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# Rachel Carson Syndrome: Jumping to Pesticide Conclusions in the Global Frog Crisis

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"Actually, when I first heard about the Minnesota [deformed frog] situation I immediately suspected a chemical substance. That's the first thing everybody thinks of. You see a screwed-up animal in the field and that's the conclusion to jump to."

*Dr. Stanley Sessions, Hartwick College*[i]

## Introduction

For years some ecologists have been convinced that pesticides are contributing to a decline in amphibian species around the globe. From an apparent epidemic of deformed frogs in Minnesota to the near disappearance of yellow-legged frogs from California's Sierra Nevada Mountains, pesticides have been considered prime suspects.

This suspicion is no doubt rooted in the belief by the ecological community that the agricultural pesticide DDT was responsible for a serious decline of raptor bird populations in North America after WWII—a theory popularized by Rachel Carson's 1962 bestselling book, *Silent Spring*.

Over the past decade, tens of millions of dollars have been spent looking for the offending chemical or chemicals involved in the amphibian declines. Yet time after time, no evidence has been found that pesticides are involved. Four high-profile case studies show how scant the evidence against pesticides is, as well as the deep bias of the ecological research community.

Mountain yellow-legged frogs began disappearing from lakes high in the Sierra Nevada decades ago. Several groups of researchers have suspected agricultural pesticides from California's Central Valley farmland as causing the decline and conducted research on pesticide levels in frogs and the environment. Yet the culprit turned out to be the deliberate stocking of these lakes with non-native trout, starting in earnest 30 years ago. Mountain yellow-legged frog tadpoles simply get eaten by the trout. When the trout are removed from a lake, the yellow-legged frog populations rebound. Yet some researchers continue to claim that pesticides are damaging amphibian populations. Environmental activist groups have filed lawsuits in state and federal courts for inadequate testing and regulation of agricultural pesticides.

In Minnesota, the finding of a bunch of deformed frogs in a farm pond in the mid-1990s eventually led to a frantic search by the Minnesota Pollution Control Agency for the guilty agricultural pesticide or chemical. Preliminary testing even led the MPCA to distribute bottled water to some families because

of presumed farm chemical contamination. Yet after almost a decade of research by government and university researchers, the culprit was finally shown to be a natural frog parasite—possibly made more prevalent by a larger snail population.

In Missouri, concern over the recent and rapid decline in the number of Ozark hellbender salamanders has led to a flurry of research into the possible role of an agricultural pollutant or other so-called endocrine disruptor. Yet extensive dams built in the region have massively altered and destroyed the Ozark hellbenders habitat. These impacts are magnified by the fact that Ozarks hellbenders live for decades and don't reproduce until they are 5–7 years old. Moreover, the Ozark hellbenders have no instinctual fear of predatory trout. Trout were never historically in the Ozark hellbender's range, but have now been purposefully introduced throughout. There is no evidence that pesticides are involved other than the simple observation that pesticide traces have been found in Ozark waters.

Finally, a California herpetologist claims to have found evidence that a widely used agricultural herbicide causes abnormal sex organ development in northern leopard frogs and damages amphibian populations. Other scientists have been completely unable to replicate his laboratory experiments, however, and evidence from his field studies is contradictory and demonstrates no impacts of the herbicide on frog populations.

In short, there is no evidence that any agricultural pesticides have or are contributing to amphibian population declines or frog deformities in the U.S. The question is: When will ecologists begin following the evidence, rather than trying to get the evidence to follow them?

Or will they continue to relive the environmental debates of the 1960s, when DDT was blamed—without evidence—for the decline of raptor bird species in North America?

We've come up with a name for this knee-jerk, immediately-blame-a-pesticide attitude among some ecologists and many environmental activists: Rachel Carson Syndrome.

#### DDT Reality

Rachel Carson, of course, was the author of *Silent Spring*, the book that launched the modern environmental activist movement. In her 1962 book, Carson blamed the pesticide DDT for long-term declines in some predatory bird populations—which she foretold would eventually lead to a future silent spring without any birdsong. Since the publication of her book, no amount of contrary scientific evidence has been able to shake DDT's bird-killing image. The cost in human lives has been enormous.

The global Carson-inspired anti-DDT attitude led African nations to stop using the cheap, effective, and long-lasting insecticide to combat malaria. This occurred even though spraying small amounts of DDT on the inside walls of huts for malaria control poses no ecological risks. (DDT is unique in that it prevents the spread of malaria more by repelling mosquitoes than by killing them.[ii])

As a result, malaria continues to kill an estimated one to three million people each year, mostly young children in Africa. A further 300 to 400 million people have been left chronically debilitated because those who survive malaria are afflicted for life. [iii]

The main claim against DDT was that widespread use by farmers caused the near extinction of some birds, particularly bald eagles, peregrine falcons, and brown pelicans. DDT, we were told, thinned the birds' eggshells and drastically lowered breeding success. This led the populations of these birds to plummet.

But consider the following facts compiled by Dr. J. Gordon Edwards, an entomologist at San Jose State University in California who testified at the 1971–1972 Environmental Protection Agency hearings on DDT.

DDT wasn't manufactured anywhere in the world before 1943 and wasn't used in North America until 1945. Yet bald eagles were threatened with extinction in the lower 48 U.S. states as early as the 1920s.[iv] There were no bald eagles in New England by 1937.[v] Same for peregrine falcons, with only 170 breeding pairs estimated to exist in the eastern U.S. in 1940.[vi]

Even after 15 years of heavy agricultural use of DDT in the U.S., Audubon Society ornithologists counted 25 percent more eagles per observer in 1960 than during the pre-DDT 1941 Audubon census.[vii]

In Canada, peregrines reproduced successfully came into wide use there. Canadian peregrines also contained 30 times more DDT than the Midwestern U.S. peregrines allegedly wiped out by the chemical.[viii] The peregrine falcon found with the highest DDT residues ever recorded (2,435 parts per million) was found feeding three healthy young.[ix]

The populations of brown pelicans also had been devastated prior to DDT's introduction. Texas saw its brown pelican population drop from a high of 5,000 in 1918 to only 200 in 1941, three years prior to the first use of DDT in North America.[x]

Why were these specific bird populations so low

before the invention and manufacture of DDT? We deliberately killed them, trapped them, and stole their eggs. Hunters, farmers, landowners, and fishermen regularly shot eagles, peregrines, and pelicans. Raptor birds and pelicans preyed on farm animals, game animals, and fish that we humans wanted for ourselves. Peregrines also were captured in significant numbers by falconers, who regularly took eggs and fledglings from nests and captured adults in traps.

In 1913, Dr. William Hornaday of the New York Zoological Society referred to peregrines as birds that "deserve death, but are so rare that we need not take them into account."[xi] This statement conveys both the anti-predator attitude of even zoologists at the time, as well as the impact of this societal attitude on predator populations.

State and federal governments, in fact, encouraged such behavior. Starting in the 19th century, bounties were paid by federal and state governments for dead eagles, falcons, coyote, wolves, and other predators. Between 1917 and 1942, the state of Alaska alone paid over $100,000 in bald eagle bounties.[xii] This is the equivalent of roughly $1 million in 2002 dollars. The bounties paid by other states over this same time period were far lower because eagle populations in these states had already been devastated. Even as late as the mid-1970s, illegal shooting continued to be the leading direct cause of bald eagle deaths in the United States.[xiii]

The administrative law judge who conducted the U.S. Environmental Protection Agency's DDT hearings recommended in 1972 that it not be banned because studies failed to show that DDT (or its break down products DDE and DDD) thinned eggshells, even at levels many times higher than actually found in nature. He was overruled by the EPA's Administrator, a political appointee, who banned DDT apparently on the basis of overwhelming negative public opinion resulting from Rachel Carson's book and heavy lobbying by environmental activist groups. This was the Rachel Carson Syndrome in action for the first time, but certainly not the last

#### Global Frog Crisis: Silent Spring Déjà vu

In the tradition of blaming pesticides for ecological problems, despite the scientific evidence or lack thereof, the following case studies on amphibians offer a window into the prejudices and preconceived notions that taint and hinder the scientific process. They waste scarce research funds and too often lead to flawed public policy.

The decline of frog species in some areas of the globe has been noted by amphibian researchers since the late 1980s. Scientists and ecologists have been at a loss to adequately explain many of these population declines. Many conservation groups have therefore declared a global amphibian crisis—and pesticides have been a top suspect.

#### Rachel Carson Syndrome Case 1: The Disappearance of Yellow-legged Frogs

In the High Sierras of the U.S. west, the populations of mountain yellow-legged frogs (*Rana muscosa*) have been in decline for several decades. Many high mountain lakes in the Sierra Nevada formerly brimming with yellow-legged frogs have seen them disappear almost completely. The question has always been why? Several theories have been presented, including increased UV radiation caused by the "ozone hole," frog parasites, fungal disease, and, of course, pesticides.

In the mid and late 1990s, some ecologists began exploring the possibility that pesticides from the intensive fruit, vegetable, and nut tree farms in California's San Joaquin Valley to the west of the Sierra Nevada Mountains were being blown up into the mountains where they could damage frogs' nervous systems and lead to frog population declines.

Why did they suspect pesticides? They don't say, other than that they "may play a very important role in population declines of amphibians in this area."[xiv] Mind you, these frog population crashes were in high-altitude alpine lakes, not farmlands.

To test their theory, a team of researchers with the U.S. Geological Survey led by Dr. Donald Sparling looked for pesticide traces in the tissues of Pacific tree frogs (*Hyla regilla*) thriving in the same areas where the populations of the yellow-legged frogs had so drastically declined— the high-mountain lakes in Yosemite National Park, Sequoia National Park, and the Lake Tahoe Basin. They compared the mountain tree frogs' pesticide levels to those of tree frogs from two coastal sites and a northern mountain area isolated from farmlands.

It is ironic that these ecologists would approach the yellow-legged frog declines by looking for traces of pesticides in tree frogs that were abundant in the same environments where pesticides were suspected to be ravaging yellow-legged frogs.

They found pesticide traces just slightly more often and at slightly higher levels in tree frogs from the Sierras east of the Central Valley than frogs from California's coast and northern mountains. However, the pesticide traces were so tiny at all

sites, including the Sierra Nevadas, they were mostly too low to measure—i.e. below 1 to 2 parts per billion (equal to one second in 32 years). Thus, the pesticide residue differences were insignificant.[xv]

As they note in their research report, published in the journal *Environmental Toxicology and Chemistry*, "because of the frequency of samples below analytical detection limits at all study sites, no significant differences were found in [pesticide] residues of either age [tadpoles or adults] or across sites, even after substituting 1/2 detection limits for values below detection limits."[xvi]

They also found that activity of a nervous system enzyme was "depressed" in tree frog tadpoles from the mountains when compared to tree frog tadpoles from the coast. However, no differences were seen in adult tree frogs. Yet the abundant tree frog populations at all locations indicate that the slightly "depressed" tadpole enzyme activity was not physiologically or reproductively important.

Based on the statistically insignificant differences in pesticide traces and the slightly reduced enzyme activity in tree frog tadpoles from the mountains, the researchers made the sweeping assertion that "pesticides are instrumental in declines of [red and yellow-legged frog] species."[xvii]

Dr. Sparling, then at the U.S. Geological Survey's Patuxent Wildlife Research Center, told the *Los Angeles Times* in 2000, "Unfortunately, there now appears to be a close correlation between declining populations of amphibians in the Sierra Nevada and exposure to agricultural pesticides."

Californians for Alternatives to Toxics, The Center for Biological Diversity, and other anti-pesticide groups seized on the baseless assertions of Sparling and his group. These activists have filed suit against the California Department of Pesticide Regulation and the federal EPA for failing to review the impacts of pesticides on California frogs and other endangered amphibians. Collectively, these lawsuits have already cost hundreds of thousands of dollars and are far from over.

However, at the same time that Sparling and his colleagues were chasing chemical phantoms, other researchers were following up on a far more plausible explanation: predatory non-native trout were eating frog tadpoles.

Starting more than a century ago, anglers began stocking western streams, rivers, and lakes with brook trout, rainbow trout, golden trout, and other non-native fish. Even as far back as the 1880s, miners used mules to haul trout fingerlings in milk cans to lakes in the Sierras. There are hundreds of glacial lakes in the Sierras suitable for trout.

The effects on frogs from the trout stocking didn't take long. As early as 1924, wildlife biologists had noted that mountain yellow-legged frog tadpoles and trout were rarely seen in the same lakes and strongly suspected trout predation.[xviii] Biologists had observed and reported trout preying on mountain yellow-legged frogs in 1938.[xix]

Nevertheless, in the 1960s, the fish stocking went large-scale and Sierra-wide. Fingerlings were dropped by the thousands via aircraft into nearly every available lake, including dozens of previously inaccessible lakes at the top of the watersheds. Thus, lakes that had never previously had fish were suddenly teeming with tadpole predators.

By the mid-1990s, some researchers were logically looking at the introduced trout as the main cause of the significant decline in mountain yellow-legged frogs in the Sierras. Dr. Martin Knapp with the University of California and Kathleen Matthews with the U.S. Forest Service noted that the mountain yellow-legged frogs spend two to four years in the tadpole stage, compared to most frogs that spend only one season as tadpoles. This confines yellow-legged frogs to the same habitats that support the introduced trout—larger lakes that don't dry out or freeze solid in winter. In contrast, tree frog tadpoles become frogs in only one season, and, therefore, can thrive in smaller, temporary pools that harbor no fish.

In the late 1990s, Knapp and Matthews examined more than 1,000 lakes in Kings Canyon National Park in the Sierras, where fish stocking was phased out between 1977 and 1991, and compared them to 1,200 lakes in the adjacent John Muir Wilderness where fish stocking has continued. Because these two areas border each other, levels of airborne pollutants, UV light, and other environmental factors are essentially the same—including levels of pesticides drifting up from the San Joaquin Valley.

They found that lakes with non-native trout generally have no yellow-legged frogs, whereas lakes with frogs generally have no fish. They also found that yellow-legged frogs existed in relative abundance in the national park where fish stocking was eliminated, while they remained virtually absent from the wilderness area where fish stocking continued.[xx]

Taking this research another step further, Dr. Vance Vredenburg at the University of California, Berkeley, spent eight years in Kings Canyon National Park running carefully controlled experi-

ments confirming that fish were the cause of Sierra Nevada frog declines.

In research published in May 2004, Vredenburg explained how he used gill nets to remove all the fish from several lakes where frogs had disappeared. The frogs then experienced a three-year "population explosion," in Vredenburg's own words. In lakes that were never stocked and large frog populations remained, Vredenburg and his team moved a few trout into small, fenced-off areas to see what impacts the newly introduced fish would have. The fish quickly consumed all the frog tadpoles they could reach.[xxi]

As Dr. Vredenburg told the *San Francisco Chronicle* in an article published on May 13, 2004, "There are at least 10,000 lakes in the High Sierra. Ninety percent to 95 percent of them hold introduced species of trout but no more frogs at all. And there may be 200 lakes that have plenty of frogs, but few or no fish. So the answer is pretty straightforward, and it doesn't get much simpler: with no trout you get an immediate and dramatic response—the threatened frogs return, and some of the High Sierra's natural biodiversity returns, too."

Unfortunately, none of the research demonstrating the overwhelming impact of trout has changed the anti-pesticide mantra of some researchers and environmental groups.

In December 2004, only six months after the publication of Dr. Vredenburg's research, ecologist Carlos Davidson at California State University, Sacramento, published yet another paper in an ecology journal claiming "a strong association between upwind pesticide-use and amphibian declines" and advocating, surprise, yet more research.[xxii]

When I asked Dr. Sparling, one of the leading anti-pesticide researchers, about the implications of Vredenburg's research on his published statements that "pesticides are instrumental in declines of [red-legged and yellow-legged frog] species," he said he didn't see any conflict between the two. "They could both contribute to frog population declines," he said.

Yet this view overlooks the fact that the lakes where frog populations exploded following the removal of the trout still had the same tiny traces of pesticides that Sparling, Davidson, and their colleagues claim are causing frog declines. The only change was the presence of predatory fish.

Dr. Sparling even cited to me the activist lawsuits against the EPA and California Department of Pesticide Regulation as further support for his theory that pesticides cause frog population declines, ignoring the fact that his groups' claims spurred the lawsuits in the first place.

In the May 13th *San Francisco Chronicle* article reporting on Vredenburg's research, reporter David Perlman incorrectly noted that "pesticides and herbicides drifting into the mountains from Central Valley farmlands *are a known cause of declining frog populations*." [emphasis added] When I asked Perlman where he got the false impression that pesticides are a "known cause" of frog declines, he cited Dr. Sparlings' studies among others.

This past October several wildlife conservation groups released the Global Amphibian Assessment (GAA). The brand new report details the state of known amphibian populations, highlighting threatened species and the suspected or known causes of their declines. The GAA lists the mountain yellow-legged frog of the Sierra Nevada as in "enigmatic decline," meaning that ecologists don't yet know the cause.

Ironically, Dr. Vredenburg was one of the amphibian experts relied upon for the GAA report. When I asked him why the GAA lists the yellow-legged frog's decline as "enigmatic" when his own research has so convincingly demonstrated the overwhelming cause to be non-native trout, he said he didn't know. He referred me to the head of the GAA project, Dr. Stuart Simon.

Dr. Simon's reply was itself enigmatic. He said that "introduced trout are the pre-eminent historic cause of the decline of [mountain yellow-legged frogs]. However, current declines are more enigmatic . . ."

Current declines? There has really only been one long decline over the past 50 years, not several declines, and the evidence indicates clearly that non-native trout are the driving factor in the overall population decline.

When I asked Dr. Vredenburg and Dr. Stuart for evidence indicating other factors besides introduced trout, both cited evidence for a possible role of a disease-causing fungus in some isolated yellow-legged frog populations. Neither said pesticides.

Yet the GAA's "enigmatic decline" designation leaves the door open to a possible role for pesticides. Therefore, it is likely that researchers who have publicly implicated pesticides in the yellow-legged frog's decline will use the ambiguous GAA designation to stump for more research funding to search for a pesticide cause. And activist lawsuits against government pesticide agencies are sure to continue given the ambiguity of the GAA designation.

The policy morass, in essence, continues. Meanwhile, the public is distracted from the one action that could truly help the frogs—removing non-native trout from Sierra Nevada lakes.

### Rachel Carson Syndrome Case 2: Minnesota Deformed Frogs

In 1995, a group of Minnesota middle school kids found some malformed norther leopard frogs (*Rana pipiens*) in a farm pond and posted pictures of them on the internet. Their teacher, Cindy Reinitz, also reported the seemingly high rate of deformities to the Minnesota Pollution Control Agency. She said her students asked her about the cancer rate in the area. If so many frogs were deformed, would the water cause cancer in the families living nearby?

The pictures of the malformed frogs set off an environmental firestorm. Others in Minnesota began reporting finding deformed frogs. Not long after these initial reports, the Minnesota Pollution Control Agency went into overdrive to find "the cause." Based on preliminary research, MPCA scientists indicated that the culprit was likely a chemical pollutant of the water, such as farm pesticide runoff.

As *Washington Post* reporter William Souder wrote in his book, *A Plague of Frogs*:

> The MPCA . . . was working on the assumption that an environmental insult of some kind was causing the deformities. [The MPCA's Dr. Judy Helgen] could not believe this was any sort of natural event; . . . Helgen said she strongly suspected farm chemicals were involved. Because of the constant introduction of new pesticide products into the market . . . it seemed reasonable to infer that some recently introduced active ingredient was involved.[xxiii]

Testing of the water from several ponds with high numbers of northern leopard frog deformities began in 1997. The MPCA worked with the National Institutes of Environmental Health Sciences (NIEHS) to test the water using a FETAX test, which stands for Frog Embryo Teratogenesis Assay *Xenopus*. The test uses the African clawed frog, *Xenopus laevis*, which is a popular aquarium pet and is commonly used in amphibian research because of their rapid development.

Basically, *Xenopus* eggs are collected just after fertilization, stripped of their protective jelly, and then placed in the water or solution being tested. The embryos are allowed to develop for four days, by which time they will have hatched. Then they are killed and examined under a microscope for malformations and abnormal development.

In September 1997, after two months of initial testing, the MPCA held a press conference to announce that the FETAX results showed there was a problem with the water. As an indication of the fevered level of interest, that event was covered by ABC News "Nightline."

"We're here because we've found something in the water," began the MCPA commissioner. Then an NIEHS scientist announced ominously, "We found that water from sites where malformed frogs have been reported was very potent in deforming frogs in this laboratory experiment."[xxiv]

Not only did water from the ponds cause malformations in the FETAX test, but in a worrying development, so did well water from the homes next to the ponds. In an interview on PBS television's "Newshour," Dr. Helgen said, ". . . is the [contaminated water] going from the pond into the groundwater, or is it going from groundwater into the pond? You know, is this just a very local thing? We're beginning to think now that we have to look at the hydrology of the sites in a very important way."

Indeed, the MPCA began giving bottled water to some families soon after they announced the FETAX test results.

Yet by late October of the same year, scientists with the U.S. EPA demonstrated that it was simply a natural lack of calcium and other salts in Minnesota water that was deforming the lab-raised African clawed frog embryos, not a chemical contaminant.

The EPA scientists first ran the FETAX test using water from one of the same affected ponds that the MPCA researchers said caused frog deformities. They saw exactly the same frog deformities as the MPCA/NIEHS team. Then they added calcium, magnesium, sodium, and potassium to the water in amounts recommended for rearing African clawed frogs and they developed normally.[xxv]

In other words, the Minnesota water simply didn't have enough salts in it for normal development of the African clawed frog, a species particularly sensitive to salt deficits.

As lead EPA researcher Joe Tietge said, "You could probably take tap water from almost any county in Minnesota and get results like this. In science, spurious correlations happen all the time," and they are "one of the weakest forms of evidence to support a hypothesis."[xxvi]

As another EPA researcher said, "Results don't mean anything if they aren't interpreted properly. Anybody with a tropical fish aquarium knows that if you fill it with tap water it will kill the fish. That

doesn't mean your tap water isn't safe to drink."[xxvii]

But two years later, the MCPA and NIEHS researchers published their flawed FETAX research anyway despite the EPA's findings—in a scientific journal sponsored by the NIEHS—and continued to claim that a chemical in the water was responsible for deformities of northern leopard frogs in Minnesota ponds. As the MCPA's Judy Helgen stated, "We're still open to other hypotheses, but it certainly looks from these papers like chemicals or some combinations that include chemicals are responsible for different types of malformations."

Six years after these statements, the MPCA has still not identified a chemical or chemicals responsible for the frog malformations. In fact, there is still not a shred of evidence that chemical contaminants, such as pesticide traces, had anything to do with malformations seen in northern leopard frogs in Minnesota or anywhere else.

Instead, the evidence for a natural cause of the frog malformations has become overwhelming. Northern leopard frogs in the wild are afflicted at an early age by tiny parasitic flatworms called trematodes. These parasites are shed by snails into pond water where they swim to the developing frog embryos and burrow into the tissues where the hind limbs are formed. As they burrow, they dislocate cells from their normal locations. These cellular dislocations in the rapidly developing tadpole surprisingly result in extra or missing limbs, feet, and all of the other malformations seen in the Minnesota ponds. In fact, many biologists believe that these limb malformations increase the chances that a parasite-infected frog will be eaten by birds, allowing the trematode to pass into its next alternate host in the parasite cycle, aquatic birds. That completes the full parasite lifecycle from birds, to snails, to frogs, and back to birds.

When the parasite explanation was voiced early in the controversy by Dr. Stanley Sessions at Hartwick College in New York, it was received with tremendous skepticism by most other researchers. There was outright derision from more than a few. They argued that parasites simply couldn't cause the range and extent of deformities seen. They argued that parasites were rarely detected in deformed frogs.[xxviii] They argued all kinds of reasons why parasites were an unlikely cause and why a contaminant was the most likely.

But we now know that frog deformities simply don't occur without exposure to the parasites.[xxix] We know that trematodes can be, and often are, eliminated from a frog's body after they have caused the deformities—leaving few signs that they were ever there.[xxx] We now know that trematodes are present across North America and have impacted frogs for at least 300 years, if not thousands.

We also now know that the extent and frequency of the frog deformities seen at the so-called "affected ponds" is not all that unusual. Surveys of museum frog specimens collected 100 years ago show similar rates and kinds of deformities.[xxxi]

We do know that deformity rates vary considerably from pond to pond and from year to year, depending on the factors that affect parasite abundance and the timing of their release from snails. When trematodes are shed by the snails at a time that coincides with frog tadpole metamorphosis, you get high numbers of deformed frogs. If not, you don't. Thus, we should expect occasionally high levels of malformations in some ponds.

If, indeed, frog malformations have increased in recent years, some research suggests that agricultural activities that increase *nutrient* levels in ponds may increase the relative abundance of the larger snail species that are the intermediate hosts of the parasite. Thus, more intensive agriculture could increase parasite loads and frog malformation levels.[xxxii]

Regardless, the parasite explanation has become so well accepted that now some "pesticide theory" researchers are examining ways in which pesticides could affect the abundance and impact of the parasites. In other words, they've simply switched their Rachel Carson Syndrome from direct pesticide impacts on frogs to indirect pesticide impacts on frogs. This despite the complete lack of evidence indicating pesticides have anything to do with the Minnesota frog deformities.

Why? According to Dr. Sessions, the motivation may be research funding. As explained by *Washington Post* reporter William Souder, "[Sessions] thought it was possible that the whole frog investigation was being manipulated—and important evidence ignored—so as to promote further research funding. In essence, he seemed to think other investigators were tilting their hypotheses toward a chemical contaminant in an effort to catch the endocrine disruption wave."[xxxiii]

#### Case 3: Missing Missouri Hellbenders

Few Americans realize that the U.S. is home to one of the world's largest and oldest salamander species, the hellbender (*Cryptobranchus alleganiensis bishopi)*. Hellbenders grow up to two feet long and can live for 30 years or more in the wild. They live in cold, fast flowing mountain streams in the

eastern U.S. and feed at night on crayfish, insects, and other critters. There are two subspecies of hellbenders. The eastern hellbender subspecies lives from southern New York down through northern Georgia and west through Tennessee and the Ohio River valley. The Ozark hellbender subspecies lives only in the Ozark region of southern Missouri and northern Arkansas.

While eastern hellbender populations have been hurt by dams, logging, and human settlement throughout their range, in recent decades their numbers have remained relatively stable in most places. Overall, eastern hellbenders are not considered endangered or in serious decline.

Ozark hellbender populations, however, have declined significantly in over the past 15 years. A 1991 survey conducted by the Missouri Department of Conservation seemed to indicate abundance, with 150 Ozark hellbenders found during a two-day search. Yet a 1998 survey found a nearly 80 percent decline compared to the 1991 survey. Worse, there were almost no young hellbenders found during the 1998 survey, indicating that Ozark hellbenders aren't reproducing successfully or their young are dying very early.

Biologist Ron Goellner, curator of the St. Louis Zoo, says the first signs of trouble for hellbenders appeared in the late 1980s and early 1990s, when hellbender numbers in the Spring River in northern Arkansas began to decline. The alarm was quickly raised. By March of 2003, the Missouri Department of Conservation declared the Ozark hellbender an "endangered species" in the state. Scientists created the Hellbender Workshop Group to share and discuss theories as to why Ozark hellbender numbers have dropped so quickly.

There are several theories but the one currently getting the most attention and resources is "endocrine disruption" by a chemical pollutant, such as an agricultural pesticide. One reason a chemical pollutant is suspected is that the sperm counts of Ozark hellbenders appear to be about 20 percent lower than the sperm counts of hellbenders in areas where their populations are stable, such as Georgia and the Carolinas.

Another reason to suspect a chemical pollutant is location. Ozark hellbenders live in closer proximity to cattle, poultry, hog, and crop farms than the Georgia and Carolina hellbenders. As University of Missouri professor Dr. Yue-wern Huang and graduate students write, "Streams where [Ozark hellbenders] inhabit have become polluted with animal manure runoff, human wastes, fertilizers, herbicides, and pesticides. Some of these pollutants have been shown to possess similar activity to 17,-estradiol, a female hormone which plays a major role in secondary sex organ development, behavior, fertility, and reproductive capacity."

So far, his team has identified some chemicals that mimic hormones in Missouri streams. However, they have not correlated the relatively low levels of these pollutants with any effects on Ozark hellbenders. Thus, it is an open question whether endocrine disruption is contributing to the decline of Ozark hellbenders.

There is no question, however, that there are other, major factors causing the Ozark hellbender declines, including massive habitat alterations and the introduction of non-native predatory trout. The petition to list Ozark hellbenders as a Missouri endangered species states:

> Much of the hellbender habitat was destroyed by the series of dams constructed in the 1940s and 1950s on the upper White River, including Beaver, Table Rock, Bull Shoals, and Norfolk dams. **The remaining habitat would be suitable for Ozark hellbenders, except for the extreme fluctuations in water levels that result whenever the generators are being run**. [emphasis added] . . . The precipitous decline of the Ozark hellbender in Missouri and Arkansas is most likely the result of habitat degradation and other environmental changes caused by human activities. Hellbenders are habitat specialists that "achieve an evolutionary stability within their habitat that magnifies the effects of rapid habitat alteration. The hellbender is confined to a narrow niche . . . [that] makes it acutely vulnerable to man's effects" (Williams et al 1981, 95). Therefore, even minor alterations to a stream habitat are likely detrimental to hellbender populations, and unfortunately, major alterations have been made throughout much of its habitat. **As a long living species, it was not readily apparent at first that a decline in hellbender populations was in progress. Thus, the initial event that would have triggered this lack of recruitment must have taken place 20–30 years ago, possibly around the time of the construction of the dams and the rise of recreational activities in the area**.[xxxiv] [emphasis added]

The dams and resulting reservoirs literally drowned a significant portion of the rivers and

streams where Ozark hellbenders live, destroying those habitats completely. These dams also cut off hellbender populations from each other and blocked in and out migration.

Moreover, farming, logging, and housing developments have resulted in millions of tons of sediment entering Ozark rivers and streams. These sediments bury the large rocks that hellbenders like to live under, further degrading the remaining hellbender habitat.

Finally, the Missouri Department of Conservation has been stocking Missouri rivers, streams, and lakes with non-native trout that are potentially devastating to Ozark hellbender young because they have no natural fear of this introduced predator.

Eastern hellbenders in Georgia and the Carolinas co-evolved over the millennia with native North American trout species. Thus, when eastern hellbenders smell trout, including introduced European trout species, they hide or avoid movement that would attract attention. Ozark hellbenders, however, did not co-evolve with trout and have no instinctual fear. They don't hide or freeze in the presence of trout, making them especially vulnerable. Moreover, hellbenders remain in the larval stage for two years and don't reach sexual maturity until they are six to eight years old, which translates into plenty of opportunity for them to become trout food.

The Missouri endangered species petition dryly notes that "There have been recent reports that the stocking of trout in the Lake of the Ozarks region could have played a significant role in the decline of the Ozark hellbender. 'Larval amphibians are extremely vulnerable to vertebrate and invertebrate predators' (Alford 1999, 136). . . . Though no published reports specifically mention this threat, **the increased abundance of game fish in the historical habitat of the Ozark hellbender could be more than a coincidence**." [emphasis added]

This threat has since been deemed to be real enough that ecologist Alicia Mathis with Southwest Missouri State University has a grant from the U.S. Fish and Wildlife Service to try and teach 150 captive Ozark hellbenders to fear trout when they smell them. The hope is that after they're trained to fear trout, they'll have a better chance at survival when they're returned to the wild. But it seems somewhat fruitless if the fear of trout is a learned behavior, rather than a genetic, instinctual one as in the eastern hellbenders. The offspring of the trained hellbenders will still lack this fear and remain vulnerable. (Ecologists have ruled out hybridizing the Ozark hellbenders with eastern hellbenders to try and breed in an instinctual fear of trout because it would dilute the unique genetic make-up of the Ozark hellbender subspecies.)

No one is suggesting removing trout from the Ozarks or busting the dams that so radically altered the hellbender's habitat. Instead, the focus is on finding a man-made chemical pollutant (and pesticides are at the top of the suspect list) that might be cutting sperm counts by 20 percent—or not. So far, all we know is that some of these chemicals are present in Ozark waters.

#### Case 4: Leopard Frogs and Atrazine Accusations

Finally, there is the research of Dr. Tyrone Hayes, a California researcher who is the newest media darling in the supposed "global frog crisis." Over the past four years, Hayes has been profiled by *National Geographic* magazine, *Discover* magazine, National Public Radio, and virtually every major newspaper in the country.

Hayes argues that traces of atrazine, one of the most widely used farm weed killers in North America, are affecting frogs from California to the Carolinas. The media has run with this theory, placing it at the heart of all supposed frog ills. As a 2003 editorial in the *Baltimore Sun* newspaper stated, "Frogs have been trying to tell us something for quite a while now. Each spring there seem to be fewer of them, while increasingly those that do appear are severely deformed; . . . A leading culprit is believed to be the widely used weed killer atrazine."[xxxv]

Yet Hayes doesn't argue that atrazine kills frogs or causes deformities. Instead he says that atrazine feminizes male frogs, chemically castrating them. Therefore, Hayes argues, atrazine "likely has a significant impact on amphibian populations" and should be banned.[xxxvi]

But even Hayes can't explain why after 30 years of extensive atrazine use, frog populations are still thriving in the areas where it is heavily used. Nor can he provide any field evidence that atrazine has harmed a single frog species anywhere.

Hayes says research in his laboratory shows that at 0.1 parts per billion (ppb) atrazine, 36% of males at metamorphosis suffer from under-developed testes. At 25 ppb atrazine, only 12% of males at metamorphosis have under-developed testes.[xxxvii] Hayes says that the greater effects of atrazine at lower concentrations are not unusual for endocrine-disrupting chemicals. Yet even Hayes admits that these frogs were simply delayed in their

sexual development and would continue normal development after metamorphosis.

Hayes also found 29% of male frogs "displayed varying degrees of sex-reversal" at 0.1 ppb atrazine, whereas only 8% of males showed some sex-reversal at 25 ppb atrazine.[xxxviii] However, scientists from four universities have been unable to reproduce Hayes's laboratory results. Dr. Ronald Kendall at Texas Tech University, a Hayes critic, says, "validated information should be replicable."

In response, Hayes accuses the other researchers of outright lying.

> This is a group of individuals whose sole goal is to prove me wrong and to keep atrazine on the market. Their science is so poor, yet they continually try to damage or hurt my findings by saying they can't reproduce my work under the pretense that they're doing real science. I thought only criminals and desperate people lied, not educated people. My 11-year-old looks over their experiments and sees that they have no controls. They can't be that dumb, so they're lying.[xxxix]

These are incredibly strong words for a scientific debate, where research usually is left to speak for itself and to sort out such debates. If experimental results cannot be replicated, their validity is understandably questioned. Instead, Hayes has resorted to ad hominem attacks, using a word that is almost never used in science debates: Liar. And Hayes is leveling that charge against an entire group of researchers from several institutions.

If the laboratory results are in contentious dispute, what, if anything, is happening to frogs out in the real world? Even according to Hayes's field research, not much.

Hayes conducted field studies in Utah, Wyoming, Nebraska, and Iowa—in places where atrazine is used regularly and in places where it is presumably never used. Hayes found traces of atrazine at all but one location, a wildlife preserve in Iowa. One water testing lab found no atrazine in the Iowa preserve's water and another found only trace levels at the limit of detection.

Bottom line: Hayes could show no correlation between atrazine levels and "gonadal abnormalities" in northern leopard frogs at any of the sites. While more than 90 percent of male frogs from one site in Wyoming had some "gonadal abnormalities," three other sites with equivalent atrazine levels had no or low levels of abnormalities. Atrazine levels were the same in sites in Utah and Wyoming, yet there is approximately a 900 percent difference in the incidence of abnormalities between the sites. At one Utah site, no abnormalities were seen despite the presence of the same atrazine levels seen at the high-incidence site in Wyoming. Moreover, the Iowa site where no atrazine was found had the same frequency of abnormalities as four other sites where atrazine levels were both low and high.

In short, none of the field data makes any sense if atrazine really has an impact on male frog sexual development. Most damning of all, Hayes had no trouble finding northern leopard frogs at any of the field sites he studied. Frogs were abundant at all locations. So much for Hayes's claims that atrazine "likely has a significant impact on amphibian populations."

Rather than finding an ecological problem in frogs and then searching for a cause, Hayes seems to have found a laboratory effect from atrazine and is now searching for an ecological problem.

Finally, Dr. Hayes made a startling statement in a recent paper he wrote about the lack of scientific evidence that DDT harmed raptor birds. Dr. Hayes recently wrote that "Years have passed since DDT was banned in the United States, but it is unclear how much policymakers and the public have learned from the case of this dangerous pesticide. DDT was banned on the basis of even less scientific evidence than currently exists for the negative impacts of atrazine." [xl]

Considering the lack of evidence on the "negative impacts of atrazine," this says volumes about the DDT paradigm that is currently driving a sector of the ecological research community and about the ultra-conservative regulatory stance of the EPA.

## Conclusion

Over the past 30 years our knowledge of the presence and persistence of man-made chemicals has become far more detailed and precise. We can now detect most chemicals in the parts per billion range, and some we can detect at the parts per trillion level (one second in 31,000 years!).

With this incredible detection capability, it is all too easy to see chemical boogeymen at every turn. Anywhere we find an ecological problem, we can surely find traces of man-made chemicals to blame and demonize. But finding traces of chemical X or Y at a "problem site" is simply not enough to make the leap to causation. Yet that is exactly what has happened again and again since *Silent Spring* was published in 1962.

In fact, we often lack the ecological knowledge to

even declare when actual problems exist. As ecologist Dr. Kathy Converse of the U.S. Geological Survey said to me, "over the past decade, we've learned an incredible amount about the natural parasites and pathogens that afflict frogs that we never knew about before. That never would have happened without the pesticide scares."[xli] While this may be good for our understanding of frog ecology, how high is the cost of this chemical crisis mentality? Surely there must be a better way to gain an understanding of frogs and their ecology than to needlessly frighten entire communities about the safety of their water and the environment. Not to mention the potential costs to the farming community in lost farm inputs that are important in modern farming systems that prevent soil erosion and reduce greenhouse gas emissions.

This Rachel Carson Syndrome has become an epidemic within the ecological research community, even though the charges made by Ms. Carson have not stood up well to careful scientific scrutiny.

Rachel Carson Syndrome has led to activist lawsuits accusing irrigated farmers in the Pacific Northwest of contributing to salmon declines in the Columbia River. We now know that aside from the direct impacts on salmon from the dams and altered river hydrology, salmon populations have been fluctuating due to a natural 50-year cycle in ocean currents linked to the Pacific Decadal Oscillation.

Rachel Carson Syndrome has needlessly frightened women about breast cancer risks from their drinking water and lawns, and diverted their attention from the real cancer risks of cigarette smoking, genetic predisposition, and late child-bearing.

The list of Rachel Carson Syndrome phantoms seems almost endless, and the victims number in the billions, never mind the direct costs in wasted research budgets and needless regulations. It is currently being used against agricultural biotechnology, even though it is now abundantly clear that this technology will allow us to substantially reduce even the phantom pesticide risks some ecologists have chased for 40 years.

The question is: How long will we allow public policy to be led by innuendo and flawed theories from the past? How long will the ecological research agenda be driven by unsupported scares that distract from real ecological threats? When will ecology become a science again?

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