The trouble with neonicotinoids

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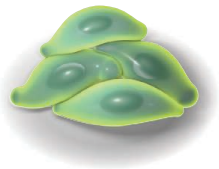

PERSPECTIVES

ENVIRONMENTAL SCIENCE

The trouble with neonicotinoids

Chronic exposure to widely used insecticides kills bees and many other invertebrates

By **Francisco Sánchez-Bayo**

Four decades ago, DDT and other pesticides that cause environmental harm were banned. Since then, newly developed pesticides have had to conform to stricter environmental standards. Yet, recent studies highlight the subtle but deadly impacts of neonicotinoids—the most widely used insecticides in the world—on ecosystems (*1*–*3*). In contrast to other insecticides, neonicotinoids are systemic, meaning that they are highly soluble and thus absorbed by the plant. They produce delayed mortality in arthropods after chronic exposure to sublethal doses but are not very toxic to vertebrates. It has taken more than a decade to unravel some of the mechanisms through which neonicotinoids affect the integrity of ecosystems. Although gaps in knowledge remain, there is a strong case for stricter regulation of these pesticides.

Neonicotinoids are mainly applied as granules into the soil or as seed-dressings during crop planting. Seeds are coated with 1 to 17 mg per kg, depending on crops and compounds. As plants grow, they take up 2 to 20% of the insecticide and distribute it to all parts of the plant, including leaves, flowers, pollen, and nectar. The resulting concentrations of 5 to 10 μg per liter [parts per billion (ppb)] in the sap are sufficient to control sucking and chewing insect pests (see the figure). However, pollinators such as bees, butterflies, moths, and hoverflies are equally exposed; where neonicotinoids are used, 11 to 24% of pollen and 17 to 65% of nectar is contaminated with these insecticides (*3*).

Soon after the neonicotinoid imidacloprid was introduced in France in 1994, beekeepers noticed that their honey bee colonies were weakening or disappearing. The ensuing investigation found that this and another systemic insecticide (fipronil) were

Hidden killers. Neonicotinoids applied to seeds or soils spread into the environment, killing many nontarget anthropods.

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particularly toxic to bees, with acute dietary LD_{50}'s (dose to kill 50% of bees) of 2.5 to 5 ng per bee (*4*). Forager bees do not die immediately after visiting flowers in treated crops because residue levels are below their acute LD_{50} and bees only ingest part of what they collect; the rest is taken to the hive. It is the daily sublethal doses the pollinators ingest that are the problem. Effects include olfactory learning, memory, and locomotory impairment and inhibited feeding (*5*). In a laboratory study, chronic ingestion of 4 to 8 ppb imidacloprid resulted in 50% survival of honey bee workers after 30 days (*6*).

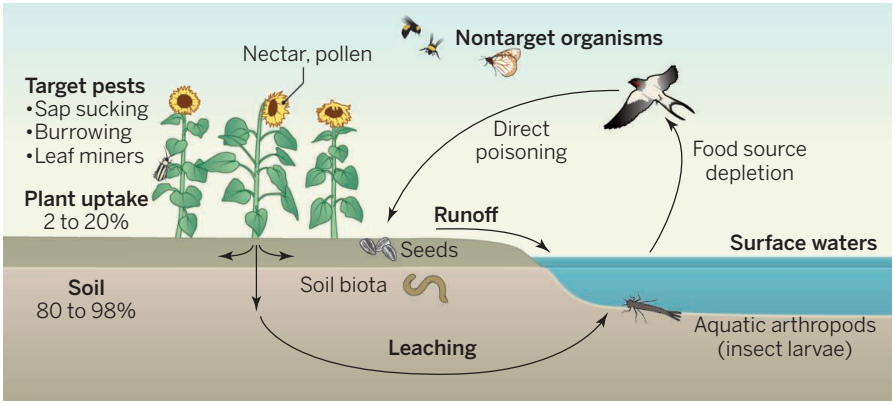
Whether these observations apply to bees in the natural environment has been a contentious question, because the performance of the hives does not change significantly. There are several reasons for this apparent lack of effect. The amount of honey produced is usually higher in contaminated hives because feeding inhibition and death of workers result in excess honey stores. Also, some undetectable sublethal effects cause mortality after a time lag (*1*). Finally, honey bee colonies compensate forager losses by producing hundreds of new workers daily; colonies thus usually overcome the initial effects during spring and summer and may survive the winter apparently unscathed. However, colony growth is usually hampered by queen failure in the next season (*7*), indicating that the queen suffers the effects of long-term intoxication. Bumble bees produce 85% fewer queens per colony when exposed to field-realistic concentrations of imidacloprid (*8*).

Concurrent with the widespread use of neonicotinoids, honey bees have experienced an increase in viral diseases, some of which are propagated by a mite parasite (*Varroa destructor*) that undermines bee health. Pollen from monoculture crops also weakens the bees' immune system, making them more susceptible to pathogens such as Nosema. These confounding factors can be blamed for the declines in honey bees but cannot account for the parallel decline in wild and bumble bees.

Di Prisco *et al.* (*1*) have established that sublethal doses of two neonicotinoids (clothianidin and imidacloprid) cause bee immune deficiency that triggers viral infections. This causal link helps to explain the time lag between initial exposure and mortality: The cascade of effects prompted by the insecticides involves irreversible biological pathways that are not observable until death takes place (*9*).

Although bees have captured most of the attention, neonicotinoids are equally toxic to ants, termites, parasitoids, and aquatic insect larvae, particularly mayflies, caddisflies, stoneflies, and midges. They are also toxic to decomposer amphipods, woodlice, and most crustaceans, but water fleas are very tolerant (*4*).

Because most neonicotinoids persist in soils for a year or more and are water soluble, 80 to 98% of residues remaining in the soil of treated crops eventually move into surface waters or leach into groundwater. Recent surveys from nine countries show 80% of surface waters contaminated with neonicotinoids at levels of 0.14 to 18 ppb, which are sublethal to aquatic arthropods (*10*, *11*). However, as in bees, chronic toxicity in all these organisms involves delayed and cumulative lethal effects over time (*12*). Experiments in aquatic model ecosystems treated with single or repeated dosages of imidacloprid confirm this: midges, ostracods, and mayflies disappear; their populations do not recover while residues in water are above 1 ppb (*13*). After 8 years of field monitoring, Van Dijk *et al.* (*11*) reported that imidacloprid concentrations as low as 0.01 ppb led to significant reduction of macroinvertebrates in surface waters.



Fate of neonicotinoids and pathways of environmental contamination.

Feeding inhibition has been observed in several decomposer organisms exposed to chronic, sublethal concentrations of imidacloprid, but starvation alone is insufficient to explain the lack of recovery and increased mortality with time (*14*). Continuous contamination of the aquatic environment with neonicotinoids may undermine the invertebrate resource base of aquatic ecosystems (*11*), thereby indirectly reducing populations of fish, birds, bats, frogs, and other animals that feed on them. Indeed, the steady decline of five species of birds in the Netherlands over the past two decades correlates with imidacloprid contamination of surface waters during the same period (*2*).

The effects of neonicotinoid residues on soil biota remain largely unknown, but the extreme efficiency with which these insecticides eliminate grub populations in turf is worrisome (*4*). This issue requires more study, because the ecosystem services provided by soil organisms are essential for sustainable agricultural production (*15*). Scattered seeds coated with high concentrations of neonicotinoids may also pose a risk to birds and rodents, despite the higher tolerance of vertebrates due to their distinct nicotinic receptor subunits (*4*).

Mechanisms that underpin chronic neonicotinoid effects on terrestrial and aquatic arthropods include immune suppression and feeding inhibition. While these and other issues are investigated further, current knowledge calls for a reconsideration of current prophylactic seed treatments with neonicotinoids. Such treatments are the main source of soil and water contamination; are often unnecessary, as they either do not increase yields or are not profitable; and go against the principles of integrated pest management (*15*). ■

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ILLUSTRATION: P. HUEY/*SCIENCE*

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