

Summer 2010

The Toll of Toxics: Investigating Environmental Contaminants

Donald Sparling


U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center

Barnett A. Rattner

Patuxent Wildlife Research Center, brattner@usgs.gov

John S. Barclay

Follow this and additional works at: <http://digitalcommons.unl.edu/usgsstaffpub>

 Part of the [Geology Commons](#), [Oceanography and Atmospheric Sciences and Meteorology Commons](#), [Other Earth Sciences Commons](#), and the [Other Environmental Sciences Commons](#)

Sparling, Donald; Rattner, Barnett A.; and Barclay, John S., "The Toll of Toxics: Investigating Environmental Contaminants" (2010).
USGS Staff -- Published Research. 949.

<http://digitalcommons.unl.edu/usgsstaffpub/949>

This Article is brought to you for free and open access by the US Geological Survey at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in USGS Staff -- Published Research by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

The Toll of Toxics

Investigating Environmental Contaminants

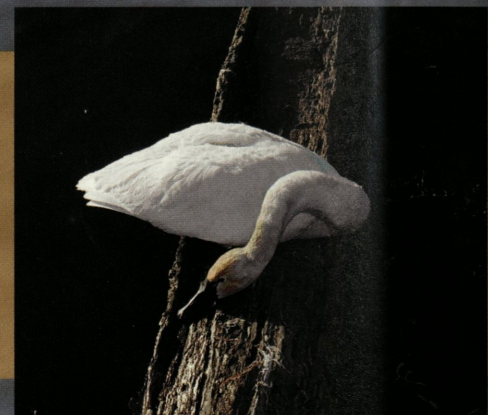
On Earth Day of this year, the British Petroleum-operated Deepwater Horizon oil drilling rig exploded in the Gulf of Mexico, 41 miles off the Louisiana coast. The blast killed 11 workers, injured 17, launched a massive oil spill, and triggered an environmental catastrophe—the full impact of which may not be realized for years.

As the U.S. government, BP, private contractors, and hundreds of volunteers battle to contain the spill, the outlook for wildlife in the region grows dire. Tens of thousands of birds reside in or migrate through coastal habitats along the Gulf, and these birds can ingest oil when they preen. Marine mammals can be exposed to the toxic slick when they surface to breathe. Endangered sea turtles can consume food tainted by petroleum. And delicate wetland habitats and fisheries can become poisoned.

By Donald W. Sparling, Ph.D., Barnett A. Rattner, Ph.D., and John S. Barclay, Ph.D.

A line of boom floats near the shore of what locals call Cat Island in Alabama's Mobile Bay, where dozens of pelicans and other birds stand oblivious to the growing oil slick offshore.

Credit: Mozart DeDeaux/National Audubon Society



Credit: Brian Spears/USFWS

Despite the known risks, the full environmental toll of the spill won't become clear until researchers emerge from the initial crisis and start to analyze impacts. They will begin to learn whether Gulf Coast wildlife populations and their habitats prove resilient in the face of seen and unseen contamination. "There will be people spending much of their careers doing follow-up on this," says Peter Albers, emeritus research wildlife biologist with the U.S. Geological Survey.

Dangers Far Beyond Oil

Less than six months prior to the spill, in December 2009, conservationists were closing a chapter on another environmental disaster. North American copper-mining conglomerate American Smelting and Refining Company (Asarco) agreed to pay the federal government \$1.79 billion as a settlement for contaminating more than 80 sites across the United

States—including a huge swathe of Idaho's Coeur d'Alene Basin—with lead and other toxic metals (EPA 2009). In Idaho, osprey (*Pandion haliaetus*) became poisoned from ingesting prey with metal residue in their tissues, and thousands of waterfowl, including tundra swans (*Cygnus columbianus*), died from ingesting heavy metals in sediments and plant material (Blus *et al.* 1991, Farag *et al.* 1998). Some \$194 million of the record settlement will fund wildlife, habitat, and other natural resource restoration projects to mitigate and compensate for the impacts of toxic metals on natural ecosystems (FWS 2009).

These two recent events bring to the fore the work of wildlife toxicologists. Focusing on amphibians, reptiles, birds, and mammals, wildlife toxicology is a component of ecotoxicology—the study of toxic effects caused by natural or synthetic pollutants on

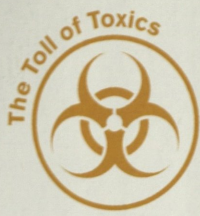


Credit: Donald W. Sparling



Credit: Marathon Turtle Hospital

A tundra swan lies dying near Idaho's Coeur d'Alene Lake, a region exposed to decades of harmful mining emissions. Twisted bones show the effects of lead poisoning in a lab frog exposed to 1,620 milligrams of lead per kilogram. Far higher doses can exist in nature. Tumors on a green sea turtle indicate fibropapilloma, a disease often found in polluted waters. It may be linked to organochlorines, found in many pesticides.



Southern Illinois University graduate student Tab Bommarito measures the intensity of ultraviolet light applied to tanks of spotted salamanders (*Ambystoma maculatum*) exposed to varying concentrations of petroleum-derived asphalt sealant. Though petroleum hydrocarbons alone can harm amphibians and other creatures, UV light can increase the toxicity of these chemicals by nearly 100 times.



Credit: Brian Hetrick

living organisms and other constituents of ecosystems (Truhaut 1977). Now a distinct discipline within the wildlife profession—practiced by members of The Wildlife Society’s own [Wildlife Toxicology Working Group](#), among others—wildlife toxicology has become increasingly important as human populations and industry have spread, causing contaminants to multiply.

Growth of a Discipline

The methods wildlife toxicologists employ to understand how wildlife is affected by the presence of chemicals in the environment have evolved over the past century. Among notable developments in the field:

Late-1800s to 1920s: Researchers studied birds dying from oil spills, alkali poisoning, and ingesting lead shot and predator control agents such as lye, arsenic, and rat poisons.

1930s: Pesticides—including arsenic, lead, pyrethrum, nicotine, mercurial fungicides, and dinitro-*o*-cresol—became commonly available, and crop-dusting aircraft greatly facilitated their application. With the discovery of the insecticidal properties of DDT in 1939 and related compounds shortly thereafter, use of such chlorine-based pesticides increased dramatically.

1940s and '50s: Following World War II, researchers documented wildlife mortality and chronic effects following pesticide application in agricultural and forest habitats (Barnett 1950). The widespread hazards of spent lead shot (Bellrose 1959) and industrial activities to free-ranging

birds and mammals were described and evaluated through controlled exposure studies (Coburn and Treichler 1946, Coburn *et al.* 1950).

1960s: Rachel Carson’s *Silent Spring* came out in 1962, implicating pesticides and pollution in the decline of some wildlife species (Carson 1962). The U.S., United Kingdom, and Canada established long-term contaminant-monitoring programs, such as the National Contaminant Biomonitoring Program and the Predatory Bird Monitoring Scheme. Studies linked DDT to eggshell thinning and population declines in bald eagles (*Haliaeetus leucocephalus*), brown pelicans (*Pelecanus occidentalis*), and other raptorial and fish-eating birds (Hickey and Anderson 1968, Heath *et al.* 1969). In 1963 Congress passed the Clean Air Act, which included provisions for a cleaner environment.

1970s: The White House and Congress established the U.S. Environmental Protection Agency (EPA) in 1970 to regulate environmental contaminants, among other responsibilities. A growing awareness of the toxic effects of contaminants on wildlife captivated the interest of society—helping spur the environmental movement—and intrigued scientists, who developed collaborative projects to research these effects. In 1972 Congress passed the [Clean Water Act](#), which today has water guidelines to ensure fish and wildlife safety. The 1976 [Resources Conservation Recovery Act](#) mandated tracking of hazardous waste “from cradle to grave.”

1980s: Wildlife toxicologists studied heavy metal pollution from mining and smelting, pesticide-induced die-offs, and the toxic results of disasters such as the 1986 Chernobyl nuclear reactor meltdown and the 1989 *Exxon Valdez* oil spill. Established in 1980, the Superfund program mandated that polluters cover the costs of clean-up at severely contaminated sites.

1990s: Scientists refined methods of determining the effects of contaminants, and expanded the use of biochemical biomarkers (indicators of contaminant exposure and effect, such as the induction or inhibition of enzyme activity). Other new areas of interest included research on endocrine disruption and population modeling. By the late 1990s concern about declining amphibian populations led to an increased interest in effects of contaminants on herpetofauna.

2000s: Today scientists are unraveling the mysteries of emerging contaminants such as



Donald W. Sparling, Ph.D., is an Associate Professor at Southern Illinois University’s Cooperative Wildlife Research Laboratory.

Coauthors

Barnett A. Rattner, Ph.D., is an Ecotoxicologist at the U.S. Geological Survey’s Patuxent Wildlife Research Center.

John S. Barclay, Ph.D., is Director of the Wildlife Conservation Research Center at the University of Connecticut.

pharmaceuticals and nanomaterials, extremely small particles used in some industrial and household products. Researchers are also studying how an individual's genes and proteins respond to toxic substances—a discipline called toxicogenomics. In addition, they're examining the interplay among toxicants, ecological integrity, and human health, and trying to determine the relative role of environmental contaminants as one of many stressors affecting wildlife populations.

Playing Chemical Catch-up

With more than 80,000 man-made chemicals produced and used in the U.S. each year, wildlife toxicologists have a seemingly endless task. New chemicals are constantly finding their way into the environment. Some may be harmless, some dangerous at even minuscule concentrations, and others only toxic at high concentrations. The dose makes the poison—which is why toxicity studies are so crucial.

According to the Toxic Substances Control Act of 1976 (TSCA), if a company wants to make, distribute, use, or sell a new chemical in the U.S., it must contact the EPA and report what is known about that chemical. Depending on the information provided, the EPA may ask for more tests. But "TSCA is not a strong piece of legislation," says Anne Fairbrother, senior managing scientist at the Exponent scientific-consulting firm and a former EPA assistant laboratory director. The European Union has recently passed a stronger piece of legislation called REACH, which requires companies to conduct toxicity studies on rodents before use of a chemical is approved.

In the U.S., the EPA requires all pesticides to be registered for use. The Pest Management Regulatory Agency serves a similar function in Canada. These regulatory agencies have established protocols that involve a battery of testing on species from aquatic invertebrates through fish, birds, mammals, and humans, depending on how the chemicals will be used and where they are likely to be found.

These tests, however, cannot duplicate the complexity of a natural ecosystem. For example, the EPA mandates that pesticides be tested on certain species of mammals, birds, fish, and zooplankton, but not on amphibians, which have been shown to be extremely sensitive to these chemicals. Some pesticides are allowed to be used on a provisional basis, before complete testing. In addition, lab-based tests cannot fully reflect natural conditions of exposure. For example, risk-assessment tests evaluate how much of a pesticide a bird could safely eat, but they

Amphibian Population Effects

It's difficult to prove that a contaminant has caused a population to decline or disappear. Almost always, contaminants are just one of many factors affecting populations, along with habitat loss, food availability, predation, disease, competition, and climate. So instead of asking "Did contaminants wipe out this population?" one can reframe the question to "Did contaminants substantially contribute to the decline of this population, or would it have died out anyway?"

Many scientists are asking this about amphibians, no doubt in response to worldwide declines in amphibian populations. Overall, scientists agree that habitat loss or degradation and the chytrid fungus have been key drivers of amphibian population declines and perhaps even extinction events, but contaminants also remain a concern.

Though contaminants do not typically trigger conspicuous die-offs of amphibians, they may affect populations in subtle ways. For example, toxics have been shown to reduce growth rates of tadpoles, leading to mortality if ponds dry up before metamorphosis occurs. Toxics slow tadpole response times or swimming ability, making them more vulnerable to predators and less able to find food. Contaminants can also interfere with sexual development, reproduction, and thyroid functioning, which may cause tadpoles to grow but not undergo metamorphosis. Finally, toxics can impair immune functions, making tadpoles or juvenile frogs more vulnerable to disease.

Most amphibian studies have been conducted in labs, yet emerging research demonstrates that contaminants have very different effects on animals in ecological communities than they do on single species. Researchers from Rick Relyea's lab at the University of Pittsburgh, for example, have shown that herbicides can reduce the availability of phytoplankton, resulting in food shortages for tadpoles (Relyea in press). (See profile on pg. 23.)

The Puzzle in California

Linking the effects of contaminants to a population decline is like solving a huge jigsaw puzzle. Several investigators from universities and government agencies are trying to put the pieces together in California's Sierra Nevada Mountains, where substantial evidence suggests that pesticide contamination is a principal factor in amphibian population declines.

California's Central Valley is a major agricultural region with a diversity of crops and pesticides. Winds carry insecticides into the Sierras, where they are deposited into ponds and rivers. Some of these chemicals are extremely toxic. Studies have shown that a few parts per billion of endosulfan, for example, is sufficient to kill more than half of exposed tadpoles of the foothill yellow-legged frog (*Rana boylei*) (Sparling and Fellers 2009). In addition, the metabolites and some breakdown products of the other insecticides are 10 to 100 times more toxic than their parent chemicals (Sparling and Fellers 2007).

Ongoing research is combining field and lab studies to elucidate the threat pesticides pose to amphibians in the Sierras. This research and other such studies may show if contamination can have a lasting effect on a wildlife species.



Credit: Jamie Bettaso/USFWS

The foothill yellow-legged frog, which makes its home near rocky streams and rivers, has disappeared from nearly half of its California range. Pesticides blown from the vast agricultural fields of California's Central Valley are one likely culprit for the species' decline.

U.S. Fish and Wildlife Service biologist Sherry Krest constructs an enclosure in Delaware's Prime Hook National Wildlife Refuge to test how tadpoles are affected by proximity to a "hot spot" of lead contamination. Lead shot from a nearby gun club wound up in the refuge, reaching a concentration high enough to cause serious developmental problems or death in amphibians.



Credit: USFWS

do not examine how a bird is affected by absorbing the chemical through its skin. Pierre Mineau, a senior research scientist for Environment Canada, and others have shown that skin absorption, which could occur if a bird landed on a recently sprayed field, can be highly detrimental. Yet "the regulatory system is slow to change," says Mineau.

The Biggest Players

The thousands of compounds wildlife may be exposed to cover the chemical spectrum, but more commonly occurring chemical contaminants fall into the following broad categories:

Pesticides. Many chemicals used to control unwanted species including weeds, insects, fungi, and rodents have had unintended adverse effects on wildlife (see page 28). Fifty years ago, for example, many pesticides, such as DDT, contained chlorine. Because bacteria have a difficult time breaking carbon-chlorine bonds, these organochlorine pesticides can persist for decades in the environment. Such pesticides were sprayed at low concentrations, but they accumulated in animals' tissues and built up in concentration at higher levels of the food web, where they caused reproductive problems or outright mortality in fish-eating birds and some other species. Most organochlorines have been banned in the U.S. by the EPA, but some are still being used legally (and illegally) in developing countries.

Modern pesticides generally degrade more quickly than the organochlorines. But many of these can be toxic to animals in the short term, or they may inhibit growth, alter behavior, or affect sexual development. Atrazine, for example, is the second most widely used herbicide in the U.S., with an estimated 80 million tons applied to crops each year. This compound, which has been banned in the European Union, has reputedly been found to turn

A Poisonous Dilemma

When rodenticides move beyond their target species

The use of rodenticides Rozol and Kaput-D to control populations of black-tailed prairie dogs (*Cynomys ludovicianus*) illustrates the inherent complications of using toxic compounds to control wildlife.

After six states authorized the use of Rozol to control prairie dogs on rangeland in 2009 that authorization was extended to the rest of the species' range, totaling 10 states. The state authorizations are considered controversial because Rozol contains chlorophacinone, an anticoagulant that causes internal hemorrhaging and that can be passed to animals that eat poisoned prairie dogs, posing a threat to bird and mammal species including ferruginous hawks (*Buteo regalis*), bald eagles (*Haliaeetus leucocephalus*), swift foxes (*Vulpes velox*), and American badgers (*Taxidea taxus*) (Defenders of Wildlife 2009).

Kaput-D, another anticoagulant rodenticide, contains the chemical diphacinone, which the manufacturer claims has lower concentrations of toxicant and, therefore, carries a lower risk

of secondary poisoning. According to reports, Kaput-D's registration through the entire range of the black-tailed prairie dog is pending under the Federal Insecticide, Fungicide, and Rodenticide Act.

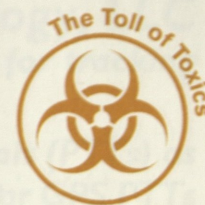
These chemical uses have garnered opposition. In June 2009, the World Wildlife Fund (WWF) called for the suspension of Rozol to control prairie dog populations until an Avian Reproduction Study could be completed (Federal Register 2009). Also in 2009, Defenders of Wildlife and Audubon of Kansas sued the Environmental Protection Agency (EPA) for allowing the use of Rozol and Kaput-D, claiming that their use violates the federal Endangered Species Act (ESA), the Migratory Bird Treaty Act, and the Bald and Golden Eagle Protection Act. The suit claims that the EPA failed to "heed warnings from the U.S. Fish and Wildlife Service (FWS) that registrations of the chemicals chlorophacinone and diphacinone be discontinued or rescinded because of known and potential impacts to wildlife" (Defenders of Wildlife 2009).

"The biggest concern is secondary hazards. Recent studies show that raptors are much more sensitive to some of these anticoagulants

than we previously thought," says Kathleen Fagerstone, research program manager with the USDA's National Wildlife Research Center. Equally if not more worrisome is the potential threat of the toxicant to the prairie dogs' key predator, the highly endangered black-footed ferret (*Mustela nigripes*).

Because Rozol is a first-generation rodenticide—less toxic than second-generation rodenticides—it requires higher concentrations to be effective. Rozol "takes multiple feedings...to administer a lethal dose," says Moira McKernan, pesticides and birds program director at the American Bird Conservancy. This means that a prairie dog may feed on Rozol for a week or two before the toxicant kills it. "By the time it expires or its behavior is inhibited because of dying, it's carrying...a very toxic dose to a bird or another predator that might feed on it," she says.

To reduce the risk of predators feeding on poisoned carcasses, people applying feed grains laced with Rozol are legally required to place the rodenticide at least six inches inside prairie dog burrows. Although application requirements vary, the federal label requires those who use Rozol to return to the site within 10 days after the first application and



male frogs into females and to chemically castrate others, at concentrations of as little as 0.1 parts per billion (Hayes *et al.* 2010). The EPA concluded in 2007 that atrazine had not been proven conclusively to negatively affect amphibian sexual organ development (EPA 2007), but is currently conducting a review that could increase restrictions on its use.

Metals and metal-like elements. Lead, cadmium, mercury, copper, zinc, selenium, and other metals can interfere with an organism's enzyme-mediated biological functions and can produce neurological disorders and bone deformities. These metals typically accumulate in higher trophic levels—in species at the top of the food web. Metal contamination problems are common and range from widespread pollution, as in the Coeur D'Alene Basin, to smaller-scale but still significant threats (TWS 2008).

In the 1990s, for example, ecotoxicologists confronted a lead-contamination problem at Prime Hook National Wildlife Refuge in Delaware. For almost 40 years, members of a neighboring gun club conducted target practice in an area up range of a refuge wetland. Sherry Krest and other FWS biologists investigated the wetland's health. After finding

lead shot on the ground nearby, they covered the contaminated area with a "cap"—essentially a high-tech tarp to prevent birds from consuming the spent lead and prevent the lead from flowing into the wetland's sediment.

"Before the clean-up I would go into these wetlands and I would hear frogs calling and see adults, but I wouldn't see tadpoles or egg masses," says Krest. Though she did not do reproductive studies in the field, she suspected that lead contamination might have been interfering with the frogs' reproduction. The FWS researchers found lead levels in the wetland up to 5,800 parts per million (ppm). Laboratory studies confirmed that even at levels as low as 75 ppm, southern leopard frog (*Rana sphenoccephala*) tadpoles experienced severe skeletal malformations (Sparling *et al.* 2006). The concentration found in the wetland caused 100 percent mortality of tadpoles in the lab. In 2000, FWS issued the gun club a cease-and-desist order from depositing lead and other shot onto Prime Hook. Since then, Krest has continued to monitor the wetland. Natural sedimentation is expected to cover the toxic sediments with layers of non-contaminated sediments. Her



Credit: Colorado Division of Wildlife

follow up over two to three weeks to dispose of dead animals. FWS has asked the EPA for a more stringent application process. Says FWS biologist Nancy Golden, "We would like to see the follow-up much more often."

Better Alternative?

When the WWF called for Rozol's suspension in 2009 it noted that a non-anticoagulant alternative, zinc phosphide, was effective at controlling prairie dogs. Zinc phosphide can also be toxic to birds and small mammals, however, it doesn't accumulate in the body

An endangered black-footed ferret peers out of a prairie dog burrow—perhaps not the safest place to hide. Ferrets prey on prairie dogs, but the use of the rodenticide Rozol to control prairie dog populations has led to concerns that ferrets could suffer secondary toxicity from eating poisoned prey.

tissues like Rozol does, so "there's very little risk of secondary toxicity," says McKernan.

In 2004, EPA biologists William Erickson and Douglas Urban released a study on the potential risks of nine rodenticides—including chlorophacinone and zinc phosphide—to birds and non-target animals (Erickson and Urban 2004). In addition to data collected from lab and field studies, liver-residue analyses confirmed the presence of rodenticide in birds and non-target mammals such as hawks, eagles, and deer. The study also showed that the rodenticides were not selective to their target species, putting non-target birds and mammals at risk of primary and secondary exposure.

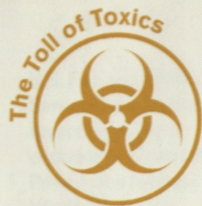
In 2008, the EPA released its final ecological risk mitigation decision, which placed some restrictions on the sale, distribution, and packaging of brodifacoum, difethalone, bromadiolone and difenacoum—all second generation anti-coagulant rodenticides (Final

Risk Mitigation Decision for Ten Rodenticides). Because of this, according to Barnett Rattner, an ecotoxicologist with the USGS-Patuxent Wildlife Research Center, other available rodenticides will probably be used more often and in greater quantities, including diphacinone and chlorophacinone.

A handful of new studies are underway to assess how chlorophacinones, diphacinones, and other ingredients in rodenticides affect non-target species. Scientists at the USDA's National Wildlife Research Center (NWRC) are testing the toxicity of Rozol to assess the residue level of chlorophacinone in prairie dogs after exposure. "It's a time-course study to look at how quickly chlorophacinone residues disappear from prairie dog tissue," says John Eisemann, registration manager at the NWRC, which is also testing whether the addition of bird repellents to certain rodenticide baits may reduce non-target consumption of the baits.

EPA's public comment period closed in the first week of November last year. The case is currently under litigation.

By Divya Abhat
Production Editor/Science Writer for
The Wildlife Society



findings indicate the frogs are recovering—slowly—but they still have lead residues in their tissues (Krest personal communication).

Organohalides. These are very persistent organic molecules that contain chlorine, bromine, or fluorine in their structure and include chlorine-based pesticides, polychlorinated biphenyls (PCBs), some fire retardants, and other compounds. Organohalides have been shown to disrupt hormone function, affecting reproduction and causing thyroid problems. They have also been linked to adverse effects on the immune system. PCBs—which were commonly used as insulating material for electrical transformers and similar products in the 1940s and 50s—are thought to cause skin rashes and, more seriously, liver problems and cancer in humans. Wildlife is similarly at risk. In 1979 PCB production was banned in the U.S. but, because of the chemicals' extreme persistence, they can still be found everywhere from the Great Lakes to the Arctic.

As early as the 1960s, mink (*Neovison vison*) farmers began noticing decreased reproduction among animals fed coho salmon (*Oncorhynchus kisutch*) from the Great Lakes. Studies showed that PCBs in the fish were the primary culprit, causing reduced feeding, gastric bleeding, and liver and kidney degeneration in mink (Kirk 1971, Aulerich and Ringer 1977). In addition, researchers found that PCBs could be passed from parents to their more-sensitive offspring. Further studies have shown that extended consumption of as little as 0.05 ppm of one form of PCB in the diet caused 50 percent mortality among captive mink (DeGuise *et al.* 2001), findings with obvious implications for wild mink that consume PCB-tainted fish from the Great Lakes region.

Emerging Threats

Emerging Environmental Contaminants (EECs) include an array of chemicals and substances that are discharged into the environment. According to the U.S. Geological Survey Toxic Substances Hydrology Program (2006), EECs include veterinary and human antibiotics, human drugs, industrial and household wastewater products, and sex and steroidal hormones. Beyond these four groupings, some experts also include phthalates that are used as plasticizers, chemicals used for disinfection in homes and industries, flame retardants, and extremely small particulates or nanomaterials (GRAC 2008, Sadler *et al.* 2003).

Since 2000, research on EECs in North America have reported links to cancer in humans and detected residues in numerous animal species, citing repro-

ductive abnormalities (e.g., gender alteration), and population declines or other problems in river otters (*Lutra canadensis*), mink, wood ducks (*Aix sponsa*), double-crested cormorants (*Phalacrocorax auritus*), alligators (*Alligator mississippiensis*), American toads (*Bufo americanus*), and many fish species. A recent study of native white suckers (*Catostomus commersonii*) in a stream exposed to municipal wastewater effluent, for example, found that the fish had taken up chemicals from anti-depressants excreted by humans, which eventually made their way into the fishes' neural tissue (Schultz *et al.* 2010).

With so many anthropogenic substances entering natural environments, EECs and their effects may not be detected soon enough to allow for meaningful corrective actions (Rattner 2009). Federal and state agencies, academic institutions, independent commissions and organizations, and private industries are developing assessment, monitoring, and evaluation procedures, particularly with wastewater effluents, to determine the importance of EECs to human health and the environment.

A Future for Research on Toxics

Studies by wildlife toxicologists have only skimmed the surface of how the thousands of chemicals in the environment affect wildlife, and new regulations and novel applications of old laws are constantly changing how toxicologists approach their work. Recent lawsuits brought against the EPA by the Center for Biological Diversity, for example, note that pesticides used on the landscape may be impacting endangered species in violation of the ESA (CBD 2010). "That's really driving a lot of EPA attention right now," says Exponent's Anne Fairbrother, "and I think that's likely to continue." Wildlife toxicologists will help determine the impacts on at-risk species. With so many questions to answer about the ecological effects of contaminants on wildlife, wildlife toxicologists have more than enough work for many decades of productive scientific research. ■

Any use of trade, product or firm names is for descriptive purposes only and does not imply endorsement by the U.S. Government.

This article has been reviewed by subject-matter experts.



See this article online at www.wildlife.org to read about pursuing a career as a wildlife toxicologist and to access a full list of references.