Cultivating Plants that Poison
Bees, Butterflies, and Birds

Photo by Carol Allaire.

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Executive Summary

In the winter of 2006-07, U.S. beekeepers lost nearly 40 percent of their bee hives, documenting some of the first reports of "abnormally heavy losses" in managed honey bee colonies.[1] Nearly a decade later, losses in managed bee colonies remain elevated, with recent 2014-15 data showing record high losses of 42.1 percent.[2]

The preponderance of independent science links a class of insecticides known as neonicotinoids (or neonics) with the dramatic decline of pollinators and other wildlife. Bees, butterflies, birds, and a range of soil and aquatic organisms essential to healthy ecological systems are imperiled by the use of these systemic and persistent pesticides. Systemic pesticides are chemicals that can be taken up by the vascular system of a plant, and then expressed throughout the plant, including pollen, nectar, and guttation droplets, indiscriminately exposing target pests and non-target organisms alike. While several classes of pesticides since the outset of the chemical-intensive agricultural era are systemic, neonicotinoids have attracted substantial scientific and public scrutiny because their appearance and proliferation in the market coincided with dramatic die-offs and the decline of honey bees around the world.

Over the last 20 years, public and regulatory pressure to limit human exposure to toxic pesticides on farms and in surrounding communities, compounded by issues of insect and weed resistance to these chemicals, has resulted in an increased demand for new pesticides with lower human exposure and toxicity. This has led to the emergence of systemic pesticides. These pesticides can be applied with a variety of methods, including foliar sprays, granules, soil drenches, and tree injections. However, insecticide or fungicide-coated seeds are among the most popular method of treating the target insect or fungal disease, and account for the vast majority of seeds for major crops and ornamental plants in the U.S. (Stokstad, 2013; Douglas and Tooker, 2015; Friends of the Earth, 2013).[3,4,5] With these application methods, and given the toxicity, longevity or persistence of these chemicals in soil and waterways, broad ecological impacts have and continue to be documented (Chagnon et al., 2015).[6]

In addition to the ecological harm caused by neonicotinoid use, the effect of systemic pesticide use on bees and other pollinators has clear economic and human health impacts. Insect pollination has been shown to enhance crop yield, thus contributing to agricultural productivity by up to 71 percent, depending on the crop.[7] The total global economic valuation of pollination services is estimated to be 9.5% of global food production value (2005), or about $190 billion.[8] In the U.S., the value of crops directly reliant on insect pollinators is $15.12 billion (2009), with the value attributed to honey bees alone at $11.68 billion.[9] Pollinator-dependent crops are also a source of nutrients critical to human health, accounting for one-third of the U.S. diet.[10,11] The continuation of pollinator decline, therefore, threatens the stability of the global economy and food supply, along with ecosystems and human health.

The empirical and scientific data on the adverse effects of systemic pesticides document the failure of the U.S. Environmental Protection Agency (EPA) to prevent the introduction and proliferation of substances that are devastating to the stability of healthy ecosystems. While various factors, including parasites, such as varroa mites, and habitat loss, have been identified as contributors to pollinator decline,[12] only now has EPA begun to accept the growing scientific consensus that neonicotinoids are harming bees. As a result of systemic pesticides' environmental impacts and emerging human toxicological profiles, EPA must begin removing these chemicals from the market, rather than embracing them as "reduced risk" in comparison to older toxic pesticides. Organic agriculture, which now supports a nearly $40 billion industry,[13] does not depend on neonicotinoids or other toxic pesticides, demonstrating that it can supply the world's food needs while building soil health and nurturing biodiversity.

This report documents the rise and use of systemic insecticides and the threat that they present to the environment and health, in light of regulatory inertia. The findings make the case that the use of persistent and systemic pesticides is inherently unsustainable at a time when protection of the biosphere is crucial.

Systemic Herbicides

Insecticides are not the only type of pesticides that can behave systemically. Some of the most widely-used herbicides on the market, such as glyphosate, are also able to translocate throughout a plant. Glyphosate is shown to translocate well in a plant, from roots to foliar tissues, and can be exuded by roots into soil.[14] Glyphosate application to glyphosate-tolerant cotton is associated with poor pollen deposition on the stigma of male plants, as well as inhibition of pollen maturation, but has the potential to translocate into developing pollen grains.[15] Residues of glyphosate and its metabolite AMPA have been detected in the grains of glyphosate-resistant soybeans.[16]

The Rise of Systemic Insecticides

Outside the context of complex ecological systems and impact associated with non-target effects, systemic insecticides might otherwise present an attractive technology for reaching target pests by conferring long-lasting toxicity throughout the plant. While applicator, farmer, and farmworker exposure may be reduced, the creation of other sources of exposure have been documented, including that of fugitive dust contaminated with seed coatings, lubricants that escape off of seed planters, and drift from planted fields.[17]

EPA defines systemic pesticides as pesticides that "can be moved (translocated) from the site of application to another site within the plant or animal where they become effective."[18] Systemic behavior is exhibited in different types of pesticides, such as insecticides, herbicides, and fungicides. However, the exact behavior of these chemicals, their relationship and impact to target and non-target organisms, and the implications of their use can differ significantly.

Washing Produce from Pesticides –Implications for the Concerned Consumer

Traditionally, consumers have been taught to carefully wash or peel their produce to remove traces of pesticide residues. However, the concept of washing or peeling away surface residues of pesticides does not apply to systemic pesticides. In fact, systemic insecticides, having been translocated within the plant, can exist within the fruit and leaves of treated crops, unable to be washed off.[19,20,21]

Systemic insecticides were first formally synthesized in the 1940s and 1950s. The benefits of these chemicals, documented in studies and patent documents from this period, include their distribution throughout plant tissues, increased persistence after initial application,[22,23] and the ability to confer insecticidal properties to new plant growth and hard-to-treat areas (such as the underside of the leaf) subsequent to application. Earlier generations of insecticides, including organophosphate (OP) compounds such as schradan, demeton, dimethoate, demeton-S-methyl, mevinphos, and phorate, exhibit systemic action.[24,25] Carbamates with systemic properties, such as aldicarb and carbofuran, were developed in the 1960s, followed by systemic insect growth regulators in the 1980s and 1990s.[26] Many of these older systemic insecticides are no longer available commercially as a result of voluntary cancellations by manufacturers

What Makes Pesticides Systemic?

A log octanol/water partition coefficient (K_{OW}) is a measure of likely systemic translocation within the vascular system of the plant,[31] with a value of two or less indicating a higher ability to translocate. Log K_{OW} may also demonstrate the extent to which a chemical translocates into the pollen of plants, with lower values correlated with an increased likelihood.[32] The table below illustrates the relationship between log K_{OW} values and water solubility with a handful of systemic and non-systemic chemicals.

Insecticide	Chemical Class	Solubility in Water (mg/L)	Log K_{OW}
Non-systemic			
Chlorpyrifos	Organophosphate	1.05 (low)	4.7
Malathion	Organophosphate	148 (moderate)	2.75
Carbaryl	Carbamate	9.1 (low)	2.36
Permethrin	Pyrethroid	0.2 (low)	6.1
Cyfluthrin	Pyrethroid	0.0066 (low)	6
Systemic			
Aldicarb	Carbamate	4,930 (high)	1.15
Acephate	Organophosphate	790,000 (high)	-0.85
Dimethoate	Organophosphate	39,800 (high)	0.704
Oxamyl	Carbamate	148,100 (high)	-0.44
Carbofuran	Carbamate	322 (moderate)	1.8
Imidacloprid	Neonicotinoid	610 (high)	0.57
Thiamethoxam	Neonicotinoid	4,100 (high)	-0.13
Fipronil	Phenylpyrazole	3.78 (low)	3.75
Chlorantraniliprole	Anthranilic Diamide	0.88 (low)	2.86
Sulfoxaflor	Sulfoximine	568 (high)	0.802
Flupyradifurone	Butenolide	3,200 (high)	1.2
Herbicide	**Chemical Class**	**Solubility in Water (mg/L)**	**Log K_{OW}**
Systemic			
Glyphosate*	Phosphanoglycine	10,500 (high)	-3.2

(Source: University of Hertfordshire, Pesticides Properties Database)
**See box on previous page.*

Benefits of Neonicotinoids –Fact or Fiction?

Although neonicotinoids are marketed as highly beneficial and advantageous, there has been a growing body of evidence that renders these claims suspect. Arguments in favor of these chemicals assert that they provide continuous protection from insect pests to the plant throughout the majority of the growing season, without the need for repeat applications. However, a 2014 EPA report offers evidence that neonicotinoid use in soybeans does not translate to better yields, with the finding that soybean seeds coated with the neonicotinoids imidacloprid, thiamethoxam, and clothianidin "provide negligible overall benefits in [yield] in most situations" when compared with no insect control treatment. Furthermore, neonicotinoid seed coatings are only bioactive for a period within the first 3 to 4 weeks of planting, which EPA states "does not overlap with typical periods of activity for some target pests of concern."[33] Thiamethoxam use is found to reduce predaceous beetles that feed on pest slugs by 60 percent, resulting in a decrease in soybean density (19%) and yield (5%) (Douglas, Rohr, and Tooker, 2015).[34] Similarly, there is evidence that foliar and subterranean herbivores and key pests of sunflowers are unaffected by seeds coated with thiamethoxam, and there is no significant difference in sunflower yield between treated and untreated fields.[35] A 2015 study finds that the use of aldicarb soil insecticide and thiamethoxam-coated seed does not reduce cutworm damage, and that, instead, plots treated with these insecticides had a higher percentage of defects when compared to untreated plots (Difonzo et al.).[36] Other studies, including a 2014 Center for Food Safety review of the literature on the subject, finds that evidence is weighted toward lack of efficacy and agricultural yield benefits.[37] Additionally, according to the UK's Agriculture and Horticulture Development Board (AHDB) Final Harvest Summary for 2015, which provides the first harvest results of crops planted without neonicotinoid treatments, yields of all cereals were 7 to 14 percent above the ten-year average.[38] These strong numbers bolster the position that the use of neonicotinoids is unnecessary.

A Historical Note

Organophosphates (OPs) were originally introduced into commerce as a safer alternative to organochlorine pesticides, such as DDT, chlordane, heptachlor, aldrin, and dieldrin, with the claim that they would breakdown in the environment quickly, rather than bioaccumulate. The OPs immediately proved to be disastrous to honey bees (see *History of Bee Kills*, p22), and some of the early thinking was disproved as the aggregate effect of dietary and non-dietary exposure raised safety questions.[39,40]

and/or because of adverse human health effects and environmental contamination. Some, including the insecticides dimethoate and aldicarb, are still registered, but are used under restricted or limited circumstances.[27,28] From the 1990s onward, insecticides, like fipronil and neonicotinoids, slowly began replacing older systemic pesticides that were shown to be more toxic to humans.[29,30] In the last several years, other systemic insecticides have been registered by EPA. These include chlorantraniliprole (from the chemical class known as anthranilic diamides), sulfoxaflor (sulfoxamine), and flupyradifurone (butenolide).

Neonicotinoid pesticides are often presented as needing fewer repeat applications due to their persistence, reducing exposure to those working on the farm, and having a range of versatile application techniques, such as seed coatings, tree injections, and soil drenching.[41,42] Additionally, many newer systemics like neonicotinoids are accepted to be less toxic to mammals,[43] although new evidence continues to emerge that disputes this (see box p16). At the same time, the efficacy of these chemicals has been called into question by numerous studies, including a 2014 report by EPA (see box, *Benefits of Neonicotinoids –Fact or Fiction?* on p13).

How do they work?

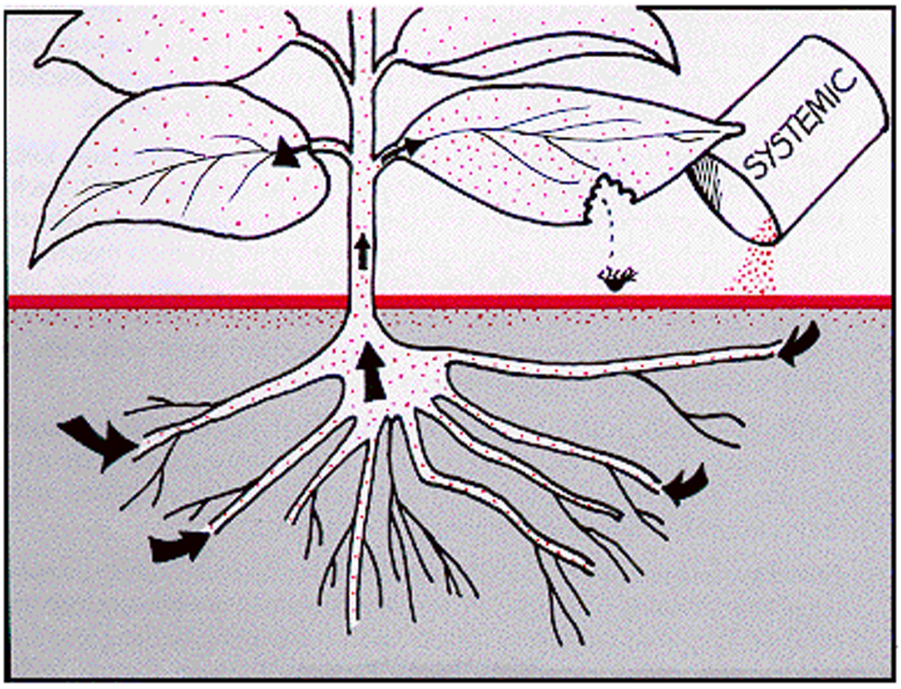
Systemic insecticides generally work via a sequence of two processes: absorption and translocation. Absorption of an insecticide across cell membranes can occur in seeds, roots, or leaves –generally all external surfaces of the plant are able to absorb some material depending upon certain conditions.[44] Systemic distribution of the chemical throughout the plant structure can be achieved following foliar application, root uptake from soil application, seed coating, trunk injections, drip irrigation, and other application methods, which can significantly influence the uptake, behavior, and persistence within a plant.[45] Once absorbed from the plant surface, the systemic chemical typically travels upward through the plant's vascular system via the xylem to various parts of the plant where it can be expressed in newly growing shoots.[46] During foliar applications, the chemical penetrates into the leaf where it is expressed throughout due to translaminar activity, including the underside of the leaf. Additives in pesticide formulations help to increase the flow of the substance into the plant.[47,48]

Following absorption, translocation of the pesticide substance can occur through the connecting vascular tissues of the plant: the xylem and phloem. The translocation process varies according to the chemical and the site of absorption.[49] Systemic pesticides typically move through the xylem, which transports water, but they can also, less frequently, be transported within the phloem, which conducts carbohydrates.[50] As with absorption, highly water-soluble chemicals are the most likely to be taken up by plant roots and translocated to shoots.[51,52]

Systemic Chemicals of Concern

Organophosphates (OP) and Carbamates

The older systemic insecticides include some organophosphorus (OPs) compounds (e.g. dimethoate) and carbamates (e.g. aldicarb and oxamyl) and were first registered in the 1950s. OPs and carbamates inactivate the acetylcholinesterase enzyme (AChE), commonly found in nervous tissues, brain cells, red blood cells, and muscle tissues, which results in the continuous stimulation of muscle or nerve fibers. Because AChE is present in most animals, these compounds are toxic to many different types of organisms.[53] Many of these systemic chemicals, including mevinphos,[54] dimethoate,[55] carbofuran,[56] aldicarb,[57] and oxamyl,[58] continue to have wide food and/



The behavior of soil applied systemic pesticides when taken up plant roots. Image by Cornell University, 2012.

or non-food (i.e. structural) uses, or are slowly being voluntarily cancelled by manufacturers.

Neonicotinoids

Chemically similar to nicotine, neonicotinoids are nicotinic acetylcholine (nACh) receptor agonists that activate neuronal receptors and disrupt many sensory and cognitive processes in invertebrate organisms. The binding of neonicotinoids to the nicotinic acetylcholine receptor is irreversible in arthropods.[59,60] Thus, they are highly toxic to insects and other invertebrates.[61,62]

In the early 1990s, imidacloprid was launched by Bayer CropScience and soon became one of the most popular insecticides in the world. By 2008, imidacloprid became the world's largest-selling insecticide, second only to glyphosate (Roundup) in all pesticide sales.[63] There are now seven neonicotinoid compounds that are commercially available worldwide: imidacloprid, thiacloprid, clothianidin, thiamethoxam, acetamiprid, nitenpyram, and dinotefuran. Neonicotinoids have been registered in more than 120 countries around the world as of 2011[64] and now dominate the market with 27 percent of insecticide sales, nearly as much as organophosphates, carbamates, and pyrethroids combined (31%).[65]

Fipronil

Fipronil, belongs to a chemical class known as phenylpyrazoles and inhibits the γ-aminobutyric acid (GABA)-gated chloride channel, exhibiting selective neurotoxicity in insects compared to mammals.[66] Fipronil is registered for use in the U.S. to control a variety of insects, both indoor and outdoor, as well as for in-furrow treatment, seed treatment, soil injection, baits, and for spot treatment on pets. Citing concerns about ecological risks, EPA converted the major remaining outdoor use products to a restricted use classification, which takes it off retail store shelves, but allows commercial applicators and farmers to continue to apply it.[67] Although EPA states that fipronil is non-systemic, there is evidence that the chemical undergoes root uptake and transport within plants (Aajoud, A., et al., 2006).[68] Fipronil and its metabolites have also been detected in the pollen of plants.[69] Fipronil is relatively mobile in soils, degrades to form persistent metabolites, and is a pervasive water contaminant.[70,71] It has been found to be toxic to non-target organisms, including honey bees[72] and various aquatic species.[73]

New and Emerging Systemic Insecticides

Over the past decade, newer classes of systemic insecticides have emerged. Many of these emerging chemicals are a response to a demand for novel modes of action due to increased pest resistance to pesticides already on the market.

Anthranilic Diamides

Anthranilic diamides make up a new class of insecticides that activate the ryanodine receptor, which is responsible for regulating muscle contraction. Their insecticidal mode of action targets insect over mammalian receptors, making them less hazardous to mammals.[74,75] Diamides now account for a little over eight percent of total global insecticide sales, a number that has been gradu-

The Growth of Seed Coatings– Implications and Consequences for Pollinators

In chemical-intensive agriculture, many consider seed coatings to be a "beneficial" treatment that reduces loss of seeds and seedlings from early season pests,[79] provides targeted applications with the potential to reduce pesticide delivery rates, decreases applicator exposure,[80] and minimizes exposure to non-target organisms in comparison to foliar application.[81] Since the onset of the use of seeds coated with systemic pesticides, the market has more than tripled in size between 1990 and 2005, with neonicotinoids representing 77 percent of the total share for coated seeds.[82] More recent estimates show that from 2000 to 2012, virtually all neonicotinoids applied to corn, soybeans, and wheat crops were applied as seed coatings. These same estimates show that neonicotinoid use increased dramatically between 2003 and 2011 with a marked shift toward large-scale, preemptive insecticide use via coated seeds. Seed coatings were used on 34-44 percent of soybean acres and 79-100 percent of corn acres in 2011, contradicting claims that insecticides are used on fewer corn acres today than a decade or more ago.[83] Systemic fungicide seed treatments, which, like their insecticide counterparts, are taken up by the growing plant and can thus persist for longer periods of time, have also grown in popularity over the past 20 to 40 years.[84]

The growth in the use of seeds coated with systemic chemicals is problematic for pollinators. For instance, guttation droplets, a source of water for bees, from corn plants grown from neonicotinoid-coated seeds exhibit high levels of thiamethoxam and clothianidin, up to 100 mg/L, and imidacloprid, up to 200 mg/L, levels near or even higher than those commonly applied in field sprays.[85] Contamination of the surrounding soil from chemicals not taken up by the coated seed is a concern, as more than 90 percent of the active ingredient from a neonicotinoid-coated seed can contaminate the soil after application.[86] One study shows that only a small proportion of the systemic insecticide, between 1.8 to 6.8 percent, is taken up by the plant itself (El-Hamady, Kubiak, and Derbalah, 2008).[87] Coated seeds also lead to exposures to non-target organisms as a result of drift from abraded seed coatings and fugitive dust from contaminated lubricants used in mechanical planters. These residues have been shown to result in deposits on the soils of planted and unplanted fields, on nearby plants visited by foraging bees (indicating uptake, deposition, or both), and in pollen collected by honey bees and stored in the hive.[88]

The Newest Systemics –Reduced Risk?

The new systemic pesticides that EPA has registered over the last decade, those that have been found to be highly toxic to bees, are nonetheless listed by the agency under the misleading category of "reduced risk pesticides," because of their relatively low acute toxicity to mammalian species. In addition to many of the neonicotinoids, this category includes the newest chemicals cyantraniliprole and flupyradifurone. However, they, too, are highly toxic to non-target organisms, and their long-term impacts on terrestrial and aquatic ecosystems are not fully understood.[89,90]

The Food Quality Protection Act (FQPA) requires EPA to expedite the registration of "reduced risk" pesticides, defined as those that "may reasonably be expected to accomplish one or more of the following: (i) reduce the risks of pesticides to human health; (ii) reduce the risks of pesticides to nontarget organisms; (iii) reduce the potential for contamination of groundwater, surface water, or other valued environmental resources; or (iv) broaden the adoption of integrated pest management [IPM] strategies, or make such strategies more available or more effective."[91]

Since FQPA requires that a "reduced risk" pesticide must meet at minimum one of the above criteria, and because neonicotinoids and other newer systemics may be less toxic to humans than organophosphate insecticides, they meet the criteria for "reduced risk," in spite of higher ecological risks. Because of the prophylactic (applied in advance of a pest problem) nature of systemic pesticide use –the chemical is essentially an inoculant that becomes an inherent part of the seed or seedling– they are not compatible with an integrated pest management (IPM) system that relies on monitoring and thresholds for economic damage before the application of a pesticide, but may be considered consistent with "IPM" seen as pesticide chemical rotation.

While companies are not allowed to put a reduced risk claim on their pesticide labels, EPA "believes that companies use the conventional reduced risk pesticide status to their marketing advantage," with additional advantages possible at the state level (e.g. New York State waives reporting requirements for reduced risk uses).[92] Reduced-risk products can be recommended for IPM programs on the label. For example, the label for Acelepryn, active ingredient chlorantraniliprole, states that the product is "recommended for IPM programs on turf and landscape ornamentals because it does not directly impact natural arthropod predator and parasitoid populations including ladybird beetles, lacewings, minute pirate bugs and predatory mites."[93]

"Reduced risk" pesticides should not be confused with "minimum risk" pesticides, which EPA has determined pose little to no risk to human health or the environment and are exempt from registration under the *Federal Insecticide, Fungicide and Rodenticide Act* (FIFRA).[94] While minimum risk pesticides provide a real least-toxic alternative, the term "reduced risk pesticide" does not.

ally increasing and will likely continue to grow with the potential addition of new diamides to the market.[76] Chlorantraniliprole, an anthranilic diamide insecticide, was registered in 2008 by DuPont for agricultural and non-agricultural uses. Chlorantraniliprole is characterized as persistent and mobile in terrestrial and aquatic environments, and is expected to accumulate in soil from year to year.[77] Its cousin, cyantraniliprole, was registered in 2014, and like chlorantraniliprole, impairs regulation of muscle contraction, causing paralysis and eventual death of the insect. Cyantraniliprole is classified by EPA as a "reduced risk" pesticide, despite data gaps, and is highly toxic to honey bees.[78]

Sulfoximines

Designated under a new class of insecticides known as sulfoximines, sulfoxaflor is a systemic insecticide that, like neonicotinoids, acts on nACh receptors. Sulfoxaflor was first registered by EPA in 2013. Dow AgroSciences, sulfoxaflor's manufacturer, put forth the claim that sulfoxaflor is distinct from neonicotinoids, and as such is the first chemical belonging to a class of insecticides known as sulfoximines. Industry scientists at Dow AgroSciences claim that sulfoxaflor's very high efficacy at nACh receptors, coupled with its chemical structure, lack of cross-resistance, and metabolic stability, prove that it is a novel insecticide.[95,96] However, other industry scientists argue that sulfoxaflor has a pharmacological profile consistent with that of imidacloprid, suggesting that the chemical is in fact a neonicotinoid.[97] As of now, EPA classifies sulfoxaflor as a sulfoximine.[98] Sulfoxaflor has relatively low toxicity to mammals,[99] and lacks cross-resistance between it and other neonicotinoids,[100] but concerns have been raised regarding its high toxicity to honey bees and other non-target beneficial organisms.[101] The U.S. Court of Appeals in September 2015 concluded that EPA violated federal law and its own regulations when it approved sulfoxaflor under an unconditional registration without reliable studies regarding the impact that the insecticide could have on honey bee colonies.[102]

Butenolides

The recently registered systemic insecticide flupyradifurone was approved for use in 2015, and is classified as a "butenolide" insecticide. Like neonicotinoids, flupyradifurone targets the nACh receptor, but differs from other chemicals with a similar mode of action, given how it binds to the receptor and the extent to which it is metabolized.[103,104] These differences present another attempt to address growing resistance to similar pesticides. Flupyradifurone is considered an alternative insecticide to certain pyrethroids,

neonicotinoids, organophosphates, and avermectins. EPA classifies flupyradifurone as a "reduced risk" pesticide based on its relatively more favorable human health toxicity profile, but this pesticide is also highly toxic to honey bees.[105]

Environmental Fate and Contamination

Soil

Insecticide residues in soil can come from a variety of different routes, such as sprays or dusts from application onto foliage, or insecticides applied directly to the soil.[106] Seed coatings constitute a major source of soil contamination. Typically, more than 90 percent of the active ingredient from a neonicotinoid-coated seed ends up in the soil after application,[107] while a small proportion of the systemic insecticide, between 1.8 to 6.8 percent, is taken up by the plant.[108] In certain soil drench treatments, pesticides can directly leach below the root zone of the treated plants.[109] For granular formulations, it is estimated that 1 to 15 percent of the pesticide remains on the soil surface, which is a source of exposure for birds and other terrestrial organisms and could runoff into surface water.[110] While the breakdown of pesticides in the soil can occur through microbial degradation, these pesticides may also adversely affect these communities, depleting the beneficial organisms needed to sustain a healthy soil ecosystem.

The level of persistence of pesticide residues in soil typically depends on factors such as soil type, formulation, concentration, moisture, temperature, and pH, so half-lives of various systemic insecticides may vary significantly with respect to these factors.[111] Neonicotinoids, for instance, have half-lives in soil that vary greatly among compounds, soil type, and across studies (see table, right), with reported half-lives ranging from 1 to nearly 7,000 days.[112] Fipronil remains in treated soil for at least 30 months, and the chemical leaches into non-treated areas directly below treated soil, both at levels toxic enough to kill termites.[113] Chlorantraniliprole has soil half-lives varying from 8 days to 16 days, with residues persisting up to nearly 60 days,[114,115] and is characterized by EPA as persistent (half-life greater than 60 days) and mobile in terrestrial environments, with extended use expected to cause accumulation of residues in soil from year to year.[116,117] Cyantraniliprole also has half-lives ranging from 2 days to 3 months in terrestrial environments, with some degradates being more persistent and more mobile than the parent compound.[118] Flupyradifurone is characterized as being persistent to very persistent with half-lives greater than 60 days and 180 days in soil, sediment, and even water.[119] Sulfoxaflor's breakdown products (which, along with the breakdown products of other systemic insecticides, can be more toxic and persistent than the parent compound), are expected to be highly persistent in aerobic soil systems.[120]

Estimated Half-lives of Systemic Insecticides in Various Soils

Chemical	DT_{50} (half-life)* in Days
Aldicarb	2.4-35
Carbofuran	12.8-47
Chlorantraniliprole	6.5-597
Clothianidin	17.2-6,932
Cyantraniliprole	9-92
Dimethoate	2.6-16
Fipronil	68-217
Flupyradifurone	37.5-401.0
Imidacloprid	28.7-187
Oxamyl	3.19-43.31
Sulfoxaflor	2.2-6.03
Thiacloprid	1.3-19.1

**Refers to time it takes for pesticide to degrade or dissipate to half of original value*

Systemic insecticides can be highly mobile in certain soils (with high water content) due to their high solubility in water. Twenty percent of systemic insecticides are prone to leaching, while 45 percent are mobile in wet soils.[121]

Water

The presence of pesticides in water is hazardous for aquatic and terrestrial organisms, and can contaminate drinking water. According to a 2006 U.S. Geological Survey (USGS) report, the most frequently detected systemic insecticide carbofuran was found at levels at or above 0.1 µg/L in both streams and groundwater, often exceeding EPA's aquatic-life safety benchmarks, along with methomyl and phorate.[122] Another USGS report found that fipronil was detected in streams from 17 to 63 percent of the time from 2002-2011 and frequently found at levels of potential concern for aquatic organisms in urban streams.[123] In 2015, USGS also found neonicotinoids to contaminate half of all U.S. streams,[124] while another found the chemicals to persist in Midwest waterways from applications from previous years.[125] Similarly, widespread use of neonicotinoids has resulted in their worldwide contamination of aquatic media. A 2015 review examining the concentrations of neonicotinoids in surface waters around the world found that water-borne neonicotinoid occurrences are frequent and long-term, often exceeding several existing water quality guidelines. Eighty-one percent and 74 percent of global surface water studies reporting maximum and average individual neonicotinoid concentrations, respectively, exceed recommended ecological thresholds for neonicotinoids in water.[126] Newer systemic insecticides, with their high mobility, also have a potential to contaminate water. Flupyradifurone, for example, is characterized as moderately mobile and thus has a potential to reach aquatic environments, including surface and groundwater for several months after application.[127]

The detection of high concentrations of these chemicals in groundwater and surface water is becoming more widespread.

Given their toxicity to aquatic organisms, monitoring the levels of these chemicals in water bodies becomes increasingly important to determine the environmental risk they present.

Effects on Non-Target Organisms

Exposure of systemic insecticides to non-target organisms can occur via contaminated soil, water, and air, and by multiple exposure routes. For instance, exposure to pesticide drift from foliar spraying or from contaminated dust plumes from treated seed can kill or impair many species.[128,129] Potential exposure from systemic pesticides applied to soil includes runoff/erosion of soil residues onto surface water and/or from root uptake into target and non-target plants from residues in the soil to pollen, nectar, and other exuded substances.[130] Seed-eating vertebrates, like birds, are exposed to lethal doses if they consume coated seeds[131] or granular pesticides on the soil surface.[132,133,134] Organisms that drink or live in contaminated waters, or soil dwellers like earthworms are all at risk from exposure. For organisms like bees, poisoned pollen and nectar can lead to long-term sublethal impacts on individual bees and the colony. These exposures can increase the organisms' vulnerability to disease and parasites.[135,136]

Impacts on Managed and Wild Bees

There are many routes of exposure by which bees are exposed to systemic pesticides, including contaminated pollen and nectar that they forage and ingest, as well as guttation droplets they drink from plants. EPA's preliminary imidacloprid pollinator risk assessment, released in January 2016, confirms harmful residues of imidacloprid in crops where pollinators forage.[137] A Harvard study by Chensheng Lu et al. (2015), found that 73 percent of pollen and 72 percent of honey samples contained at least one detectable neonicotinoid. Forty-nine percent, 20 percent, and four percent of pollen samples contained one, two, and three neonicotinoids respectively,[138] while 57 percent and 15 percent of honey samples contained one and two neonicotinoids respectively. According to EPA's *Pollinator Risk Assessment Guidance*, avenues of potential exposure to bees and other pollinators from the foliar application of systemic pesticides include direct deposition on bees, deposition on plants and soil (after which residues of the chemical contaminate plant surfaces, soil, pollen, and nectar), and deposition onto surface water. A 2012 study by Krupke et al. finds that bee-attractive plants, like dandelions, that are adjacent to treated fields can also be contaminated by pesticide drift and thus expose bees. Contaminated field dust, which results from the sowing of treated seeds, is another source of exposure that puts bees at risk.[139] Soil dwelling/burrowing bees, like the mining bee, are also at risk from soil contamination. Bees given a choice between dimethoate-treated and untreated syrup displayed no aversion or preference to either, suggesting that not only are levels of dimethoate found in nectar toxic, but they are also not repellent.[140] Similarly, honey bees cannot taste neonicotinoids and are not repelled by them, and instead prefer food containing neonicotinoids even though the pesticides caused them to eat less food overall.[141]

Photo by Cameron Crane, 2014.

Several acute and chronic impacts on bees have been attributed to exposure to systemic insecticides found in the pollen and nectar of treated plants. Dimethoate residues are found in the pollen of alfalfa plants at levels toxic to honey bees as much as two weeks after initial treatment, with residues increasing from 0.5 parts per million (ppm) in pollen one day after application, to 3 ppm in nectar one week later and 1 ppm in nectar two weeks later. Bees exposed to these levels displayed inhibited cholinesterase activity and reduced survival (Barker, Lehner, and Kunzmann, 1980).[142]

Toxic effects resulting from exposure to very low levels of systemic insecticides are extensive. Systemics, like fipronil, have been demonstrated to impair the olfactory learning and sucrose perception of honey bees, even at very low levels (0.5 ng/bee and 1 ng/bee, respectively).[143] Continuous exposure to a sub-lethal dose of thiamethoxam was found to damage organs used during the metabolism and detoxification of the insecticide.[144]

Photo by Anneliese Markle.

Ecological Risk Assessment

Like its health risk assessments, EPA's ecological risk assessments suffer from a framework that ignores the precautionary principle. As part of the pesticide registration process, EPA performs an ecological assessment that "evaluates the likelihood that exposure to one or more pesticides may cause harmful ecological effects"[148] based on data submitted on the potential hazards to non-target fish and wildlife species. The effects can be direct or indirect, short-term (acute) or long-term (chronic), as shown by tests on organisms that broadly represent non-target organisms. EPA "may require testing for effects in insect pollinators, such as honey bees, when the typical end-use product (TEP) is intended for outdoor use and honey bees may be exposed to the pesticide."[149] The environmental fate of the pesticide –the interaction with soils, air, sunlight, surface water, and ground water– is also studied.[150]

EPA's characterization of ecological risk is based on a comparison (Risk Quotient, or RQ) of the expected environmental concentration (EEC) of the pesticide and certain threshold concentrations, which vary for different types of risk. For example, the Risk Quotient for dietary exposures are:

- Acute dietary RQ = EEC/LC_{50}
- Chronic dietary RQ = EEC/NOEC

The LC_{50} is the median lethal dose –the dose that kills 50% of the test animals, and the NOEC is the no observed effect concentration– the highest level that did not produce an effect in test animals. Unless threatened or endangered species are known to be exposed, the LC_{50} and NOEC used are those of the most sensitive animal tested. The RQ is compared to a level of concern (LOC) for the particular type of risk. The predicted level of the pesticide in the environment would reach the "level of concern" for acute impacts on birds, for example, when the estimated environmental concentration is half the median lethal concentration.

EPA's ecological assessments for pesticides have been critiqued and challenged in court by many stakeholder and citizen groups, especially with regard to endangered species. The courts have ruled that EPA's ecological assessments failed to adequately protect critical species like salmon and steelhead,[151] the California red-legged frog,[152] and most recently, honey bees.[153]

A 2013 National Academy of Science (NAS) report, *Assessing Risks to Endangered and Threatened Species from Pesticides,* identified deficiencies in EPA's ecological assessments and outlined recommendations to improve assessments and consultations with other federal agencies.[154] Since then, EPA has worked with other federal agencies (e.g. U.S. Fish and Wildlife Service) to develop shared scientific approaches that reflect the advice provided by NAS.

NAS identified important issues that affect EPA's ability to produce a scientifically defensible assessment of risk to plants and animals, but the critique is based on the risk assessment paradigm of the National Research Council (NRC) report, *Risk Assessment in the Federal Government: Managing the Process*.[155] This paradigm, the basis of all of EPA's risk assessments, is anti-precautionary, built on the premise that releasing toxic chemicals into the environment is acceptable unless shown to present a high level of risk. The biodiversity and climate of the planet are in critical condition, and a new framework for decision making is needed –one based on the precautionary principle and the premise that human action should heal the planet, not merely slow its demise.

The best decision-making framework that exists in U.S. law today is the one embedded in the *Organic Foods Production Act* (OFPA). OFPA and organic regulations require that organic farmers build and protect habitat for organisms that provide natural support for the agroecosystem. If such biodiversity conservation measures fail to provide an environment in which crops can thrive, then organic producers may use materials that (1) do not harm humans, other species, or the environment; (2) are necessary; and (3) are compatible with organic and sustainable production. This kind of precautionary approach should be adopted by EPA in deciding what risks are "unreasonable."

The scientific literature on honey bee and pollinator decline indicates that current required eco-toxicology reviews do not adequately evaluate, as necessary, complex biological communities in nature. Rather, the requirements allow the registration of materials that have

Researchers in the UK found low, field-realistic exposure (0.7 ppb in sugar water and 6 ppb in pollen) to imidacloprid resulted in a reduction in bumblebees' ability to gather food by 57 percent (Feltham, Park, and Goulson, 2014).[145] Exposure to imidacloprid has led to both acute and chronically impaired pollen foraging performance, resulting in effects that include a decrease in pollen foraging efficiency and a change in flower preference and/or impairment in ability to find flowers in exposed bumblebees.[146] A 2013 study by Smagghe et al. reports that at levels representing 1/100 of the concentration recommended for use in the field, chronic exposure to chlorantraniliprole-contaminated pollen causes bumble bees and their offspring to become lethargic. The same study also showed severe sub-lethal effects on reproduction in bumblebee colonies exposed via the same route.[147]

Impacts on Other Beneficial Insects and Microorganisms

While bee declines have been the most visible of non-target impacts, other beneficial insects such as ladybugs, beetles, and predators of agricultural pests are also negatively affected by the use of systemic insecticides. The loss of these and other non-target organisms pose serious concern for ecosystem diversity and natural control of other pests. *Macrolophus pygmaeus*, a predatory mirid from the Mediterranean used to control whiteflies, thrips, aphids, mites, and eggs and larvae of other pests, was found to undergo 100 percent mortality when exposed to thiacloprid, while chlorantraniliprole exposure resulted in a decrease in plant feeding.[156] The beneficial predatory insect *Orius insidiosus* (insidious flower bug) experienced reduced egg viability, delayed oviposition (egg laying), reduced nymph and female survival, and delayed development when exposed to sunflowers grown from seed treated with thiamethoxam and chlorantraniliprole.[157] The predatory mite *Neoseiulus fallacis* is susceptible to several systemic insecticides, including clothianidin and the now-cancelled spirotetramat. Exposure to these two systemics caused 61.4 and 40.2 percent mortality of adult *N. fallacis*, as well as significantly reduced fecundity.[158] *Microplitis croceipes*, a wasp parasitoid of caterpillar pests, was found to have impaired foraging ability after feeding on extrafloral nectar from cotton treated with various systemic insecticides. Nectar from imidacloprid-treated cotton reduced the flight response to host-associated odors of the parasitoid for four days, acephate for six days, and aldicarb for as long as 18 days after application. Wasps that fed on nectar from plants treated with all three of these insecticides were also found to have reduced longevity (Stapel, Cortesero, and Lewis, 2000).[159] Secondary poisonings of other non-target organisms also occur. Slugs exposed to thiamethoxam in fields, while unaffected by the chemical, poisoned the predaceous beetles that fed on them, resulting in a 60 percent population reduction in the beetles. The loss of predatory beetles contributed to a reduction in soybean densities by 19 percent and yield by five percent.[160] Exposure to systemic insecticides may also adversely affect soil organisms. Fallen leaves from neonicotinoid-treated trees were found to result in reductions in feeding rates by insects and earthworms, inhibition of aquatic and terrestrial microbial decomposition activity, and a decrease in leaf decomposition.[161,162]

Harmful to Humans and Mammals, Too?

Due to their mode of action, many of the newest systemic insecticides, including neonicotinoids, are touted as being less toxic or non-toxic to humans and mammals. However, evidence is emerging that this may not be the case. A study by Japanese researchers (2012) found that acetamiprid, imidacloprid, and nicotine exert similar effects at mammalian nACh receptors, suggesting that excitation or desensitization or both of nACh receptors by neonicotinoids affect the developing human and mammalian nervous system, as is known to occur with nicotine.[163] These findings were cited when, in 2013, the European Food Safety Authority (EFSA) proposed that guidance levels for acceptable exposure to the two neonicotinoids be lowered, while further research is being carried out to provide more reliable data on developmental neurotoxicity.[164]

EPA identifies sulfoxaflor as having "Suggestive Evidence of Carcinogenic Potential" based on the preputial gland tumor response seen in rats. According to regulatory documents, sulfoxaflor targets the nervous system and liver, resulting in developmental toxicity, liver toxicity, and other effects. Developmental and offspring toxicity, in the form of skeletal abnormalities and neonatal deaths, were seen in rat studies. Liver toxicity expressed as changes in liver weight and enzymes, hypertrophy, proliferation, and tumors in subchronic and chronic studies have also been observed. Other effects include a decrease in food consumption and body weight, as well as changes in the male reproductive system.[165] Preliminary studies on cyantraniliprole describe changes in the liver, thyroid gland, and renal cortex, and altered thyroid functioning in mammals. Dogs are found to be more sensitive to cyantraniliprole than both rats and mice.[166]

Impacts on Aquatic Organisms

Systemic insecticides present an especially large threat to aquatic systems.[167] Older systemics like dimethoate are highly toxic to juvenile carp, with exposure resulting in adverse effects that include erratic swimming, increased surfacing, reduced agility, and the inability to maintain normal posture and balance with increasing exposure time, as well as a decrease in oxygen consumption.[168] Methomyl is also toxic to fish and aquatic invertebrates, and can significantly decrease the number of molts, total neonates, malformed neonates, fertility and sex ratio index in aquatic invertebrates like *Daphnia obtusa*.[170] There is evidence that carbofuran adversely affects reproduction in freshwater fish, reducing the number of eggs as well as hatching percentage (Adhikari et al., 2008).[171]

A study investigating fipronil and three of its environmental metabolites found very high toxicity to crayfish (Schlenk et al., 2001).[172] Aquatic insects in waters containing fallen leaves from trees that were treated with imidacloprid exhibited reduced feeding rates.[173] While chlorantraniliprole has been found in some studies to have relatively low toxicity to certain fish,[174] other studies have found the chemical to be highly toxic to various types of aquatic organisms on both an acute and chronic level.[175] Amphibians, which have been facing an existential threat with staggering declines in populations across species over the past few decades,[176,177] have also been shown to be sensitive to systemic insecticide exposure. Researchers in Argentina found that imidacloprid exposure to the Montevideo tree frog tadpoles resulted in acute effects, such as increased genetic damage (Pérez-Iglesias et al., 2014).[178] Sub-lethal exposure to spirotetramat was found to result in oxidative stress and peroxidation in tadpoles of the Chinese toad (*Bufo bufo gargarizans*).[179]

Red Munia Amandava amandava. Photo by Dr. Raju Kasambe, 2011.

Population level effects, such as a reduction in biodiversity, have also been documented. A study in Japan found that zooplankton, bottom-dwelling, and water surface aquatic communities in imidacloprid-treated rice fields had a significantly reduced abundance of species. There are also differences in abundance of swimming organisms in both imidacloprid- and fipronil-treated paddies, as well as an impairment of the growth of Japanese rice fish, in both adults and their offspring (Hayasaka et al., 2012).[180] Another study found that locations where high concentrations of imidacloprid are detected have less abundant communities of aquatic macroinvertebrates (Van Dijk, Van Staalduinen, and Van der Sluijs, 2013).[181]

Impacts on Birds

Ingestion of treated seeds or granular pesticides present a source of exposure for some species of birds. A report by the American Bird Conservancy (2013) found that neonicotinoids are lethal to birds, as well as the aquatic systems on which they depend for food. The report also found that a single corn kernel coated with a neonicotinoid can kill a songbird, and even a tiny grain of wheat or canola treated with imidacloprid can poison a bird. Furthermore, as little as 1/10 of a corn seed per day during egg-laying season can affect reproductive endpoints, such as fertility and embryonic development, with any of the registered neonicotinoids.[182] A separate study found oral exposure of male quails to clothianidin adversely affects the reproduction of the birds through fragmentation of DNA in

Botanical Systemic Insecticides

Some naturally-occurring or naturally-derived insecticides also have systemic action. Azadirachtin, an active ingredient derived from the oil found in neem tree seeds, is one such example. Studies have indicated the ability of this chemical to translocate through treated plants, with a relatively short half-life ranging from 5.1 to 12.3 days.[187] Evidence has shown that azadirachtin repels bumblebees, although exposure to the chemical can also result in adverse reproductive effects (i.e. inhibited egg laying and decrease in ovarian length).[188] Thus, more research is needed to determine whether, and under which circumstances, azadirachtin could be used in a way that does not threaten non-target organisms and ecosystems.

Other biologically-based chemicals that induce systemic responses include elicitors, which are chemicals from various sources that induce plant defenses. They include biologically generated volatile organic compounds (VOCs) released by plants as well as materials like laminarin, salicylic acid, and chitosan that may be applied to plants.[198] The toxicological and ecological impacts of these materials have not been adequately studied.

History of Bee Kills – Temporary Fixes for a Persistent Problem

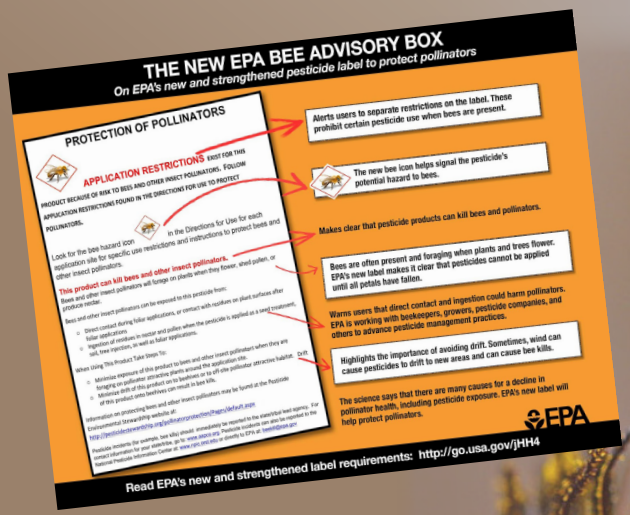
As long as there has been pesticide-intensive agriculture, beekeepers have been faced with bee die-offs associated with exposure to toxic chemicals, highlighting a persistent flaw in the U.S. approach to pesticide regulation. In order to compensate beekeepers for these type of burdens, the government has historically created indemnification programs. Indemnification programs have served to compensate those harmed by regulatory action[190] –in this case, allowing the use of pesticides more toxic to bees following the cancellation of DDT. The Beekeeper Indemnity Payment Program was enacted by Congress in the *Agricultural Act of 1970* after commercial beekeeping operations in California and Arizona were virtually destroyed due to a spray program to control pink bollworm on cotton. A 1976 USDA document, *Report on the Beekeeper Indemnity Program*, stated that the "Beekeeper Indemnity Program has kept pollination costs low by indirectly subsidizing crop producers through direct payments to beekeepers providing pollination services," in face of large-scale use of artificial fertilizers and pesticides.[191] The report concluded:

> *Unless Federal and State governments act to regulate and caution applicators of toxic pesticides, colony damage will continue to be a major problem for beekeepers. However, most government officials emphasize that farmers and spray applicators are already confronted with enough regulations… the current development of stronger and longer-lasting pesticides…is creating an environment entirely unsuitable for honey bees in many parts of the U.S. These areas will find it harder to maintain the present level of bee population regardless of an Indemnity Program or higher honey and pollination prices.*[192]

The risks of earlier pesticides to bees and other pollinators through systemic action were never a focus of pesticide regulation. Instead, EPA (and before that, USDA) used label instructions to try to minimize direct contact of domestic honey bees with insecticides. Methyl parathion, an organophosphate insecticide highly toxic to birds, mammals, aquatic organisms, as well as bees and other beneficial insects, was first registered for agricultural use in 1954.[193] The change from the spray form of the chemical to an encapsulated form known as Penncap-M was blamed for the loss of several thousand colonies, as bees were shown to carry the capsules to the hive along with pollen.[194] Although beekeepers were aware of the highly toxic nature of methyl parathion, they were not prepared for the lengthened period over which the encapsulated chemical would kill bees.[195] Penncap-M was classified as a restricted use pesticide (RUP) due to residual effects on avian species and bees, and in 2010 all methyl parathion product registration uses were cancelled.[196]

EPA uses language on pesticide product labeling in an attempt to reduce exposure of toxic chemicals to bees and other organisms. Penncap-M contained specific language warning about the dangers of this product to bees, stating that the "product is highly toxic to bees exposed to direct treatment or residues on blooming crops or weeds. Do not apply this product or allow it to drift to blooming crops or weeds if bees are visiting the treatment area," and outlined various prohibitions to use based on crop types and times when bees are foraging.[197] However, label language does little to prevent exposure to chemicals that are persistent in the environment, and is limited in its application to the domesticated honey bee. It is also often difficult to determine whether or not an applicator is in violation of a label. Recent EPA label changes for neonicotinoids include wording such as "Do not apply this product while bees are foraging. Do not apply this product until flowering is complete and all petals have fallen… ."[198] This language has similar limitations and does not address issues such as persistence or the use of neonicotinoids as seed coatings.

Advances in pesticide technology have often done more disproportionate harm to beekeepers and the bees they manage. Indemnification programs have propped up the hazardous chemicals, eventually taken off the market, and changes to labeling do little to protect the health of bees. More importantly, as essential as commercial beekeeping is to agriculture, the ecosystem services provided by wild pollinators, predators, parasites, and soil organisms are of equally important and cannot be addressed by compensation or avoidance through labeling.



germ cells and the inhibition or delay of embryonic development (Tokumoto et al., 2013).[183] Ingestion of imidacloprid-treated seeds by red-legged partridges at the recommended application rate was found to result in 100 percent mortality, while dosage at 20 percent of the recommended application rate resulted in adverse effects including reduced nutrient levels in plasma, as well as reduced clutch size, delayed egg-laying, and depressed T-cell immune response in chicks.[184] Researchers in the Netherlands (2014) found that local insect-eating bird population numbers were inversely associated with higher surface-water concentrations of imidacloprid. Bird populations declined by 3.5 percent on average annually in areas where imidacloprid concentrations of more than 20 ng/L were found (Hallmann et al., 2014).[185] Low dose exposure of imidacloprid in the diet of Red Munia (*Amandava amandava*) was found to bring about changes in thyroid functioning.[186]

New Generation Pesticides and the Shortcomings of Pesticide Regulation

The current approach to assessing the risks posed by systemic pesticides and others is designed to accept harm and uncertainty. Risk assessment calculations under the *Federal Insecticide, Fungicide, and Rodenticide Act* (FIFRA) –the law that regulates pesticide registration and use– overlook many real world scenarios, leading to an underestimation of hazards posed to human health and the environment. Furthermore, pesticide registration decisions employ risk/benefit analyses that assume certain market or economic "benefits" without determining whether the products are in fact necessary, or whether safer alternative are available. EPA issues determinations on the reasonableness of risk, FIFRA's legal standard, or assessments of acceptable risk without data on the availability of less or non-toxic alternative practices or materials.

Neonicotinoids and their impacts to pollinators and the wider environment exemplify the problems with the current risk assessment process that does not fully account for the sublethal and indirect effects of pesticides and impacts on ecological communities. New advances in pesticide technologies and application methods expose the inadequacies of the regulatory system.

In 2014, the White House issued a Presidential Memorandum directing federal agencies to create a federal task force to come up with recommendations to "reverse pollinator losses and help restore populations to healthy levels." The memorandum recognized the severe losses in the populations of the nation's pollinators, including honey bees, wild bees, monarch butterflies, and others.[199] The final report, issued a year later, identifies key recommendations to protect pollinator populations, but falls short of necessary regulatory action, including the cancellation and suspension of bee-killing systemic pesticides. Concerned groups, environmentalists and beekeepers believe that without the suspension of neonicotinoid and other systemic insecticide use, the expansion of pollinator habitat recommended by the federal plan will remain a source of pesticide exposure and poisoning.

Coated Seeds and the Treated Article Exemption

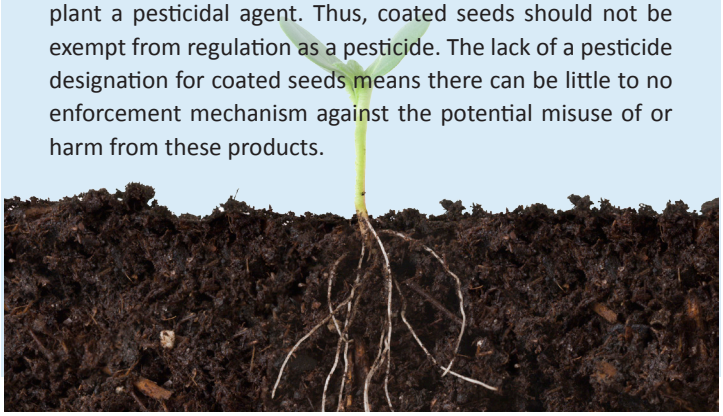
Seeds of corn, soybean, canola, and others are typically coated with pesticides before they are planted. Pesticide manufacturers believe the applied pesticides (insecticides, fungicides, etc.) protect the seed before and after germination from soil pests. Despite being coated with pesticides, these seeds are not regulated under FIFRA or any other federal law. As such, there is no federal product labelling or federal/state enforcement and oversight around the cultivation or subsequent environmental contamination from their use. This is because, the seeds are categorized as treated articles and fall under the treated article exemption "loophole."

According to 40 C.F.R. § 152.25, the exemption states that a treated article is:

> *[...an] article or substance treated with, or containing, a pesticide to protect the article or substance itself (for example, paint treated with a pesticide to protect the paint coating, or wood products treated to protect the wood against insect or fungus infestation), if the pesticide is registered for such use.*[200]

The interpretation of this exemption when it comes to seeds is subject to challenge.

Pesticides that are systemic and used as seed coatings translocate throughout the plant, essentially making the entire plant a pesticidal agent. Thus, coated seeds should not be exempt from regulation as a pesticide. The lack of a pesticide designation for coated seeds means there can be little to no enforcement mechanism against the potential misuse of or harm from these products.



EPA's Steps to Address the Pollinator Decline Are Insufficient

EPA has proposed product label amendments, a moratorium on new neonicotinoid pesticide products, and restrictions on foliar applications of bee-toxic pesticides during bloom when managed bees are on-site and under contract. For all other situations, states have been tasked with creating their own pollinator protection plans that rely heavily on notification requirements and best management practices for farmers, placing continued responsibility on beekeepers. The federal government's emphasis on creating "physical and temporal space" between the use of pesticides and the presence of pollinators does little to address the chronic, sublethal threat of systemic, neonicotinoid pesticides.[201]

"By their very nature, chemical controls are self-defeating, for they have been devised and applied without taking into account the complex biological systems against which they have been blindly hurled. The chemicals may have been pretested against a few individual species, but not against living communities . . . To assume that we must resign ourselves to turning our waterways into rivers of death is to follow the counsel of despair and defeatism. We must make wider use of alternative methods that are now known, and we must devote our ingenuity and resources to developing others."
–Rachel Carson, Silent Spring, 1962

New systemic insecticides that are highly toxic to bees continue to be registered. The latest, flupyradifurone, was registered in 2015, much to the dismay of beekeepers and concerned groups. We argue that these new systemics will compound the already dire pollinator crisis. Label amendments, which apply to all bee-toxic pesticides, focus only on foliar applications, but do not cover seed treatments or other applications, making no attempt to address the systemic nature of these chemicals. Environmental groups, beekeepers, and others have taken action challenging EPA's continued approval of systemic pesticides, including lawsuits filed against the registrations of sulfoxaflor, cyantraniliprole and some neonicotinoids.

U.S. Fish and Wildlife Phases Out Neonicotinoids

Distinguishing itself from EPA, the U.S. Fish and Wildlife Service (FWS) announced in the spring of 2014 that it will ban neonicotinoid insecticides from all wildlife refuges nationwide by January 2016,[202] making it the first federal agency to adopt policy stating that the prophylactic use of toxic materials with broad spectrum impacts is inconsistent with IPM policy.[203] The White House Council on Environmental Quality (CEQ) also announced new guidelines for federal agencies to incorporate pollinator-friendly practices at federal facilities and on federal lands, including recommendations to avoid treating plants with systemic insecticides.[204]

Conclusion

Systemic pesticides present an increased intensification of the threat to biodiversity that has not been observed since catastrophic wildlife declines associated with DDT. The pesticide treadmill –the continuous push to find new chemistry when, inevitably, chemicals in use face resistance from target pests– brings with it trade-offs and impacts that are unacceptable at a period in history when toxic chemicals are not needed in productive, profitable, and cost-effective organic production systems. Coupled with a piecemeal response to problems –label warnings or cancellation of particular "problem" pesticides– it ratchets up the threat to biodiversity and ecological systems. The perceived advancements in the protection of those who handle systemic insecticides cannot be traded for wreaking havoc with critical ecosystems that provides the foundation for all life. The very characteristics that enable the unsupported economic benefits of systemic pesticides –high solubility in water and persistence– are traits that make them a devastating threat to the ecosystem. A precautionary approach is needed when dealing with substances whose ecological fate and impact are not fully understood. The reign of systemic pesticides must end –if only to save pollinators from extinction. With these special organisms as a bellwether for change, it is time to take a hard look at how the systems we use to grow food and manage pests creates a pesticide dependency that only serves to endanger vital ecosystems. The biodiversity and climate of the planet are in critical condition, and a new framework that embraces and builds on certified organic systems for decision making is needed –one based on the precautionary principle and the premise that human action should heal the planet, not merely slow its demise.

Beyond Pesticides Staff joins other environmental groups to rally at the White House to demand that the federal government ban neonicotinoid pesticides.

International Efforts to Protect Pollinators

International efforts to protect pollinators from highly toxic systemics pesticides have had more success restricting neonicotinoids. In 2013, the European Union (EU) took critical steps to protect pollinators from the hazards associated with their use. Despite attempts by agrichemical corporations to delay or reverse the decision, the two-year (still continuing), continent-wide ban on bee-harming pesticides (clothianidin, thiamethoxam, and imidacloprid) went into effect. The ban came several months after the European Food Safety Authority (EFSA) released a report identifying "high acute risk" to honey bees from uses of certain neonicotinoid chemicals.[206] EFSA is continuing to update its assessment of the risks to bees posed by the three neonicotinoid chemicals, which will take a look into their use as seed treatments and granules. The assessment is slated for completion by January 2017.[207]

A fully cited version of this is available at bit.ly/pesticidesandyou.

Nichelle Harriott, Terry Shistar, PhD, and Jay Feldman contributed to this piece.

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