

**REVIEW**

The Impact of Pesticides on Honey Bees: A review

MOHAMED R. FOUAD

Department of Pesticide Chemistry and Technology, Faculty of Agriculture, Alexandria University, Aflaton St., 21545, El-Shatby, Alexandria, Egypt


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Corresponding author

Mohamed R. Fouad 
Department of Pesticide Chemistry
and Technology, Faculty of Agriculture,
Alexandria University, Aflaton St., 21545,
El-Shatby, Alexandria, Egypt.
E-Mail: mohammed.riad@alexu.edu.eg
mohammed.reid@yahoo.com

Abstract

The review highlights the critical relationship between pesticide exposure and honey bee health. It discusses how various classes of pesticides, including neonicotinoids, carbamates, organophosphates, pyrethroids, and others, adversely affect bee behavior, reproduction, and immune systems. The paper reviews recent scientific findings indicating that sub-lethal doses can impair navigation, foraging efficiency, and colony stability, leading to colony collapse disorder. Emphasizing the ecological and agricultural significance of honeybees as pollinators, the abstract highlights the need for sustainable pesticide use and integrated pest management strategies to protect bee populations and maintain environmental health.

Introduction

Honey bees (*Apis mellifera*) are vital pollinators that play a crucial role in maintaining biodiversity and supporting agricultural productivity worldwide (Jacob et al., 2019; Khalifa et al., 2021; Walker et al., 2022; Iqbal et al., 2024; Al Dhafar et al., 2025). Their pollination activities are essential for the reproduction of many flowering plants and the production of a significant portion of the crops consumed by humans. However, in recent decades, the health and populations of honey bees have been increasingly threatened by a range of environmental stressors, among which pesticides are considered a major factor. Pesticides, designed to control pests and enhance crop yields, have raised concerns due to their potential adverse effects on non-target organisms, particularly honey bees. Here, I examine the intricate relationship between pesticide exposure and honey bee health, aiming to understand how pesticides affect bee behavior, immunity, and colony sustainability, and to emphasize the

importance of sustainable pest management practices. Honey bees are significant insects from both ecological and economic perspectives (Pham-Delègue et al., 2002; Abd-Eldaim et al., 2023; Li et al., 2023; Fouad et al., 2025a-b; Mostafa et al., 2025). Honey bees are the sole pollinator responsible for 35% of global food production (Bommuraj et al., 2021; Khalifa et al., 2021; Omelchun et al., 2025). In addition to producing nutritious foods such as honey, wax, pollen, propolis, royal jelly, and venom, honey bees are also highly valuable for boosting revenue and producing pharmaceuticals (Lee et al., 2016; Souza et al., 2024; Al Dhafar et al., 2025). Honey, apiculture's main product, has many benefits, including a long shelf life, concentrated form, and high market value (Khan et al., 2020; Iqbal et al., 2024). Task distribution is essential to social behavior; individual bees carry out distinct tasks based on their age, genetic make-up, and environmental circumstances (Pham-Delègue et al., 2002; Fisher & Rangel, 2018; Straub et al., 2021). Younger bees typically assist with hive tasks, including cleaning, cell construction, and



nursing, before some become guards and older bees begin to forage (Pham-Delègue et al., 2002). Particularly in rural areas, especially those with steep terrain, they are a tremendous help in promoting economic growth, nutrition, and healthcare (Iqbal et al., 2024). Therefore, a colony with poor health or foraging skills can have a detrimental effect on the sale of honey and other bee products, as well as on vital pollination services (Souza et al., 2024). Farmers have been compelled to use more pesticides to produce higher yields due to the rising demand for food (Migdał et al., 2018; Fouad et al., 2024a-c; Al Dhafar et al., 2025; Omelchun et al., 2025). Insecticide use in crops is considered one of the primary causes of the decline in bee populations worldwide (Ulziibayar & Jung, 2019; Al Dhafar et al., 2025; Omelchun et al., 2025). Additionally, by altering gene expression and disrupting enzyme activity, pesticide exposure can cause harm to cells, the body, and their appearance (Yordanova et al., 2022; Chen et al., 2025). Depending on how a pesticide enters the body, honeybees may experience harmful consequences. Some, like neonicotinoids, are more harmful when consumed, whereas others are toxic upon contact (Pashte & Patil, 2018; El-Aswad et al., 2019; De Souza et al., 2024; Shepherd et al., 2024; Omelchun et al., 2025). Honeybees are at risk of contracting pesticide poisoning not only from direct contact poisoning but also by consuming certain contaminated nectar, pollen, and water, as well as from bringing contaminated objects into the hive (Radwan et al., 2020). Repeatedly exposing bee colonies to small amounts of pesticides can have detrimental long-term effects, such as hive bee population decline or even colony loss (Suchail et al., 2001). Therefore, before being used in agriculture, both active ingredients and prepared pesticides are currently subjected to numerous studies to determine the risks they pose to honey bees (Laurino et al., 2011; Radwan et al., 2020). Additionally, when regulatory criteria or best practices for applying pesticides are not followed, the danger of affecting bee health increases (Radwan et al., 2020). Because of their decreased capacity for pollination, the presence of pesticide residues in their honey, their mortality, and the inhibition of enzymes in their tissues when exposed to harmful levels of pollutants, bees may continue to serve as a trustworthy biomarker for environmental contamination (Badawy et al., 2015; Migdał et al., 2018; Ulziibayar et al., 2021). In addition to field studies using bees collected directly from the wild, there is a laboratory study that allows for the simpler duplication of variables that negatively impact the insects (Migdał et al., 2018).

Materials and Methods

Literature search strategy

A comprehensive literature review was conducted to assess the impact of pesticides on honey bees (*Apis mellifera*). Relevant studies were identified through systematic searches of electronic databases, including PubMed, Web of Science,

Scopus, and Google Scholar. The search strategy employed a combination of keywords, including “pesticides,” “honey bees,” “*Apis mellifera*,” “bee toxicity,” “neonicotinoids,” “pesticide exposure,” and “bee health.” These keywords were used in various combinations to maximize coverage. The search was restricted to articles published up to August 2025 to ensure inclusion of the most recent and relevant research (Toselli & Sgolastra, 2020; Lehmann et al., 2021; Shamsan et al., 2023; Abd Ul-Malik et al., 2024; El-Aswad et al., 2024a-c; Fouad & Abdel-Raheem, 2024; Arany & Czucz, 2025; Ferreira et al., 2025; Galczyńska et al., 2025).

Inclusion and exclusion criteria

Studies were included if they investigated the effects of pesticides on honey bees (whether through laboratory, field, or semi-field experiments) and reported quantitative data on outcomes such as mortality, behavior, or physiological effects. Only peer-reviewed journals, conference proceedings, or credible reports were considered. Conversely, studies focusing solely on other pollinators without specific data on honeybees, commentaries and opinion pieces without original data, or publications not available in English were excluded from the review.

Data extraction and synthesis

Data from selected studies were extracted independently and included information on pesticide type, exposure methods, doses, duration, bee life stages affected, and observed effects. Discrepancies were resolved through discussion. The findings were synthesized qualitatively, highlighting patterns, similarities, and differences across studies (da Costa et al., 2024).

Quality assessment

The methodological quality of the included studies was assessed based on criteria such as experimental design, sample size, control groups, and statistical analysis. Studies with significant methodological limitations were noted accordingly.

Data presentation

Results were organized thematically to discuss different classes of pesticides (e.g., insecticides for insects, herbicides for weeds, fungicides for fungi), routes of exposure, and observed impacts on honey bee health and behavior (Shi et al., 2019; Schuhmann et al., 2022; de Souza et al., 2024).

Results and discussion

Acute toxicity of pesticides against honey bees

Median lethal concentration (LC_{50})

LC_{50} is a standardized measure used in toxicology to assess the toxicity of a substance. It represents the

concentration of a chemical, pollutant, or other harmful agent that causes death in 50% of a test population within a specified period under controlled conditions (Abdel-Raheem et al., 2023; El-Aswad et al., 2023a; Fouad et al., 2023a). Widely used in environmental risk assessment, pesticide regulation, and chemical safety testing. The data indicate that among the tested compounds, spinosad exhibited the highest toxicity to honey bees with an LC_{50} value of 7.34 ppm. Oxymatrine showed slightly lower toxicity with an LC_{50} of 10.68 ppm, whereas chlorfluazuron was the least toxic, with an LC_{50} of 2526 ppm (Rabea et al., 2010). The LC_{50} values for clothianidin and thiamethoxam were 0.081 and 0.134, 0.077 and 0.126, and 0.075 and 0.123 ppm after 24, 48, and 72 h of exposure, respectively (Laurino et al., 2011). Lee et al. (2016) found that LC_{50} were 0.006, 0.006, 0.006, and 5.167 ppm for thiamethoxam, imidacloprid, clothianidin, and carbaryl after 12 h of treatment. The LC_{50} value (ppm) of *A. mellifera* larvae at 72 h was 494.27 for amitraz, 15.39 for chlorpyrifos, 90.01 for coumaphos, 27.69 for fluvalinate, and 138.84 for imidacloprid. Chlorpyrifos is the most toxic among these, given its lowest LC_{50} , while amitraz is the least toxic at the tested concentrations (Dai et al., 2017). According to Heard et al. (2017), after 240 hours of exposure, the relative toxicity ranking, from most to least toxic, was: clothianidin, dimethoate, and 2,4-D. The statement highlights the relative toxicity of emamectin benzoate compared to other insecticides based on LC_{50} values. Specifically, emamectin benzoate has an LC_{50} of 0.66 ppm, indicating high toxicity. It is 3.27 times more toxic than abamectin, 31.74 times more toxic than spinosad, and 7.56 times more toxic than spinetoram, according to Abdu-Allah and Pittendrigh (2018). This information can be useful for assessing the relative safety and environmental impact of these chemicals. The LC_{50} values for the worker bees were 385, 370, 125, 70, and 266 ppm for dimethoate, cypermethrin, fipronil, imidacloprid, and Indoxacarb, respectively. According to the safety index, imidacloprid, followed by fipronil, is the least safe to bees (Pashte & Patil, 2018). The LC_{50} of formulated clothianidin was 0.53 ppm after 48 h for larvae (Yao et al., 2018). The median LC_{50} values estimated for acetamiprid, imidacloprid, and thiacloprid were 189.62, 22.78, and 142.31 ppm of diet for *A. mellifera* (Jacob et al., 2019). Bommuraj et al. (2021) found that the LC_{50} for acetamiprid, dimethoate, imidacloprid, and thiacloprid were 277.3, >0.8, 2.2, and 194.3 ppm after 48 h of exposure. Ulziibayar et al. (2021) found that LC_{50} values were 4911.8, 3.7, 12.5, and 40.7 ppm after 72 h of exposure to acetamiprid, lufenuron, acequinocyl, and fluxametamide. The estimated imidacloprid LC_{50} was 1.44 ppm for larvae at 72 h (Carneiro et al., 2022). The 168 h toxicity test results indicated that thiamethoxam exhibited the highest toxicity, with an LC_{50} value of 0.25 ppm. It was followed by methomyl and dimethoate, which had LC_{50} values of 4.19 ppm and 5.30 ppm, respectively. In contrast, other pesticides such as cypermethrin, cyfluthrin, permethrin, esfenvalerate, and tetraconazole demonstrated relatively lower toxicity, with

LC_{50} values ranging from 33.78 to 1125 ppm (Li et al., 2023). In the acute toxicity test, haloxyfop-p-methyl exhibited the highest toxicity to forager workers after 96 h of oral exposure, with an LC_{50} value of 0.95 ppm. This was followed by clethodim, and the mixture of the two compounds was found to be the least toxic among the tested substances (Mohamed et al., 2023). Saad et al. (2023) studied the toxic effects of emamectin benzoate, imidacloprid, chlorpyrifos, indoxacarb, lambda-cyhalothrin, glyphosate, and thiophanate-methyl on honeybee (*A. mellifera*) workers. The highest toxic pesticide during the exposure period 24, 48, and 72 h was emamectin benzoate with LC_{50} values (0.247, 0.047, 0.020 ppm), respectively, and the least toxic one was glyphosate with LC_{50} values of (6861.151, 3366.968, and 2477.267 ppm) after 24, 48, and 72 h of exposure, respectively. However, chlorpyrifos ranked as the third most toxic pesticide at 24 and 48 hours of treatment, with LC_{50} values of 10.226 and 2.731 ppm. Al Dhafar et al. (2025) found that thiamethoxam was extremely toxic to *A. mellifera* adult workers (LC_{50} = 0.006 ppm) followed by lambda-cyhalothrin (LC_{50} = 0.053 ppm) and fenpyroximate (LC_{50} = 2.29 ppm) after 24 h of treatment; however, hexythiazox was relatively less toxic to bees (110.09 ppm). As for the toxicity after 48 h of exposure, lambda-cyhalothrin and thiamethoxam were obviously the most harmful to honey bees (LC_{50} = 0.001 & 0.002 ppm), while hexythiazox showed the lowest harm to the foragers (LC_{50} = 16.735 ppm), and fenpyroximate was relatively low in toxicity to bees (LC_{50} = 1.49 ppm). LC_{50} was decreased to 0.0001, 0.0004, 0.09, and 3.12 ppm for lambda-cyhalothrin, thiamethoxam, fenpyroximate, and hexythiazox, respectively, after 72 h of oral exposure (Table 1).

Median lethal dose (LD_{50})

LD_{50} is a standard measurement used in toxicology to assess the acute toxicity of a substance. It represents the dose required to kill 50% of a tested population. LD_{50} values facilitate the comparison of toxicity among different substances and are essential for risk assessment, regulatory decisions, and safety evaluations. Lower LD_{50} values indicate higher toxicity, meaning a smaller amount of the substance can cause death. The acute toxicity of pesticides exhibited similar patterns in both feeding and contact tests (Table 2). However, feeding tests demonstrated greater toxicity to honey bees compared to contact tests (Ulziibayar & Jung, 2019). Imidacloprid's LD_{50} values were 37 ng/bee at 72 h and 57 ng/bee at 48 h (Suchail et al., 2001). The oral LD_{50} at 72 h reached 57.25 and 0.11 μ g a.i./bee for propiconazole and dimethoate, respectively (Ladurner et al., 2005). The acute oral LD_{50} is calculated at 24, 48, and 72 h for clothianidin and thiamethoxam by Laurino et al. (2011). Dinotefuran was identified as the most harmful to honey bees, with an LD_{50} of 0.0006 μ g/bee. In contrast, pyridalyl exhibited the lowest toxicity to foragers, with an LD_{50} of 6.16 μ g/bee. Pymetrozine and acetamiprid displayed intermediate toxicity levels, with LD_{50} values of 0.16 μ g/bee and 1.69 μ g/bee, respectively,

Table 1. LC₅₀ of pesticides against *Apis mellifera* by oral method

Pesticides	Stage	Time exposure (h)	LC ₅₀ (ppm)	References
Chlorfluazuron, Oxymatrine & Spinosad	Adult	24	2526, 10.68 & 7.34	Rabea et al., 2010
		24	0.081 & 0.134	
Clothianidin & Thiamethoxam	Adult	48	0.077 & 0.126	Laurino et al., 2011
		72	0.075 & 0.123	
Thiamethoxam, Imidacloprid, Clothianidin & Carbaryl	Adult	12	0.006, 0.006, 0.006 & 5.167	Lee et al., 2016
Amitraz, Chlorpyrifos, Coumaphos, Fluvalinate & Imidacloprid	Larvae	72	494.27, 15.39, 90.01, 27.69 & 138.84	Dai et al., 2017
		48	0.104, 2.42 & >900	
Clothianidin, Dimethoate & 2,4-D	Adult	96	0.055, 1.16 & >900	Heard et al., 2017
		240	0.017, 0.016 & >900	
Emamectin benzoate, Abamectin, Spinosad & Spinetoram	Adult	24	0.66, 2.19, 20.95 & 4.99	Abdu-Allah and Pittendrigh, 2018
Dimethoate, Cypermethrin, Fipronil, Imidacloprid & Indoxacarb	Adult	24	385, 370, 125, 70 & 266	Pashte and Patil, 2018
Clothianidin	Larvae	24	0.53	Yao et al., 2018
Acetamiprid, Imidacloprid, Thiacloprid & Dimethoate	Adult	24	189.62, 22.78, 142.31 & 0.75	Jacob et al., 2019
Acetamiprid, Dimethoate, Imidacloprid & Thiacloprid	Adult	48	277.3, >0.8, 2.2 & 194.3	Bommuraj et al., 2021
Acetamiprid, Lufenuron, Acequinocyl & Fluxametamide	Larvae	72	4911.8, 3.7, 12.5 & 40.7	Ulziibayar et al., 2021
Imidacloprid	Larvae	72	1.44	Carneiro et al., 2022
		48	0.53, 312.7, 182.9, 9.01, 12.50, 321.6, 82.07 & 1343	
		96	0.33, 258.7, 156.9, 6.54, 6.59, 245.6, 51.82 & 1212	
Thiamethoxam, Tetraconazole, Cypermethrin, Dimethoate, Methomyl, Cyfluthrin, Permethrin & Esfenvalerate	Adult	168	0.25, 216.8, 149.8, 5.30, 4.19, 182.2, 33.78 & 1125	Li et al., 2023
		24	1.95, 5.02 & 6.02	
		48	1.52, 4.11 & 5.19	
		72	1.21, 3.57 & 3.69	
Haloxypop-p-methyl, Clethodim & Haloxypop-p-methyl + Clethodim	Adult	96	0.95, 3.15 & 3.24	Mohamed et al., 2023
		24	0.247, 2.437, 10.226, 14.436, 96.967, 829.476 & 6861.151	
Emamectin benzoate, Imidacloprid, Chlorpyrifos, Indoxacarb, Lambda-cyhalothrin, Thiophanate-methyl & Glyphosate	Adult	48	0.047, 0.397, 2.731, 5.672, 38.504, 445.531 & 3366.968	Saad et al., 2023
		72	0.020, 0.329, 1.985, 2.086, 25.029, 367.229 & 2477.276	
Lambda-cyhalothrin, Hexythiazox, Fenpyroximate & Thiamethoxam	Adult	24	0.053, 110.09, 2.29 & 0.006	Al Dhafar et al., 2025
		48	0.001, 16.735, 1.49, 0.002	
		72	0.0001, 3.12, 0.09 & 0.0004	

24 hours after exposure, relative to dinotefuran (Badawy et al., 2015). The acute oral LD₅₀ values of imidacloprid and clothianidin for *A. mellifera* were 8.6 and 2 ng/bee, respectively (Li et al., 2017). For topical application, emamectin benzoate was the most toxic (LD₅₀ = 0.00006 µg a.i./bee), with a potency 133.3, 750, and 38.33-fold more toxic than abamectin, spinosad, and spinetoram, respectively (Abdu-Allah & Pittendrigh, 2018). Domatskaya et al. (2018) found that LD₅₀ values were 98.28, 1.376, 0.304, and 71.3 µg a.i./bee after 24 h of exposure to acetamiprid, ivermectin, fipronil, and chlorfenapyr. The findings by Ulziibayar and Jung (2019) indicate that certain

pesticides exhibit significant variations in their toxicity levels. Organophosphate insecticides like fenitrothion and neonicotinoids (imidacloprid, dinotefuran, clothianidin, thiamethoxam) tend to be more toxic, whereas cyanoneonicotinoids (thiacloprid, acetamiprid) are comparatively less harmful. The acaricide amitraz shows an intermediate toxicity level, while the herbicide glyphosate and fungicide metconazole have minimal impact. This differentiation is important for understanding the environmental risks associated with these chemicals. The LD₅₀ values were 9.5, 10.45, 3.68, 18.33, and 12.55 µg/bee for methomyl, cyhalothrin,

Table 2. LD₅₀ of pesticides against *Apis mellifera*

Pesticides	Stage	Method exposure	Time exposure (h)	LD ₅₀	References
Imidacloprid	Adult	Oral	48	57 ng/bee	Suchail et al., 2001
			72	37 ng/bee	
Dimethoate	Adult	Contact	24	0.19 µg a.i./bee	Ladurner et al., 2005
			48	0.16 µg a.i./bee	
Propiconazole & Dimethoate	Adult	Oral	24	61.67 & 0.15 µg a.i./bee	
			48	59.43 & 0.13 µg a.i./bee	
Clothianidin & Thiamethoxam	Adult	Oral	72	57.25 & 0.11 µg a.i./bee	
			24	2.844 & 4.679 ng/bee	Laurino et al., 2011
48	2.689 & 4.411 ng/bee				
72	2.608 & 4.316 ng/bee				
Acetamiprid, Dinotefuran, Pymetrozine & Pyridalyl	Adult	Topical	24	1.69, 0.0006, 0.16 & 6.16 µg/bee	Badawy et al., 2015
Imidacloprid & Clothianidin	Adult	Oral	24	8.6 & 2 ng/bee	Li et al., 2017
Emamectin benzoate, Abamectin, Spinosad & Spinetoram	Adult	Topical	24	0.00006, 0.008, 0.045 & 0.0023 µg a.i./bee	Abdu-Allah and Pittendrigh, 2018
Acetamiprid, Ivermectin, Fipronil & Chlorfenapyr	Adult	Contact	24	98.28, 1.376, 0.304 & 71.3 µg a.i./bee	Domatskaya et al., 2018
Imidacloprid, Clothianidin, Thiamethoxam, Fenitrothion & Amitraz	Adult	Feeding	24	0.109, 0.003, 0.01, 0.162 & 7.082 µL/bee	Ulziibayar and Jung, 2019
Thiamethoxam, Fenitrothion & Amitraz	Adult	Feeding	48	0.005, 0.162 & 3.273 µL/bee	
Thiacloprid, Imidacloprid, Dinotefuran, Clothianidin, Thiamethoxam & Fenitrothion	Adult	Contact	24	6.109, 1.79, 0.138, 0.05, 0.019 & 0.011 µL/bee	
Methomyl, Cyhalothrin, Deltamethrin, Chlorpyrifos & Profenfos	Adult	Topical	48	33.105, 0.82, 0.14, 0.022, 0.034 & 0.092 µL/bee	Radwan et al., 2020
			24	0.95, 1.045, 6.36, 1.83 & 1.25 µg/bee	
			72	0.54, 0.71, 0.24, 1.23 & 0.73 µg/bee	
Acetamiprid, Imidacloprid & Thiacloprid	Adult	Oral	48	0.62, 0.51, 0.1, 0.75 & 0.61 µg/bee	Bommuraj et al., 2021
			72	63.1, 0.08 & 37.8 µg/bee	
Acetamiprid, Lufenuron, Acequinocyl & Fluxametamide	Larvae	Feeding	72	982.3, 0.4, 10 & 8.1 µg/larva	Ulziibayar et al., 2021
Sulfoxaflor & Azoxystrobin	Adult	Oral	48	41.04 & > 60 µg/bee	Barascou et al., 2022
Iponazole, MCPA, Cycloate, Prosulfuron, Acetochlor, Forchlorfenuron, Pinoxaden, Amicarbazone, Acetamiprid, Abamectin, Tetraniliprole, Tolfenpyrad, Propargite, Fenpyroximate & Sulfoxaflor	Larval	Oral	24	22, 34.6, 10, 26, 26.5, 15, 52, 45, 1.16, 0.0011, 0.013, 0.044, 25.31, 0.2 & 2.65 µg/larva	Farruggia et al., 2022
Cyantraniliprole & Sulfoxaflor	Larval	Feeding	72	0.047 & 11.404 µg a.i./larva	Kim et al., 2022

deltamethrin, chlorpyrifos, and profenfos, respectively, after 24 h of exposure. After 48 h of topical exposure, the LD₅₀ values range from 2.44 to 12.32 µg/bee (Radwan et al., 2020). Bommuraj et al. (2021) found that the LD₅₀ for acetamiprid, imidacloprid, and thiacloprid were 63.1, 0.08, and 37.8 µg/bee after 48 h of exposure. Ulziibayar et al. (2021) found that LD₅₀ values were 4982.3, 0.4, 10, and 8.1 µg/larva after 72 h of exposure to acetamiprid, lufenuron, acequinocyl, and fluxametamide, respectively. Barascou et al. (2022) found that the LD50 was 41.04 µg/bee and greater than 60 µg/bee for sulfoxaflor and azoxystrobin, respectively, after 48 hours. Farruggia et al. (2022) found that the LD₅₀ of the following pesticides: ipconazole, MCPA, cycloate, prosulfuron, acetochlor, forchlorfenuron, pinoxaden, amicarbazone, acetamiprid, abamectin, tetraniliprole, tolfenpyrad, propargite, fenpyroximate, and sulfoxaflor ranged between 0.0011 and 52 µg ai/larval/day. The 72-h LD₅₀ values of cyantraniliprole and sulfoxaflor were 0.047 and 11.404 µg/larva, respectively (Kim et al., 2022).

Impact of pesticides on enzyme activity in honey bees

Acetyl cholinesterase (AChE)

AChE in honey bees is an essential enzyme involved in nerve function, responsible for breaking down the neurotransmitter acetylcholine after nerve signals are transmitted. Its proper activity is crucial for bees' neural communication, behavior, and overall health. However, AChE is also a target for certain pesticides, such as organophosphates and carbamates, which inhibit its activity, leading to the accumulation of acetylcholine, neural overstimulation, paralysis, and potentially death in bees. Because of this, measuring AChE activity in honey bees serves as a biomarker for exposure to neurotoxic chemicals and environmental stressors. Understanding AChE's role helps researchers monitor the impacts of pesticides, assess environmental risks, and develop strategies to protect bee populations, which are vital for pollination (Badiou et al., 2008; Boily et al., 2013; Tavares et al., 2017; Pervez & Manzoor, 2021). AChE activity levels differ significantly among forager bees depending on age, season, pesticide type, and environmental conditions (Pandey et al., 2023). Tu et al. (2009) reported that AChE has been widely used as a biomarker for assessing general exposure to pollutants, especially organophosphate and carbamate pesticides (Al Dhafar et al., 2025). The toxicity of acephate is primarily due to its bioactivation into methamidophos, which functions as an AChE inhibitor. Exposure to a median lethal dose of 6.97 ppm of acephate resulted in notable suppression of AChE activity in honey bees, exemplifying the typical symptoms of organophosphate poisoning in these insects (Yao et al., 2018). Ayoub et al. (2024) observed a significant inhibition of AChE activity in bees exposed to chlorpyrifos, dimethoate, and profenophos. The study found that exposure to chlorpyrifos alone significantly decreased AChE activity in

honey bees compared to untreated bees and those exposed to atrazine alone. When bees were exposed to both atrazine and chlorpyrifos, AChE activity was further reduced relative to bees exposed to each chemical individually and to untreated bees. Interestingly, exposure to chlorpyrifos-oxon alone did not alter AChE activity compared to untreated bees, but resulted in lower activity than bees exposed to atrazine alone. Furthermore, combined exposure to atrazine and chlorpyrifos-oxon increased AChE activity relative to chlorpyrifos-oxon alone; however, the levels were significantly lower than those with atrazine alone and were similar to those of untreated bees. These findings suggest complex interactions between these pesticides affecting neural enzyme activity in honey bees (Fellows et al., 2022). The study by Pervez and Manzoor (2021) using probe-based real-time quantitative polymerase chain reaction demonstrated that treatments with imidacloprid and chlorpyrifos significantly increased AChE levels, whereas carbaryl treatment led to a decrease in AChE levels. Bees exposed to neonicotinoids, atrazine, and glyphosate showed altered AChE activity, suggesting potential neurotoxicity. The extent of enzyme activity changes varied depending on the chemical and dose (Boily et al., 2013). The combined biochemical and in silico data suggest that imidacloprid impairs honeybee neural function by both inhibiting AChE and interacting with nicotinic acetylcholine receptor (Ali et al., 2024a). AChE was depressed in *A. mellifera* during exposure to imidacloprid (Li et al., 2017). Imidacloprid, thiamethoxam, clothianidin, and acetamiprid exhibited inhibitory activity on AChE to varying degrees, with some compounds showing significant inhibition at certain concentrations (Györi et al., 2017). The AChE activity was strongly inhibited by dinotefuran followed by acetamiprid, pyridalyl, and then pymetrozine in descending order (Badawy et al., 2015). The study indicates that sub-lethal concentrations of lambda-cyhalothrin, hexythiazox, fenpyroximate, and thiamethoxam all led to a reduction in AChE activity in *A. mellifera* (adult workers). Among these, lambda-cyhalothrin and thiamethoxam caused the most significant inhibition of AChE activity, surpassing the effects observed with fenpyroximate, hexythiazox, and the control group. This suggests that lambda-cyhalothrin and thiamethoxam may pose a higher neurotoxic risk to honeybees at sub-lethal doses (Al Dhafar et al., 2025). Deltamethrin exposure led to significant alterations in AChE activity, suggesting that this enzyme could serve as a sensitive indicator of sublethal pesticide exposure in honeybees (Badiou et al., 2008). Spinosad significantly inhibited AChE activity across various organs of honey bee workers, with the highest inhibition percentage observed in the enzyme isolated from the thorax (Rabea et al., 2010).

Glutathione-s-transferase (GST)

The impact of pesticides on GST in honey bees is a significant area of research, given the crucial role this enzyme plays in detoxification and protecting bees from environmental

stressors. Pesticides can modulate GST activity in honey bees, either inducing or inhibiting its function depending on the type, dose, and exposure duration. These changes impact bees' ability to detoxify harmful substances, influencing their overall health and survival. GST plays a role in preventing oxidative stress induced by thiamethoxam in *A. mellifera* bees (Badiou-Bénéteau et al., 2012; Li et al., 2017). Imidacloprid exposure for a brief period increased enzyme activity; however, as the activity could not withstand prolonged exposure, the protective efficiency decreased (Ali et al., 2024b). Exposure to dimethoate, malathion, quinalphos, and sulfoxaflor results in significant alterations in GST activity, suggesting potential implications for bee health and resilience against environmental toxins (Kumar, 2017; Ibrahim et al., 2023; Al Dhafar et al., 2025). Zhu et al. (2017) found that there is a positive correlation between the toxicity of lambda-cyhalothrin and the activity of the GST enzyme. Exposure to glyphosate-based herbicide Roundup® induces significant alterations in GST activities, indicating oxidative stress in bees (Chen et al., 2023).

Esterase (EST)

Esterases are a group of enzymes involved in detoxification processes in honey bees (*A. mellifera*). They help metabolize and neutralize various xenobiotics, including pesticides. Changes in EST activity can reflect the exposure and physiological response of bees to pesticides. Many pesticides, particularly organophosphates and carbamates, inhibit ESTs, impairing detoxification pathways and increasing bees' susceptibility to toxins. Some pesticides may induce EST activity as a defensive response, which could lead to increased metabolic capacity but also potential energy costs. Even at sublethal doses, pesticides can alter EST activity, affecting bee health, behavior, and lifespan. Chronic exposure may lead to enzyme inhibition or overexpression, impacting bees' ability to detoxify other environmental toxins. Understanding these biochemical effects can inform safer pesticide usage and bee conservation strategies. The study by Attencia et al. (2005) investigates the impact of methyle-parathion and malathion on EST enzyme activity in *A. mellifera*. Results demonstrate that exposure leads to significant alterations in EST activity levels, indicating potential sublethal effects that could compromise bee health and resilience. Chlorpyrifos alone and the mixture of clothianidin and chlorpyrifos significantly suppressed esterase EST activity (Yao et al., 2018).

Mixed function oxidase (MFO)

Certain pesticides can induce the activity of MFO enzymes, thereby enhancing their detoxification capacity. This is often observed with sub-lethal exposures, where bees upregulate MFO activity to cope with chemical stress. Some pesticides may inhibit MFO enzymes, impairing detoxification processes, which can increase susceptibility to toxins and contribute to toxicity. Pesticide exposure can alter the gene expression levels of cytochrome P450 enzymes, affecting

the metabolic capacity of bees. Over-activation may lead to increased production of reactive metabolites, potentially causing oxidative stress.

Polyphenol oxidase (PPO)

PPO is an essential enzyme in honey bees that participates in the immune system by catalyzing the oxidation of phenolic compounds, leading to melanin formation, which helps in pathogen defense and wound healing. Pesticide exposure, particularly to insecticides such as neonicotinoids and organophosphates, can affect PPO activity, potentially compromising bee immunity. Certain pesticides may decrease PPO activity, weakening immune responses and increasing susceptibility to pathogens. Laboratory studies have shown that exposure to pesticides like imidacloprid reduces PPO activity in bee hemolymph. Chronic or sub-lethal doses can lead to dysregulation of PPO, affecting bee immunity over time. Variations depend on the type of pesticide, dose, exposure duration, and the bee's developmental stage. Reduced PPO activity can lead to increased vulnerability to diseases such as American foulbrood and Nosema. Altered immune enzyme activity can compromise colony health and productivity. Dinotefuran significantly inhibited PPO activity in both the head and thorax (Badawy et al., 2015). The findings indicate that exposure to lambda-cyhalothrin, thiamethoxam, fenpyroximate, and hexythiazox at their respective LC₂₅ concentrations resulted in activation of the PPO enzyme in *A. mellifera* (adult workers). In contrast, when bees were treated with the LC₅₀ concentration of these pesticides, PPO activity was suppressed. This suggests that sub-lethal doses may stimulate immune-related enzyme activity, whereas higher, more toxic doses impair it (Al Dhafar et al., 2025).

Carboxylesterase (CCE)

CCEs are enzymes that hydrolyze ester bonds in various compounds, including pesticides like organophosphates and pyrethroids. Pesticides can modulate CCE activity in honey bees, either inducing or inhibiting these enzymes depending on the compound, dose, and exposure duration. Changes in CCE activity influence bees' detoxification capacity and overall health, emphasizing the importance of understanding these interactions for sustainable pest management and pollinator conservation. Studies have shown that exposure to specific pesticides, such as chlorpyrifos (an organophosphate), can alter CCE activity levels. Sublethal doses of pesticides may lead to increased enzyme activity initially but could cause enzyme inhibition or enzyme exhaustion with prolonged exposure. Acetamiprid, pymetrozine, and pyridalyl were found to significantly stimulate carboxylesterase activity, potentially as a response mechanism to detoxify or cope with these compounds. Conversely, dinotefuran markedly inhibited the enzyme's activity, which could impair detoxification processes and increase susceptibility to toxicity (Badawy et al., 2015).

Histological observations

Histological studies of honey bees exposed to pesticides typically reveal various tissue damages that can impair their health and functions. Common observations include; 1) damage to the cells lining the digestive tract, including vacuolization, disorganization, and cell death, which can impair digestion and nutrient absorption, 2) structural degeneration or hypertrophy affecting excretion and osmoregulation, 3) reduction in size and cellular degeneration in hypopharyngeal glands, impacting brood food production, 4) neuronal degeneration, vacuolation, and disrupted synaptic connections in the brain, affecting learning, navigation, and foraging behavior, 5) degeneration of ovarian tissues in queens, leading to reduced fertility, 6) degeneration and atrophy of flight muscles, impairing flight capacity, and 7) decrease in immune cells, compromising immune responses. These histological alterations contribute to the decline in bee vitality, foraging efficiency, and colony health, ultimately impacting pollination services. The severity and type of damage depend on the pesticide class, exposure duration, and concentration. Tapparo et al. (2012) conducted research demonstrating that bees exposed orally to neonicotinoid pesticides exhibited cytoplasmic vacuolization and a decrease in their regenerative cell populations. Similarly, Pervez and Manzoor (2021) also reported these adverse cellular effects, highlighting the potential risks that neonicotinoids pose to bee health and the broader implications for pollinator populations. Prolonged contact with imidacloprid causes significant histopathological damage in key reproductive tissues, including the midgut, ovaries, and spermathecae, which store spermatozoa. These damages could impair the queen's health and reproductive capacity, potentially affecting colony viability (Moreira et al., 2022). The study by Carneiro et al. (2022) describes significant histopathological changes in the midgut epithelium of bees exposed to imidacloprid at its LC_{50} concentration (1.44 ppm). Observed alterations include the formation of cytoplasmic vacuoles, expansion of intercellular spaces, disorganization of the striated border, and nuclear pyknosis. In imidacloprid, carbaryl, and chlorpyrifos-treated groups, the epithelial tissue cells were deformed and had abnormal nuclei as compared with the control (Pervez & Manzoor, 2021). Microscopic analysis revealed damage to the honeybees' tissues, especially in the midgut and hypopharyngeal glands. Structural damages included degeneration of epithelial cells, vacuolation, and tissue disorganization, suggesting that sulfoxaflor causes cellular and tissue-level harm (Ibrahim et al., 2023). Chronic exposure to teflubenzuron causes significant structural damage to the bees' midgut tissue, including cellular degeneration, epithelial disorganization, and vacuolization. These histopathological alterations suggest that teflubenzuron exerts toxic effects on bee digestive health, potentially impairing nutrient absorption and overall bee vitality (Oliveira et al., 2024). Exposure to

a strobilurin causes histopathological (tissue-level) changes in key organs of honey bees, specifically the midgut and Malpighian tubules, which are vital for digestion and excretion (Batista et al., 2020). Azoxystrobin exposure induced significant histopathological alterations in the midgut tissue, including cellular degeneration and tissue disorganization. Cytotoxic effects were observed, such as damage to epithelial cells and the appearance of apoptotic features. These changes suggest that azoxystrobin, although classified as a fungicide, can have deleterious effects on non-target organisms, such as honey bees (Serra et al., 2023).

Genetic

Understanding how pesticides influence honey bee genetics is crucial for developing sustainable pest management practices and conservation strategies. Reducing the use of harmful pesticides and promoting bee-friendly alternatives can help preserve genetic diversity and promote colony health. Some pesticides have mutagenic properties, meaning they can cause mutations in bee DNA. These mutations may lead to developmental issues, reduced lifespan, or other health problems that can affect the colony's stability. Monitoring genetic markers over time can reveal shifts in population genetics due to pesticide exposure. The study by Forfert et al. (2017) investigates the impact of neonicotinoid pesticides on honeybee colonies. The researchers found that exposure to these pesticides can lead to a reduction in the genetic diversity within honeybee colonies. This decline in genetic variability may have implications for the resilience and health of bee populations, potentially making them more vulnerable to diseases and environmental stresses. Conversely, the genetic variation within bee populations plays a crucial role in determining their capacity to withstand pesticide exposure; populations with higher genetic diversity are often better equipped to adapt and survive, as they possess a broader range of genes that may confer resistance or tolerance. Studying these genetic interactions enables researchers to develop more effective strategies for protecting bee health and ensuring the sustainability of pollination services, which are essential for ecosystems and agriculture (Rinkevich et al., 2015; Astolfi et al., 2025).

Impact of pesticides on the behavior of honey bees

The impact of pesticides on honey bee behavior is a significant area of research, given the crucial role bees play in pollination and ecosystem health. Pesticides, especially neonicotinoids and other neurotoxic chemicals, can adversely affect honey bee behavior in several ways: impaired navigation and foraging, reduced communication, altered learning and memory, changes in activity levels, and disruption of social behaviors. Overall, pesticide-induced behavioral changes can contribute to colony decline and have broader ecological consequences. Sublethal doses of

pesticides, such as neonicotinoids and organophosphates, can alter bee behavior (Pham-Delègue et al., 2002). Cabirol and Haase (2019) investigate how neonicotinoid pesticides affect honeybee behavior through neurophysiological mechanisms. The study reviews evidence indicating that neonicotinoids bind to nicotinic acetylcholine receptors in the bee's nervous system, disrupting normal neural signaling. This disruption impairs key behaviors such as foraging, navigation, learning, and memory, which are vital for colony health and survival. The authors highlight that these neurophysiological effects contribute to the decline in bee populations and emphasize the need for further research to understand the impacts of pesticides and develop strategies to mitigate harm to pollinators. The study by Ayoub et al. (2024) investigates the effects of chlorpyrifos, dimethoate, and profenophos on honeybees (*A. mellifera*). The researchers found that exposure to these insecticides leads to significant behavioral alterations in bees, such as impaired movement and foraging activities. Understanding the behavior of pesticides in the environment is crucial for developing safer and more sustainable agricultural practices. This includes studying their environmental fate (such as degradation, mobility, bioaccumulation, and persistence) which informs efforts to improve their synthesis and formulation. By gaining insights into these aspects, researchers can design pesticides that are effective against pests but less harmful to non-target organisms and the environment, ultimately leading to improved synthesis methods that prioritize environmental safety and reduce ecological impact (El-Aswad et al., 2023b; Fouad, 2023a-e; Fouad et al., 2023b-c; Fouad et al., 2024d-h).

Counteracting the negative effects of pesticides on beehives

Counteracting the negative effects of pesticides on beehives has become an important area of research in apiculture. One approach involves implementing management measures that help bees recover and maintain their health despite exposure to pesticides. For instance, administering probiotics to bees can enhance their gut microbiota, which may be disrupted by chemical contaminants, thereby improving their immune response and resilience. Additionally, providing supplementary diets rich in essential nutrients can help strengthen bees' overall health and detoxification capacity. These strategies aim to mitigate the sub-lethal effects of pesticides, which can impair foraging behavior, reduce colony productivity, and increase mortality rates. Researchers are exploring various probiotic strains and nutritional formulations to optimize their protective effects. The integration of these management practices into regular hive maintenance offers a promising way to support bee populations amid ongoing pesticide use. Several studies and references highlight the potential benefits of such interventions, providing a scientific foundation for developing sustainable beekeeping practices that safeguard bee health in pesticide-affected environments (Chmiel et al., 2020; Syama and CV, 2022).

Integrated pest and pollinator management

Integrated pest and pollinator management (IPPM) is a comprehensive approach that aims to optimize crop health by balancing pest control with the conservation of pollinators. This strategy involves the simultaneous management of pest populations while protecting beneficial insects such as bees, butterflies, and other pollinators that are essential for crop productivity. IPPM emphasizes the use of environmentally friendly methods, including biological control, habitat manipulation, and selective use of pesticides, to reduce negative impacts on pollinator populations. By monitoring pest levels through regular scouting and employing targeted interventions, farmers can minimize chemical inputs and prevent unnecessary harm to pollinators. The approach also encourages the planting of pollinator-friendly habitats, such as flowering cover crops and wildflower strips, to support pollinator health and diversity. Education and awareness are key components of IPPM, helping farmers understand the importance of pollinators and how to protect them. This integrated management reduces the reliance on broad-spectrum pesticides, which often cause collateral damage to beneficial insects. It promotes sustainable agriculture by maintaining ecological balance and enhancing crop yields. Furthermore, IPPM aligns with environmental conservation goals and supports biodiversity within agricultural landscapes. Overall, the implementation of IPPM leads to healthier ecosystems, more resilient farming systems, and increased productivity through the harmonious coexistence of pest control and pollinator conservation (Egan et al., 2020; Lundin et al., 2021; Mukhtar & Shankar, 2023; Phan et al., 2025).

Conclusions

The impact of pesticides on honey bees has significant ecological and economic implications. Evidence indicates that certain pesticides, particularly neonicotinoids and other systemic chemicals, can impair honey bee navigation, foraging behavior, reproduction, and immune systems. These effects contribute to colony declines and increased vulnerability to diseases and environmental stresses. The decline of honey bee populations poses a threat to pollination services, which are essential for the production of many fruits, vegetables, and nuts, thereby affecting global food security. To mitigate these impacts, it is crucial to implement integrated pest management practices, restrict or ban the use of harmful pesticides during key pollination periods, and promote the development of bee-friendly pest control alternatives. Protecting honey bees from pesticide-related harm is vital for maintaining biodiversity, ecosystem health, and sustainable agriculture.

Data availability

The datasets generated and analyzed during the current study are available upon request to the corresponding author.

Declaration of Competing Interest

The author has no conflicts of interest to declare.

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