



Pesticide Use Harming Key Species Ripples through the Ecosystem

Boulder Creek,
Boulder, Colorado

**Regulatory
deficiencies cause
trophic cascades
that threaten
species survival**

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DREW TOHER

Despite a growing body of scientific literature, complex, ecosystem-wide effects of synthetic pesticides are not considered by the U.S. Environmental Protection Agency (EPA). Beyond direct toxicity, pesticides can significantly reduce, change the behavior of, or destroy populations of plants and animals. These effects can ripple up and down food chains, causing what is known as a trophic cascade. A trophic cascade is one easily-understood example of ecosystem-mediated pesticide effects.

In determining legal pesticide use patterns that protect ecosystems (the complex web of organisms in nature) EPA requires a set of tests intended to measure both acute and chronic effects. An ecological risk assessment considers the lethal concentration at which 50% of a population of a given species will die (LC50), and the chronic impacts associated with environmental exposure.

The concept of a trophic cascade is well illustrated by the local extinction and subsequent repopulation of wolves in Yellowstone National Park (YNP). The loss of wolves from YNP in the 1930s due to overhunting led populations of their primary prey, elk, to explode. Without a check on their growth, elk browsed heavily on aspen, cottonwood, and willow. Denuded willow stands stunted populations of beavers that rely on the trees to make it through the winter. When wolves were reintroduced in the mid-1990s, beaver populations began to improve. Elk still had other predators—bears, cougars, coyotes—but only wolves kept elk skittish enough to avoid browsing willow on streambanks. This led to a resurgent willow population, providing new habitat for songbirds. Beaver dams further altered the landscape by reducing runoff and

stabilizing the water table, and both worked in tandem to provide cool, deep, shaded water for native fish.

When a predator higher up on the food chain is eliminated, that predator's prey is released from predation, often causing a trophic cascade that throws the ecosystem out of balance. It is not always the top-level predator that creates a trophic cascade. The loss or reduction of populations at any trophic level—including amphibians, insects, or plants—can result in changes that are difficult to perceive, but nonetheless equally damaging to the stability and long-term health of an ecosystem. Salient research on the disruptive, cascading effects that pesticides have at the ecosystem level must lead regulators to a broader consideration of the indirect impacts caused by the introduction of these chemicals into complex living systems.

PESTICIDE-INDUCED TROPHIC CASCADES LEAD TO DISEASE OUTBREAKS

Pesticides create disruptions in ecosystems because of the interplay between toxicity and indirect impacts. But it is not always possible to observe these effects in the wild, as researchers did in YNP. To study how food webs respond to human-made stressors like pesticides, scientists use mesocosms—small structures containing various plants and animals intended to mimic the natural environment.

In many cases, researchers will use mesocosms to investigate observations in the field, as Rohr et al. (2008) did when studying the impacts of the herbicide atrazine on the health of northern leopard frogs, a once-abundant species in the U.S. that is now in decline. In the field, atrazine was found to be associated with an increase in the number of parasitic flatworms in leopard frog habitat. However, the ecological process behind this phenomenon was murky.

Figure 1: **Yellowstone's Trophic Cascade**



TROPHIC CASCADE: No wolves leads to more elk, which eat more willow, which eliminates food sources for beavers, shaded areas needed for native fish, and habitat for songbirds.



STABLE ECOSYSTEM: More wolves leads to fewer elk browsing streambanks, which increases the willow population, subsequently providing more food for beavers, and habitat for songbirds and native fish.

Flatworms have a complex life history that brings them through several hosts in the food web, with the endpoint of infecting a predator, which will release its eggs into the water when defecating. Aquatic flatworm eggs hatch into free-swimming larvae, and further develop by using snails as an intermediate host. After infecting snails, the larvae can then infect tadpoles. Adult frogs infected with flatworm larvae as tadpoles exhibit limb malformations, kidney damage, and complete exhaustion, making them easy prey for predators. Field observations of higher levels of periphyton (attached algae), a major food source for snails, in atrazine-contaminated waters led Rohr et al. (2008) to hypothesize that atrazine was increasing snail abundance. To verify this hypothesis, researchers created a series of 1,100 liter mesocosm tanks containing, among other flora and fauna, phytoplankton (free floating algae), periphyton, snails, and leopard frog tadpoles. (The mesocosm also contained zooplankton, beetles, water bugs, and dragonfly larvae, however impacts to these species as a result of atrazine exposure were not analyzed by researchers.) Some tanks were dosed with real-world levels of atrazine, while others acted as a control. Snail abundance increased significantly in the atrazine-contaminated tank, over four times compared to an unexposed mesocosm. This was indicative of a trophic cascade, which took the following route: atrazine killed off most free floating phytoplankton algae, leading to increased water clarity and light penetration, which jump-started the production of periphyton, which subsequently increased the population of snails that could carry flatworm parasites. Not only did atrazine increase the parasite load, it also had the direct effect of making frogs more vulnerable to infection through immunosuppression.

The effects of this trophic cascade are not limited to sensitive amphibian species. Humans are also at risk of infection from parasitic flatworm larvae, particularly in intensive agricultural areas in developing countries. Using a mesocosm experiment that analyzed both the aforementioned species as well as snail predators (water bugs, crayfish), Halstead et al. (2017) investigated the effects of environmentally relevant concentrations of both atrazine and the insecticide chlorpyrifos on the transmission of schistosomiasis, a human disease caused by flatworm parasites. Schistosomiasis can result in rashes, itchy skin, fever, chills, cough, headache, belly pain, joint pain, muscle aches, and in severe cases impair organ or nervous systems.

In mesocosms tested, atrazine created a trophic cascade similar to that observed by Rohr et al. (2008), reducing free-floating algae and increasing attached algae, leading to higher snail populations due to increased food availability. When chlorpyrifos, often applied to agricultural fields alongside atrazine, was added to the mesocosm, snail predators declined significantly, releasing snails from predation that would otherwise suppress their population.

Halstead et al. (2017) incorporated these data into epidemiological models to determine the risk of disease transmission in real world scenarios. It was determined that while atrazine caused a 28% increase in schistosomiasis transmission risk by indirectly increasing snail populations, the loss of crayfish and water bug predators were catastrophic for human health, leading to a 10-fold expected increase in parasitic infection. On the other hand, in healthy mesocosms unexposed to

either pesticide, predator populations were able to adequately maintain snail numbers below thresholds for disease transmission.

While regulators have acknowledged sublethal effects of atrazine on amphibians, this intricate process whereby atrazine use increases debilitating parasitism in amphibians is not taken into account. Nor is there any mechanism through which EPA considers the potential for multiple pesticides to trigger a trophic cascade that magnifies the risk of human disease transmission. A wealth of independent mesocosm research, however, is providing a road map for regulators to begin evaluating these complex interactions.

TROPHIC CASCADES IN AQUATIC ENVIRONMENTS UNDERSCORE ECOSYSTEM COMPLEXITY

Pesticides can make their way into aquatic ecosystems through run-off from a single application (such as a “pulse” from an agricultural area), or in low doses over the course of several weeks (as is common in mosquito control efforts). Relyea and Dieks (2008) hypothesized that differences in application timing, amount, and frequency would lead to different impacts on a pond ecosystem. To test this idea, researchers created a series of mesocosm tanks comprised of phytoplankton, periphyton, zooplankton (herbivorous phytoplankton algae eaters), and wood frog and leopard frog tadpoles. Some tanks received one large single dose of the organophosphate insecticide malathion, while others had the pesticide applied at low amounts over seven weeks.

In both instances, malathion’s impact on zooplankton caused a trophic cascade. By depressing the zooplankton population, phytoplankton flourished. The increase in free-floating algae clouded water, decreased light penetration, and led to reduced periphyton growth. Decreases in periphyton algae, the primary food source for tadpoles, retarded growth and development in leopard frogs, which prevented many from metamorphosing before the vernal pool in which they resided dried up (though wood frogs were generally unaffected). While zooplankton in the single-application mesocosm eventually experienced a population rebound, it took nearly a month and a half before this occurred. Overall, frogs in single-application mesocosms fared slightly better than those in chronically exposed tanks, which experienced an ongoing state of disruption that never permitted zooplankton populations to bounce back.

Hua and Relyea (2012) sought to find out whether pond ecosystems from different regions respond the same way to malathion contamination. This was tested by creating mesocosms with different food web assemblages—one from the east coast and one from the west coast. Though both food webs still contained zooplankton and algae, west coast tanks included northwestern salamander and cascade frog tadpoles, and east coast tanks contained spotted salamanders and wood frogs. As hypothesized, both communities produced

Issues with EPA’s Current Ecological Risk Assessment

Under 40 CFR 158.630, EPA is required to evaluate how pesticide use patterns impact a range of non-target species. Risk assessments are conducted on test organisms that represent a class of animal, including birds, mammals, aquatic organisms, such as fish and invertebrates, and insect pollinators. The agency generally assumes that the response of these test species will be indicative of how the class responds.

Under EPA Guidelines for Ecological Risk Assessment issued in 1998, the agency acknowledges the potential for “cascading adverse effects” as a result of pesticides’ indirect effects. However, when these evaluations are conducted, they are usually simplified in a way that considers how a single chemical’s impact on one sensitive species may cause a less sensitive species to decline.

EPA’s evaluation generally does not take into account the real-world complexities that are required for a full analysis, including multiple trophic levels, longer exposure periods, behavioral and developmental factors, pesticide mixtures, species resistance, and “pass-through” effects.

Strengthening regular integration of complex ecological assessments into EPA’s review process may offer more information, however risk assessments do not take into account all potential adverse effects that could occur after the release of a chemical into an ecosystem, ultimately necessitating the adoption of alternative practices that do not require pesticide use.

similar trophic cascades in response to malathion input. However, while malathion’s effect on zooplankton reduced the growth and development of their salamander predators, both frog species fared well. The researchers indicate this was likely because, when compared to leopard frogs tested by Relyea and Dieks (2008), cascade frogs and wood frogs are quicker to metamorphosize, and able to complete their transition to adulthood before the trophic cascade limited their food supply.

The sum of these studies have important implications for regulators. Beyond direct toxicity to a single species, the timing and frequency of a pesticide application can determine whether an ecosystem may be able to recover from a trophic cascade event. Further, even when generalizations can be made about trophic cascades, effects on different species of the same animal can vary based upon differences in physiology and life history. This additional complexity underlines the fact that there is much more to understand about the broader effects of pesticides on the environment.

PESTICIDE HAZARDS CAN AFFECT THE TERRESTRIAL ENVIRONMENT, ECOSYSTEM SERVICES

Pesticide-induced trophic cascades can affect a range of aquatic ecosystems, and these impacts can translate to terrestrial food webs. In addition to still-water ponds, researchers

can also craft mesocosms to mimic a runoff event of a pesticide into a stream ecosystem. In a study conducted by Rodgers et al. (2016), the synthetic pyrethroid insecticide bifenthrin created a trophic cascade that rippled both up and down the food chain. Bifenthrin caused significant downward population pressure on larval macroinvertebrates (such as mayflies, stoneflies, and caddisflies) at concentrations lower than previously recorded in literature. The loss of these periphyton-eating species initiated a trophic cascade from the top-down, causing a bloom in attached algae. Bifenthrin's impact on the endocrine (hormonal) system of macroinvertebrates also caused those that remained to speed up their time to metamorphosis, emerging smaller and earlier than usual. This can lead to bottom-up trophic effects on terrestrial insects, amphibians, reptiles, and birds that rely on a healthy population of aquatic invertebrates as a food source. Extrapolating the data gained from the mesocosm experiment and comparing it to the U.S. Geological Survey's Midwest Stream Quality Assessment of pesticide contamination, Rodgers et al. (2016) determined that 40% of streams are at risk of altered food web dynamics, and 7% are at risk of a trophic cascade leading to an algae bloom.

In a rare instance for trophic cascade science, Thompson et al. (2016) investigated what happens in the real world when a pesticide contaminates a stream environment. After a significant chlorpyrifos spill into a UK stream, researchers set up a comparison control site to evaluate changes in

aquatic communities at the affected site. The contaminated stream underwent significant trophic reshuffling, precipitated by population declines of important macroinvertebrates and detritivores. The loss of these animals resulted in less food for species like trout higher on the food chain, which researchers indicated can then flow upwards to affect other predator species, such as birds, otters, and other mammals. Down the food chain, although bacteria and other microbes proliferated in an attempt to make up the work, the loss of amphipods and other detritivores led to lower rates of decomposition overall, indicating a potentially significant impact on nutrient cycling, a critical ecosystem service provided by natural environments.

In both still and fast-moving aquatic environments, pesticides act powerfully on the foundational levels of the food web. Although algae blooms are usually considered the result of excess nutrient input, it could also be the case that a recent insecticide application eliminated all of the herbivorous grazing macroinvertebrates. Likewise, declines in threatened predators like otters could be related to impacts two steps down the food chain, if the fish on which they rely have declined due to pesticide-induced reductions in their prey.

RESISTANCE MEASURES

Given clarity that pesticides lead to trophic cascades, researchers have begun to investigate whether there may be ways to buffer their deleterious impact on the environment.



Figure 2: **Atrazine-Initiated Trophic Cascade Increases Parasite Infection Rate in Frogs**



STABLE ECOSYSTEM (no pesticide): In a stable, uncontaminated ecosystem, phytoplankton blocks sunlight so that periphyton does not grow out of control. This maintains low populations of snails that transmit parasites to frogs, and allows frog populations to remain stable.



TROPHIC CASCADE (atrazine contaminated): The influx of atrazine into the ecosystem kills off free floating algae, which increases populations of attached algae, providing more food for snails and increasing their population, and the parasite load they can carry, causing more frogs to become infected, reducing their population.

Boone and Sullivan (2012) found that adding leaf litter to a mesocosm, set up similar to Relyea and Dieks (2008) but contaminated with the insecticide carbaryl (rather than malathion), increased the survival of green frog tadpoles. When the trophic cascade occurred, the nutrients in the leaf litter facilitated the growth of periphyton, which increased food resources and hastened the metamorphosis of tadpoles into frogs. Similarly, Brogan and Relyea (2015) found that macrophytes (submerged aquatic plants) had the ability to mitigate trophic cascades caused by malathion contamination by shielding the zooplankton population. According to researchers, this buffer effect occurred because macrophytes take up carbon dioxide, which reduces carbonic acid in the water and thus raises pH, which subsequently results in faster degradation of malathion. Also, through nutrient competition and natural allelopathy, macrophytes can suppress the growth of light-reducing phytoplankton algae even if zooplankton decline in the habitat.

Nature may even be taking steps to slow down trophic cascades without human intervention. Randall and Relyea (2014) found that in agricultural areas, high percentages of zooplankton populations are resistant to the insecticide chlorpyrifos. By inserting these resistant zooplankton into a mesocosm, Bendis and Relyea (2016A) found that they had the ability to mitigate trophic cascades by maintaining their numbers and preventing phytoplankton blooms, leading to increased survivorship of leopard frogs in a mesocosm. Bendis and Relyea (2016B) also found that this pesticide

insensitivity translates to other insecticides with similar modes of action. While resistant zooplankton were unaffected by other nerve inhibitors like carbaryl and malathion, exposure to another class of insecticides, synthetic pyrethroids, still led to a trophic cascade ending with a phytoplankton bloom.

These studies provide some indication that ecosystems can adapt to the effects of trophic cascades, but in no way do they nullify the original contamination caused by pesticide use. In fact, these data underscore the need to adequately evaluate the complexity of pesticide impacts on the environment. Regulators can begin to get a handle on these impacts by including mesocosm studies in pesticide registration requirements. As the next section reveals, this research should also be paired with agricultural case-studies evaluating the overall effectiveness of pesticide use.

TROPHIC CASCADES IN AGRICULTURE CANCEL OUT ANY PESTICIDE "BENEFITS"

In more simplified ecosystems, such as those seen in agricultural fields, changes in trophic structure can be particularly pronounced. Mesleard et al. (2005) found that the insecticide fipronil, used to control midge pests in conventional rice fields, causes a trophic cascade that reduces the nutritional value of the area for waterfowl. Comparing a chemical-intensive rice field to one managed organically, the trophic cascade ultimately neutralized the efficacy of synthetic pesticide use in the first place.

Direct toxicity from fipronil reduced the number of invertebrate predators in chemical-intensive rice fields. This led to a trophic cascade that allowed herbivorous animals to flourish. On the surface, organic and chemically-managed rice fields both contained the same amount of invertebrate biomass. However, in chemical-intensive fields, this biomass was primarily in the form of gastropods (snails and slugs). When researchers surveyed the fields in late summer, only 12% of the invertebrate community were predators, while in organic fields that proportion was 70%. Slugs and snails are not a major food source for the most common waterfowl in the region studied, the heron, making organic plots a more valuable source of sustenance. As evidenced by Rohr et al. (2008), if flatworm populations are present they may also be leading to higher disease loads in amphibians and other aquatic wildlife. Critically, researchers identified relatively equal numbers of midge pests between organic and conventional fields, leading the authors to deem fipronil use “inefficient,” as the trophic cascade that occurred in conventional fields depressed natural predation of midge pests by its macro-invertebrate predators.

Many researchers have made similar determinations about efficacy after looking into the trophic impacts of systemic pesticides like fipronil, and the neonicotinoid class of insecticides. These chemicals are increasingly employed to address outbreaks of invasive species, potentially creating more problems than they solve. Analyzing case studies aimed at managing the Asian longhorned beetle and emerald ash borer in Maryland and New York City’s Central Park, Szczpaniec et al. (2011) found that the use of the neonicotinoid imidacloprid indirectly causes a predator-prey reversal. Mites feeding on neonicotinoid-contaminated leaves accumulated the insecticide in their bodies, but did not die. Because of this, the mites successfully killed their insect predators, a result of a “pass-through” effect. Not only did they reverse the predator-prey relationship, laboratory tests also found that spider mites that ate these contaminated leaves laid more eggs, which researchers attributed to a physiological change in the infested trees after imidacloprid application. Prior research by Raupp et al. (2004) had also identified higher rates of spider mite infestation when using imidacloprid to treat for hemlock wooly aldergid.

The “pass-through” phenomenon in trophic cascades is best exemplified in the study conducted by Douglas et al. (2014). Field crops, such as soybeans, will often be coated with a variety of pesticides, with those in the neonicotinoid class the most frequent. The intent of these seed treatments is to reduce risk of crop damage to young seedlings, however Douglas et al. (2014) found this practice to be counterproductive.

Starting in the lab, researchers provided slugs, a primary pest in soybean fields, neonicotinoid-treated seedlings to eat. The slugs experienced no adverse effect on survival, behavior, or physiology as a result of eating the contaminated seedlings. Researchers then placed these slugs in an enclosure with a ground beetle predator. Beetles that ate unexposed slugs fared well, but those that ate slugs that had dined on neonicotinoid-treated seedlings experienced impairment ranging from reduced motor function to paralysis or death. This process also translated to field conditions. During the first month after seed treatment in a soybean field, slug predation was reduced by 33%, slug activity increased by nearly 70%, and, over the course of the season, soybean yields were down 19%. Contrary to claims by the pesticide industry that seed treatments are “targeted” and do not impact non-target organisms, this study reveals an enormous flaw in the pesticide review process.

In a follow-up meta-analysis on the impacts of neonicotinoid seed treatments, Douglas and Tooker (2016) determined that natural predators were reduced by 16% in agricultural fields where these seeds were used, comparable to what would occur if their use was replaced with synthetic pyrethroid insecticides.

HOW REGULATORS CAN DO BETTER

The studies analyzed only begin to touch on the multitude of ways that pesticides can upset and imbalance ecosystem health and stability. Findings related to increased risk of disease transmission, dangers to declining species, algae blooms, the loss of ecosystem services like nutrient cycling, and importantly, ineffective pest management, establish the critical need for EPA to consistently assess ecosystem level trophic effects as part of the pesticide registration process. In the absence of a truly precautionary system where independent science is adequately considered by regulators, and pesticides are removed from the market when found to cause trophic cascades or other ecosystem disruption, the agency must develop a “No Observed Adverse Effect Level” for ecosystems, in addition to individual species. The agency must require manufacturers to conduct mesocosm experiments on typical aquatic communities. Pesticides placed on the market should be continuously monitored for their ability to create “pass-through” impacts in target pests that lead to trophic cascades. Case studies in agricultural fields are also needed to confirm the efficacy of pesticide use in the first place. When chemicals are found to be associated with ecosystem level effects, they should be immediately suspended until research can determine whether there is a threshold at which no adverse effects on ecosystems are seen. If regulators begin to consider the complex, ecosystem-wide impacts of pesticide contamination in our environment, we can get a true assessment of pesticides’ adverse effects and prevent future disruptions through the adoption of alternative practices.

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