Chapter 2

Chemical Threats to Nature: Life

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Abstract:

Pesticide exposure has been shown to cause a 42% loss in species richness in Europe, even at levels considered environmentally acceptable by present standards. In contrast to insecticide-treated regions, untreated or organic crops have a far higher species richness of beneficial insects including bees, spiders, and beetles. Pesticides are frequently used in agriculture that depends on chemicals.

A chemical-intensive approach to pest management pales in comparison to organic pest management in terms of ecological stability and longevity. The survival of wild species and biodiversity are greatly impacted by this discrepancy. Understanding how diverse land management strategies affect the interconnectedness of life is essential to preserving the delicate balance and benefits of nature. The adoption of organic pest control techniques rather than chemical-intensive ones is the most crucial step in decreasing the harmful effects of pesticides on animals and protecting the planet's remaining biodiversity.

Keywords: pesticides, environment, biodiversity, ecology,

INTRODUCTION

Millions of pounds of harmful chemicals have been released into the environment as a result of the industrial revolution. Multiple sorts of pollution can have an impact on any ecosystem. Toxins have an impact on plant and animal health, just as they do on human health. Toxic compounds have been discovered in pristine woods and Arctic animal blood. Toxic chemicals and organic and inorganic pollutants enter the body through the skin or through the intake of contaminated food and water. These poisons build in increasing and higher amounts as animals move up the food chain. Bio-magnification is the term for this. Diseases, birth deformities, genetic alterations, and other negative impacts are more likely to occur when higher-level predators have larger levels of toxins. Our water, air, and soil are increasingly contaminated with pollution, synthetic chemicals, pesticides, heavy metals, and even compounds from everyday items. This noxious legacy is poisoning animals as well as our future.

There is ample evidence of widespread toxic pollution of ecosystems and animal exposure. Pesticides have been found in more than 90% of the nation's tested waterways and fish, according to a 10-year government investigation. These pollutants also find their way into people's drinking water, posing a serious health risk. In the blood of neonates, an independent study has discovered over 200 compounds, many of which are recognised poisons. Toxic chemicals raise cancer rates, create reproductive issues, and lead to a broad range of other health problems in both humans and animals. Chronic poisoning is defined as a long-term, recurrent, or continuous exposure to a toxin that does not cause symptoms immediately or after each exposure. The individual grows unwell gradually or after a long time of latent illness. Exposure to toxins that bioaccumulate or are biomagnified, such as mercury, gadolinium, and lead, is the most prevalent cause of chronic poisoning (Bank *et al.* 2005). Several nonmetallic inorganic organisms have the potential to be hazardous to ecosystems. Small streams have been briefly sterilised by spills of cyanide ions CN- from mining activities. In the stomachs of ruminant animals and newborns, nitrate ions NO^{-3} in polluted well water may be converted to nitrite ions NO^{2-}. The nitrite changes the iron (II) in blood haemoglobin to iron (III),

resulting in methemoglobin, which is ineffective in transporting oxygen throughout the body. Fatalities have occurred in cattle and human newborns in severe situations.

Plants and wildlife are poisoned by pesticides for purposes. Water quality has been contaminated as a result of their usage, deterioration, and combinations in the environment. The Pesticides Reduction Campaign at the Center aims to obtain programmatic improvements in the pesticide registration process as well as prevent hazardous pesticides from entering fish and animal habitats. Polar bear poisoning is a fantastic indication of how widespread pesticide pollution has gotten. Pesticides that have been permitted for use and application in developed countries and developing countries are transferred through the atmosphere, the ocean, and biological routes, eventually ending up in the Arctic, endangering the ecosystem's health.

Predation, particularly by Eurasian sparrowhawks; electromagnetic radiation from mobile phones; and diseases such as avian malaria have all been postulated as possible causes for the substantial decreases in the population of the house sparrow (*Passer domesticus*). A lack of nesting sites due to changes in urban building design is likely a problem, and conservation groups have urged the use of special sparrow nest boxes. Insufficient bug food for nestling sparrows appears to be a major contributor to the reduction. Increased monoculture crops, extensive pesticide usage, the replacement of native plants in cities with alien plants and parking areas, and probably the introduction of unleaded gasoline, which produces harmful chemicals such as methyl nitrite (Summers *et al.* 2007), all contribute to insect population declines.

The chemical makeup of soil and land is altered by acid rain, which is caused by the mixing of sulphur dioxide in the air with water vapour and its precipitation. It causes fish to die, and it has an impact on tree growth on land. It affects both animals’ and humans' respiratory systems. Acid rain has an impact on the aquatic ecology when it falls and runs into rivers and ponds. It creates water pollution by changing the chemical composition of the water to a state that is damaging to the aquatic ecosystem's ability to exist. Acid rain also causes water pipelines to corrode, resulting in heavy metals such as iron, lead, and copper seeping into drinking water.

Large amounts of floating detritus usually made up of plastic, are known as garbage patches in the ocean. These gather in ocean gyres, which are brought hither by ocean and wind currents. They contaminate the waters, harm coral reefs, and trap small and large species in the debris. Plastic trash in the waters affects whales, seals, walruses, dolphins, manatees, and penguins. Plastic waste is consumed by even minute species like krill and zooplankton, which pass it up the food chain.

Fracking, or high-pressure hydraulic fracturing, is a controversial and dangerous oil-drilling technology that has been connected to water contamination and methane production. According to recent studies, fracking has caused over 1,000 recorded occurrences of groundwater contamination, either as a result of fracking fluids and methane leaking into groundwater or as a result of aboveground leaks of polluted wastewater affecting flora and fauna.

Uranium mining has left a poisonous and radioactive legacy across most of the Western United States. Exploration, mining, processing, and disposal of uranium all pose unique dangers to species, ecosystems, and human groups. Uranium enters the food chain and pollutes the environment in an irreversible manner. It has the potential to contaminate aquatic ecosystems for hundreds of years, posing harm to downstream communities, fish, and wildlife. Even trace levels of uranium can kill fish, build up in the food chain, and cause malformations and reproductive issues in aquatic animals.

Endocrine disruptors are substances that interfere with normal hormone functioning and have an impact on fish and animal reproduction, development, and growth, as well as on humans. Endocrine disruptors include pesticides, medicines, plasticizers, cleaning agents, cosmetics, antibiotics, and medications that are introduced into streams and aquatic environments. The impacts on endangered species are substantial since endocrine disruptors are increasingly and needlessly contaminating drinking water supplies and aquatic animal habitats. The Center works to fight endocrine disruptors and their harmful effects on the environment. Pollutants in the air can poison wildlife by disrupting endocrine function, causing organ injury, making them more vulnerable to stresses and diseases, reducing reproductive success, and even killing them. Changes in any species' abundance as a result of air pollution can have a significant impact on the abundance and health of dependent species. For example, the loss of some fish species

due to greater aluminium levels may allow bug populations to grow, which may help particular types of ducks that eat insects. However, eagles, ospreys, and other species that rely on fish for food may suffer as a result of the loss of fish.

Amphibians, such as frogs and salamanders, are particularly vulnerable to the impacts of water pollution due to their extremely sensitive skin. They have the unusual capacity to absorb oxygen via their skin, but this also makes them vulnerable to hazardous substances. Pesticides, nitrogen-based fertilizers, and heavy metal pollutants are all known to be harmful to these animals. Following severe rains, these contaminants frequently find their way into water systems via runoff. In addition to killing frogs directly, pollution can impair their immune systems (as was the case with the Monteverde golden toad's extinction) and produce morphological malformations or anomalies. (Marian *et al.* 1983). Amphibians are far from the only animals affected by pollution in water. Fish and invertebrates also end up with these contaminants in their systems as well. Though small amounts might not kill the fish, those chemicals stay in their system. This poses a danger to the predators in the food web, such as birds of prey.

Peregrine Falcon populations decreased as a result of the pesticide DDT use, demonstrating the hazard. The falcons ate fish and small mammals that had been exposed to DDT in their habitat, and the chemicals in their prey bioaccumulated inside the falcons. Peregrine Falcons were unwell, and breeding females developed thin eggshells; reproductive success dropped, and the birds became endangered. Birds of prey are not the only predators impacted by the bioaccumulation of pollutants. Sea lions and other marine mammals are yet another example of animals affected by pollution. In the case of sea lions, fertilizer runoff results in harmful algal blooms. This alga releases a neurotoxin known as domoic acid. Fish eat these harmful algae and accumulate them in their bodies, and the sea lions eat the fish.

The sea lions swallow significant levels of toxic domoic acid as a result of bioaccumulation, culminating in domoic acid toxicosis. The illness causes neurological problems, seizures, miscarriages, and death if left untreated. Reduced pesticide and fertilizer use can help safeguard sea lions and their ecology by reducing dangerous algal blooms. Chemical pollution is far from the only dangerous form of ocean pollution, and sea lions and other marine mammals are far from the only sea species affected by it. Marine debris is any man-made object that has been dumped and has found its way into the water, and it poses a threat to a variety of marine organisms.

Oceanic animals are harmed by a variety of marine debris kinds. Discarded fishing gear, such as nets and lines, frequently entangle marine mammals, birds, turtles, and sharks. Many animals confuse plastic objects for food, and they eat them. Plastic bags are one of the most problematic types of marine litter. Large volumes of plastic waste are ingested by sea turtles in particular. Plastic bags are particularly problematic because they resemble jellyfish or algae, which are the major diet of some sea turtle species. In fact, studies estimate that more than half of the world's marine turtles have eaten plastic at some point.

Ingesting plastic might cause food digestion problems as well as intestinal blockage. The plastic cannot pass through their system and, if not treated, usually leads to death. Plastic recycling can help minimize marine trash in the oceans, protecting sea turtles and other animals. Dolphins are affected by a range of contaminants all throughout the world. Chemical pollution, plastic pollution, and noise pollution are just a few of the many risks that dolphins face as a result of human activity. Oil spills, red tides, stray plastic bags blocking digestive systems, entrapment by discarded fishing gear, and disorientation by underwater sounds are all threats to these marine creatures. Diverse types of contaminants have different effects on dolphins, whales, and other marine species.

Pesticides, nitrogen-based fertilizers and heavy metal contaminants all pose a direct danger to these creatures. These pollutants often find their way into water systems via runoff after heavy rain. In addition to directly killing amphibians, these pollutants can also weaken their immune system (as may have been the case in the extinction of the Monteverde golden toad) and cause physical deformities or abnormalities.

Lead continues to enter the food chain through the widespread use of lead hunting ammunition and fishing equipment, harming wildlife and posing a health risk to humans. The Get the Lead Out initiative, launched by the Center, aims to replace lead-based ammunition and fishing equipment with safer, non-toxic alternatives. Each year, lead-based paint at government facilities on Midway Atoll kills up to 10,000 Laysan albatross eggs, putting the

endangered Laysan duck in jeopardy. Pollution of the environment by agricultural chemicals and industrial waste disposal has a negative impact on the reproduction of exposed birds. Pollutants like lead have a wide range of physiological impacts, including direct effects on breeding adults as well as developmental effects on embryos. Through methods of hormonal mimicking of estrogens, the consequences on embryos include mortality or decreased hatchability, the inability of chicks to flourish (wasting syndrome), and teratological effects such as skeletal deformities and poor differentiation of the reproductive and neurological systems. Acute death, sublethal stress, lower fertility, suppression of egg development, eggshell thinning, poor incubation and chick-rearing behaviour are all examples of chemical impacts on adult birds (Fry 1995). Organochlorine pesticides and industrial pollutants, organophosphate pesticides, petroleum hydrocarbons, and heavy metals include mercury (Hg), cadmium (Cd), arsenic (As), chromium (Cr), thallium (Tl), and lead (Pb), and, in a smaller number of instances, herbicides and fungicides have all been linked to reproductive consequences. Environmental estrogens impacting gull populations mating in polluted "hot spots" in southern California, the Great Lakes, and Puget Sound have been identified as o,p'-DDT, polychlorinated biphenyls (PCBs), and combinations of organochlorines. Because the avian reproductive system is estrogen-dependent, estrogenic organochlorines are an important class of toxicants for birds.

Mercury, once released into the atmosphere, settles on the Earth and accumulates in our rivers and soils, where it is converted to methylmercury, a very poisonous form that accumulates in the tissues of wildlife and humans. It's a very toxic neurotoxin that affects the function and development of the central nervous system in both humans and animals. Mercury poisoning is especially dangerous for pregnant and lactating women, as well as infants because mercury is most damaging during development. Scientists have discovered worrying amounts of mercury deposition in a variety of wildlife species, posing a threat to reproductive and brain health. Fish have a hard time schooling and have a lower chance of spawning. Birds lay fewer eggs and have trouble caring for their chicks. Mammals have impaired motor skills that affect their ability to hunt and find food. In addition, some evidence indicates elevated mercury levels can adversely affect species' immune systems. All these effects combine to create a severe threat to wildlife survival. Two-thirds of the lethal mercury in our waters is due to pollution from metal manufacture, pulp industries, waste processing, and coal generation. Mercury itself is naturally occurring, but the amounts in the environment have been on the rise from industrialization. The metal can make its way into soil and water, and eventually to animals like fish. The most prevalent cause of this sort of poisoning is eating mercury-containing foods. Mercury poisoning is particularly dangerous to children and fetuses. Mercury poisoning is toxicity caused by mercury ingestion. Mercury is a hazardous metal that may be found in many forms in the environment. Consuming too much methylmercury or organic mercury, which is connected to eating seafood, is the most prevalent cause of mercury poisoning. Methylmercury (organic mercury) poisoning is commonly associated with the consumption of seafood, particularly fish. Toxicity from fish is caused by two factors: eating mercury-containing fish and eating too much fish. The water in which fish dwell absorbs mercury. Mercury may be found in almost all varieties of fish. Because they feed on other mercury-containing fish, larger fish can accumulate greater levels of mercury. The most prevalent of them are sharks and swordfish. High mercury levels can also be seen in bigeye tuna, marlin, and king mackerel. With each step up the food chain, the concentration of mercury rises. As a result, mercury levels in large predator fish like walleye and trout can be a million times higher than in the surrounding water. As a result, people and wildlife who consume high-mercury fish or other species are at danger of major health problems.

Mercury is found in small levels in ordinary foods and items, and it is unlikely to harm your health. However, too much mercury might be harmful. It's also vital to look for new strategies to avoid mercury poisoning and raise awareness about the problem. Mercury is most commonly consumed by humans through contaminated seafood, with tuna accounting for one-third of all mercury consumption (Albers *et al*.2007). The Food and Drug Administration will enforce stronger requirements to safeguard women, children, and all people who eat fish from mercury contamination in seafood. The usage of mercury in the past created a hazardous legacy that can be revived utilising contemporary mining techniques.

Suction dredge mining in streams not only harms delicate, fragile, and endangered fish and frogs directly, but also has the potential to contaminate rivers with toxic mercury plumes leftover from the Gold Rush. Suction dredge mining was declared unlawful through lawsuits and legislation. Air pollution from coal-fired power plants, trash

from coal-mining tailings, and coal ash slurry, in addition to the direct physical implications of coal mining, have severe negative consequences. These dangers are addressed in order to reduce coal's detrimental effects. Mercury, selenium, CO_2, nitrogen dioxide, sulphur dioxide, and methane, among other toxic coal combustion byproducts, contaminate the environment, worsen climate change, and contribute to smog and acid rain, to mention a few.

Wildlife is valuable as a source of food, clothing materials, and other needs and frills. There is also a strong desire to see animals in their natural environment. Ecotourism is a burgeoning sector, particularly in countries with rare or unusual animals or magnificent ecosystems. Sport and trophy hunting relies on wildlife as well. The government's responsibility is to protect wildlife from overhunting and to safeguard the environment that wildlife needs to feed and reproduce; this role is implemented by a number of laws and regulations. To preserve animals from human disruptions, additional legislation and enforcement mechanisms may be implemented.

Wildlife can be an indirect target for commercial operations that could occur in more remote areas and such attacks could have an immense impact on wildlife including fish and other aquatic organisms. The release of toxic water into the headwaters of large river systems was reported to have a devastating impact on wildlife (Cunningham, 2005). One can only imagine the public and political reaction that would have occurred if such an incident was proven to be an act of terrorism. Fish and other aquatic organisms can be impacted by runoff water from firefighting. This water could contain toxic substances and these substances could have negative impacts on wildlife. Run-off waters from burnt over lands draining into waterways also are high in toxic substances.

Many poisonous compounds are only dangerous indirectly, through toxication. "Wood alcohol," or methanol, is an example of a non-toxic substance that is chemically transformed into hazardous formaldehyde and formic acid in the liver. Many pharmacological molecules become toxic in the liver, and the genetic variability of key liver enzymes causes the toxicity of many chemicals to vary significantly between people. Radiation poisoning can occur as a result of exposure to radioactive chemicals, which is a separate occurrence. Theobromine poisoning in dogs and cats and mushroom poisoning in humans are two prevalent causes of acute natural poisoning. Even though dogs and cats are not natural herbivores, a chemical defense generated by Theobroma cacao can be lethal. Because many omnivores, including humans, readily ingest edible fungus, many fungi have evolved to be inedible, non this instance as a direct defensive mechanism. Persistent Organic Pollutants (POPs) are manmade harmful substances, such as PCBs, DDT, and dioxins, that are easily dispersed by wind or water and can last a long time in the environment. They can build up in the tissues of plants, animals, and people, and can be passed down the food chain from one species to the next. Chemicals get more concentrated as they ascend up the food chain (a process known as "biomagnification"). Raptor populations, such as the bald eagle, plummeted in the 1950s and 1960s as a result of DDT, which thinned the shells of their eggs, preventing them from producing offspring.

ILLICIT AND RESTRICTED SUBSTANCES

Concerns have been raised about the usage of illegal chemicals/pesticides for nefarious purposes (Whitlow *et al.*, 2005). Because of their severe toxicity, the use of several drugs has been restricted throughout time, and in some circumstances, they have even been deregistered. Because residual levels in corpses can kill animals that feed on them, these substances are highly harmful to wildlife.

Diclofenac is a medicine that reduces swelling (inflammation) and pain. It's used to treat aches and pains, as well as problems with joints, muscles and bones. These include rheumatoid arthritis and osteoarthritis sprains and strains in muscles and ligaments back pain toothache migraine gout ankylosing spondylitis – this causes inflammation of the spine and other parts of the body, Vultures were prevalent until the 1980s, and they are now struggling for survival. Currently, seven African species and eight Indian species are on the verge of extinction. White-backed Vulture, Long-billed Vulture, and Slender-billed Vulture populations in India have declined by 99 percent. The populations of the Red-headed and Egyptian Vultures have likewise plummeted by 91% and 80%, respectively. The usage of diclofenac, a non-steroidal anti-inflammatory medicine (NSAID), in veterinary treatment throughout the 1990s has been blamed for this precipitous fall. When vultures feast on the carcass of an animal that has been treated with diclofenac 72 hours before death, they are exposed to diclofenac (Abprez *et al.* 2021). The drug is extremely toxic to vultures and impacts their kidneys and they die of visceral gout.

Despite the fact that the drug's veterinary usage was banned in 2006, human dosages were utilised to treat animals in the nation. Over the years, the incidence of diclofenac and other harmful medicines has been constantly tracked. Despite the fact that diclofenac is no longer widely utilised in India, it is nevertheless abused in some areas. Vulture populations are vulnerable because they are too tiny to recover fast and will not survive another catastrophic occurrence. This susceptibility is exacerbated by the fact that vultures are sluggish breeders, depositing just one egg each year and remaining immature for longer after fledging. Without adverse circumstances, the remnant population will only double in 10-15 years. Other NSAIDs, including aceclofenac, ketoprofen, nimesulide, and others, are toxic to vultures but are nevertheless accessible in India for veterinary use (Kanaujia *et al.* 2011) .

The government plans to establish eight new captive-breeding centres (eight are already operational) in the revised National Vulture Conservation Action Plan (2020-2025), but without a check on the toxicity of these non-steroidal anti-inflammatory drugs (NSAID) and their use (misuse of diclofenac), releasing captive-bred populations into the wild will not be considered feasible.

Furthermore, they die from NSAID poisoning rather than retaliatory poisoning, electrocution, or starvation. As a result, the government and other stakeholders must ensure that the environment is free of vulture-toxic medications. Veterinary treatment includes the misuse of diclofenac. The use of aceclofenac (which metabolises into diclofenac and is considerably more harmful to vultures), and another medicine called nimesulide (which has been discovered to be poisonous to vultures). The increase in neglected carcasses has a direct impact on human health. The corpse can serve as a breeding ground for a variety of pathogens, increasing the risk of direct and indirect infections including anthrax and rabies. Pathogens and vectors connected with putrefying carrion enhance the risk of diseases affecting animals, such as brucellosis and tuberculosis. Unattended rotting carrion or carcasses pollute the environment (air, soil, and water), potentially increasing the risk of anthrax and other water-borne infections in people. In addition, when vulture populations drop, alternative scavengers such as feral dogs, other canine species, and rodents become more prevalent. This condition raises the risk of zoonotic illnesses like rabies and bubonic plague spreading. In India, a significant increase in dog populations was seen in 1997, with an estimated population of 29 million dogs. Human rabies is common in India, with dog bites accounting for almost 95 percent of human rabies mortality. (Markandya et al., 2008)

Dichlorodiphenyltrichloroethane, commonly known as DDT, crystalline chemical compound, an organochloride. Originally developed as an insecticide, The classic persistent organo- halide compound is DDT. which was widely used as a very effective insecticide during the mid-1900s. DDT is an excellent example of a chemical with substantial ecotoxicological consequences. It has few acute toxicological effects and is safe to use near people and other animals. Unfortunately, it persists across food chains, accumulating at high levels in top-of-the-food-chain birds of prey, leading them to lay thin-shelled eggs that shatter before hatching. Before DDT was outlawed for broad usage, eagles, ospreys, hawks, and other species were nearly wiped off. Several related organohalides used as pesticides, such as dieldrin and aldrin, have also been linked to cancer. These were first widely utilised in the 1950s when they were sprayed on fields to combat insect pests. Unfortunately, it was discovered after a few years that the number of birds of prey was fast falling, and thousands of seed-eating birds were dying. The previously abundant sparrow hawk has become an uncommon bird in Britain by the 1960s. The seed-eating birds that the sparrow hawk hunted were carrying toxic pesticides in their bodies. As a result, the chemical sprays poisoned countless sparrow hawks and other birds of prey indirectly. Many birds laid thin-shelled eggs that cracked readily as a result of the organo-chlorines. Chemicals were found to be entering food chains and being stored in the fat of the animals' bodies, according to research. Snails that have eaten a sprayed cabbage plant, for example, may be devoured by thrush. The thrush may not have consumed enough poison to be killed, but a sparrowhawk consuming numerous thrushes consumes enough poison to be killed. As the chemicals move up the food chain, they become more concentrated. Despite the fact that organo-chlorines had been phased out of use by 1976, most persons still carry modest levels of these compounds in their fatty tissues. However, they are still utilised in some Third World countries. The sparrowhawk population had recovered considerably by the 1980s, and it is now a common bird.

Tetramethylenedisulfotetramine (TETS, DSTA, also called tetramine) is a toxic organic chemical. Its chief application is as an effective rodenticide (rat poison). Tetramethylenedisulfotetramine (TETS) is a highly toxic convulsant and a potent antagonist of γ-aminobutyric acid (GABA), leading to excitation. This organic compound

was developed as an effective rodenticide, but the high toxicity to mammals led to it being banned in most countries Tetramethylenedisulfotetramine is a new neurotoxin on the market (Barrueto *et al.,* 2003; Whitlow *et al*., 2005). Tetramine is a tasteless odourless white crystalline powder that is radially soluble in water, combines well with feedstuffs, and has no odour. TETS works by binding to the chloride channel on the g-aminobutyric acid receptor in a selective and irreversible manner, disrupting chloride regulation in the neuron. Inhibitory activity in the central nervous system (CNS) is reduced, and clinical symptoms include seizures that begin soon after consumption of TETS. Differentiate TETS toxicity from another pesticide poisoning. The modifications that have been discovered are all nonspecific. The blood of individuals with severe acute TETS poisoning showed elevated levels of bilirubin, aspartate aminotransferase (AST), lactate dehydrogenase (LDH), and creatine kinase (CK). The severity of the poisoning is connected to these numbers. Muscle contraction occurs during convulsions, which causes a rise in CK. Hematuria and proteinuria have both been recorded. In most acute TETS poisoning patients, white blood cells are elevated (Zolkowska (2013).

Tetramethylenedisulfotetramine has a higher potency than sodium monofluoroacetate (SMFA). The oral LD50 of TETS is 0.1 to 0.3 mg/kg of body weight in most animals, with a total fatal dosage of w10 mg for humans. Tetramine levels (0.07 to 0.238 mg/g) were detected in the liver, kidney, heart, and lungs of rabbits given 0.4 mg tetramine/kg body weight and died 1 hour later (Xiang *et al.*, 2001).

Tetramine is eliminated in the urine, which can be exploited for forensic purposes (Zeng *et al.,* 2006). Tetramethylenedisulfotetramine is stable in tissues, therefore it can cause a chain reaction or secondary poisoning. Because TETS is stable in tissues and bodily fluids, it can be poisoned by scavenging animals and birds. The human pathophysiology of TETS has been documented (Zhou *et al.,* 1998). Oedema of the brain, haemorrhages in the brain stem, and cardiac degeneration in the papillary muscles were all observed in poisoned humans. TETS may be detected in human tissues and urine (Xiang *et al.,* 2001; Zeng *et al.,* 2006). It's plausible to assume that identical disease exists in other animals.

Ingestion or, less often, inhalation are two ways to be exposed to sodium monoflouroacetate. After ingesting enough sodium monofluoroacetate to produce poisoning, clinical signs generally appear between 30 minutes to 2.5 hours after exposure (Eason, 2002). They might, however, be delayed for up to 20 hours. Sodium monoflouroacetate poisoning is characterized by metabolic, cardiovascular, and neurologic signs and symptoms. Acute poisoning can cause metabolic acidosis, hypotension, dysrhythmias, seizures, coma, and respiratory depression, among other things. Until the early 1970s, sodium monofluoroacetate was widely used as a toxin (Proudfoot *et al.,* 2006). SMFA and similarly related chemicals are discovered in Dichapetulum spp. plants, in addition to being synthetic. In many countries, the use of SMFA as a poison to kill undesirable animals and birds is prohibited. SMFA is used to regulate imported animal species that disturb and imperil local wildlife in countries with small indigenous mammal populations (Eason, 2002). The usage of SMFA can be restricted to the elimination of problematic predators. In certain areas, for example, SMFA has been confined to SMFA-containing sheep collars. The SMFA is housed within bladders that are ruptured during a predator assault in these applications (Burns and Connolly, 1995).

Fluoroacetamide has toxicity comparable to SMFA (Osweiler et al., 1976). The fluorine atom replaces the hydrogen on the methyl group in fluoroacetamide, a chemical molecule based on acetamide. It was employed as a rodenticide and is a metabolic toxin that affects the citric acid cycle. It has a high-water solubility and is quite volatile. It is very poisonous to animals and has a high bio-concentration potential. It's neurotoxic with the potential to harm reproduction and development. Birds are very poisonous to fluoroacetamide, whereas fish are mildly harmful. The sensory organs are unable to detect sodium monofluoroacetate. It's a white powder that looks like flour or sugar and may be stored for a long time. SMFA can withstand temperatures of up to 200°C. It is water-soluble but ethanol and lipids are insoluble. It can be destroyed by microbes in water. The gastrointestinal tract, respiratory tract, mucous membranes, and wounds all absorb compound 1080. Different exposure methods have no discernible influence on toxicity. The blockade of the Krebs cycle is SMFA's mode of action. The creation of fluorocitrate, which is known as fatal synthesis, is essential for metabolic activation. Fluoroacetate is transformed to fluoroacetyl-CoA, which is subsequently turned to fluorocitrate by the enzyme citrate synthase. Fluorocitrate inhibits aconitase, which catalyses the conversion of citrate to isocitrate. The Krebs cycle is stopped when aconitase is inactive. Fluorocitrate also inhibits citrate transport across the mitochondrial membrane. The heart and the central nervous

system are both targets for SMFA. Scavengers, opportunists, and carnivores are all at risk when animals are poisoned by SMFA. Aconitase is inhibited by fluoroacetate and its poisonous metabolite fluorocitrate. Both chemicals are preferentially taken up by glial cells in brain tissue, resulting in suppression of the glial TCA cycle. However, it's vital to remember that the glia-specificity of these drugs is dependent on both the dosage and the model employed. Fluorocitrate's glia-inhibitory action in vivo, established by intracerebral microinjection, is reversible within 24 hours. Fluoroacetate systemic treatment needs a fatal dosage to significantly block the glial TCA cycle. When the glial aconitase enzyme is inhibited, citrate accumulates and glutamine production is reduced. While the former is likely to be responsible for the main toxic effect of these compounds, possibly through chelation of free calcium ions, the latter has gotten the most attention in the study of glial-neuronal interactions, as glutamine is an important precursor for transmitters glutamate and GABA.

The consequences of SMFA poisoning on the CNS and heart cause clinical symptoms (Osweiler *et al.,* 1976; Proudfoot *et al.,* 2006). Herbivores may have greater heart symptoms. Arrhythmias, tachycardia, ineffective pumping (weak pulse), and death occur in this order when the heart fibrillates. Ataxia, collapse, and a brief period of agonal striving are some of the clinical indications that have been documented (Robinson, 1979). The discovery of a dead animal with the appearance of rapid collapse and scant evidence of agonal struggle is typical of field observations. Hyperesthesia, aimless roaming, frantic running, vocalisation, incoordination, emesis, opisthotonus, unconsciousness, and death are common neurological indications in carnivores. Ingestion of SMFA causes death within 1 to 24 hours. Birds either fall from the sky and die immediately after impacting the ground, or they display nervous system indications and lose their capacity to fly. Animals poisoned by SMFA are devoured by other vertebrates, a phenomenon known as relay poisoning. Acute cardiac failure and hypoxia are pathological findings in SMFA poisoning patients. Intoxication with SMFA might lead to hypocalcemia (Proudfoot *et al.,* 2006). Gas chromatography can be used to test tissues for SMFA (Okuno *et al.,* 1984).

Cyanide is a rapidly acting, potentially deadly chemical that can exist in various forms. Cyanide can be a colourless gas, such as hydrogen cyanide (HCN) or cyanogen chloride (CNCl), or a crystal form such as sodium cyanide (NaCN) or potassium cyanide (KCN). Cyanide is a chemical compound that contains the group C≡N. This group, known as the cyano group, consists of a carbon atom triple-bonded to a nitrogen atom. In inorganic cyanides, the cyanide group is present as the anion CN^-. Sodium cyanide and potassium cyanide are exceedingly deadly soluble salts. Many cyanides are extremely poisonous. The cyanide anion inhibits the enzyme cytochrome c oxidase (also known as aa3), which is situated in the inner membrane of eukaryotic cells' mitochondria and is part of the electron transport chain's fourth complex. It binds to the iron present in this protein. The cyanide binding to this enzyme limits electron transfer from cytochrome c to oxygen. The electron transport pathway is interrupted as a result, and the cell can no longer create ATP for energy aerobically. The central nervous system and the heart, which rely heavily on aerobic respiration, are particularly vulnerable. This is a case of histotoxic hypoxia in action (Nelson *et al.*2000). In New Zealand, cyanide is also used to control pests, particularly possums, an imported mammal that threatens native species and spreads tuberculosis among livestock. Possums can become bait shy, but bait shyness can be reduced by using cyanide pellets. Native species, especially the rare kiwi, have been reported to succumb to cyanide poisoning. The Dama wallaby, another invasive marsupial pest in New Zealand, is likewise controlled with cyanide (Shapiro *et al.* 2011). Ships are fumigated using cyanides as pesticides. The less-lethal poison arsenic is more frequent; however, cyanide salts are utilised for killing and have been employed as rat poison in some regions.

The most hazardous compound is hydrogen cyanide, which is a gas that kills by inhalation. For this reason, an air respirator supplied by an external oxygen source must be worn when working with hydrogen cyanide. Hydrogen cyanide is produced by adding acid to a solution containing a cyanide salt. Alkaline solutions of cyanide are safer to use because they do not evolve hydrogen cyanide gas. Hydrogen cyanide may be produced in the combustion of polyurethanes; for this reason, polyurethanes are not recommended for use in domestic and aircraft furniture. Oral ingestion of a small quantity of solid cyanide or a cyanide solution of as little as 200 mg, or exposure to airborne cyanide of 270 ppm, is sufficient to cause death within minutes. Cyanides are illegally used to capture live fish near coral reefs for the aquarium and seafood markets. (Dzombak *et al.* 2006) The practice is controversial, dangerous,

and damaging but is driven by the lucrative exotic fish market. Poachers in Africa have been known to use cyanide to poison waterholes and to kill elephants for their ivory.

Organic nitriles do not readily release cyanide ions, and so have low toxicities. By contrast, compounds such as trimethylsilyl cyanide (CH3)3SiCN readily release HCN or the cyanide ion upon contact with water Almost every animal species get poisoned by cyanide. Water bait, for example, can include cyanide. Other types of cyanide, such as sodium and potassium cyanide, can also be utilised. If the impoundment is breached, tonnes of cyanide might be released into rivers, lakes, and streams. cyanide-contaminated water impoundments. When gold and silver ores are extracted, cyanide is utilised. Although the cyanide used in the process is recycled, some cyanide escapes and ends up in the tailings pond. These tailing ponds have poisoned bats and other creatures who drink from them. (Barber *et al*. 2003).

A dam containing a tailing pond has been known to be breached by a weather event, spilling tonnes of cyanide into river systems and causing an ecological calamity. Cyanide has been employed to manage undesired species in the past and is hazardous in the ocean. The symptoms of cyanide poisoning in birds have been documented (Wiemeyer *et al.*, 1986). Ataxia, eye blinking, head bending, wing droop, greater ataxia, convulsions with tail fanning, opisthotonos gasping, and death are the clinical indications of cyanide poisoning in vultures. Kestrels, owls, and quail have more rapid development of clinical symptoms. The amount of time between consumption and death is dose-dependent.

A cyanide spill can have long-term effects on a water supply (Lakatos *et al.*, 2003). Run-off water from firefighting and run-off from burned regions can contain cyanide (Barber *et al.*, 2003). Free cyanide sensitivity is high in freshwater fish. Free cyanide sensitivity is high in freshwater fish. At< 20 mg/l, fish can die (Eisler and Wiemeyer, 2004). Water temperature and physical activity affect the fatal amount of cyanide in rainbow trout (McGeachy and Leduc, 1988). The effects of cyanide on the thyroid gland in young fish can potentially be dangerous (Brown *et al.*, 2004). Rainbow trout that are sexually developing generate less viable eggs when exposed to cyanide (Lesniak and Ruby, 1982).

Ricin is a poison found naturally in **(Ricinus communis)** and **(Ricinus communis**) castor beans (Coppock, 2009). Castor beans have no distinct clinical indications of poisoning and might be mistaken for other acute causes of mortality in ducks (Jensen and Allen, 1981). Castor bean-related deaths of Canadian geese have been reported, however, the evidence is mixed. Intoxication from castor beans may have killed 10,000 waterfowl in Texas in 1967 and 2,000 ducks in 1969 and 1970. In Texas in 1971, 1,673 ducks were killed. In one duck's inspection, a castor bean was discovered. Castor beans have been farmed in the region previously.

Despite the lack of clear evidence, this article implicates castor bean intake as a cause of mortality in ducks and geese. Clostridium botulinum toxins and pathogenic microorganisms have not been detected. Haemorrhage entering the gut was shown to be a sign of catarrhal enteritis. Scrapings of the proventriculus wall revealed seed components that resembled Ricinus communis. Hepatocytes showed signs of fatty deterioration. Whole castor beans were force-fed to ducks (Jensen and Allen, 1981). Passage of blood-streaked mucus, leg paresis, loss of motion with wings, sitting, prone recumbency, and death were all indicators of severe intoxication. Castor bean wholes and pieces were discovered during the autopsy, as well as hepatic congestion. Severe fatty degeneration of hepatocytes, granulocytic infiltration into portal regions, pulmonary congestion, and peribronchial haemorrhage was seen on histopathology, while the spleen showed necrosis and haemorrhage. According to these studies, certain ducks swallow castor beans, which can lead to castor bean intoxication. The fact that the duck is the most likely to consume castor beans suggests that castor beans are used against wild ducks.

PESTICIDES

Pesticides kill beneficial insects, soil bacteria and fish along with unwanted pests. Insecticides have little effect on birds or other animals (Schafer *et al.,* 1983). In a wide range of species, pesticide exposure has been related to cancer, endocrine disruption, reproductive impacts, neurotoxicity, kidney and liver damage, birth abnormalities, and developmental alterations. Pesticide exposure can also change an organism's behaviour, making it more difficult for it to survive. Certain herbicides, for example, can impair bird singing skills, making it difficult to attract partners and

reproduce. Hormone-imitating chemicals designated as endocrine disruptors have been linked to several deformations. Frogs with hermaphroditic malformations, pseudo-hermaphrodite polar bears with penis-like stumps, panthers with atrophied testicles, and intersex fish in rivers across the United States are all effects of these substances. Mammalian, avian, reptile, fish, and mollusc reproductive problems have been documented at low levels of exposure. Some pesticides can kill or sicken wildlife after only a few minutes of exposure.

Fish deaths caused by pesticide residues conveyed to ponds, streams, or rivers by surface runoff or spray drift, and bird die-offs induced by feasting on pesticide-treated plants or insects, or by eating pesticide-treated granules, baits, or seeds are examples of acute wildlife poisoning. Poisonings of this nature may usually be proven by examining the tissues of affected animals for the suspected pesticide or looking into the effects on biochemical systems (e.g., cholinesterase levels in the blood and brain tissue). Acute poisoning of animals, in general, occurs over a short period of time, affects a small geographic region, and is tied to a single chemical. Pesticides can also impair a bird's capacity to care for offspring, resulting in the death of its young. Systemic insecticides have sublethal effects on bee movement, feeding behaviour, and navigation, even at "near-infinitesimal" doses. Insecticides might be used against a specific animal population, according to observations from both registered and unlawful usage of insecticides. When pesticides are sprayed according to the registered label, vertebrates might become nontarget species (Poppenga, 2007). Insecticides used illegally or off-label have resulted in the death of vertebrates. Models can predict the number of birds that will die as a result of pesticide treatment in the field (Mineau, 2002). Pesticides that enter water systems through surface water can harm birds, animals, fish, and other aquatic life. Chronic poisoning can come from long-term exposure of wildlife to pesticide levels that aren't instantly fatal. The organochlorine pesticide DDT's (through the metabolite DDE) influence on reproduction in certain birds of prey is the most well-known example of a chronic effect on wildlife. Bird mortality has been linked to prolonged exposure to DDT and other organochlorine pesticides such as dieldrin, endrin, and chlordane. Reduced organochlorine residues in most places have resulted from the reduction of these chemicals in the 1970s and early 1980s, and bald eagle reproduction has substantially improved. Organochlorine insecticides applied in some foreign nations may endanger migratory birds that overwinter in those countries. Pesticides can cause secondary poisoning in animals when an animal eats prey that has pesticide residues. (1) Birds of prey becoming sick after eating an animal that is dead or dying from acute pesticide exposure, and (2) the buildup and transportation of persistent chemicals in wildlife food chains are examples of secondary poisoning.

Pesticides, particularly insecticides, can poison waterways and flood regions, resulting in bird mortality (Elliott *et al.,* 1996). Carbofuran granules found in flooded fields have been linked to duck fatalities as well as mortality in raptors scavenging dead or crippled ducks (Hunt *et al.* 1995). Illegal pesticide usage has also been documented, resulting in the killing of wildlife (Allen *et al.,* 1996). Hormone disruption is a possible side effect of chemical contamination. Male fish get feminised and begin to produce eggs in their testes, which is the most well-known impact. Crustaceans are very poisonous to brominated flame retardants. They've been discovered in sperm whales and, more recently, peregrine falcon eggs. Bisphenol A, which is used to create plastic bottles and a variety of other plastic items, has induced sex reversals in the broad-snouted caiman, a South American alligator. In quail and chicken embryos, it has caused reproductive abnormalities. Hundreds of pet birds are said to be killed each year by Teflon-coated goods' fumes and particles. Certain pesticides have been shown to reduce the variety of aquatic creatures and predatory insects when they are introduced to aquatic habitats.

Conclusion

Pesticide exposure has been proven to cause a 42 per cent decrease in species richness in Europe, even when exposures are at levels regarded ecologically acceptable by existing regulations. Beneficial insects such as bees, spiders, and beetles have a far higher species richness in untreated or organic crops than in insecticide-treated areas. In chemical-dependent agriculture, the use of pesticides is frequent.

In terms of ecological stability and durability, organic pest management stands in stark contrast to a chemical-intensive strategy. This disparity has massive implications for biodiversity and wild species' survival. Diverse land management approaches have different impacts on the web of life; understanding this is critical to maintaining nature's delicate balance and life-sustaining advantages. The most important step in reducing the detrimental effects

of pesticides on animals and maintaining the planet's remaining biodiversity is to use organic pest management rather than chemical-intensive treatments.

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