesis, he implanted resin beads of approximately the same size as the metacercarial cysts into the limb buds of young frogs. The types of mutations observed when resin beads were implanted were consistent with the types of mutations observed in Trematode-infested frog populations (Sessions & Ruth, 1).

Research done by Pieter T. Johnson, et al. has both linked pond snails and frogs with limb deformities (2), and has shown through laboratory tests that the Trematode species *Ribeiroia* (4-7) causes deformities in frogs that match the type and distribution of frog deformities observed in the wild. The case for Trematodes is far from open and shut, but the Trematode hypothesis is currently the most probable means to explain the deformed limbs observed in frog populations.

Trematodes are the most likely cause of limb deformities, but the predation hypothesis is the best explanation scientists have to explain the missing limbs observed in frog populations. But before the predation hypothesis can be discussed, it's necessary to discuss the remarkable regenerative abilities of amphibians; these regenerative abilities are critically important to understanding frog deformities. Frogs exhibit a unique ability to spontaneously regenerate their limbs at early stages of their life (Sessions web page, "Regenerative Decline"). The hallmark of frog's natural limb regeneration process is a cartilaginous spike at the end of limb. If a frog's limb is injured early on in its life, odds are that the frog will be able to re-grow an entirely new limb. However, if the injury

occurs later in the frog's life, the frog will most likely be unable to regenerate the entire limb and will instead grow only a meager stump. The regenerative process is not perfect; even if are young frog's limb is severed, the frog may not re-grow a limb, and a small nip near a leg may cause a new, third leg (Sessions web page, "Predation").

These nips and scratches are key to understanding the predation hypothesis, the second most likely explanation for frog deformities. The premise is simple: bites or scrapes on the tadpole may trigger the frog's regenerative response and induce the formation of additional limbs. Missing limbs can be explained by unsuccessful predation of adult frogs, or unsuccessful predation of young frogs where the limb did not regenerate.

The background of the predation hypothesis is fairly solid. It is common knowledge that many predators feed on frogs in all stages of life, but there is also a solid body of evidence showing that many nips, bites, scratches, and wounds in tadpoles are the result of cannibalism. Sessions grew bullfrog tadpoles in overcrowded conditions and observed the tadpoles attempting to devour one another, and the resultant deformities caused by these attacks were similar to the deformities observed in the wild (Sessions web page, "Predation"). When I raised frogs this summer, I too observed the cannibalistic tendencies of frogs; shortly after two tadpoles died, I observed several of the larger tadpoles fighting while consuming the bodies of their two unfortunate comrades. Sessions' own research has shown that causing physical trauma (i.e. implanting resin beads) will spur leg regeneration (Sessions & Ruth, 1). Additionally, other research has shown that surgical rotation of limb buds, or even movement of cells between different

regions of the limb bud can prompt the development of extra limbs (Johnson, 2). The predation hypothesis appears to be perfectly plausible, but I could find no published research that more than mentioned the predation hypothesis in passing. Relying solely on Sessions' unpublished work is dangerous, but the quality of Sessions previous research has been extremely high, and there are no other probable hypotheses to explain limb amputations.

Sessions has conducted unpublished research to determine the veracity of the predation hypothesis. He reasons that if limb amputations and deformities are caused by a chemical or other external factor, one would expect there to be missing limbs from the earliest stages of tadpole development, and no sign of trauma to the limbs. However, research from the field shows the opposite: longitudinal studies of tadpoles have shown that they can suddenly lose a leg, a tail, or even an eye at any stage of life (Sessions web page, "Predation"). Sessions admits that there is a lot more work to be done before the predation hypothesis is a foregone conclusion, but he feels that it is a highly probable cause for observed frog deformities and leg amputations.

The final two hypotheses, UV-B rays, and chemical pollutants, are the least likely hypotheses. The main proponent of the UV-B radiation theory is Andrew Blaustein of the University of Oregon. He has put together a convincing case that increased levels of UV-B rays increase the rate of embryo mortality in a given cluster of eggs (Blaustein, 1), but his case for UV-B rays causing deformities in adult frogs is weak at best. When UV-B rays harm a frog, they typically harm it so much that it dies almost immediately; few

embryos damaged by UV-B radiation live to adulthood. Therefore, while UV-B radiation is a serious concern for frog populations, it cannot explain limb amputations or deformities (Blaustein, 3).

The viability of UV-B radiation as a possible hypothesis is further diminished by the fact that UV-B radiation is predicted to cause a very specific subset of limb deformities in frogs. Specifically, existing amphibian research shows that UV-B radiation typically produces deformities that are "both bilateral and symmetrical" (Sessions web page, "UV-B Radiation"). It's not necessary to know exactly what "bilateral and symmetrical" means, other than to understand that it represents a specific type of deformity that has never been observed in the field. Scientists may be fairly certain that UV-B radiation is not the root cause of limb deformities, but what about limb amputation (i.e. a missing limb)? The key to solving this specific puzzle lies in the previously discussed cartilaginous spikes; they form as part of the natural response of an adult frog to a limb trauma, and are known to be suppressed by UV-B radiation. A large percentage of the frogs observed with missing legs have cartilaginous spikes, which rules out UV-B radiation (Sessions web page, "UV-B Radiation").

Both cartilaginous spikes and specific deformation patterns also hold the key to debunking the retinoid hypothesis. Retinoids are, without a doubt, harmful chemicals; they are known to cause birth defects in humans, and have numerous side effects. It has also been shown that retinoids can produce limb deformities. However, the limb deformities produced by retinoids are of several very specific types; they are typically

posterior mirror image duplications (PMIDs) and proximal-distal duplications (PD duplications) (Maden, 1). It's not important to understand exactly what "PMIDs" and "PD duplications" are, other than to know that they are widely recognized types of deformities, and have never been observed in the field. This therefore precludes them as a possible cause of limb deformities in frogs. Retinoids are not a plausible hypothesis for limb amputations because they, like UV-B radiation, are known to inhibit the formation of cartilaginous spikes.

There is a considerable amount of evidence that the frog / Trematode relationship is a completely natural one – one in which the frogs and the Trematodes are regulating each other's populations. Trematode-infested frogs with deformed limbs are highly susceptible to predation. This is desirable for the Trematodes, as their end hosts are birds, key frog predators. However, if birds gobble up too many frogs, then the Trematodes lose their primary carrier. Frogs who are resistant to Trematodes are less likely to be eaten by predators, and are therefore are more likely to live long enough to procreate – in evolutionary jargon, these frogs are "selected." Trematodes, in turn, adapt to infect these resistant frogs. The conflicting evolutionary pressures exerted by Trematodes and frogs keep populations in a natural balance.

Ironically, it seems that the most probable causes of frog deformities and population declines are completely natural. While this may be depressing for naturalists like myself, who live in areas where there are now virtually no frogs, it's important to remember that animal population declines – and even extinctions – are nothing new; in fact, they predate

the first human being by approximately 4 billion years. While there's no doubt that pollution, pesticides, and other human-caused maladies are the root cause of countless environmental problems, population fluctuations are natural phenomenon.

Yet no newspaper article mentioned the fact that Trematodes and frogs exist in a natural homeostasis; in fact, very few of them even mentioned Trematodes, despite the fact that Sessions' widely accepted and peer-reviewed research on Trematodes had been published more than five years before the majority of the articles were written. But you can't lay all of the blame with the authors of articles on frog deformities, or with the producers of TV shows on declining frog populations; when dealing with any serious public health issue, it's easy – actually, it's perfectly natural – to become panicked or overly dire, and to misrepresent the facts. Such errors aren't malicious, but are products of a highly deadline-centric media system that precludes all but the most meager research, and are also simply human nature. The media firestorm about deformed frogs didn't do any harm – in fact, increased public consciousness regarding any environmental issue is unilaterally a good thing – but one doesn't need to look far or think very hard to imagine a situation where exaggeration, confusion, and slanted perspectives could cause irrevocable harm. With this in mind, I believe that there is an enduring lesson that we can all take away from the frog fiasco: whenever confronted with what seems to be an obvious problem with an obvious cause and an obvious solution, take care to remember that a good, strong dose of skepticism has never killed anyone.

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Section: A SECTION
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Tocris: “Retinoids are derivatives of vitamin A and bind to nuclear retinoic acid receptors (RARs) and retinoid X receptors (RXRs) to directly modulate gene expression”