After slogging through 101 ponds and wetlands in five western states, scientists on the trail of a mysterious outbreak of deformities in frogs have settled on a microscopic parasitic flatworm as the prime suspect.

Linked with existing laboratory studies showing that the trematode known as Ribeiroia ondatrae can cause the frogs to sprout extra legs, the new field work closes the loop by showing a direct correlation between the prevalence of the parasite and the number of deformed frogs, scientists said.

"There's still work to do, but this nails it," said Stan Sessions, associate professor of biology at Hartwick College in Oneonta, N.Y., who did not take part in the study, but has been working on the mystery since 1990.

The study was published in the May issue of Ecological Monographs, the journal of the Ecological Society of America.

Since the late 1980s, scientists have been concerned about catastrophic declines in populations of frogs, toads, salamanders and other amphibian species, particularly in Australia, South America, Central America and high-altitude regions of the American West.

In the 1990s, reports started popping up around the United States of large numbers of deformed frogs. Scientists suspected pesticides, ultraviolet light and trematodes as causes.
The reason the deformities are becoming more common appears to be a chain reaction related to human changes to ecosystems, especially fertilizer and cow manure washing into the ponds, said Andrew Blaustein, professor of zoology at Oregon State University and one of the study’s authors.

"We think the fertilizer is causing more algae," Blaustein said. "More algae means more snails to eat the algae. More snails means more parasites (because they live in the snails). More parasites means more malformed frogs."

The trematodes go through three different hosts in their life cycle. When their eggs hatch, the larvae infest aquatic snails. As they mature, they move on to frogs, where they burrow in, forming cysts at the bud where legs sprout when a frog transforms from a tadpole. When the frog is eaten by a bird, the trematodes lay their eggs, which are turned loose in the bird's droppings.

"What we think the trematode is doing is increasing its chances for survival by attacking tadpoles and destroying their hind limbs, so they are easier to be eaten by a primary host, which is a bird," said Sessions.

Pieter Johnson, a graduate student at the University of Wisconsin and senior author of the study, said more surveys need to be done in the Midwest and East, where frog deformities are also prevalent, to conclusively put the blame on trematodes.

More research is also needed to reach hard conclusions about fertilizer as the trigger for the chain reaction leading to the malformations, Johnson added.

But the work already done showed 80 percent of the ponds with more than 5 percent malformations in one or more species of amphibians were built to water cattle, he said.

Deformities were found in 12 different species of amphibians, and at one pond near Corvallis, 80 percent of the Pacific tree frogs were deformed, though the deformities did not seem to cause an overall reduction in the local frog population, Blaustein said.

The researchers tested water samples for pesticides, but found no statistical link to the prevalence of malformations. They did not directly test for ultraviolet light levels, other than to note the elevations of the sites. The Environmental Protection Agency has shown UV radiation can induce deformities in laboratory tests.

"Chemical companies might be breathing a sigh of relief, but this does not necessarily put pollution off the hook," Sessions said. "We still have to explain why this is happening now."

Besides fertilizer runoff, Sessions said there could be two other explanations. Habitat improvements could be resulting in more birds, such as great blue herons, which serve as the final hosts for the trematodes, giving a boost to trematode
populations. And some sort of pollution or ultraviolet light could be diminishing the immune systems of frogs, so that the trematodes have an easier time infesting them.

The quest for an explanation will not only benefit frogs, said Joseph Kiesecker, a disease ecologist at Pennsylvania State University.

Funded by a $2.1 million grant from the National Institutes of Health, Kiesecker and David Skelly of Yale University are cutting down trees and tossing fertilizer in ponds to see how urbanization affects disease dynamics in wildlife, particularly diseases related to trematodes.

"There seems to be some sort of general link between environmental stress that ultimately can be traced back to human-induced problems and the incidence of disease," Kiesecker said.

Diseases such as hanta virus, West Nile virus, Lyme disease, and perhaps even AIDS have moved from wildlife to humans, Kiesecker noted.

"If we are changing the environment in ways that influence disease dynamics in wildlife populations like amphibians, are we doing different things that influence the disease dynamics of human populations?" Kiesecker asked.

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