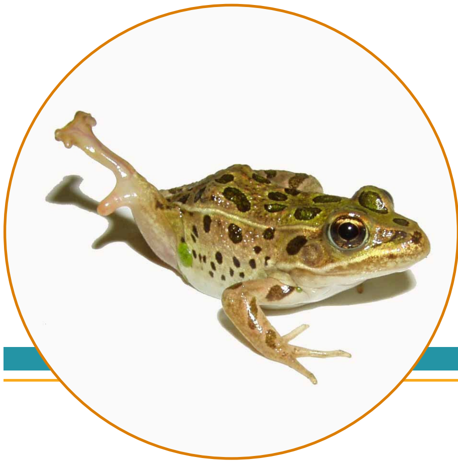
WILDLIFE FUTURES PROGRAM

PARTNERS IN AMPHIBIAN AND REPTILE CONSERVATION

FACTSHEET



FROG DEFORMITIES

Photo Pieter Johnson, University of Colorado at Boulder

OVERVIEW OF MALFORMATIONS

Over the past two decades, increasing observations of frogs with severe deformities, including extra limbs, missing limbs, and misshapen limbs, have garnered widespread attention and concern. This phenomenon predominantly affects the hind legs of tadpoles and young, recently metamorphosed frogs, in part because deformed animals do not survive into adulthood. While a small fraction of individuals are likely to be abnormal in any population (typically <2%), recent cases often affect 10% or more of emerging frogs, and up to 80% of all frogs. Because of their reduced capacity to feed, jump, swim, and avoid predators, deformed frogs can face additional mortality risks. Within the United States, most observations of deformities – which have been detected in dozens of amphibian species, including those known to be threatened or endangered– are reported in the western, midwestern, and northeastern regions of the US. It is not known whether deformities have increased among amphibian populations, their geographic range, across a larger geographic range, or within individuals.

Image source: David Herasimtschuk, Freshwaters Illustrated. A northern leopard frog from a malformation hotspot in Minnesota.

CAUSES: TREMATODE PARASITE INFECTION

Debate over the causes of amphibian deformities has been extensive, including discussions over the roles of pesticides, UV radiation, invasive predators, endocrine disruption, and parasites. Here we focus on the role of parasite infection. Evidence from field surveys and experiments implicates infection by a trematode parasite (*Ribeiroia ondatrae*) as the most widespread driver of limb deformities in amphibians. Based on experimental infection studies, exposure to *R. ondatrae* induces missing, extra, or twisted limbs in a dose-dependent manner. Thus, low levels of infection probably cause little harm, but when amphibian larvae (tadpoles) are exposed to large or persistent numbers of infectious parasites, the risk of death or deformity increases. Among surveys of North American amphibian populations, there is often a functional link between the abundance of *R. ondatrae* and the frequency of severe limb deformities among juvenile amphibians that can range as high as 80-100%.

OVER THE PAST TWO DECADES, INCREASING OBSERVATIONS OF FROGS WITH SEVERE DEFORMITIES, INCLUDING EXTRA LIMBS, MISSING LIMBS, AND MISSHAPEN LIMBS, HAVE GARNERED WIDESPREAD ATTENTION AND CONCERN.

continued ▸

LIFE CYCLE

Ribeiroia is a digenetic trematode (flatworm) that cycles through freshwater snails, amphibians, and birds. Infected snails release a free-swimming stage of the parasite that invades the developing limbs of tadpoles, sometimes causing them to develop abnormally. The parasites encyst just below the skin, typically near the hind limbs. Water birds such as herons and ducks can become infected when they eat infected amphibians. As birds fly across the landscape, they release eggs of the parasite with their feces that then become infectious to a new crop of snails. Deformities in frogs may be beneficial to the parasite by increasing the chance an infected frog is eaten. Indeed, deformed frogs show inhibited jumping ability, slower swimming, and reduced feeding. Although many different species of water birds can become infected with *Ribeiroia*, the parasite is highly specific in the types of snails it infects, as only rams horn snails (genus *Helisoma*) are suitable hosts.

SPECIES AFFECTED

Ribeiroia has been recorded in over 30 species of frogs, toads, and salamanders. Primarily, these are species that breed in still-water (lentic) habitats, such as lakes and ponds. Whether an individual exhibits a deformity depends, at least in part, on the number of infectious *Ribeiroia* to which it has been exposed. High levels of exposure can lead to severe limb deformities or mortality. Deformities are most often reported in recently metamorphosed frogs, likely because deformed animals do not survive to adulthood. Based on experimental studies, frogs and toads with smaller body sizes and faster development (i.e., a fast 'pace of life') tend to be more vulnerable to infection and deformities relative to large-bodied frogs or salamanders.

DISTRIBUTION

Deformed amphibians have been observed in many species and countries, likely stemming from a wide range of causal factors. Parasite-induced deformities, such as those caused by *Ribeiroia*, have primarily been reported in North America although other trematodes have recently been linked to frog deformities in Europe and India. *Ribeiroia*-infected amphibians are most commonly observed in wetlands, lakes, and pond habitats, especially along the major migratory bird flyways (e.g., the Pacific, Mississippi and Atlantic flyways). Within the United States, particular hotspots have been reported from the West, the Upper Midwest, and the Northeast. Deformities also are more frequently reported in northerly latitudes. Some studies have suggested that parasite-induced deformities are more common in agricultural habitats or wetlands with nutrient enrichment, which can be beneficial for the snail host.

CLINICAL SIGNS

Deformities in frogs associated with *Ribeiroia* infection almost always involve the limbs. It is important to recognize that many factors, both natural and anthropogenic, can cause abnormal limb development. Most amphibian populations exhibit a low frequency of abnormalities, typically around 3% or less. For populations infected by *Ribeiroia*, the frequency of limb malformations in recently metamorphosed frogs can often exceed 10%, in rare cases even exceeding 50% of examined individuals. The types of limb deformities tend to vary somewhat among amphibian species, but some of the most commonly observed are: extra limbs or digits, twisted or misshapen limbs, and abnormal skin webbings. While deformities are much more common in the hind limbs, some amphibian species (e.g., certain toads, bullfrogs) often exhibit abnormalities in the front legs.

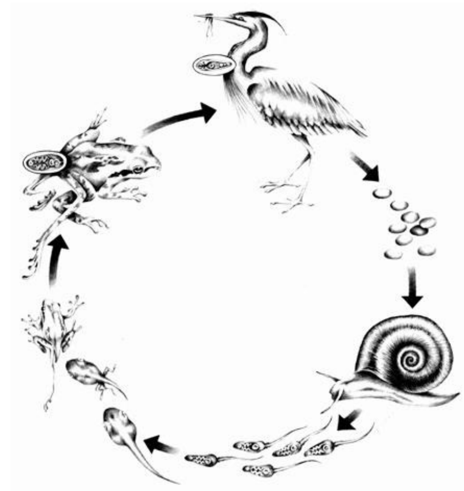
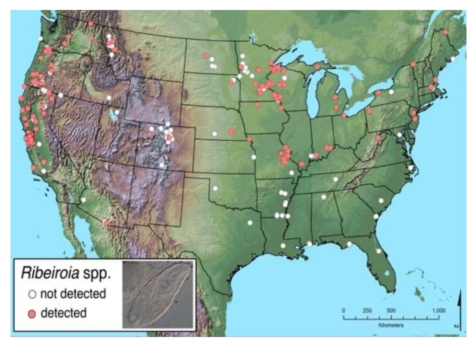


Image Source: Johnson et al. 2004. Life cycle of the trematode Ribeiroia ondatrae. Rams horn snails (genus Helisoma) release free-swimming infectious stages (cercariae) that burrow into the limb tissue of developing tadpoles. Encysted parasites (metacercariae) mature into adult worms only after an infected amphibian is eaten by a predatory water bird (such as a heron). Sexual reproduction occurs in the bird, resulting in eggs that are released along with the bird's feces into other ponds and lakes. Hatching parasite eggs infect and cause castration in susceptible snails.



Cartography by Sarah Haas. Map showing geographic distribution of locations where the trematode Ribeiroia ondatrae has been detected in the United States.

continued ▸

CITIZEN SCIENCE

Members of the public – and especially children – have played an essential in detecting and reporting observations of deformed frogs. If you observe a deformed animal, please take a digital photo and record relevant information about the location and number of animals affected. Data can be uploaded to Malformation Nation (deformedfrogs.org) or to the Johnson Laboratory at the University of Colorado (johnsonlaboratory.com). Confirming the presence of Ribeiroia generally requires dissection of the amphibian to detect encysted metacercaria that most often occur around the hind limbs, although at times, they may be visible through the skin with a dissecting microscope. Frogs can be infected by many different larval trematodes, so the identification of *Ribeiroia* specifically requires morphological or molecular diagnostics. In addition, it is useful to note of the presence of aquatic snails in the habitat, with a focus on rams horn (planorbid) snails. Careful recording of the frequency and types of deformities (digital imaging) in an amphibian population can be extremely informative.

MANAGEMENT

The long-term consequences of *Ribeiroia* infection and parasite-induced deformities in amphibians are unknown. There is evidence that, in areas where infection is high and persistent, *Ribeiroia* has the potential to cause population-level consequences for some amphibian species. This may be of particular concern when the affected amphibian species are threatened or endangered. To date, there is no evidence that *Ribeiroia* is a non-native parasite, and too little research has been conducted to know whether infection is increasing in load or geographically. Multiple anthropogenic factors have been shown to amplify or exacerbate parasite-induced deformities, including nutrient runoff that can which can promote snail densities, pesticide exposure which may inhibit amphibian immune function, the introduction of non-native species, and native biodiversity losses, that can lead to increases in the successful transmission of parasites. Little is known as well on ways to limit or control infection from a management perspective. The parasite's use of birds allows it to be widely dispersed. Some have advocated reducing snail populations in select ponds, although their high reproductive rates and compensatory growth make this challenging.

SUGGESTED READING

Lunde, K. B., et al. (2012). A practical guide for the study of amphibian malformations and their causes. *Journal of Herpetology* 46: 429-441.

Haas, S. E., et al. (2017). Continental-extent patterns in amphibian malformations linked to parasites, chemical contaminants and their interactions. *Global Change Biology* 24: e275-e288.

Johnson, P. T. J., et al. (2010). A decade of deformities: advances in our understanding of amphibian malformations and their implications. Chapter 16 (pp. 511-536) In D. W. Sparling, G. Linder, C. A. Bishop, and S. K. Krest (eds), *Ecotoxicology of Amphibians and Reptiles*, 2nd edition. SETAC Press, Pensacola FL.

Johnson, P. T. J. et al. (2008). Effects of environmental change on helminth infections in amphibians: exploring the emergence of Ribeiroia and Echinostoma infections in North America. Chapter 11 (pp. 249-280) in Fried, B. and R. Toledo, *The Biology of Echinostomes, From the Molecule to the Community*. Springer. 105(44): 17034-17093.