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Gut Microbiome Disruption by Pesticides: Converging Evidence Across Human and Animal Systems

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Abstract

The gut microbiota, a complex ecosystem of bacteria, archaea, fungi, and viruses, plays a vital role in regulating metabolism, immunity, barrier integrity, and neuroendocrine signalling. Emerging evidence indicates that pesticides—chemical agents targeting essential biological pathways in pests—can inadvertently disrupt gut microbial communities across humans, animals, and environmental species. Many microbial taxa share enzymatic pathways with pesticide targets, such as the shikimate pathway inhibited by glyphosate, making them susceptible to compositional and functional disturbances. Experimental studies in rodents and insects have shown that pesticide exposure induces dysbiosis, depleting beneficial taxa, enriching xenobiotic-degrading microbes, and altering the production of key metabolites including short-chain fatty acids, bile acids, and aromatic amino acid derivatives. These microbial changes have been linked to systemic metabolic, immune, and neurobehavioral effects. In pollinators and other insects, microbiota-mediated pesticide degradation contributes to both detoxification and resistance, while dysbiosis reduces immunity and ecological stability. Although human data remain limited and primarily correlational, biomonitoring and dietary studies suggest that chronic exposure to common pesticides such as organophosphates, pyrethroids, and glyphosate-based herbicides may influence microbial diversity and function. Notably, gut microbes are not passive targets; they actively metabolize pesticides via hydrolytic, oxidative, and reductive reactions, producing metabolites with distinct toxicological profiles. This two-way interaction complicates traditional toxicokinetic models and positions the microbiome as both a mediator and modifier of pesticide effects. Integrating microbial endpoints into toxicological and regulatory frameworks is essential for understanding and mitigating the health and ecological impacts of pesticide exposure.

1. Introduction

The gastrointestinal (GI) tract harbors a vast and dynamic ecosystem of microorganisms—comprising bacteria, archaea, viruses, and fungi—that collectively contribute to human physiology and homeostasis (Lloyd-Price et al., 2016; Thursby & Juge, 2017). This complex microbial community, known as the gut microbiota, is intricately involved in processes such as nutrient metabolism, immune system modulation, protection against pathogens, and xenobiotic biotransformation (Nicholson et al., 2012; Rowland et al., 2018). The microbial genome, or "microbiome," provides metabolic capabilities that complement host functions, influencing lipid metabolism, vitamin synthesis, and neurotransmitter production (Valdes et al., 2018).

Disturbances in the gut microbial ecosystem—termed *dysbiosis*—have been implicated in a spectrum of chronic diseases, including obesity, diabetes, inflammatory bowel disease (IBD), allergies, and

neurodevelopmental and neurodegenerative disorders (Carding et al., 2015; Fung et al., 2017). Dysbiosis can result from several factors: dietary shifts, antibiotic exposure, infection, stress, and increasingly, environmental toxicants such as pesticides (Mesnage & Antoniou, 2020; Wang et al., 2021). Since the gut microbiota acts as a first-line interface between environmental exposures and the host, alterations in microbial community structure or function can significantly influence health outcomes through immune, metabolic, and neuroendocrine pathways (Sharon et al., 2016).

Pesticides constitute a diverse class of xenobiotics—including herbicides, insecticides, fungicides, acaricides, and rodenticides—designed to target and disrupt essential biological processes in pest organisms (Aktar et al., 2009; Damalas & Eleftherohorinos, 2011). However, because many microbial taxa share conserved biochemical pathways with pesticide targets, these compounds often exhibit unintended antimicrobial or metabolic effects on non-target organisms, including commensal gut bacteria (Joly Condette et al., 2015; Wang et al., 2021). Indeed, pesticide exposure can occur through multiple routes—dietary intake of residues, contaminated water, inhalation, and dermal absorption (Rizzati et al., 2016). As a result, the gut microbiota is continuously exposed to low levels of these compounds, raising concern over chronic impacts on microbial composition and function.

Pesticides can perturb the microbiome through direct antimicrobial action, selective inhibition of metabolic pathways, oxidative stress induction, and modulation of bile acid metabolism (Mesnage & Antoniou, 2020; Rizzati et al., 2016). These disruptions can, in turn, affect host physiology. For example, altered microbial metabolites—such as lipopolysaccharides (LPS), short-chain fatty acids (SCFAs), and tryptophan derivatives—may trigger systemic immune activation, disrupt intestinal permeability, or influence central nervous system function through the gut-brain axis (Rueda-Ruzafa et al., 2019; Sharon et al., 2016). The growing body of evidence linking pesticide-induced dysbiosis with metabolic and neurological effects underscores the need to incorporate gut microbiome endpoints into toxicological risk assessments (Mesnage et al., 2021).

Collectively, these findings reveal that the gut microbiota represents both a target and a mediator of pesticide toxicity. Understanding these interactions offers a crucial bridge between environmental exposure and systemic disease pathogenesis. This review examines specific pesticide classes, experimental models, and mechanistic pathways that underpin these microbiota-mediated effects.

2. Mechanisms by which pesticides can affect gut microbiota

Understanding the mechanisms through which pesticides alter gut microbial communities is crucial for moving beyond correlation toward causation. Pesticide-induced changes in the gut microbiome arise from several interconnected processes, including direct antimicrobial activity, selection for resistant taxa, host-mediated effects, microbial biotransformation, and community-level functional disruption (Mesnage & Antoniou, 2020; Wang et al., 2021).

2.1 Direct antimicrobial effects

Many pesticide active ingredients and their co-formulants exhibit intrinsic antimicrobial or antifungal properties that directly influence microbial growth or metabolism. Glyphosate, for example, inhibits the enzyme *5-enolpyruvylshikimate-3-phosphate synthase* (EPSPS) in the shikimate pathway—a key biosynthetic route in many bacteria, fungi, and plants but absent in mammals (Mesnage et al., 2021). Inhibition of this pathway disrupts the synthesis of aromatic amino acids, thereby constraining microbial proliferation and altering the overall composition of gut communities. Experimental studies in animal models have shown that long-term glyphosate exposure can reduce beneficial bacterial groups such as *Lactobacillus* and *Bifidobacterium*, while favouring potentially opportunistic taxa capable of utilizing alternative carbon sources (Motta et al., 2018; Mesnage & Antoniou, 2020).

2.2 Selection for resistant or tolerant taxa

Chronic, low-dose pesticide exposure can act as an ecological filter that suppresses sensitive microorganisms and selects for those capable of tolerating or metabolizing toxic compounds. Microbes equipped with efflux pumps, hydrolases, or oxidoreductases involved in pesticide degradation can thrive under such conditions. In insects, for instance, gut bacteria possessing organophosphate-hydrolase or carboxylesterase activity enable partial detoxification of ingested pesticides, indirectly contributing to host resistance (Joly Condette et al., 2015). Similar adaptive shifts in microbial composition have been observed in mammalian studies following exposure to chlorpyrifos or pyrethroids, reflecting how pesticide stress can reconfigure microbial ecosystems.

2.3 Indirect host-mediated effects

Pesticides may influence the gut microbiota indirectly through physiological alterations in the host. Damage to the intestinal epithelium, oxidative stress, or immune dysregulation can create a microenvironment that favors specific microbial taxa (Rizzati et al., 2016). Organophosphate-induced oxidative stress, for example, has been linked with lower microbial diversity and an elevated Firmicutes-to-Bacteroidetes ratio in rodents (Tu et al., 2019). Changes in bile acid pools and mucosal immunity further affect microbial colonization and metabolism, demonstrating the reciprocal relationship between host condition and microbial community structure.

2.4 Microbial biotransformation of pesticides

Gut microorganisms possess a variety of enzymes capable of transforming pesticide molecules via hydrolysis, oxidation, reduction, or conjugation reactions. These transformations can either detoxify pesticides or generate secondary metabolites with altered toxicity. In agricultural pests, microbial degradation of compounds such as imidacloprid and chlorpyrifos contributes to pesticide resistance. In mammalian systems, microbial hydrolysis of organophosphates can yield intermediates that interact with host metabolic and signaling pathways (Joly Condette et al., 2015; Mesnage & Antoniou, 2020). Thus, microbial metabolism acts as a double-edged sword—mediating both detoxification and potential bioactivation of xenobiotics.

2.5 Community and functional network disruption

Beyond compositional changes, pesticide exposure can alter microbial functional capacities. Metagenomic and metabolomic studies demonstrate that exposure to herbicides and insecticides such as glyphosate and atrazine modifies microbial production of short-chain fatty acids (SCFAs), tryptophan-derived metabolites, and bile acid conjugates (Mesnage et al., 2021; Wang et al., 2021). These metabolic perturbations can influence host immune tone, energy metabolism, and neuroactive signalling through the gut-brain axis. Therefore, disruption of community function—rather than taxonomic shifts alone—may represent a key mechanistic bridge between pesticide exposure and systemic health effects.

3. Evidence from Non-Human Model Systems

Model organisms have been indispensable for understanding how pesticides affect gut microbiota, offering mechanistic insights under controlled exposure conditions. By examining microbiome alterations across insects, rodents, and aquatic species, researchers have been able to delineate conserved patterns of dysbiosis, microbial detoxification, and host physiological responses. Pesticide exposure induces complex microbiome shifts that can, in turn, modulate host metabolism, immunity, and resistance phenotypes.

3.1 Insects

Insects provide particularly useful systems for studying microbiome–pesticide interactions due to their ecological relevance, rapid generation times, and well-characterized gut microbial communities. Numerous studies have shown that the gut microbiota of insects contains taxa capable of enzymatically degrading pesticides, especially organophosphates, carbamates, and neonicotinoids (Kumar et al., 2018; Kikuchi et al., 2012). Bacterial species such as *Pseudomonas*, *Enterobacter*, and *Bacillus* have been found to express esterases, oxidases, and phosphatases that metabolize pesticide residues, thereby reducing toxicity to the insect host (Li et al., 2018).

This microbial degradation has direct ecological and evolutionary implications. For instance, in pest insects like *Plutella xylostella* (diamondback moth) and *Helicoverpa armigera*, gut bacteria can hydrolyze chlorpyrifos and fenitrothion, leading to decreased mortality rates upon pesticide exposure (De Almeida et al., 2017). In turn, selective enrichment of these pesticide-degrading microbes under chronic exposure can confer partial pesticide resistance to the host population, affecting pest management outcomes (Kikuchi et al., 2012). Such findings underscore the dual role of microbiota—as both mediators of detoxification and as dynamic participants in host adaptation.

Pollinator studies, particularly those involving honeybees (*Apis mellifera*), have demonstrated that herbicides such as glyphosate can perturb gut microbial composition, often reducing the abundance of beneficial *Snodgrassella alvi* and *Gilliamella apicola* while increasing opportunistic bacteria (Motta et al., 2018). These alterations impair immune defences and increase susceptibility to pathogens like *Serratia marcescens*, linking microbiota disruption to colony health and resilience. Therefore, beyond detoxification, pesticide-driven dysbiosis in pollinators represents a major ecological concern due to potential cascading effects on pollination and ecosystem services.

3.2 Rodents

Rodent models—especially rats and mice—offer the advantage of precise dosing control and detailed microbiome–metabolome integration. Chronic pesticide exposure produces reproducible microbial alterations, often linked to metabolic dysfunction and systemic effects (Rizzati et al., 2016; Mesnage & Antoniou, 2020).

One of the most comprehensive analyses was conducted by Mesnage and colleagues (2021), who employed shotgun metagenomics and metabolomics to assess the impact of glyphosate and glyphosate-based herbicides in rats. Their findings revealed significant compositional shifts in both bacterial and fungal communities within the caecum, including depletion of beneficial fermentative taxa and enrichment of opportunists. Metabolomic profiling identified accumulation of shikimate pathway intermediates, consistent with inhibition of 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS), and altered short-chain fatty acid (SCFA) profiles. Importantly, some formulations containing surfactant co-formulants induced greater disruptions than glyphosate alone, suggesting that non-active ingredients contribute meaningfully to gut microbiota toxicity.

Other rodent studies have corroborated these findings with different pesticide classes. Chlorpyrifos, an organophosphate insecticide, has been shown to reduce microbial diversity and impair intestinal barrier integrity, while simultaneously inducing low-grade inflammation (Joly Condette et al., 2015). Such effects are often associated with metabolic disturbances and immune dysregulation. Furthermore, multi-omics investigations integrating 16S rRNA sequencing, metabolomics, and transcriptomics have begun to link pesticide-induced dysbiosis to downstream changes in lipid metabolism, bile acid homeostasis, and neurotransmitter pathways (Tu et al., 2019).

3.3 Aquatic and Other Vertebrates

Aquatic species and non-mammalian vertebrates offer complementary perspectives on how environmental pesticide contamination influences gut microbial ecology. Fish, amphibians, and other aquatic organisms are often directly exposed to pesticide residues in water, sediments, and prey, allowing assessment of dose-dependent microbiome responses under environmentally relevant conditions.

Studies in teleost fish, including zebrafish (*Danio rerio*) and common carp (*Cyprinus carpio*), have shown that pesticide exposure can markedly shift intestinal microbial communities (Zhang et al., 2017; Jin et al., 2017). Organophosphates such as chlorpyrifos and pyrethroids like cypermethrin consistently reduce beneficial taxa, including *Lactobacillus* and *Cetobacterium*, while increasing potentially pathogenic *Aeromonas* and *Vibrio* species (Li et al., 2019). These microbial alterations have been linked to oxidative stress, intestinal inflammation, and decreased digestive efficiency.

Herbicides such as atrazine and paraquat have also been reported to disrupt aquatic microbiota, altering nitrogen and carbon cycling functions (Sun et al., 2019). In amphibians, pesticide exposure during larval stages leads to reduced microbial diversity and impaired immune development, suggesting long-term consequences for survival and metamorphosis (Kohl et al., 2016). Despite interspecies variability, the overall trend across aquatic and semi-aquatic vertebrates indicates that pesticide exposure consistently induces dysbiosis, metabolic alteration, and impaired host function.

However, the aquatic evidence base remains fragmented due to methodological differences in exposure duration, concentration, and sequencing approaches. While results collectively reinforce the plausibility of pesticide-induced dysbiosis across taxa, they also highlight the need for standardized experimental protocols to allow cross-species synthesis and risk extrapolation.

4. Evidence in Humans

Compared to the wealth of animal and in vitro studies, evidence directly linking pesticide exposure to gut microbiota alterations in humans remains relatively limited. However, emerging observational and translational studies have begun to uncover associations between pesticide exposure and changes in microbial composition and metabolic activity, supporting the plausibility of microbiota-mediated health effects. These investigations—while mostly cross-sectional and exploratory—highlight potential biological pathways connecting environmental exposure to metabolic, immune, and neurobehavioral outcomes.

4.1 Dietary Exposure and Microbiome Composition

The general population is exposed to pesticides primarily through dietary intake of residues on fruits, vegetables, and grains. Dietary exposure assessments have been correlated with urinary pesticide metabolites

to approximate internal dose levels (Lu et al., 2018; Ye et al., 2020). A few human microbiome studies have integrated such biomonitoring data with faecal microbial profiling. For instance, one pilot investigation in adults consuming predominantly conventionally grown produce found lower gut microbial diversity and altered representation of key taxa such as *Bacteroides* and *Prevotella* compared to individuals consuming organic diets, which are associated with lower pesticide residue intake (Mueller et al., 2017). Although causality could not be established, this observation aligns with preclinical findings that link pesticide exposure to shifts in bacterial diversity and metabolic capacity.

Other studies have used agricultural exposure indices rather than biomarker data. Rueda-Ruzafa et al. (2019) summarized that individuals living in rural or agricultural areas may exhibit distinct gut microbial signatures compared to urban populations, possibly due to higher chronic pesticide exposure alongside differences in diet, hygiene, and co-exposures. Such observations underscore the complex interplay between environment, microbiota, and host physiology. Nonetheless, most human datasets to date lack longitudinal sampling and mechanistic endpoints, making it difficult to attribute microbiome differences directly to pesticide exposure.

4.2 Occupational and Environmental Exposures

Occupational settings—such as pesticide manufacturing, mixing, and application—represent scenarios of higher and more sustained exposure. Although numerous epidemiological studies have linked occupational pesticide exposure with health outcomes including neurotoxicity, endocrine disruption, and metabolic disorders (Kim et al., 2017; Mostafalou & Abdollahi, 2017), only a small subset has examined gut microbiome alterations.

Indirect evidence suggests that chronic pesticide exposure can induce systemic inflammation and oxidative stress—conditions known to influence the gut ecosystem. For example, agricultural workers exposed to organophosphates and carbamates have been reported to show elevated serum inflammatory markers and altered bile acid profiles (Bassil et al., 2007; Badr et al., 2019). These physiological disturbances could mediate microbiome perturbations, though direct microbiota sequencing data from occupational cohorts remain sparse.

In one exploratory study, volunteers with documented pesticide exposure histories exhibited fecal microbial changes resembling those observed in rodent models exposed to similar compounds (Mesnage & Antoniou, 2020). Specifically, enrichment of *Proteobacteria* and reduction of butyrate-producing taxa suggested a state of low-grade dysbiosis potentially linked to xenobiotic stress. However, given small sample sizes and reliance on self-reported exposure data, these findings require replication using objective exposure biomarkers and longitudinal designs.

4.3 Biomarker-Based Human Studies

Human biomonitoring efforts have increasingly focused on integrating pesticide exposure assessment with molecular markers of health status. Urinary metabolites such as 3-phenoxybenzoic acid (for pyrethroids) or dialkylphosphates (for organophosphates) provide reliable indicators of recent exposure. When combined with microbiome sequencing or metabolomic profiling, such data can reveal potential correlations between pesticide burden and microbial activity.

For example, a cross-sectional study in children from agricultural regions found associations between urinary organophosphate metabolite levels and reduced faecal microbial diversity (Cox et al., 2019). Similarly, research on adolescents with higher estimated pyrethroid exposure revealed altered faecal concentrations of short-chain fatty acids (SCFAs) and bile acids, consistent with microbial metabolic disruption (Zhang et al., 2020). Although causation could not be inferred, these biomarker-based analyses provided valuable evidence that real-world pesticide exposure levels may be sufficient to influence gut microbial metabolism.

Another avenue of inquiry involves secondary analyses of dietary intervention studies. For instance, switching from conventional to organic diets has been shown to substantially reduce urinary pesticide metabolites within days (Oates et al., 2014), implying that the gut microbiota may experience fluctuating xenobiotic inputs based on dietary sources. Integrating microbiome endpoints into such designs could clarify whether these exposure reductions translate to microbial recovery.

5. Case Studies and Representative Pesticide Classes

Although the literature varied in methodological quality and scope, four major pesticide groups—glyphosate-based herbicides, organophosphate insecticides, neonicotinoids and pyrethroids, and fungicides—emerged as the most studied in relation to gut microbiota. Together, these case studies provide an integrated view of how diverse agrochemicals, despite differing primary targets, can converge on similar outcomes of microbial imbalance and altered host–microbe interactions.

5.1 Glyphosate and Glyphosate-Based Herbicides

Glyphosate, the most widely used herbicide globally, is the best-characterized compound in terms of microbiome-related mechanisms. Mechanistically, glyphosate inhibits 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS), a key enzyme in the shikimate pathway. Because this pathway is present in many bacteria and fungi but absent in mammals, the gut microbiota is considered a plausible off-target site for glyphosate action (Mesnage & Antoniou, 2020). Computational modelling and in vitro experiments confirmed that several commensal bacteria are sensitive to glyphosate, particularly those dependent on aromatic amino acid biosynthesis through EPSPS.

Animal models strengthened this mechanistic plausibility. Mesnage and colleagues (2021) used multi-omics approaches—including metagenomics and metabolomics—in rats exposed to glyphosate and representative formulations. They reported shifts in both bacterial and fungal community composition, notably an enrichment of taxa capable of xenobiotic metabolism and a reduction in short-chain fatty acid (SCFA)-producing species. Metabolomic analyses revealed accumulation of intermediates upstream of the shikimate pathway blockade, supporting a functional impact on microbial metabolism. Importantly, co-formulants present in commercial products sometimes produced qualitatively distinct effects from glyphosate alone, suggesting that formulation additives can modulate microbiome responses.

Outside of mammalian systems, pollinator studies offered compelling ecological evidence. For instance, honeybee gut microbiota exposed to glyphosate displayed reduced diversity and decreased abundance of beneficial *Snodgrassella* and *Gilliamella* species, accompanied by increased vulnerability to opportunistic pathogens such as *Serratia marcescens* (Motta et al., 2018). Such findings linked microbiome disruption with impaired immunity and colony health. Despite this extensive experimental work, human data remains scarce. Observational studies focused primarily on dietary exposure estimates and urinary biomonitoring, emphasizing mechanistic plausibility rather than direct causal evidence of microbiome perturbation.

5.2 Organophosphates and Cholinesterase-Inhibiting Insecticides

Organophosphates (OPs) represent another intensively studied class due to their neurotoxicity and widespread agricultural use. While their canonical target is acetylcholinesterase, experimental evidences suggest that OP exposure also perturbs gut microbial ecosystems. Animal studies revealed that sub chronic or acute exposure to chlorpyrifos, diazinon, or malathion led to reduced microbial diversity and shifts in the Firmicutes/Bacteroidetes ratio, often associated with inflammation and metabolic dysfunction (Rizzati et al., 2016).

These microbial shifts may arise from both direct and indirect mechanisms. OPs and their metabolites possess antimicrobial properties that can inhibit sensitive taxa, while host-mediated mechanisms—such as oxidative stress, intestinal barrier impairment, and immune activation—further influence microbial composition. Importantly, some gut bacteria and environmental isolates harbour organophosphate-degrading enzymes (e.g., phosphotriesterases, carboxylesterases), enabling microbial detoxification and contributing to pesticide resistance in insects and soil systems. However, these biotransformation processes can generate metabolites with altered toxicity, suggesting a dual role for the microbiota as both protector and participant in OP-induced toxicity.

In human studies, occupational exposure among agricultural workers was associated with systemic oxidative stress and altered bile acid metabolism, conditions that could secondarily affect the gut microbiome. Although microbiota sequencing data were limited, these findings supported the hypothesis that chronic OP exposure may disrupt microbial homeostasis indirectly via host physiological stress.

5.3 Neonicotinoids, Pyrethroids, and Other Insecticides

Neonicotinoids and pyrethroids, widely used as insect neurotoxins, have drawn attention for their non-target effects on pollinators and other insects. Numerous insect studies have demonstrated that sublethal exposure to imidacloprid or clothianidin altered gut bacterial composition, reducing beneficial taxa involved in digestion and immunity and increasing susceptibility to viral and fungal infections (Li et al., 2019). Similar findings in bumblebees and butterflies indicated that neonicotinoid-induced microbiome changes could impair foraging, reproduction, and colony stability.

Pyrethroids, though generally less persistent, also exhibited microbiome-mediated effects. Insects and microbial symbionts capable of metabolizing pyrethroids were shown to contribute to pesticide tolerance and resistance evolution. In mammals, animal models exposed to pyrethroids such as deltamethrin or cypermethrin exhibited gut dysbiosis characterized by decreased SCFA-producing bacteria and increased inflammatory markers, supporting cross-species parallels (Zhang et al., 2020). Collectively, these findings emphasize that

microbiota-mediated xenobiotic metabolism may represent a shared mechanism across chemically diverse insecticides.

5.4 Fungicides and Other Pesticide Classes

Fungicides have been comparatively less studied in relation to gut microbiota, yet their potential impact is conceptually significant given their antifungal mode of action. Emerging evidence from both environmental and animal studies has shown that fungicides such as azoles and strobilurins can alter fungal communities within the gastrointestinal tract (the mycobiome) and indirectly affect bacterial populations (Deising et al., 2020; Stana et al., 2021). This cross-kingdom interaction arises because fungi and bacteria engage in metabolic cooperation and competition; thus, eliminating or suppressing fungal taxa can disrupt bacterial nutrient networks, signalling molecules, and immune interactions (Huffnagle & Noverr, 2013; Richard & Sokol, 2019). For example, exposure to triazole fungicides in fish and rodent models reduced intestinal fungal diversity and led to downstream changes in bacterial composition and bile acid metabolism (Kers et al., 2020; Stana et al., 2021). In zebrafish, azole-based antifungals disrupted the balance between *Ascomycota* and *Basidiomycota*, altering microbial metabolites involved in oxidative stress response and lipid regulation (Zhang et al., 2018). Similarly, rodent experiments with strobilurin fungicides demonstrated significant shifts in short-chain fatty acid (SCFA) profiles and an increase in proinflammatory bacterial taxa, suggesting that gut microbial changes may contribute to systemic toxic outcomes (Kers et al., 2020).

6. Health Outcomes

Scientific literature had proposed several plausible health and ecological outcomes potentially mediated by pesticide-induced alterations in the gut microbiome. Experimental studies in animal models suggested that chronic dietary exposure to pesticides could contribute to metabolic dysregulation, including insulin resistance and obesity-like phenotypes, often accompanied by shifts in microbial diversity and function (Zhang et al., 2019; Liang et al., 2020; Joly Condette et al., 2015; Gao et al., 2017). While these findings supported mechanistic plausibility, causal inference in humans remained unconfirmed due to limited longitudinal and intervention data (Rueda-Ruzafa et al., 2019; Mesnage et al., 2021). Pesticide-driven microbial disruption was also linked to immune dysfunction and increased infection susceptibility, with evidence indicating that reductions in short-chain fatty acids (SCFAs), compromised epithelial barriers, and altered immune signaling could promote chronic inflammation (Claus et al., 2016; Rosas-Pérez et al., 2017; Lozano et al., 2018). Beyond mammalian models, pollinator studies provided clear examples of how pesticide exposure could impair microbial community stability, leading to increased pathogen vulnerability and colony health decline (Kakumanu et al., 2016; Motta et al., 2018; Raymann & Moran, 2018). In addition, growing attention was directed toward the potential neurobehavioral consequences of pesticide-induced dysbiosis, given the central role of the gut–brain axis in regulating mood, cognition, and neurodevelopment (Rueda-Ruzafa et al., 2019; Velmurugan et al., 2017). Although animal data suggested that microbiome alterations could modulate neurochemical pathways and behavioural outcomes, corresponding evidence in humans remained sparse and largely correlational (Mesnage & Antoniou, 2020). Finally, these microbiome-mediated effects extended beyond individual health, influencing ecological and agricultural systems. Altered pollinator microbiomes and microbial contributions to pesticide degradation or resistance carried important implications for ecosystem balance, pest management strategies, and food security (Zhao et al., 2021; Zhu et al., 2016). Pesticides can perturb host-associated microbial ecosystems in ways that may have broad physiological, ecological, and economic significance, while emphasizing the need for more causal, longitudinal, and integrative human studies.

7. Microbiota mediated pesticide metabolism

Gut microorganisms are not merely passive targets of xenobiotic toxicity but active participants in the metabolism of pesticides and other environmental chemicals. Across taxa, microbial enzymes such as esterases, hydrolases, oxidases, reductases, and transferases were shown to bio transform parent pesticide compounds into metabolites with distinct pharmacokinetic, bioavailability, and toxicity profiles (Claus et al., 2016; Rosas-Pérez et al., 2017). This biotransformation can occur through both detoxifying and activating pathways; whereby microbial metabolism either reduces the toxicity of a compound or converts it into a more reactive intermediate capable of exerting systemic effects on the host. In insects, these interactions are particularly well characterized: several studies demonstrated that gut symbionts isolated from pesticide-resistant insects, including *Spodoptera*, *Plutella*, and *Nilaparvata* species, possess enzymatic capabilities that

degrade organophosphates, neonicotinoids, and pyrethroids, thereby contributing directly to host pesticide resistance and survival (Kikuchi et al., 2012; Cheng et al., 2017; Xia et al., 2018). In honey bees and other pollinators, microbial degradation of herbicides such as glyphosate or insecticides such as imidacloprid has been linked to altered microbiome composition and increased pathogen susceptibility, underscoring the ecological significance of such microbial–chemical interactions (Motta et al., 2018; Raymann & Moran, 2018). In vertebrate systems, microbial xenobiotic metabolism can substantially influence systemic exposure to both parent compounds and secondary metabolites. For example, microbial deconjugation and reduction processes in the gut can modulate enterohepatic circulation, prolonging the bioavailability of toxic residues or influencing host metabolic responses (Gao et al., 2017; Liang et al., 2020). Moreover, the same metabolic versatility that allows microbes to detoxify environmental contaminants may also generate harmful byproducts, including reactive intermediates capable of inducing oxidative stress or immune activation. This bi-directional interaction between pesticides and gut microbes—whereby pesticides alter community structure and, in turn, microbes modify pesticide fate and toxicity—has complicated conventional exposure–effect models and underscored the necessity of incorporating microbiome-mediated processes into toxicokinetic frameworks (Mesnage & Antoniou, 2020; Zhao et al., 2021). Recognition of these reciprocal interactions marked a paradigm shift in understanding pesticide toxicity, prompting calls for integrated experimental designs that jointly evaluate microbial metabolism, host physiological outcomes, and environmental persistence within a unified mechanistic context.

8. Knowledge gaps and research priorities

Several key knowledge gaps and research priorities had been identified to advance understanding of pesticide–microbiome interactions and their implications for human and ecological health. A major emphasis was placed on the need for prospective human cohort studies incorporating repeated measures of exposure biomarkers, such as urinary or serum pesticide metabolites, along with comprehensive dietary, lifestyle, and longitudinal microbiome and metabolome profiling to establish causal inference (Rueda-Ruzafa et al., 2019; Mesnage et al., 2021). Standardisation of experimental approaches was also highlighted, with calls for well-characterised pesticide formulations, exposure doses reflective of real-world conditions, and transparent reporting of co-formulants and adjuvants to enhance reproducibility and regulatory relevance (Claus et al., 2016; Lozano et al., 2018). The integration of functional multi-omics—combining shotgun metagenomics, metatranscriptomics, and metabolomics—with host molecular readouts was viewed as essential to link taxonomic disruptions with biochemical and physiological outcomes, exemplified by the multi-omics framework proposed by Mesnage and colleagues (2021) (Mesnage & Antoniou, 2020). Another critical research priority concerned mixture and co-exposure studies that better reflect environmental reality, since humans and wildlife are typically exposed to multiple pesticides and other xenobiotics with additive or synergistic effects (Zhang et al., 2019; Liang et al., 2020). Mechanistic investigations into microbial biotransformation pathways were also recommended to elucidate enzymatic processes that either detoxify or activate pesticide metabolites and to clarify how such pathways vary across host species and microbial communities (Rosas-Pérez et al., 2017; Zhao et al., 2021). Finally, the literature stressed the need for regulatory method development, including validated microbiome perturbation assays and interpretative frameworks for integrating microbiome endpoints into pesticide risk assessment, thereby bridging mechanistic microbiome research with public health policy (Velmurugan et al., 2017; Zhu et al., 2016).

9. Conclusion:

The accumulated body of evidence supported the plausibility that pesticides can perturb gut microbial communities across diverse taxa and that such perturbations can alter microbial metabolic outputs—such as short-chain fatty acids, aromatic amino acid metabolites, and bile acids—with potential downstream effects on host metabolism, immune regulation, epithelial barrier integrity, and neurobehavioral function. Mechanistic studies in rodent models using integrated multi-omics approaches provided detailed insights into pesticide-induced disruptions of microbial pathways, including effects on the shikimate pathway and formulation-specific outcomes, while insect research demonstrated microbial roles in pesticide degradation and highlighted functional consequences for host resistance, detoxification capacity, and immune competence, particularly in pollinators. Nevertheless, direct causal evidence in humans linking environmentally relevant pesticide exposures to microbiome-mediated clinical outcomes remained limited. Key research gaps included the absence of standardised exposure metrics, a lack of prospective cohort studies with longitudinal microbiome profiling, insufficient incorporation of functional multi-omics analyses, and underrepresentation of studies

addressing realistic chemical mixtures and co-exposures. Moreover, regulatory frameworks had not yet integrated microbiome endpoints into formal risk assessment processes, and the scientific community emphasised the need for method development, assay validation, and consensus on interpretation standards before such endpoints could be routinely applied in regulatory decision-making.

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