

Pesticides & Pollinator Toxicology

Comprehensive Research Review

A curated synthesis of the most-cited and representative open-access studies, grounded in a harvested literature corpus. Every claim is traceable to a cited study; this is a curated overview, not an exhaustive review. Generated 2026-06-20 for the bee-health knowledge base at scrapeworks.net.

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Pesticides & Pollinator Toxicology — Overview

A grounded synthesis of the most-cited open-access papers on pesticides and bees. Every claim is traceable to a cited study; curated overview, not exhaustive.

Overview

Pesticides are one of the core stressors implicated in pollinator decline. The dominant concern is the **neonicotinoids** — since their introduction in the 1990s they have become the most widely used class of insecticides worldwide, applied largely as systemic seed coatings that make the whole plant, including its pollen and nectar, toxic to insects (Simon-Delso 2015; Wood 2017). But bees encounter a far wider chemical cocktail than neonicotinoids alone.

Key themes

Multiple chemical classes, multiple routes

Bees are exposed to insecticides, fungicides, herbicides and acaricides via several routes: contaminated pollen and nectar, dust from treated seed, and residues accumulated in wax comb (Stewart 2014). This is detailed under [exposure routes & risk](#).

Fungicides matter even though they don't kill bees directly

A key insight is that fungicides — over a third of the global pesticide market — are often applied during bloom when bees are foraging, and although not acutely toxic themselves, they can amplify the toxicity of insecticides and interact with stressors (Rondeau 2022; Schuhmann 2021). Real-world combinations, such as the insecticide-fungicide tank mixes used on California almonds, have been linked to brood damage (Wade 2019).

Sublethal and combined effects dominate the real risk

The harm bees actually experience is mostly **sublethal and combinatorial** rather than outright lethal: impaired learning and navigation, weakened immunity, and effects that emerge only in combination with other chemicals or pathogens. These are covered under [neonicotinoids & sublethal effects](#) and [pesticide interactions](#), and connect to the broader [multi-stressor model](#).

Conclusion

The pesticide threat to bees is not a single chemical at a lethal dose; it is chronic, multi-route exposure to mixtures whose sublethal and interactive effects conventional risk assessment underestimates. The subtopics below break this down.

Notable studies

Systemic insecticides (neonicotinoids and fipronil): trends, uses, mode of action and metabolites

Simon-Delso et al., Environmental Science and Pollution Research 2015 · 927 citations — The reference overview of systemic insecticide use and chemistry.

Fungicides and bees: a review of exposure and risk

Rondeau & Raine, Environment International 2022 · 77 citations — Why fungicides, applied at bloom, matter for bees.

Combined toxicity of insecticides and fungicides applied to California almond orchards

Wade et al., Insects 2019 · 76 citations — Real-world tank-mix toxicity to brood.

Potential exposure of pollinators to neonicotinoids from seed treatments

Stewart et al., Environmental Science & Technology 2014 · 93 citations — Quantifies field exposure routes.

Interaction of insecticides and fungicides in bees

Schuhmann et al., Frontiers in Insect Science 2021 · 36 citations — How fungicides potentiate insecticide toxicity.

Curated synthesis of representative and most-cited studies — not exhaustive. Explore via [search](#). Subtopics:
[Neonicotinoids](#) · [Interactions](#) · [Exposure & risk](#) · [Multi-stressor model](#).

Neonicotinoids & Sublethal Effects

A grounded synthesis of the most-cited open-access papers on neonicotinoids and bees. Every claim is traceable to a cited study; curated overview, not exhaustive.

Overview

Neonicotinoids are systemic insecticides that act as agonists of the insect nicotinic acetylcholine receptor (nAChR), attacking the nervous system (Simon-Delso 2015; Moffat 2016). Because they are taken up into pollen and nectar at low concentrations, the central question for bees is not acute lethality but **sublethal harm** — effects on behaviour, cognition and immunity at field-realistic doses.

Key themes

Cognitive and motor impairment

The best-documented sublethal effects are neurological. Field-realistic exposure impairs olfactory learning and memory and degrades the motor function of adult workers, undermining the foraging and navigation on which the colony depends (Williamson 2014; Li 2019). These behavioural deficits have a measurable molecular signature: environmentally relevant imidacloprid concentrations reshape the brain transcriptome of exposed bees (Christen 2018).

Distinct receptor targets

Not all neonicotinoids act identically. Different compounds target distinct nAChR subtypes and neuron populations, producing different physiological effects — which matters for predicting and regulating each chemical's risk (Moffat 2016).

Metabolism and the acute-versus-chronic puzzle

Imidacloprid is metabolised in the bee into compounds including olefin and 5-hydroxy-imidacloprid (Suchail 2004). Crucially, there is a discrepancy between acute and chronic toxicity: chronic low-dose exposure can be far more harmful than short acute tests suggest, partly because toxic metabolites accumulate — a key reason acute-only risk assessment understates the hazard (Suchail 2001).

Beyond the nervous system: immunity and survival

Neonicotinoids also weaken the bee. Thiacloprid, imidacloprid and clothianidin impair immunocompetence, rendering bees more vulnerable to parasites and pathogens (Brandt 2016), and imidacloprid reduces honey bee survival rates even at environmentally relevant exposure (Raymann 2018). The breadth of these effects is reflected at the molecular level, where field-realistic neonicotinoid concentrations reshape global gene expression in the bee brain (Christen 2018).

Conclusion

The neonicotinoid risk to bees is overwhelmingly a story of sublethal effects — impaired cognition, weakened immunity, accumulating chronic toxicity — that individually fall short of killing a bee outright but together degrade colony function and amplify other stressors. This is why they feature so heavily in the [multi-stressor model](#) and [pesticide interactions](#).

Notable studies

Imidacloprid decreases honey bee survival rates but does not affect the gut microbiome

Raymann et al., Applied & Environmental Microbiology 2018 · 65 citations — Direct evidence of reduced survival under neonicotinoid exposure.

The neonicotinoids thiacloprid, imidacloprid and clothianidin affect immunocompetence

Brandt et al., Journal of Insect Physiology 2016 · 242 citations — Neonicotinoids weaken bee immune defence.

Metabolism of imidacloprid in *Apis mellifera*

Suchail et al., Pest Management Science 2004 · 116 citations — Imidacloprid is metabolised into olefin and 5-hydroxy compounds that contribute to its chronic toxicity.

Exposure to neonicotinoids influences the motor function of adult worker honeybees

Williamson et al., Ecotoxicology 2014 · 115 citations — Sublethal motor and behavioural impairment.

Neonicotinoids target distinct nicotinic acetylcholine receptors and neurons

Moffat et al., Scientific Reports 2016 · 76 citations — Different compounds, different receptor targets.

Curated synthesis of representative and most-cited studies — not exhaustive. Explore via [search](#).

Pesticide × Disease / Varroa Interactions

A grounded synthesis of the most-cited open-access papers on pesticide interactions in bees. Every claim is traceable to a cited study; curated overview, not exhaustive.

Overview

A pesticide's effect on bees often depends on what *else* the bee is exposed to. The headline interaction is between insecticides and fungicides: many fungicides are not toxic to bees on their own but dramatically increase the toxicity of insecticides, because bees encounter them together in the field (Schuhmann 2021; Wade 2019). Understanding these interactions is essential, because regulatory testing of single compounds misses them.

Key themes

Fungicides potentiate insecticide toxicity

Certain fungicides — particularly sterol-biosynthesis-inhibiting (azole) fungicides — block the bee's detoxification enzymes, so an insecticide that the bee could otherwise break down becomes far more potent in their presence. This synergy is repeatedly demonstrated for insecticide-fungicide combinations in bees (Schuhmann 2021). Real-world tank mixes show it in practice: insecticide-fungicide combinations applied to California almond orchards during bloom were linked to brood damage that neither class produced alone (Wade 2019).

Interactions are not always synergistic

A critical nuance — echoing the broader [multi-stressor model](#) — is that combined effects are not uniformly amplifying. In some cases a fungicide can even *reduce* the impact of a neonicotinoid (Schuhmann 2025), so interactions must be measured, not assumed. Treating every combination as synergistic would be as wrong as ignoring interactions entirely.

Synergists are deliberately engineered

The synergy principle is well enough understood that it is exploited on purpose: specific compounds (e.g. IPPA08) have been designed to synergise neonicotinoid insecticides, increasing efficacy while reducing the active ingredient needed (Bao 2016). The same biochemistry that makes a designed synergist work is what makes an incidental fungicide co-exposure dangerous.

Pesticide × pathogen interactions

Beyond chemical mixtures, pesticides interact with disease. Neonicotinoid-induced immune suppression makes bees more susceptible to pathogens, and the pesticide-pathogen couplings (such as *Nosema*-virus synergies and fungicide effects on disease transmission) are detailed under the [multi-stressor model](#). The systemic, persistent nature of neonicotinoids means this co-exposure is the norm, not the exception (Simon-Delso 2015).

Conclusion

Pesticide risk to bees is fundamentally combinatorial. Fungicide co-exposure can turn a "safe" insecticide dose harmful; pathogens can do the same; and the interactions run in both directions. The practical implication is that risk must be assessed for realistic mixtures and stressor combinations — not one chemical at a time.

Notable studies

Interaction of insecticides and fungicides in bees

Schuhmann et al., Frontiers in Insect Science 2021 · 36 citations — Reviews how fungicides potentiate insecticide toxicity via detoxification inhibition.

Combined toxicity of insecticides and fungicides applied to California almond orchards

Wade et al., Insects 2019 · 76 citations — Real-world tank-mix toxicity to brood during bloom.

Specific synergist for neonicotinoid insecticides: IPPA08

Bao et al., Journal of Agricultural and Food Chemistry 2016 · 34 citations — A deliberately engineered neonicotinoid synergist.

Seed coating with a neonicotinoid insecticide negatively affects wild bees

Rundlöf et al., Nature 2015 · 570 citations — Field-scale harm against which interaction effects compound.

Systemic insecticides: trends, uses, mode of action and metabolites

Simon-Delso et al., Environmental Science and Pollution Research 2015 · 927 citations — Why systemic, persistent insecticides guarantee co-exposure.

Curated synthesis of representative and most-cited studies — not exhaustive. Explore via [search](#).

Exposure Routes & Risk Assessment

A grounded synthesis of the most-cited open-access papers on pesticide exposure in bees. Every claim is traceable to a cited study; curated overview, not exhaustive.

Overview

To understand pesticide risk you have to know how bees actually encounter the chemicals. Residue surveys consistently find a complex cocktail of pesticides in the materials bees collect and store — pollen, nectar and wax — making exposure chronic, multi-compound and partly hidden inside the hive (Damalas 2011; Böhme 2018).

Key themes

Residues in pollen and nectar

The foraging routes are the front line. Pesticide residues are routinely detected in the pollen and nectar of crops and in the pollen loads bees carry home, and surveys of bee-collected pollen reveal multiple residues per sample (Azpiazu 2023; Böhme 2018). Modelling approaches now estimate residue levels in nectar and pollen to support exposure assessment where direct measurement is impractical (Li 2022). In high-pesticide-use regions, residues in the beekeeping environment can be severe (Nai 2017).

Wax comb: the hidden reservoir

Exposure is not only at the flower. Lipophilic pesticides accumulate in **wax comb**, where multi-residue analyses detect scores of compounds (over 160 in one validated method), turning the comb into a chronic internal reservoir (García 2017). Critically, sublethal effects flow from this reservoir: pesticide residues in brood comb impaired developing workers and shortened adult worker lifespan (Wu 2011).

Does regulation keep pace?

Whether exposure stays within "safe" limits is contested. An assessment of residues in honeybee-collected pollen questioned whether EU regulation actually protects bees from harmful exposure (Kaila 2022), reflecting a broader concern that legal residue limits and real-world bee harm are not well aligned (Damalas 2011).

The surrogate-species problem

Regulatory risk assessment relies on the honey bee as a surrogate for all bees, but honey bees and non-*Apis* bees (e.g. bumble bees) differ in both exposure and susceptibility — so honey-bee-based assessment can misjudge the risk to wild pollinators (Gradish 2019).

Conclusion

Bee pesticide exposure is chronic, multi-route (pollen, nectar, dust, wax) and multi-compound, and several lines of evidence suggest standard risk assessment — single compounds, lethal endpoints, one surrogate species — understates the true hazard. Realistic risk assessment must account for mixtures, sublethal effects, and the wax reservoir.

Notable studies

Sub-lethal effects of pesticide residues in brood comb on worker honey bees

Wu *et al.*, *PLoS ONE* 2011 · 196 citations — Comb residues impair development and shorten worker lifespan.

Multiresidue method for trace pesticide analysis in honeybee wax comb

García et al., Talanta 2017 · 31 citations — Detects 160+ pesticide residues accumulating in wax.

Comparison of pesticide exposure in honey bees and bumble bees

Gradish et al., Environmental Entomology 2019 · 92 citations — The honey-bee surrogate underestimates risk to other bees.

Pesticide residue survey of pollen loads collected by honeybees

Böhme et al., PLoS ONE 2018 · 62 citations — Multiple residues in daily-collected pollen.

Pesticide residues in honeybee-collected pollen: does EU regulation protect bees?

Kaila et al., Environmental Science and Pollution Research 2022 · 20 citations — Questions whether residue limits are protective.

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