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Plastic Micro and Nanoparticles and PFAS as Emerging Threats to Managed and Wild Bees

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Article

Keywords: microplastics, nanoplastics, PFAS, honey bees, wild bees, pollinators, environmental, persistent pollutants

Posted Date: November 25th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11714423/v1

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Additional Declarations: No competing interests reported.

Abstract

Microplastics (MPs) and per- and polyfluoroalkyl substances (PFAS) are worldwide persistent pollutants now found in bees and hive products, yet their consequences for pollinator health remain poorly understood. Evidence is accumulating rapidly. This systematic review synthesises evidence from 37 studies and reveals a strong research bias toward microplastics and honey bees, with comparatively limited evidence for PFAS exposure and non-Apis bee species. Both contaminant classes have been associated with alterations in gut integrity, microbial communities and immune-related pathways, although the strength of evidence varies between contaminants. Microplastic co-exposure with pesticides and pathogens may exacerbate adverse health effects, depending on the exposure conditions. PFAS additionally impair reproduction and development across generations. This review highlights key knowledge gaps concerning wild bees, realistic exposures and contaminant mixtures to guide future research and pollinator risk assessment.

Introduction

Pollinators are essential for maintaining terrestrial biodiversity and supporting agricultural productivity by enabling the reproduction of wild flowering plants and numerous cultivated crops [1, 2]. In particular, managed honey bees (*Apis mellifera* Linnaeus, 1758) and wild bee species provide critical ecosystem services; their decline is now a major global concern, driven by the combined effects of habitat loss, climate change, pathogens, pesticides and other anthropogenic stressors [3–6]. While these drivers have traditionally received the greatest scientific attention, recent research has shifted to emerging environmental contaminants whose distribution, exposure pathways and effects on pollinator health remain insufficiently understood [6]. Among these, microplastics (MPs) and per- and polyfluoroalkyl substances (PFAS) are of particular concern because of their environmental persistence, widespread distribution and presence across multiple environmental compartments, which create several potential routes of exposure for pollinators [7–9]. Growing evidence indicates that managed bees are exposed to microplastics across diverse environmental settings, while emerging studies have also documented PFAS occurrence in bees and bee-collected pollen. [8, 9]. Environmental monitoring studies have detected MPs in foraging bees, hive bees, pollen, honey, and other hive-associated matrices from urban, peri-urban, and agricultural landscapes [9]. Together, these findings suggest that honey bee colonies can serve as effective biomonitoring systems, integrating contamination from their surrounding environment [10–13]. Similarly, PFAS have recently been detected in forager bees, hive bees and bee-collected pollen, showing that fluorinated contaminants can also enter pollinator-associated matrices [9]. Multiple exposure pathways have been proposed, including direct environmental contamination, transfer through contaminated pollen, dispersion within colonies via social interactions, and materials used in beekeeping management, which highlight the complexity of the circulation of those contaminants within bee colonies [13, 14]. Beyond documenting environmental occurrence, recent experimental studies increasingly show that MPs and PFAS act on common physiological targets in bees. Microplastic exposure has been associated with disruption of gut integrity, microbial dysbiosis, oxidative stress, altered detoxification pathways, impaired immune responses and reduced survival, and several studies have also reported effects on sucrose responsiveness, learning, memory and social recognition [15–28]. Increasing evidence further indicates that contaminant mixtures frequently modify toxicity, producing additive, synergistic or even antagonistic interactions depending on the combination of stressors and polymers involved, meaning that assessments based solely on single contaminants may underestimate or misrepresent environmental risks [19, 24]. Knowledge of PFAS toxicity in bees is more limited than for MPs, but it is expanding rapidly. Available studies demonstrate that PFAS accumulate in bee tissues and colony products, impair gut barrier integrity, alter immune

and detoxification pathways, disrupt energy metabolism, modify gut microbial communities and induce developmental, reproductive and colony-level effects. In addition, recent evidence points to intergenerational consequences despite limited short-term impacts on colony performance [29–34]. Collectively, these findings suggest that MPs and PFAS converge on common biological pathways involving the gut microbiota-immune axis, while acting through multiple environmental and colony-level exposure routes. However, differences among bee species, contaminants, experimental designs, exposure concentrations, and measured endpoints make it difficult to draw general conclusions about their effects on bee health. Despite increasing research interest, evidence for both contaminant classes remains fragmented across managed and wild bee species and across levels of biological organisation, from molecular and physiological responses to behavioural, reproductive, and colony-level effects. This systematic review, therefore, aimed to evaluate and synthesise the available scientific evidence on the occurrence, exposure pathways, and effects of MPs and PFAS on the health of managed and wild bees, and to identify major knowledge gaps and priorities for future research and pollinator risk assessment.

Material and Methods

The review protocol was developed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The review addressed the following research question: *What is the effect of exposure to microplastics and PFAS on the health of managed and wild bees?* The question was formulated using the PEO framework [35]: Population (P): managed and wild bees; Exposure (E): exposure to microplastics (MPs) and/or per- and polyfluoroalkyl substances (PFAS); Outcome (O): health-related effects. A comprehensive literature search was carried out on 13 March 2026 in three electronic databases: PubMed, Scopus and Web of Science. Search strategies were tailored to each database using combinations of controlled vocabulary (when available) and free-text terms related to bee species, MPs, PFAS, exposure, and health outcomes. Searches covered titles, abstracts and keywords (or topic fields, depending on the database).

All retrieved references were imported into Rayyan for duplicate removal and study screening. Two reviewers independently screened titles and abstracts, resolving disagreements by consensus. Potentially eligible articles then underwent full-text assessment against predefined inclusion and exclusion criteria, which were likewise performed independently by two reviewers, with disagreements resolved by consensus.

Results

The literature search identified 1779 records, including 144 from PubMed, 617 from Web of Science, and 1018 from Scopus. After duplicate removal, 1279 unique records remained for title and abstract screening. Following independent screening by two reviewers, 142 articles were considered potentially eligible and underwent full-text assessment, after which 37 studies met the eligibility criteria and were included in the qualitative synthesis (Fig. 1). Of these, 30 studies investigated microplastics (MPs) and nanoplastics (NPs), whereas 7 focused on per- and polyfluoroalkyl substances (PFASs) (Table S1).

The scientific interest in these emerging contaminants is relatively recent, as nearly all of the included studies were conducted and/or published within the last decade. The only exception was the study by [29] which evaluated the effects of PFOS exposure in *Bombus terrestris* worker bees and represents the earliest investigation identified in this review.

The included studies adopted two main methodological approaches: field monitoring (Table 1) and experimental exposure (Table 2). Twelve studies assessed environmental contamination under field conditions; only two of these investigated PFAS occurrences, whereas the remaining studies focused on MPs. In contrast, 27 studies evaluated the effects of contaminant exposure through controlled experimental assays, almost all performed under laboratory conditions and only one under field conditions. Among the experimental studies, 21 investigated the effects of MPs, confirming the greater research attention devoted to MPs than to PFAS (Table S1).

Table 1

Monitoring studies reporting the presence of microplastics and PFAS in bees. Studies are grouped by contaminants and listed from the most recent to the oldest.

Contaminant	Continent	Country	Species	Study design / analytical method	Main quantitative results	Reference
Microplastics and nanoplastics						
PET, PE, EVA, PA, PP	Asia	Türkiye	*A. mellifera* (forager bees)	Field monitoring; H_2O_2 digestion, ATR-FTIR	Urban: 0.28 ± 0.04 MPs/bee; rural: 0.15 ± 0.09 MPs/bee	[45]
PET, PAN	South America	Argentina	*A. mellifera* (forager bees, nurse bees); honey	Field monitoring + behavioural experiments; Raman	0.16 MF/forager; 0.13 MF/nurse; honey: 20.6 ± 7.5 MF/500 g	[13]
PE	Asia	Malaysia	*Heterotrigona itama* (honey); *A. mellifera* (honey)	Field monitoring; H_2O_2 digestion, SEM, ATR-FTIR	8.18 ± 2.57 MPs/g (*H. itama*); 5.52 ± 1.13 MPs/g (*A. mellifera*)	[10]
PE, PET, and others	South America	Ecuador	*A. mellifera* (forager bees)	Field monitoring; Nile Red, fluorescence microscopy, ATR-FTIR	24 MPs/50 bees (0.48 MPs/bee)	[44]
PET, PP, PE, PAN, PA	Europe	Denmark	*A. mellifera* (worker bees); pollen	Field monitoring; APITrap + honeybees; stereomicroscopy, μ-FTIR	APITrap: 39–67 MPs/sample; bees: 6–9 MPs/5 g; pollen: 6–11 MPs/5 g	[11]
PET, PA, PE, PL, PVC	South America	Brazil	*A. mellifera* (worker bees)	Field monitoring; washing + H_2O_2 digestion, Micro-Raman	505 MPs detected (307 fibres, 137 fragments, 61 filaments)	[46]
PET, PE, PA, PAN, PTFE and others	Europe	Italy	*A. mellifera* (worker bees, forager bees); honey; pollen	Field monitoring; digestion, filtration, μ-FTIR	Honeybees: 178 particles; honey: 104 particles	[8]
PET	North America	USA (Hawaii)	*A. mellifera* (worker bees); honey	Field monitoring; stereomicroscopy, Raman	Bees: 4.7 ± 2.28 MF/20 bees; honey: 11.83 ± 3.76 MF/20 g (treated colonies)	[14]
PC, PET, PE, PS	Asia	China (6 provinces)	*A. mellifera* (worker bees); *A.*	Field monitoring, experimental laboratory	66.7% positive samples; 1–2	[22]

Contaminant	Continent	Country	Species	Study design / analytical method	Main quantitative results	Reference
Microplastics and nanoplastics						
			cerana (worker bees)	exposure; digestion, Raman microscopy	MPs/0.5 g pooled bees	
PET, PE, PA, PVC and others	Europe	Denmark	*A. mellifera* (worker bees)	Field monitoring; washing, filtration, stereomicroscopy, μ-FTIR	56 confirmed MPs (125 suspected)	[7]
Per- and polyfluoroalkyl substances (PFAS)						
PFOA	Asia	China (5 provinces)	*A. c. cerana* (forager bees); *A. mellifera* (forager bees)	Field monitoring, experimental laboratory exposure; LC-MS	Occurrence confirmed; no environmental concentrations reported	[32]
PFAS (30-target panel; 9 detected)	Europe	Austria	*A. mellifera* (worker bees, forager bees); pollen	Seasonal field monitoring; LC-ESI-MS/MS (MRM), CIC	ΣPFAS: hive bees 5.29 ng/g d.w.; forager bees 1.93 ng/g d.w.; pollen 1.10 ng/g d.w	[9]

Table 2

Summary of experimental studies on the effects of microplastics and PFAS on bees. Studies are grouped by contaminants and listed from the most recent to the oldest.

Species	Contaminant and methodology	Concentration	Effects	Observations	Reference
Microplastics and nanoplastics					
A. m. ligustica (newly-emerged bees)	PE, PMMA, PS (single and combined) Lab; oral	50 mg/L	↑ food intake; gut microbiota altered; ↑ mortality (PMMA and polymer mixtures)	*L. melliventris* changed over time in all MP treatments; *B. asteroides* decreased with PE + PS+PMMA; triple co-exposure substantially affected lifespan, food consumption and gut microbiota	[20]
A. mellifera (newly-emerged bees)	PS (0.5, 5 µm) + FPF Lab; oral; 21 d	50 mg/L (FPF 4 mg/L)	↓ Body weight (5 µm); midgut damage and dysbiosis with FPF; additive effect on survival	Additive PS × FPF interaction on survival; *L. helsingborgensis* supplementation increased survival in FPF and PS + FPF groups	[17]
B. terrestris (worker bees)	PS, PMMA, mixture Lab; oral	0.5, 5, 50 mg/L	↓ Survival, learning and memory (single polymers); antagonistic effect of mixture	Antagonistic interaction: combined exposure mitigated or cancelled the negative effects of single polymers	[36]
A. mellifera (newly-emerged bees)	PS NPs (50–5000 nm) ± JNCC Lab; oral; ≤ 10 d	50 mg/L	↓ Survival; ↑ apoptosis and detoxification enzymes; ↓ *G. apicola*; effects mitigated by JNCC	JNCC alone had a prebiotic effect; PS + JNCC reduced toxicity and restored *G. apicola* and lipid metabolism; PS + JNCC + *G. apicola* gave near-complete restoration	[47]
A. mellifera (newly-emerged bees)	PS (0.5, 5 µm) Lab; oral; 21 d	25, 50 mg/L	↓ Survival and food intake; gut damage; dysbiosis; metabolic disruption	Gut accumulation of PS linked microbiota disruption and metabolic imbalance to reduced survival	[18]
A. mellifera	PS + Dif	5 mg/L (Dif 50 mg/L)	PS alone: minor effects; co-	Co-exposure produced fewer	[42]

NOTE: ↑, increase; ↓, decrease; d, days; wk, weeks; Lab, laboratory; n.r., not reported; CHC, cuticular hydrocarbons; Cy, cyfluthrin; DEGs, differentially expressed genes; DEHP, di-2-ethylhexyl phthalate; Dif, difenoconazole; DnBP, di-n-butyl phthalate; FPF, flupyradifurone; GLY, glyphosate; HPG, hypopharyngeal glands; IAPV, Israeli acute paralysis virus; JNCC, jute nanocrystalline cellulose; LC_{50}, median lethal concentration; MFs, microfibres; NPs, nanoplastics; PE, polyethylene; PET, polyethylene terephthalate; PFHxS, perfluorohexanesulfonic acid; PFOA, perfluorooctanoic acid; PFOS, perfluorooctanesulfonic acid; PMMA, poly(methyl methacrylate); PS, polystyrene; BAF, bioaccumulation factor.

Species	Contaminant and methodology	Concentration	Effects	Observations	Reference
Microplastics and nanoplastics					
(newly-emerged bees)	Lab; oral; 13–14 d		exposure: ↓ sucrose responsiveness, stronger dysbiosis	DEGs than Dif alone and partially mitigated oxidative damage	
A. c. cerana (newly-emerged bees)	NanoPS + Cy Lab; oral	0.5, 5, 50 mg/L (Cy 0.2 mg/L)	No effect on survival; midgut damage; HPG atrophy; altered immune and detoxification genes	NanoPS reduced Cy-induced mortality; co-exposure enhanced changes in P450 genes	[19]
B. atratus (forager bees)	Paint-derived MPs (pristine, photodegraded) Lab; oral; 96 h	50 mg/L	Cellular and DNA damage, ↓ body weight (mainly photodegraded MPs)	Pristine MPs caused subtle effects; photodegradation increased toxicity through release of incorporated substances (Cu, Mn and Fe exceeded safety thresholds)	[38]
A. mellifera (forager bees)	PS, PMMA, mixture Lab; oral	0.5, 5, 50 mg/L	↓ Survival (mixture); ↓ immune response; altered CHC; impaired nestmate recognition	Additive rather than synergistic effects of PS and PMMA on survival and immunity; nest guards unable to detect exposed foragers	[24]
A. mellifera (larvae)	Nylon (80 µm) + GLY Lab; in vitro larval rearing	50, 500, 1000 mg/L (GLY 5 mg/L)	MPs alone mostly no effect; synergistic ↓ survival with GLY; immune genes ↓	MPs alone mostly without significant effects; MPs enhanced GLY toxicity (synergistic effect)	[40]
A. m. ligustica (newly-emerged bees)	PS (~ 5 µm) + FPF + *N. ceranae* Lab; oral; chronic	50 mg/L	↓ Survival (strongest under triple exposure); ↑ *N. ceranae*; midgut lesions	Highest mortality risk under triple exposure; synergistic effect between biotic and abiotic stressors	[26]
A. mellifera (worker bees)	PE (1–100 µm) ± *H. alvei* Lab; oral	10^3–10^5 particles/mL	↑ Mortality (size-dependent); gut damage; dysbiosis; ↑	MPs retained 4 h in the gut and 3 days in the rectum; damage due to combined mechanical gut	[25]

NOTE: ↑, increase; ↓, decrease; d, days; wk, weeks; Lab, laboratory; n.r., not reported; CHC, cuticular hydrocarbons; Cy, cyfluthrin; DEGs, differentially expressed genes; DEHP, di-2-ethylhexyl phthalate; Dif, difenoconazole; DnBP, di-n-butyl phthalate; FPF, flupyradifurone; GLY, glyphosate; HPG, hypopharyngeal glands; IAPV, Israeli acute paralysis virus; JNCC, jute nanocrystalline cellulose; LC_{50}, median lethal concentration; MFs, microfibres; NPs, nanoplastics; PE, polyethylene; PET, polyethylene terephthalate; PFHxS, perfluorohexanesulfonic acid; PFOA, perfluorooctanoic acid; PFOS, perfluorooctanesulfonic acid; PMMA, poly(methyl methacrylate); PS, polystyrene; BAF, bioaccumulation factor.

Species	Contaminant and methodology	Concentration	Effects	Observations	Reference
Microplastics and nanoplastics					
			pathogen susceptibility	injury and microbial dysbiosis	
A. mellifera (nurse bees)	PS fragments (27, 93 µm) Lab; oral; chronic	1, 10, 100 µg/mL	No effect on survival; ↓ food intake and body weight	No effect of PS fragments on survival, but decreased feeding rate and body weight	[41]
A. m. carnica (colonies)	Polyester MFs Colony feeding; 1 month	50 mg/L	MF transfer to larvae, wax and honey; no colony-level effects	MFs incorporated into the hive from food, by ingestion or cuticle adhesion; highest proportion in wax; MF levels in honey similar to those reported in other foods	[12]
A. m. ligustica (forager bees)	PS, PMMA, mixture Lab; oral; 2–3 d	0.5, 5, 50 mg/L	↓ Sucrose responsiveness; impaired learning and memory; MPs detected in brain	Fluorescent MPs detected in the brain, especially in the optic lobes, after 3 days of oral exposure	[23]
Partamona helleri (larvae)	PS, PET Lab; larval feeding	500 ng/bee	No effect on survival; altered adult behaviour and hemocyte counts	Low larval exposure induced long-term sublethal physiological, behavioural and immune effects without affecting survival	[39]
A. m. ligustica (forager bees)	PE Lab; oral; 1 and 7 d	0.5, 5, 50 mg/L	↑ Mortality at 50 mg/L; ↓ response consistency; learning and memory unaffected	Dose-dependent effects: mortality only at high concentration; sublethal exposure induced behavioural inconsistency without impairing learning and memory	[16]
B. terrestris (worker bees)	PS Lab; oral	40 mg/40 mL	MPs visualised in crop, midgut and hindgut (methodological study)	Protocol enables study of pollinator exposure under realistic conditions and trophic transfer	[37]

NOTE: ↑, increase; ↓, decrease; d, days; wk, weeks; Lab, laboratory; n.r., not reported; CHC, cuticular hydrocarbons; Cy, cyfluthrin; DEGs, differentially expressed genes; DEHP, di-2-ethylhexyl phthalate; Dif, difenoconazole; DnBP, di-n-butyl phthalate; FPF, flupyradifurone; GLY, glyphosate; HPG, hypopharyngeal glands; IAPV, Israeli acute paralysis virus; JNCC, jute nanocrystalline cellulose; LC_{50}, median lethal concentration; MFs, microfibres; NPs, nanoplastics; PE, polyethylene; PET, polyethylene terephthalate; PFHxS, perfluorohexanesulfonic acid; PFOA, perfluorooctanoic acid; PFOS, perfluorooctanesulfonic acid; PMMA, poly(methyl methacrylate); PS, polystyrene; BAF, bioaccumulation factor.

Species	Contaminant and methodology	Concentration	Effects	Observations	Reference
Microplastics and nanoplastics					
				from contaminated nectar/pollen	
A. mellifera (worker bees)	PS (100 nm–10 µm) ± *H. alvei* Lab; oral; 15 d	10^4–10^5 particles/mL	↓ Body weight, gut damage and dysbiosis (100 nm); ↑ pathogen susceptibility	10-µm PS accumulated in the rectum (days 3–15); 100-nm PS increased susceptibility to *H. alvei*	[21]
A. mellifera, *A. cerana* (worker bees, newly-emerged bees)	PS (0.5–50 µm) ± IAPV Lab; oral	0.1, 1, 1, 100 mg/L	↓ Survival; gut lesions; translocation beyond the gut; ↑ IAPV titre	0.5 µm PS more harmful than 5 µm; both sizes crossed the gut barrier (hemolymph, tracheae, Malpighian tubules); PS increased IAPV titre > 4-fold	[22]
A. mellifera (newly-emerged bees)	PS (~ 25 µm) ± tetracycline Lab; oral; 14 d	0.5, 5, 50 mg/L	Low mortality; dysbiosis; stress-gene changes; ↑ lethality with antibiotic	MPs accumulated in the hindgut and were partially degraded; microbiota depletion by tetracycline increased MP lethality	[15]
Per- and polyfluoroalkyl substances (PFAS)					
A. m. ligustica (worker bees)	PFOS Colony; oral; 25 d	5, 10, 15, 20 µg/L	No effect on colony survival; developmental and intergenerational effects; altered protein expression	PFOS accumulated in bee tissue and was transferred to honey and faeces	[34]
A. mellifera (worker bees)	PFOA, PFHxS Lab; oral; ≤ 10 d	0.5, 5, 50 µg/L	No effect on survival; midgut injury, inflammation and apoptosis	Curcumin restored midgut integrity; environmentally relevant PFAS concentrations could compromise honey bee gut health	[33]
A. c. cerana (forager bees)	PFOA Lab; oral; 8 d	0.1, 1 mg/L	↓ Survival (1 mg/L) and body weight; gut damage; dysbiosis;	PFOA accumulated mainly in the gut (gut > thorax > abdomen > head >	[32]

NOTE: ↑, increase; ↓, decrease; d, days; wk, weeks; Lab, laboratory; n.r., not reported; CHC, cuticular hydrocarbons; Cy, cyfluthrin; DEGs, differentially expressed genes; DEHP, di-2-ethylhexyl phthalate; Dif, difenoconazole; DnBP, di-n-butyl phthalate; FPF, flupyradifurone; GLY, glyphosate; HPG, hypopharyngeal glands; IAPV, Israeli acute paralysis virus; JNCC, jute nanocrystalline cellulose; LC_{50}, median lethal concentration; MFs, microfibres; NPs, nanoplastics; PE, polyethylene; PET, polyethylene terephthalate; PFHxS, perfluorohexanesulfonic acid; PFOA, perfluorooctanoic acid; PFOS, perfluorooctanesulfonic acid; PMMA, poly(methyl methacrylate); PS, polystyrene; BAF, bioaccumulation factor.

Species	Contaminant and methodology	Concentration	Effects	Observations	Reference
Microplastics and nanoplastics					
			accumulation mainly in gut	antennae). Field data in Table 1	
A. mellifera (worker bees)	PFOS Lab; Y-maze choice	0.02, 30, 61, 103 µg/L	PFOS consumed at all concentrations; weak avoidance	Bees can display PFOS avoidance but consume PFOS at high concentrations when no alternative food is available	[31]
A. mellifera (worker bees, forager bees, honey, feces)	PFOS Field colonies; oral	0.02, 0.08, 0.2, 0.3, 0.8, 1.6 mg/L	↑ Mortality at ≥ 0.8 mg/L; brood failure and behavioural changes at ≥ 0.02 mg/L	PFOS detected in all bee tissues, honey and faeces; seven additional PFAS (including PFOA) at lower concentrations	[30]
B. terrestris (worker bees)	PFOS Lab; oral; ≤ 11 wk	1, 2, 5, 10 mg/L, 1, 10, 100 µg/L	↑ Mortality (LC_{50} = 1.01 mg/L); no drone production; bioaccumulation	Strong bioaccumulation (2184 ± 365 ng/g at 100 µg/L; BAF = 27.9); antagonistic activity on the ecdysteroid receptor	[29]

NOTE: ↑, increase; ↓, decrease; d, days; wk, weeks; Lab, laboratory; n.r., not reported; CHC, cuticular hydrocarbons; Cy, cyfluthrin; DEGs, differentially expressed genes; DEHP, di-2-ethylhexyl phthalate; Dif, difenoconazole; DnBP, di-n-butyl phthalate; FPF, flupyradifurone; GLY, glyphosate; HPG, hypopharyngeal glands; IAPV, Israeli acute paralysis virus; JNCC, jute nanocrystalline cellulose; LC_{50}, median lethal concentration; MFs, microfibres; NPs, nanoplastics; PE, polyethylene; PET, polyethylene terephthalate; PFHxS, perfluorohexanesulfonic acid; PFOA, perfluorooctanoic acid; PFOS, perfluorooctanesulfonic acid; PMMA, poly(methyl methacrylate); PS, polystyrene; BAF, bioaccumulation factor.

The geographical distribution of the studies was global, spanning five continents (Fig. 2). Asia and Europe each contributed the largest number of publications (n = 13), followed by South America (n = 8), Australia (n = 3), and North America (n = 1). By contrast, PFAS-related studies had a more limited geographical distribution, as they were conducted exclusively in China, Belgium, Austria, and Australia (Table S1).

Most studies focused on bees of the genus *Apis*, particularly *A. mellifera* Linnaeus, 1758 and *A. cerana* Fabricius, 1793. Regarding western honey bees, *A. m. ligustica* Spinola, 1806 [16] and *A. m. carnica* Pollmann, 1879 [12] were the only subspecies investigated. Bumblebees were examined in only four studies, specifically *B. terrestris* (Linnaeus, 1758) [29, 36, 37] and *B. atratus* Franklin, 1913 [38]. Wild bees were markedly underrepresented, with only *Partamona helleri* (Friese, 1900) and *Heterotrigona itama* (Cockerell, 1918) being investigated, exclusively in two studies on MPs [10, 39].

Workers were the social caste most commonly considered across the included studies. Several investigations also evaluated contaminant occurrence in larvae, pollen, honey, wax, and bee faeces, providing additional insights into contaminant distribution within colonies and hive products.

The distribution of contaminants across bee taxa and biological matrices is summarised in Fig. 3, while Fig. 4 provides an overview of the environmental exposure pathways, within-colony transfer, and main effects of MPs/NPs and PFAS on bee health.

Microplastics

Studies of MPs in pollinators included laboratory exposure experiments, field studies and environmental monitoring [7, 8, 11–13]. Experimental studies primarily investigated polystyrene (PS), polyethylene (PE), polyethylene terephthalate (PET) and polymethyl methacrylate (PMMA), either individually or in mixtures [16, 20, 24, 39]. When expressed as mass concentrations in the exposure medium, doses ranged from 0.1 to 1000 mg/L, with 50 mg/L being one of the most frequently tested concentrations [16, 20, 22, 24, 40]. Particle sizes varied considerably among studies, ranging from the nanometer to the micrometre scale [21, 22, 25, 41]. Following ingestion, MPs were detected throughout the gastrointestinal tract, including the crop, midgut and rectum [22], and smaller particles were also able to cross the gut barrier and reach extra-intestinal compartments, including the hemolymph, tracheae and Malpighian tubules [23]. Fluorescent PS and PMMA particles were also detected in brain tissues, particularly in the optic lobes [23].

In honey bees, MP exposure affected survival in a concentration-, size- and polymer-dependent manner. Reduced survival was reported following PS exposure under several experimental conditions [18, 22], whereas PE increased mortality only at the highest tested concentration [16]. Food consumption was also reduced after PS exposure and under combined MP treatments [18, 20, 41], whereas it increased under combined MP treatments [20] and after low-dose PE exposure [16]. Body-weight reductions were observed under specific PS exposure conditions [21, 41], and gut microbiota alterations were frequently reported, involving shifts in core taxa such as *Lactobacillus*, *Bifidobacterium*, *Snodgrassella* and *Gilliamella* [15, 18, 21, 25]. Histopathological damage included microvilli disruption, basement membrane alterations, thinning of the gut wall, and impaired peritrophic membrane integrity [21, 22, 26]. MPs also altered lipid, amino acid and carbohydrate metabolism and affected detoxification- and immunity-related pathways [18, 21, 22]. Co-exposure to MPs and pesticides or tetracyclines generally enhanced the negative effects on survival, midgut microbiota and midgut histology [15, 17, 40, 42], although nanoPS reduced cyfluthrin-induced mortality in *A. cerana* [19]. Conversely, supplementation with the probiotic *Lactobacillus helsingborgensis* or with jute nanocrystalline cellulose mitigated MP-induced effects [17, 22]. Co-exposure of MPs with honey bee pathogens increased pathogen load and susceptibility: PS exposure increased IAPV titres, while co-exposure with *Nosema* (= *Vairimorpha*) *ceranae* increased pathogen abundance; MP exposure also increased susceptibility to the opportunistic pathogen *Hafnia alvei* [21, 22, 25, 26]. Furthermore, triple exposure to PS, flupyradifurone and *N. ceranae* reduced bee longevity, decreased regenerative cell nests, and increased compacted chromatin [36]. In bumblebees, PS and PMMA reduced survival and impaired memory, whereas their mixture showed antagonistic effects [39]. In *Partamona helleri*, by contrast, larval exposure to PET and PS did not reduce survival but altered adult behaviour and hemocyte composition [39].

PFAS

PFAS studies predominantly focused on *A. mellifera*, with only one laboratory study on *A. cerana* [32] and two on *B. terrestris* [29, 43]. Most experimental evidence concerned PFOS, whose toxicity was first demonstrated in laboratory microcolonies of *B. terrestris*, where chronic oral exposure caused concentration-dependent mortality, reaching 100% at ≥ 2 mg/L (LC_{50} = 1.01 mg/L), and completely suppressed drone production at ≥ 1 mg/L. PFOS

also accumulated in workers (2184 ± 365 ng/g at 100 µg/L; BAF = 27.9) and significantly reduced mitochondrial activity and lipid reserves [29]. Consistently, colony-level experiments in *A. mellifera* showed increased worker mortality at ≥ 0.8 mg/L, reaching 75.6 ± 3.82% at 1.6 mg/L, while brood development ceased at concentrations as low as 0.02 mg/L, a concentration at which behavioural alterations in colony activity, temperament, hive maintenance and defence behaviour. PFOS accumulated in bee tissues and were transferred to honey and faeces. Notably, subsequent experiments at lower concentrations (5–20 µg/L) showed no overt effects on adult colony survival but revealed developmental and intergenerational effects even at 5 µg/L, including reduced body weight in newly emerged bees (0.094 ± 0.001 *vs.* 0.1066 ± 0.001 g in the controls) [34]. This potential for chronic exposure is further supported by behavioural assays, in which ≥ 80% of bees consumed PFOS-contaminated syrup, with avoidance occurring only at 61–103 µg/L when an uncontaminated alternative was available [31]. Evidence for other PFAS similarly indicated predominantly sublethal effects: PFOA (0.1–1.0 mg/L) reduced survival and body weight and impaired gut integrity, immunity and microbiota in *A. cerana* [32], while PFOA and perfluorohexanesulfonic acid (PFHxS) (0.5–50 µg/L) induced midgut injury and altered barrier, inflammatory and apoptotic markers in *A. mellifera*, despite low acute toxicity (LC_{50} = 266–331 mg/L) [33]. Field data on environmental occurrence and distribution remain very limited compared with the evidence on PFAS toxicity in bees. The most comprehensive quantitative field evidence currently comes from a seasonal biomonitoring study in Austria, in which 30 PFAS were targeted in *A. mellifera* and bee-collected pollen. Nine PFAS were detected across 90 samples collected from six sites representing different land-use types, with mean concentrations of 5.29, 1.93 and 1.10 ng/g in hive bees, foragers and pollen, respectively [9]. This monitoring evidence confirms that bees may be exposed to complex PFAS mixtures under field conditions and suggests multiple potential exposure routes.

Discussion

This review examines the effects of two classes of emerging contaminants, MPs and PFAS, on the health of managed and wild bees. Despite their distinct physicochemical properties, evidence from the studies reviewed here indicate that MPs and PFAS affect several overlapping biological pathways in bees. Across independent studies, oral exposure to MPs consistently disrupted the peritrophic membrane, induced gut epithelial degeneration, promoted oxidative imbalance and caused microbial dysbiosis [15, 18, 21, 25]. Comparable alterations were also described following oral exposure to PFAS, including impairment of gut barrier integrity, suppression of digestive enzyme activity, and changes in microbial community composition [32, 33]. Immune pathways appeared to be similarly affected by both types of exposure [22, 32]. The remarkable consistency of these responses supports the hypothesis that disruption of the gut–microbiota–immune axis constitutes a shared toxicological pathway through which chemically distinct contaminants may affect bee health.

Several studies have also investigated the effects of MPs and PFAS on bee behaviour. Collectively, their findings indicate that behavioural toxicity cannot be predicted solely by the presence of a contaminant but is strongly influenced by polymer identity, particle characteristics, exposure scenario, and contaminant interactions. For MPs, acute PE exposure had limited effects on associative learning but reduced behavioural consistency [16], whereas PS and PMMA impaired sucrose responsiveness, olfactory learning and memory, with fluorescent imaging confirming the translocation of MPs into the brain [23]. In *A. mellifera*, MPs impaired nestmate recognition [24], whereas experiments on *B. terrestris* showed that combined exposure to PS and PMMA unexpectedly mitigated some behavioural impairments through antagonistic interactions [36]. PFAS exposure also affected behaviour in honey bees, with colony activity, temperament, hive maintenance and defence adversely affected even at the lowest tested PFOS concentration of 0.02 mg/L [30].

An important contribution of recent studies is the demonstration that contaminant interactions are not uniformly synergistic. Additive, antagonistic or synergistic effects have been reported following co-exposure of MPs or NPs with pesticides, pathogens or antibiotics, resulting in increased mortality, gut damage and immune suppression [15, 17, 19, 26, 40]. In contrast, simultaneous exposure to different polymer types sometimes produced antagonistic responses that partially alleviated behavioural impairment [36]. These contrasting outcomes indicate that mixture toxicity is highly endpoint-dependent and highlight the limitations of ecological risk assessments based exclusively on single contaminants. Furthermore, the protective effects of *L. helsingborgensis* supplementation [17] suggest that host-associated factors, particularly gut microbiota composition, can substantially modulate contaminant toxicity under environmentally relevant exposure scenarios, which supports the development of microbiota-based mitigation strategies.

Beyond mixture toxicity, the available evidence also highlights important challenges in translating contaminant exposure into ecological risk. For MPs, the ingestion of polyester microfibers can be transferred from adult honey bees to larvae and hive matrices, including honey and wax [12]. However, exposure to 50 mg/L polyester microfibers for one month did not significantly affect colony population structure or honey reserves, indicating that the presence and transfer of MPs within colonies do not necessarily translate into detectable colony-level effects under these experimental conditions. Conversely, the exposure to 25-µm polystyrene MPs altered the gut microbial community of *A. mellifera*, demonstrating measurable sublethal responses at the individual level [15]. Together, these findings emphasise the need to distinguish exposure and internal contamination from biologically and ecologically relevant effects when assessing MP-related risks.

Environmental monitoring studies also revealed a high diversity of MPs in bees and hive-associated matrices, with up to 13 polymer types differing in size, colour and morphology and occurring mainly as fibres and fragments [7]. Higher MP abundance and diversity were generally associated with greater proximity to urban areas [44]. In urban wild bees, a predominance of cellulose-derived fibres over microplastics was reported, underlining the need to discriminate synthetic polymers from natural and semi-synthetic fibres when interpreting occurrence data [28].

Although PFAS have received considerably less attention than MPs, the available evidence consistently indicates that these persistent contaminants pose long-term risks extending beyond individual physiology. Experimental studies have demonstrated PFAS bioaccumulation in bee tissues, honey and faeces, indicating transfer within the colony system and raising concerns regarding contaminant persistence in pollinator-derived products [30, 34]. Seasonal biomonitoring studies further support these findings by detecting multiple PFAS compounds in forager bees, hive bees, and bee-collected pollen, confirming environmental exposure under field conditions [9]. Experimental investigations also reported impaired gut barrier integrity, inhibition of digestive enzyme activity, oxidative imbalance, altered immune and detoxification pathways, and shifts in gut microbial communities following exposure to PFOA and PFHxS [32, 33]. At the colony level, PFOS exposure reduced brood development, worker activity, food consumption, and colony maintenance, while increasing queen and worker mortality at higher concentrations [30]. Moreover, developmental and intergenerational effects were observed even under sublethal exposure conditions despite the absence of immediate impacts on adult colony performance [34], suggesting that conventional toxicity endpoints may underestimate the long-term ecological consequences of PFAS contamination. Taken together, these findings indicate that PFAS should be regarded as chronic stressors capable of compromising colony resilience through cumulative physiological, developmental, and intergenerational effects rather than through acute toxicity alone.

From a risk-assessment perspective, the studies reviewed here indicate that the occurrence of MPs and PFAS, their accumulation or transfer within bees and colony matrices, and the detection of biological effects should be considered as interconnected but distinct components of ecological risk. For MPs, evidence of transfer from adult bees to larvae, honey, and wax without detectable effects on colony population structure or honey reserves under the same exposure conditions [12] illustrates that contaminant occurrence alone does not necessarily predict colony-level consequences. Conversely, experimentally induced alterations in gut microbial communities following PS-MP exposure demonstrate that biologically relevant sublethal responses may occur at the individual level [15]. For PFAS, field detection in bees and bee-collected pollen [9], together with evidence of bioaccumulation, within-colony transfer, colony-level effects, and developmental and intergenerational responses [30, 34], provides a stronger link between exposure and biological effects. Overall, these findings indicate that ecological risk assessment should integrate environmentally realistic exposure, internal contaminant burdens and responses across multiple levels of biological organisation rather than relying primarily on contaminant occurrence or acute toxicity endpoints.

Nevertheless, substantial knowledge and methodological gaps continue to constrain such assessments. Most experimental evidence is derived from laboratory studies using *A. mellifera*, whereas data on wild bee species, colony-level exposure, and environmentally realistic contaminant mixtures remain limited. In addition, differences in particle size, polymer composition, exposure concentrations and duration, and biological endpoints reduce comparability among studies and hinder quantitative synthesis. Future research should therefore prioritise standardised experimental protocols, field-based investigations, and integrative designs linking biomonitoring with mechanistic toxicology and colony-scale responses. Particular attention should be given to contaminant mixtures and to wild bee species, whose exposure patterns and sensitivity may differ from those of managed honey bees. At the same time, emerging evidence that microbiota-targeted and nutritional interventions can partially restore gut homeostasis and mitigate contaminant-induced physiological damage [17, 22] provides a promising basis for exploring mitigation strategies alongside improved risk characterisation.

Conclusion

Microplastics and PFAS represent emerging threats to pollinator health by disrupting interconnected physiological pathways rather than acting as isolated toxicants. Current evidence identifies the gut–microbiota–immune axis as a central target linking intestinal dysfunction, oxidative stress, immune impairment, and behavioural alterations. Importantly, the studies reviewed here demonstrate that contaminant occurrence, internal exposure and biological effects do not always correspond directly, which highlights the need for ecological risk assessments that integrate environmentally realistic exposure with sublethal, developmental and colony-level responses. Future research should prioritise realistic multi-stressor scenarios, standardised approaches, colony-level investigations and greater inclusion of wild bee species, so as to better characterise the ecological consequences of these emerging contaminants and protect both managed and wild pollinators.

Declarations

Author Contribution

Conceptualization: M.B., G.C., R.R.Data curation: P.P.D., F.F., M.G., F.L., F.M., A.M., K.P., I.P., R.T.Funding acquisition: M.B.Methodology and supervision: M.B., G.C., R.R.Validation: all authors Visualization: M.B., R.R., P.P.D., F.F., I.P., R.T.Writing – original draft: all authorsWriting – review and editing: M.B., G.C., R.R.

Data Availability

All data analysed in this systematic review were extracted from previously published studies cited in the manuscript. The data supporting the findings of this review are provided in the manuscript and its accompanying Supplementary Information.

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Figures

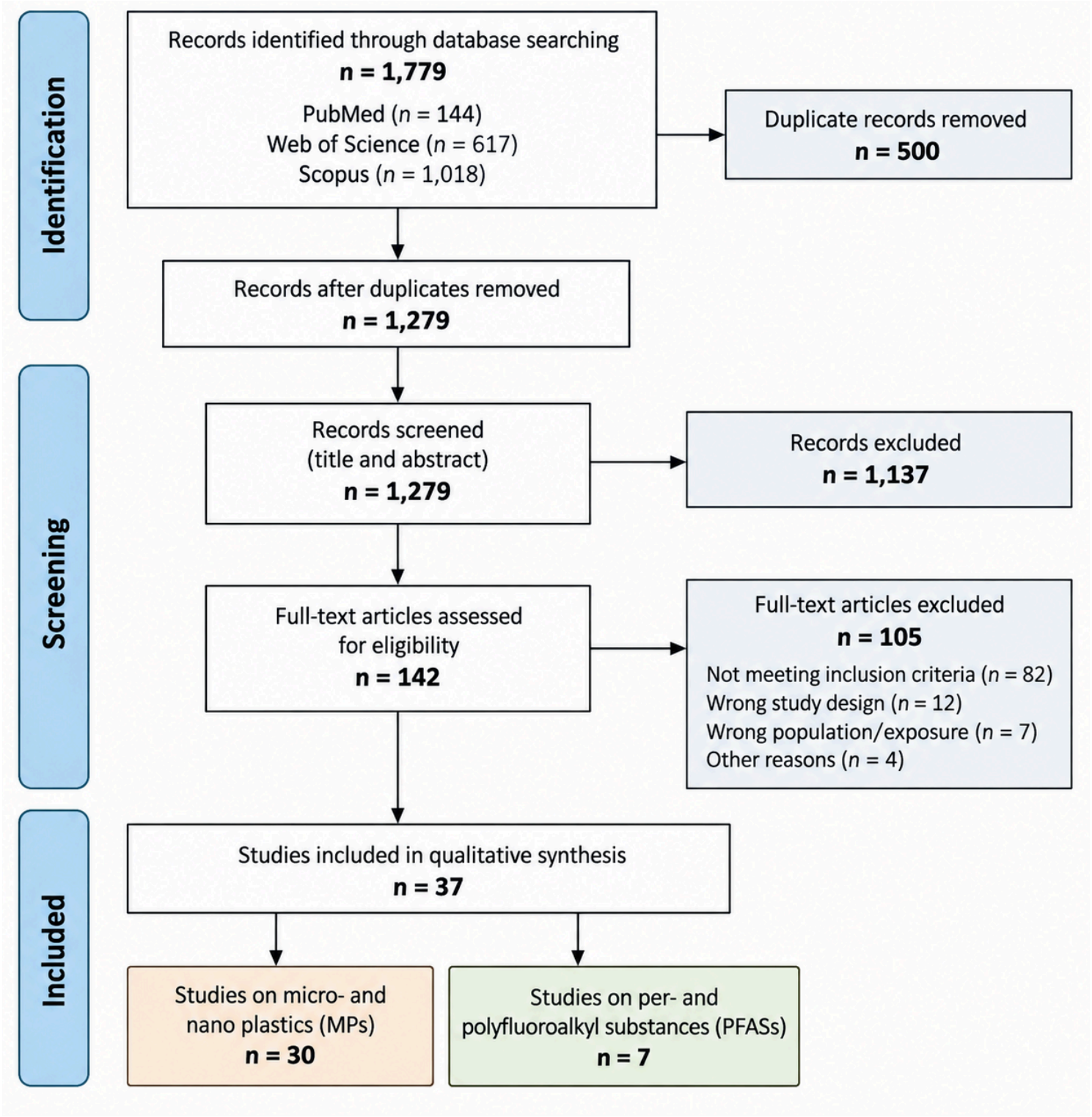

Figure 1

PRISMA flow diagram of the study selection process. The diagram summarises the identification, screening, eligibility assessment, and final inclusion of studies investigating the effects of microplastics (MPs) and nanoplastics (NPs) and per- and polyfluoroalkyl substances (PFAS) on managed and wild bees.

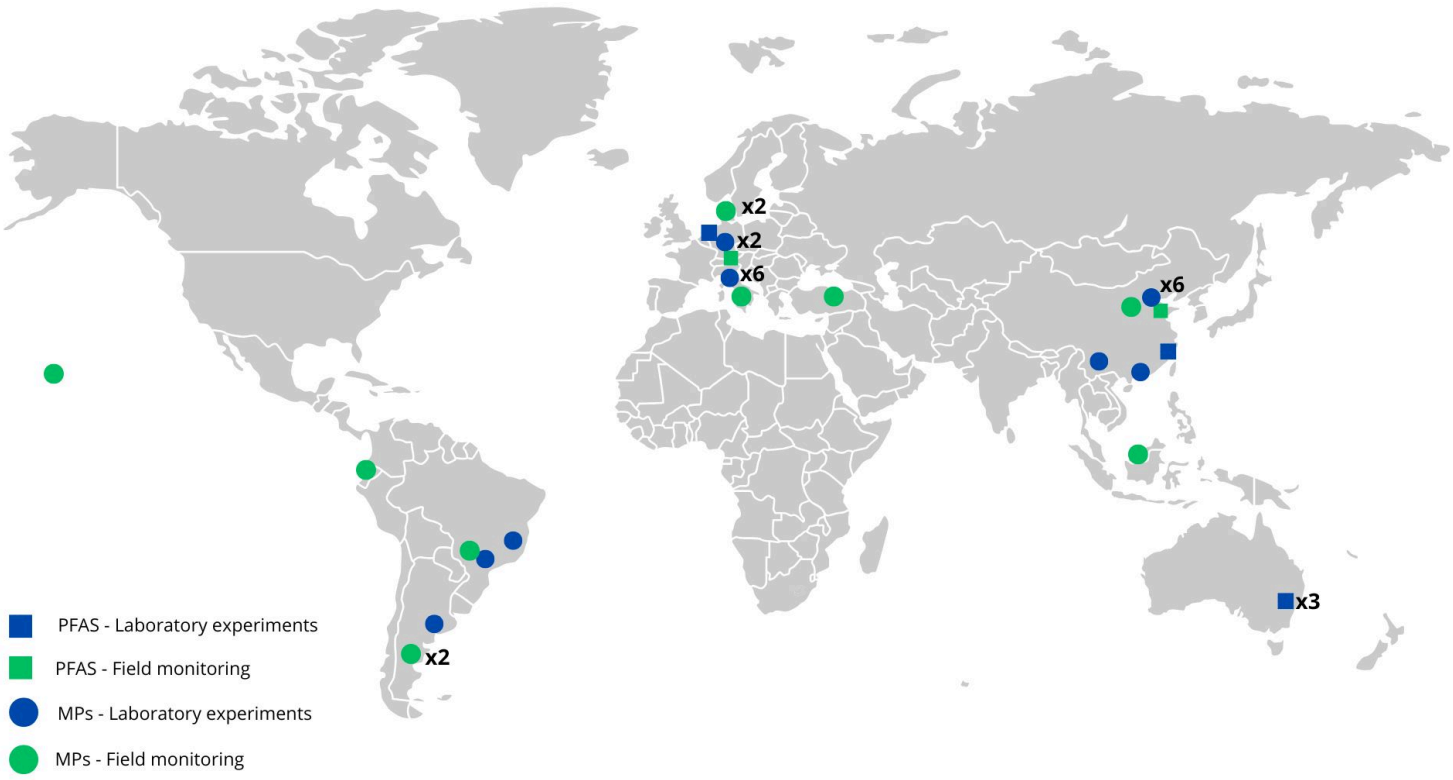

Figure 2

Worldwide distribution of studies included in this review. Circles represent studies on MPs and squares represent studies on PFAS. Blue symbols indicate laboratory exposure experiments, whereas green symbols indicate field monitoring studies. When more than one study of the same type was conducted at the same location, the corresponding symbol is followed by × n, where n indicates the number of studies.

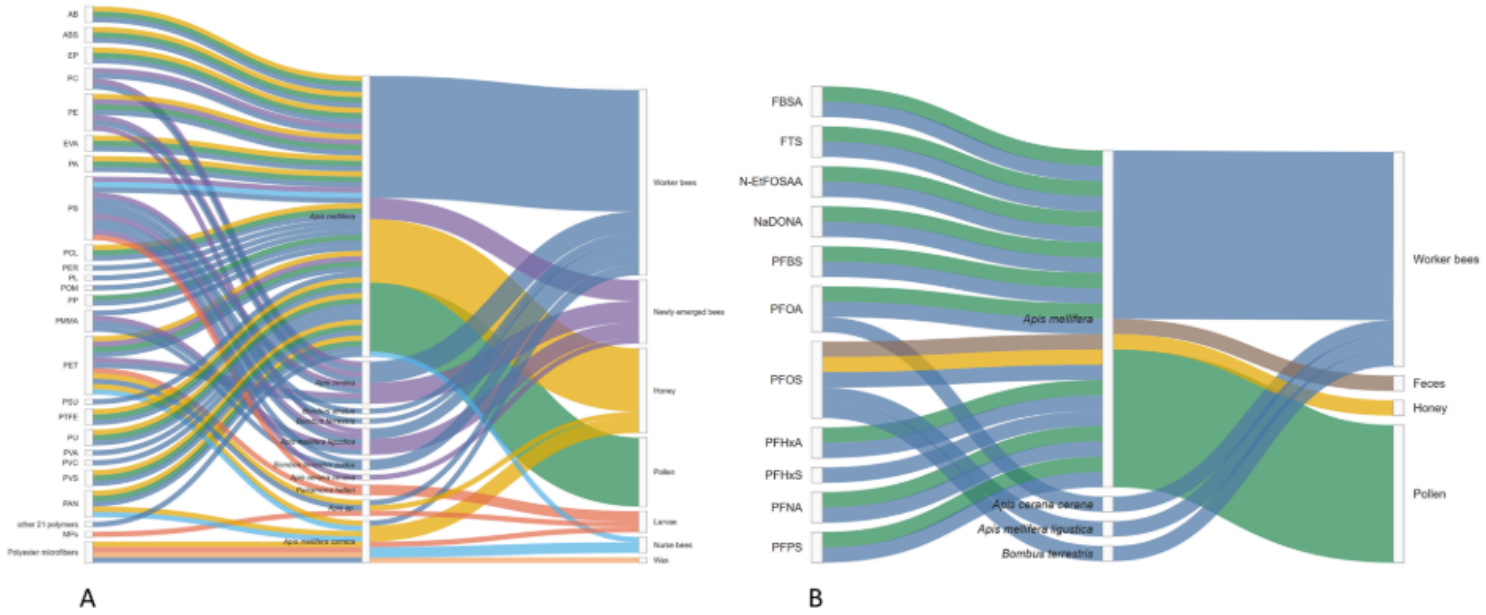

Figure 3

Alluvial representation of the contaminants, bee taxa, and biological matrices investigated in the studies included in the review. Panel A shows studies on microplastics (MPs) and nanoplastics (NPs), whereas panel B shows studies on per- and polyfluoroalkyl substances (PFAS). Flows connect each contaminant to the bee taxon and biological matrix in which it was investigated or detected. Ribbon width is proportional to the number of records reporting each contaminant–taxon–matrix combination.

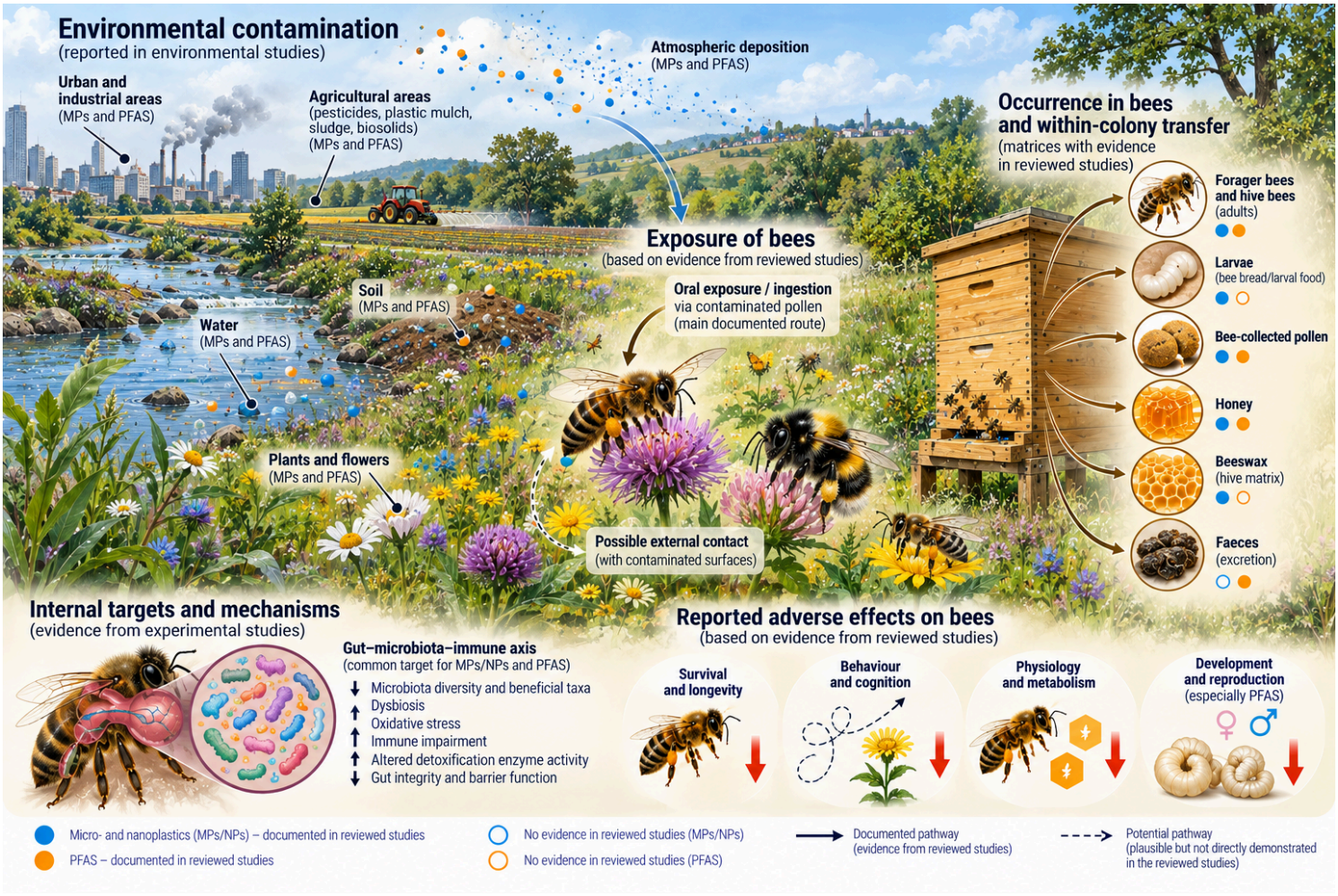

Figure 4

Schematic overview of the environmental exposure pathways and main effects of microplastics and nanoplastics (MPs/NPs) and PFAS on bee health. The figure summarises contaminant occurrence and transfer within bee colonies and the main physiological, behavioural, and developmental effects reported in the reviewed studies. This image was created with the assistance of OpenAI ChatGPT image generation and subsequently reviewed and edited by the authors for scientific accuracy and consistency with the evidence included in this review.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Table2.docx
- Table1.docx
- TableS1.xlsx