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The Side Effects of Pesticides  
on Nontarget Arthropods

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**Keywords**

pesticide–arthropod interactions, pesticide stress, sublethal effects, pesticide hormesis, ecosystem services, environmental stressors

**Abstract**

Millennia after the advent of pesticides and nearly eight decades into the widespread use of synthetic compounds, the role of such chemicals in modern society remains pivotal, despite persistent concerns over human and environmental safety. Rather than declining, pesticide use continues to expand, with shifting priorities regarding compound selection and application strategies. The growing prominence of biopesticides broadens pest management options but complicates the evaluation of their side effects. Additionally, evolving pesticide use patterns—including the increasing reliance on mixtures—introduce further complexities, as compound interactions and their effects on exposed organisms require closer scrutiny. Although pesticide risk assessment is a relatively young and evolving field, its progress remains hindered by misconceptions, biases, and oversimplifications. This review integrates ecotoxicology and stress biology into a conceptual framework to address these challenges, advocating for more precise and dynamic approaches to pesticide risk assessment.

**Biopesticide:**

pesticidal agents sourced from living organisms, including microbes, botanicals, and plant-incorporated protectants

**Natural pesticide:**

pesticides derived from natural sources, including minerals and plants, but not exclusively

**Acute toxicity:** toxic response from a single exposure, with effects observable within 24 h to a few days

**Chronic toxicity:**

toxic response from repeated or continuous exposure over an extended period, often exceeding the organism's half-life

## 1. ANTHROPOGENIC STRESSORS, PESTICIDES, AND BIOPESTICIDES

Among global anthropogenic stressors, pesticides are central to the modern chemical landscape. Since ancient times, human societies have relied on chemical interventions to manage pests—an approach that has evolved alongside technological advances and growing environmental concerns. Urbanization and agricultural intensification have driven increased pesticide use to protect crops and control disease vectors (39, 155). Yet, this dependency is counterbalanced by rising concerns over environmental and human health risks, particularly those associated with insecticides (24, 91).

Pesticides remain as ubiquitous and controversial today as they were 60 years ago, when *Silent Spring* ignited debate. Early concerns about pollination service loss spurred extensive research into pesticide side effects on nontarget insects, ecosystem services, and insect declines. Over time, the debate has shifted, shaped by innovations in pesticide chemistry, evolving pest management strategies, novel biopesticides, and regulatory changes (81, 126). Although synthetic pesticides dominate discussions, biopesticides are often perceived as safer due to their natural origins. However, natural origin alone does not ensure safety (71, 75). Large-scale applications of natural pesticides—often at concentrations exceeding natural levels or in previously unaffected ecosystems—warrant critical scrutiny. Thus, risk assessments must extend beyond synthetic pesticides to include natural pesticides, including biopesticides.

Historically, pesticide use has been framed through a utilitarian lens, prioritizing food security and human health. However, this perspective is expanding to consider broader ecological impacts, particularly those on critical ecosystem services. While pollination and biological control (biocontrol) receive the most attention, other services—such as nutrient cycling and soil health—should not be overlooked.

This review adopts a holistic approach to pesticide side effects, focusing on nontarget organisms. Using a stress response framework, it systematically examines the physiological and ecological stress induced by different pesticidal agents. Covering a range of compounds—from early inorganic formulations and traditional biopesticides to synthetic pesticides and the resurgence of biopesticides—this review provides a broader perspective on pesticide use, emphasizing ecological sustainability and environmental health.

## 2. DOSES, POISONS, AND SIDE EFFECTS

*Alle Dinge sind Gift...* begins Paracelsus's sixteenth-century adage, acknowledging that toxicity is dose dependent, but with centuries of accumulated research, it is clear that the dose alone does not tell the full story. The duration and type of exposure, as well as the context in which exposure occurs, are pivotal in determining toxicity. These effects and responses are not necessarily linear, or harmful, in their continuum (4). This is particularly true in today's chemical landscape, which now includes a surge of biopesticides and other natural compounds, sometimes applied at concentrations far above their natural levels (75).

### 2.1. Acute Versus Chronic Toxicity

Historically, toxicity assessments have overemphasized dose while neglecting exposure duration. Acute toxicity, which evaluates single doses with rapid mortality as the end point, has been the standard for arthropods, especially pest species and their natural enemies. However, this focus overlooks chronic toxicity, which arises from repeated or prolonged exposure and accumulates over time, eventually reaching a harmful threshold. This emphasis on a single exposure and quick mortality response, while suitable for early neurotoxic insecticides such as organophosphates and pyrethroids, overlooks potential chronic toxic effects. Modern insecticides and bioinsecticides,

with shorter environmental lifespans, require repeated applications, resulting in a gradual buildup of effects (75).

## 2.2. Lethal Versus Sublethal Effects

The historical focus on mortality as the primary toxicological end point has skewed pesticide assessments toward lethal effects (64). However, organisms are often exposed to sublethal doses, which may result from environmental breakdown of pesticides or the intrinsic differences in susceptibility between nontarget and pest species; other pesticidal compounds may not even cause direct mortality. For instance, certain essential oils are insect antifeedants (58, 118), while insecticide modulators of chordonotal stretch receptor neurons, such as pymetrozine and afidopyropen, compromise aphid feeding, impairing plant vector transmission (142).

## 2.3. Direct Versus Indirect Effects

Direct pesticide effects occur when an organism is directly exposed, but indirect effects are equally significant. The latter effects occur when exposure to one species cascades through ecological interactions, affecting nonexposed species. For example, in leaf-cutting ants, toxic baits transferred by foragers to the colony compromise the minor workers, indirectly harming the queen's health and reproductive capacity and leading to colony collapse (53, 54). Similar (indirect) effects cascaded among species occur when pesticides directly compromise food sources, competitors, or natural enemies, affecting nontargeted associated species, as exemplified by herbicide effects on pest natural enemies (160; for other examples, see 68, 64, 131).

## 2.4. From Individuals to Populations

Pesticides aim to manage pests by compromising key physiological systems in targeted organisms. The toxic effects observed at the individual level translate to the population when a significant number of individuals are affected. This cascading effect from individuals to populations is modeled through dose/concentration/time response bioassays, providing valuable toxicological information for both targeted and nontargeted species and for synthetic pesticides and biopesticides. However, these studies typically focus on isolated species and neglect interspecies interactions. A stress response pathway framework better captures these complex interactions, guiding decisions about pesticide use and its broader ecological consequences (70).

## 3. SIDE EFFECTS AT THE INDIVIDUAL LEVEL

The stress response pathway (**Figure 1**) is crucial for understanding pesticide effects, starting at the individual level and reaching populations and communities. It highlights complex interactions and avoids the assumption that adverse outcomes are inevitable, allowing for the possibility of neutral or even positive responses and mitigating and compensatory effects (72). Pesticides must enter the organism; must interact with a target site; and may undergo transformation, sequestration, or excretion. These processes, along with interactions at secondary sites, can result in toxic or nontoxic effects, including mitigatory responses (**Figure 2**). Synthetic pesticides, especially neurotoxic ones, are subject to heightened scrutiny (32, 55).

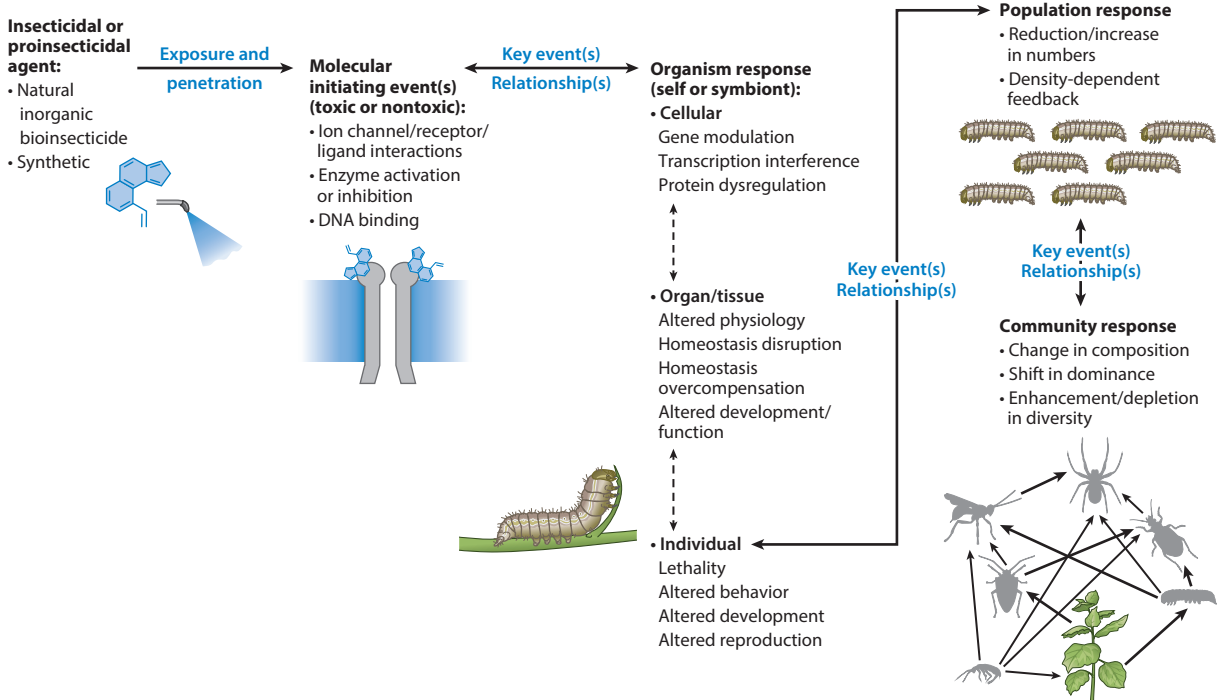
### 3.1. Biochemical Effects of Pesticides

Understanding the mode of action and transformation of pesticides in organisms is foundational for unraveling the mechanisms that induce mortality. This includes how compounds are

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**Toxicological end point:** a key measure assessing how toxic substances affect living organisms

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**Figure 1**

Diagram illustrating the general framework of stress response pathways elicited by insecticidal stress across the different levels of biological organization. The potential chain of events identifying the main components and relationships is indicated as a cascade of effects from the initial exposure to an eventual community response. The double-headed arrows interconnecting the outcomes of the different levels of biological organization emphasize both the potential stress, with its cascading effects, and mitigatory or compensatory responses.

metabolized, potentially enhancing or diminishing their toxic effects. However, pesticide interactions extend beyond the primary target site, challenging the notion that mortality is the only outcome. Viewing the organism as a symbiotic ecocosm, where nonself responses play pivotal roles in homeostasis, is pertinent (93, 130).

This perspective is especially relevant for nontarget species exposed to sublethal pesticide levels, where diverse outcomes may occur (45, 72). Research on neonicotinoids supports these assertions (154), showing that nicotinic acetylcholine receptor (AChR) subtypes and detoxification enzyme variations contribute to different stress outcomes (104, 151). The coexistence of different AChR subunits in the same organism represents primary and secondary target sites, yielding different responses based on their relative compositions (98). Variations in prevalent detoxification enzyme isoforms also contribute to neonicotinoid selectivity, evident in honeybees, bumblebees, and the *Sphingomonas* gut symbionts of cotton aphids that mediate imidacloprid detoxification (100, 102).

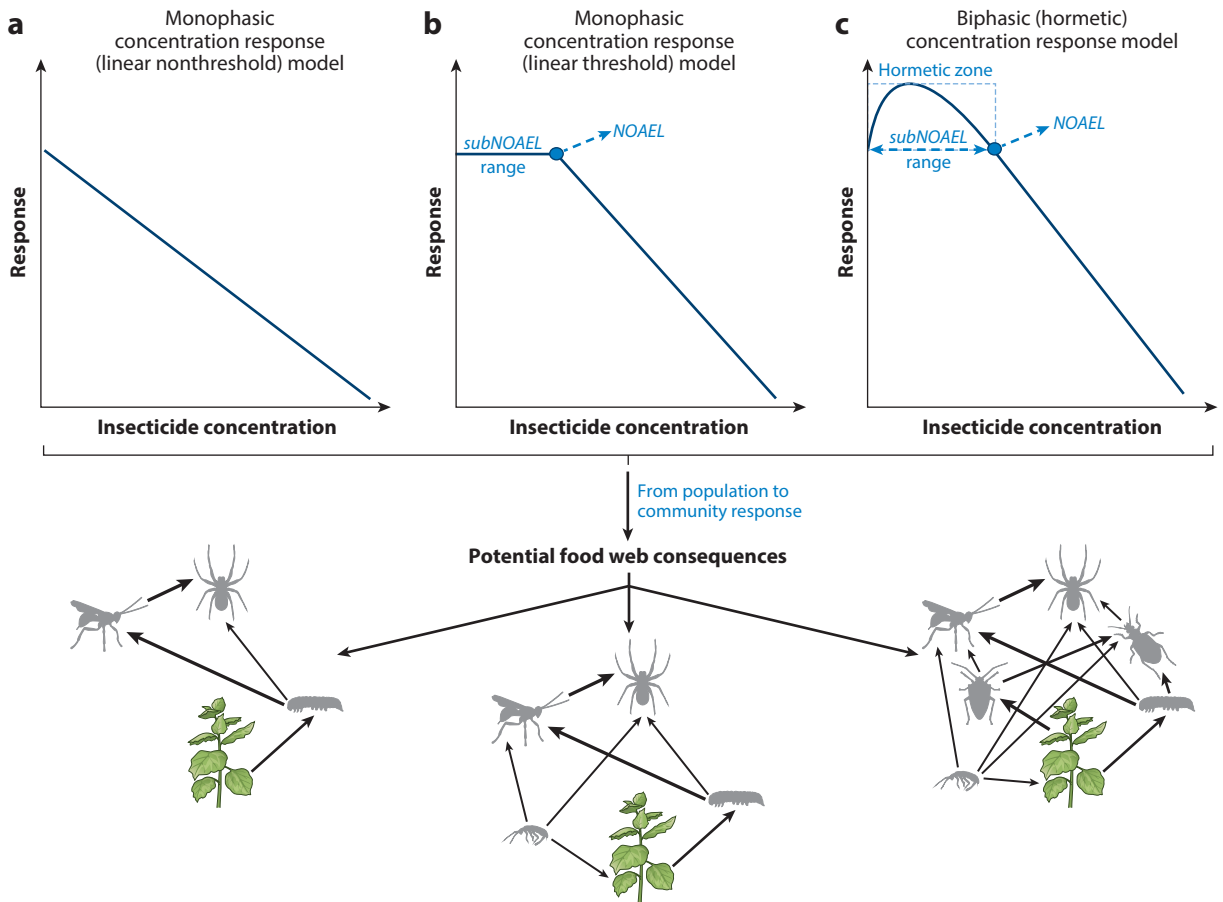
Beyond the primary target site and mode of action, neonicotinoids exert subtle effects on nontarget organisms. Imidacloprid affects stingless bee larvae brain development at low doses (150), while the neurotoxic bioinsecticide spinosad and the organophosphate temephos induce developmental malformations and gut poisoning (1, 20). Additionally, pesticides can disrupt immune responses (96, 159) and alter DNA methylation patterns, as seen in mayflies (60), highlighting the complex pathways through which pesticides induce harm.

**Symbiotic ecocosm:**

an organism serving as an ecosystem to its endosymbionts and including their closed-loop interactions

**Homeostasis:**

an organism's process of maintaining a stable internal environment suitable for sustaining life



**Figure 2**

Alternative monophasic and biphasic toxicological models indicating the hypothetical effects of insecticide stress and their potential cascading to the community, represented as food webs of diverse complexities. Pesticide stress levels are exemplified as pesticide concentration, but pesticide dose and length of exposure are also valid and complementary alternatives to consider, depending on the study design. The integration of both pesticide concentration/dose and length of exposure is particularly important, although not represented here due to aesthetic design limitations. The different population-level responses among exposed species of a food web may lead to different food web outcomes, decreasing, maintaining, or increasing food web complexity, as illustrated by the three alternative food web diagrams indicated. The different arrows and their thicknesses indicate the direction and the strength of the interaction, respectively.

### 3.2. Gene Regulation in Response to Pesticide Exposure

The biochemical alterations caused by pesticide exposure often have a genetic basis, with contributions from epigenetic modulation. For instance, the susceptibility of honeybees and bumblebees to neonicotinoids hinges not on differences in target site sensitivity but rather on variations in the functional expression of specific cytochrome P450 genes—CYP9Q3 in honeybees and its ortholog CYP9Q4 in bumblebees—that metabolize these compounds (102). Changes in gene expression driven by epigenetic mechanisms also play a crucial role in shaping the pesticide stress response (9, 108).

Environmental stressors, including pesticides, can influence phenotypic responses by interfering with the expression of heritable traits. DNA methylation, a common feature among most

**Ortholog genes:** genes in different species that evolved from a common ancestral gene through speciation

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**Reactive oxygen species (ROS):** highly reactive and pervasive molecules formed from diatomic oxygen (O<sub>2</sub>), water, and hydrogen peroxide

**Molecular chaperone:** polypeptides and proteins important for protein folding or unfolding, protein assembly or disassembly, and eventual translocations into organelles

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insect orders, involves the attachment of a methyl group to the 5-carbon position of cytosine nucleotides. The consequences of such methylation vary on the basis of its location—whether on promoter regions affecting downstream gene expression, on transposable elements suppressing their activity, or on gene exons influencing gene expression and promoting alternative splice variants (23). This epigenetic modification may persist for generations, characterizing transgenerational stress inheritance (23, 108). For example, imidacloprid exposure reduces DNA methylation of cytochrome P450 enzymes in Colorado potato beetles (*Leptinotarsa decemlineata*), enhancing detoxification enzyme activity (22).

Pesticide-induced epigenetic alterations have been documented in several insects, influencing insecticide susceptibility in honeybees and the Colorado potato beetle (22, 105, 116). These changes may either increase or decrease susceptibility or simply serve as a marker for pesticide exposure in nontargeted natural populations, as observed in the mayfly *Andesiops torrens* (60). Regardless, pesticide-mediated gene regulation translates into biochemical consequences that potentially manifest as physiological responses.

### 3.3. Physiological Consequences of Pesticide Exposure

Pesticide interactions with their primary target site are typically aimed at causing pest organism mortality. However, secondary target sites and modes of action may lead to secondary sublethal responses, and even nontoxic effects, which are significant for exposure assessment. While sublethal (toxic) responses impose direct physiological costs on the exposed organism, challenging its survival and reproduction (64, 73), nontoxic effects, despite appearing less significant, play a crucial role in exposure assessment and evoke protective responses (64). The misperception of nontoxic responses deserves acknowledgment, and the pesticide-induced expression of antioxidants and heat shock proteins exemplifies this phenomenon.

Pesticides can elevate metabolic rate and oxygen consumption in arthropods, producing reactive oxygen species (ROS) and triggering oxidative stress when ROS levels exceed antioxidant capacity (124). This results in oxidative damage to lipids, proteins, and DNA, compromising the organism's survival or reproduction (124, 157). The exposed organism's defense against oxidative stress involves an antioxidant system and heat shock proteins. The former includes antioxidants such as ascorbate, glutathione, and carotenoids, as well as antioxidant enzymes like superoxide dismutase, catalase, and glutathione transferase (95, 117). Heat shock proteins act as molecular chaperones, preserving cellular homeostasis, preventing protein denaturation, and shielding against oxidative stress. However, these protective systems incur physiological costs that may compromise individual development and/or reproduction (124, 157), counterbalanced by their potential cross-protection against other environmental stressors (25).

### 3.4. Behavioral Responses to Pesticide Exposure

Behavior is the integrated response of organism physiology to environmental conditions. Pesticides impact arthropod physiology and often lead to behavioral changes, which can serve as sensitive biomarkers for environmental contamination (64). Despite traditionally being secondary considerations, behavioral assessments are gaining attention due to new methodologies like artificial intelligence-mediated video-tracking systems and electropenetrography (12, 16, 139). Behavior serves as a source of variation in response and adaptation to pesticide stress, evidenced by avoidance behaviors observed in mosquito vectors and other pest species (30, 41, 64). It also reflects consequences of exposure, such as insecticide behavioral resistance, repellence, irritability, and resistance-associated mating preferences, which may exhibit dose-dependent or dose-independent responses (30, 37, 41, 115).

An essential consideration is that pesticide-mediated arthropod behavior is often evaluated as isolated traits rather than an integrated set of behavioral traits determining individual responses. Despite evidence supporting the importance of this integrated approach among the maize weevil *Sitophilus zeamais* and the lady beetle *Eriopis connexa* (111, 128), it remains underappreciated. This integrated approach is crucial for scaling up to population-level impacts, particularly those on nontarget species.

#### 4. SIDE EFFECTS AT THE POPULATION LEVEL

The dose response relationship, as discussed above, is a cornerstone of toxicology, but response is dictated not only by dose but also by exposure duration, which is a key determinant of pesticide penetration into individuals. Evaluating toxicity requires accounting for both exposure time and dose (or concentration), assessed in combination, as seen with fumigants like phosphine (10, 21). This approach is crucial for extrapolating individual-level effects to populations, particularly in pest management and environmental risk assessment of synthetic toxicants and natural toxins.

##### 4.1. Changes in Population Growth

Mortality, typically assessed through the median lethal dose (LD<sub>50</sub>) or median concentration (LC<sub>50</sub>) in traditional dose/concentration response models, is a crucial parameter in assessing toxicity. While this parameter is important for pest control, other life-history traits are more relevant for evaluating side effects on nontarget organisms. Population growth rate, shaped by factors such as age at first reproduction, fertility, and lifespan, provides a broader ecotoxicological end point (34, 64). However, even this metric is limited in capturing behavioral interactions, density-dependent regulation, transgenerational effects, and environmental stochasticity, requiring more complex models (64).

Pesticides influence both direct and indirect population interactions, including mating preferences and larval competition strategies. For instance, pesticide exposure affects maize weevils' life-history traits and population growth (19, 37, 63). Even passive dispersal by predatory mites can be altered, as seen in *Neoseiulus baraki*, where exposure to bioinsecticides and a synthetic pesticide compromised population size and growth at the treated site (110). Transgenerational pesticide effects and changes in the progeny's functional response in generalist predators further complicate population dynamics (2).

##### 4.2. Changes in Population Dynamics

Population growth models, integrating growth rate and environmental carrying capacity, are particularly sensitive to pesticide exposure, especially in nontarget species and biopesticide applications. Nontarget species often face sublethal exposure, while biopesticides generally induce lower mortality than synthetic pesticides (64, 72). This complexity is further evidenced in arthropod population dynamics, including metapopulations and chaotic fluctuations, which may be influenced by pesticide exposure.

Landscape structure, such as mosaic patterns, and metapopulation dynamics buffer the negative impacts of pesticides, as observed in the red mason bee *Osmia bicornis* and the Eastern monarch butterfly *Danaus plexippus* (14, 62). Chaotic population dynamics—characterized by deterministic, nonlinear fluctuations—are common in insects but are often neglected, despite their potential vulnerability to stressors such as insecticides. This has been demonstrated in species like *Tribolium* flour beetles (44, 113).

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**Median lethal dose (LD<sub>50</sub>) or concentration (LC<sub>50</sub>):** toxicity parameter referring to the estimated dose/concentration required to kill 50% of a population within a set exposure period

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**Hormesis:**

phenomenon characterized by a biphasic dose response relationship of a stressor

**Biphasic dose**

**response:** alternative toxicological model where responses shift from stimulation at low doses to inhibition or toxicity at high doses

### 4.3. Hormesis

Hormesis adds complexity to pesticide–arthropod interactions by introducing a biphasic dose response, where low doses of a stressor stimulate biological functions while high doses suppress them (3, 69) (**Figure 2**). Hormesis has become an important toxicological model, especially for examining sublethal responses, making it highly relevant when one is considering pesticide side effects on nontarget species (6, 40, 103). This phenomenon is not restricted to synthetic pesticides but also applies to biopesticides, including bacterial entomotoxins, such as Cry toxins from *Bacillus thuringiensis* (Berliner) (28, 122).

The underlying mechanisms of hormesis involve overcompensation responses to mild stress or resource reallocation, where energy is redirected between physiological pathways and life-history traits. The latter involves physiologically reallocating resources—such as surplus energy or stressor provision of scarce elements—or shifting energy between competing physiological pathways and life-history traits (3, 83). The underlying biochemical mechanisms of hormesis include the inhibition of secondary receptors, desensitization of stressor receptors, and upregulation of metabolic enzymes that neutralize toxins (40). Regardless of the mechanism, hormesis influences life-history traits and, in turn, population growth and dynamics.

### 4.4. Insecticide Resistance

Insecticide resistance emerges as an ecological consequence of genetic adaptations to selective pressure from toxicants (65, 72). This phenomenon, often resulting from the overuse of insecticides, serves both as an environmental biomonitoring tool to assess insecticide exposure and as a significant challenge for pest management, leading to control failures and increased costs (65).

Research on insecticide resistance primarily focuses on pest species subject to intensive selection due to repeated insecticide exposure. While resistance is typically associated with differential mortality, survival and reproduction under sublethal exposure also play crucial roles, as they also allow for differential selection favoring the resistant genotypes (64, 72). However, two biases, an overemphasis on mortality-based selection and a narrow focus on pest species, persist in resistance studies. Consequently, studies on nontarget organisms—such as natural enemies, detritivores, pollinators, and aquatic insects—are scarce. Resistance in natural enemies is recorded approximately ten times less frequently than in pest species, a likely result of the low number of studies and fewer cases due to the lower levels of pesticide selection for resistance among natural enemies than among (targeted) pest species (17, 72). Cases of pesticide resistance among pollinators, detritivores, and aquatic species receive even more limited attention (8, 72, 86).

The prevailing utilitarian perspective on insecticide resistance tends to focus on natural enemies and nontarget pest species rather than other nontarget organisms. Natural enemies are of particular concern due to potential conflicts between chemical and biocontrol tactics, driving research on integrating insecticides with biocontrol agents (18). Meanwhile, the focus on nontarget pest species stems from concerns about shifts in pest dominance and secondary outbreaks (66). However, recognizing sublethal exposure as a major driver of resistance underscores the need for broader research on its ecological and evolutionary implications.

## 5. SIDE EFFECTS AT THE COMMUNITY LEVEL

Toxicological and ecotoxicological testing has traditionally focused on isolated species in laboratory or field studies. However, this reductionist approach is unrealistic, as species do not exist in isolation in nature. A shift is needed from single-species tests to considering co-occurring, interacting species in ecotoxicology and impact assessments (26, 64). Embracing multiple-species

bioassays, a concept gaining attention (50, 77), is crucial, although this paradigm shift is still in its early stages, particularly in entomology and arthropod pest management (133).

### 5.1. From Shifts in Species Dominance to Outbreaks

Expanding our understanding of toxicants requires considering the simultaneous exposure of interacting species. Traditional pest–natural enemy studies with pesticides often rely on differential toxicity and neglect heterospecific interactions. Recent studies on sublethal effects have revealed pesticide impacts such as developmental delays, delayed reproduction, reduced fertility, and altered dispersal, which complicate temporal dynamics and introduce complexity through pulse interventions like pesticide applications (64, 74).

The interaction between insecticides and biocontrol agents in pest management is complex, with some studies showing synergistic effects (74, 84). This complexity extends to biopesticides, where predictions of synergy are not usually assessed in pest–predator interactions (85). Similarly, pesticide impacts on plant–insect interactions are notable, particularly with seed treatments and systemic pesticide use (59, 89, 92). Plants, common targets for insecticide applications, respond to systemic insecticides, inducing a continuum of responses from phytotoxicity to phytotonic effects (bioactivation) (68). This complexity is reflected in the plant's expression of insecticide-mediated hormesis and the intricate plant stress response through the jasmonate (JA) and salicylic acid (SA) phytohormone pathways. Neonicotinoid exposure in crop plants, for instance, triggers antioxidant defenses and induces SA production, affecting ecological relationships and potentially leading to shifts in pest species dominance while providing defense against pest insects and non-necrotrophic pathogens (5). The cross talk between this pathway and the JA pathway further complicates the defense against mites and necrotrophic pathogens, allowing for shifts in pest species dominance (68).

Surprisingly, pesticide effects on heterospecific competition are poorly documented, although they can alter ecological relationships, leading to shifts in species dominance and competitive displacement. Differences in species susceptibility to insecticides can shift ecological dominance under intermediate stress levels, as observed among grain beetles and aphids (36, 109). In cases of pesticide-mediated hormesis and insecticide resistance, the likelihood of a shift in dominance in favor of another (secondary) species increases, raising the risk of pest outbreaks (66). Such shifts can present challenges in pest management, but they can also play positive roles in ecosystems, such as facilitating biogeochemical cycling during invertebrate herbivore outbreaks, providing nutrient sources for recycling in the savanna, and favoring pest-induced tree-dieback events that lead to greater forest heterogeneity (66).

### 5.2. Changes in Species Assembly and Community Structure

Species assemblies are collections of species populations occupying the same habitat. Habitat contamination exposes co-occurring species, with effects ranging from negative to neutral and sometimes positive. This complexity is greater than in predator–prey or competitive interactions, as noninteracting species may also be exposed and influenced by the pesticide. Even at a microscale, individual arthropods host microbiota that could undergo changes in composition and dominance with consequences for the host organism (31, 156). Arthropod and other invertebrate assemblages are also subject to pesticide-mediated changes in composition (72, 76, 94), as are arthropod communities.

Communities are groups of interacting populations of different species inhabiting a given site at a given time. Pesticides not only affect communities but also may play a role in shaping the initial community and influencing species colonization of contaminated areas, an aspect

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**Phytotoxic:** adverse or toxic effects of a compound on an exposed plant

**Phytotonic:** beneficial effects of a compound on an exposed plant

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often neglected (69, 72). Few long-term studies of pesticide effects on terrestrial arthropod communities exist, with most focusing on short-term effects. Food web studies for genetically engineered crop plants that express insecticidal proteins originating from *Bacillus thuringiensis* (Bt crops) are available, but they often use low-level trophic groups, limiting analysis resolution (146). Even when such studies have higher resolution, they are typically short term, extending to two cultivation cycles, with none detecting significant pesticide effects on the trophic network and associated food web, as observed in tropical fields of maize and cotton (99, 162, 163). However, imidacloprid seed treatments in soybeans have been shown to enhance trophic network complexity, suggesting a potential community-level hormetic effect (125).

Pesticide-induced hormesis can alter arthropod communities by affecting a keystone species, by shifting community composition, or by having complex effects on multiple species. This can result in community hormesis outcomes (40, 69) (Figure 2). Examples of such effects exist among soil microbial communities, but other environments remain understudied. A major shortcoming in current studies is their focus on single stressors, while multiple stressors, including pesticides, often co-occur in nature (66). The coapplication of pesticides and under variable weather conditions makes for a complex set of (artificial and natural) stressors and responses that can lead to antagonism, potentiation, or synergism, affecting existing communities (59, 125). Thus, a more holistic approach is needed to understand stressor interactions and species dynamics in natural systems.

## 6. IMPACTS ON ECOSYSTEM SERVICES

Pesticides are major environmental stressors that disrupt biological processes, often with far-reaching consequences. Despite the ubiquity of pesticides, the focus on pesticide effects tends to center on ecosystem services, particularly regulating, provisioning, and supporting services. Ecosystem services, dating back to Plato's time (~400 BC), refer to the conditions, processes, and assets through which natural ecosystems benefit human life. The concept, popularized by the Millennium Ecosystem Assessment, emphasizes the importance of natural capital, although monetizing these services can oversimplify complex ecological relationships (88).

### 6.1. Plant Health and Yield

Crop plants serve as a cornerstone of human survival and play a central role in provisioning ecosystem services. However, they are frequently subjected to insecticide applications, notably neonicotinoids, raising concerns due to their systemic nature (90). These pesticides can cause phytotoxic (harmful) and phytotonic (beneficial) responses, complicating the impact assessment. While phytotoxicity can reduce plant health and yield, phytotonic effects may enhance plant performance, creating a dynamic interplay within ecosystems (68, 125).

The relationship between insecticides and crop plants is complex, affecting entire trophic levels. A biphasic dose response continuum, ranging from low-dose bioactivation to high-dose toxicity, underscores the profound implications for ecosystem services provided by crops (5, 68). This continuum was initially observed with herbicides and fungicides and extends to insecticides, where cascading effects may directly and indirectly influence other trophic levels and ecosystem services (5, 49, 125).

### 6.2. Biocontrol

Biocontrol is a key regulating service, helping manage pest populations. The integration of biocontrol with pesticides has received considerable attention, as pesticides can negatively affect natural enemies, thus jeopardizing biocontrol effectiveness safeguarding their ecosystem service provision

(132, 160). While augmentative and conservative biocontrol methods are well-studied, classical biocontrol, which introduces natural enemies, remains underexplored due to environmental concerns over invasive species (97, 147).

Pesticides often target pest species without considering the indirect effects on biocontrol agents. Most pesticide testing fails to incorporate pest–prey interactions, compromising ecological balance (7, 64). This oversight is especially problematic for biopesticides, which are often tested in isolation without considering their potential effects on biocontrol agents (88, 153). Comprehensive testing that accounts for these broader ecological interactions is essential for sustaining biocontrol practices.

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**FIFRA:** Federal Insecticide, Fungicide, and Rodenticide Act

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### 6.3. Pollinators

Pollination is a vital regulating service influenced by pesticides, especially neonicotinoids, which have high toxicity to pollinators like honeybees. These insecticides have drawn significant attention due to their systemic nature and the decline in pollinator populations (15, 138). Initial studies focused on adult mortality but have expanded to include sublethal effects and impacts on hive-level dynamics (15, 46). Beyond neonicotinoids, other pesticide classes, including biopesticides like spinosyns and azadirachtins, also affect pollination but have received less empirical testing (15).

While honeybees are frequently studied as flagship species, native pollinators are often overlooked in research (13, 82). Honeybees, although important for agriculture, are invasive in some regions, underscoring the need for a broader focus on native species (13, 35). The decline of these species poses significant ecological risks, highlighting the importance of considering the full spectrum of pollinators in pesticide regulation.

### 6.4. Decomposers (Soil Arthropods)

Decomposers are critical for nutrient cycling and soil health. They break down organic matter, contributing to soil fertility, water retention, and overall ecosystem functioning. Microorganisms and detritivores (soil arthropods) play essential roles in waste decomposition, yet they are vulnerable to pesticide exposure, including herbicides, fungicides, and especially broad-spectrum insecticides (119, 121).

Exposure to pesticides can lead to shifts in their composition and activity, with potential consequences for ecosystem functioning (112, 134). The consequences of pesticide-induced declines in decomposer populations extend beyond soil health, potentially affecting adjacent ecosystems such as streams associated with agricultural fields (38). However, research on the impacts of pesticides on decomposers is limited, with few studies examining the long-term consequences of their disruption. Although the role of detritivores in ecosystems is widely recognized, their vulnerability to pesticide-induced stress remains underexplored (121, 127).

## 7. RISK ASSESSMENT SCHEMES FOR PESTICIDE REGISTRATION

Pesticide risk assessment for arthropods, particularly pollinators and other nontarget species, is crucial in regulatory frameworks in industrialized regions, including the European Union (EU), United States, China, and South America. These assessments ensure that pesticides do not negatively affect beneficial species vital to ecosystem services, such as pollination, biological pest control, and nutrient cycling. While the EU operates under Regulation (EC) 1107/2009, guided by the European Food Safety Authority, the US framework is managed by the Environmental Protection Agency under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), which

also recognizes pesticide exemptions from regulatory requirements [FIFRA section 25(b)], and the Endangered Species Act.

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**Integrated pest management (IPM):** strategy combining multiple pest control methods to reduce pesticide use while maintaining economic and ecological sustainability

**EC:** European Commission

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## 7.1. Risk Assessment Frameworks

Tiered risk assessment systems to evaluate pesticide impacts on nontarget arthropods are common in the EU and United States. In the EU, first-tier assessments include lab tests on surrogate species like parasitoid wasps and predatory mites to assess acute and reproductive toxicity. For pollinators, studies focus on acute, chronic, and larval toxicity, with higher-tier evaluations considering sublethal and population-level impacts in semifield or field conditions (78, 129). Advanced methods, such as behavioral assays and population modeling, offer insights into long-term risks (45, 57, 61).

In the United States, initial tests use honeybees as proxies for other nontarget arthropods. Risk quotients are calculated on the basis of acute and chronic toxicity data and environmental exposure estimates. When thresholds are exceeded, semifield and field studies assess sublethal effects, like impaired foraging and navigation (51, 145). Mitigation strategies, such as buffer zones, timing restrictions, and integrated pest management (IPM) practices, are considered to reduce pesticide risks (47, 143). Advanced computational models are also adopted to predict long-term impacts (52, 78).

## 7.2. Advancing Sustainable Practices and Current Needs

Risk assessment frameworks attempt to balance agricultural productivity with the protection of pollinators and other nontarget arthropods. These frameworks should support sustainable farming while preserving ecological integrity. However, challenges persist, such as pesticide registration applications bypassing extensive lab or semifield assessments (Tier 2), relying instead on recolonization potential in treated fields on the basis of the debatable assumption that such fields will be naturally recolonized after pesticide impact (Tier 3). While field trials often use less intensively grown crops (29), they may fail to detect population-level impacts in large monocultures dependent on chemical pesticides.

Advanced methodologies, including molecular approaches and population modeling, continue to improve the accuracy of risk assessments (27, 123). However, effects such as sublethal ones and chronic toxicity are crucial in pesticide risk assessments for nontarget arthropods (45, 78). These effects could be evaluated through extended laboratory and field studies. Regulatory frameworks should increasingly incorporate chronic exposure data to ensure long-term ecological safety and sustainability in pest management. Moreover, biopesticides, such as botanical pesticides, should be assessed similarly to chemical pesticides, given their potential side effects on nontarget arthropods (58, 67).

## 8. CHALLENGES FOR SUSTAINABLE PESTICIDE USE

Pesticide use is a double-edged sword—an essential tool for protecting human, animal, and crop health, yet fraught with risks to human and environmental safety. The ecological repercussions, such as pesticide resistance and pest outbreaks, create a pesticide treadmill, where initial pesticide use begets more use (64, 66). This intricacy presents a challenge without a straightforward solution, prompting the ongoing advocacy for sustainable pesticide use (101), which brings us to a foundational challenge—defining sustainable pesticide use.

The European Commission (EC) defines sustainable pesticide use as reducing pesticide risks without compromising pest management efficacy (EU Directive 2009/128/EC). The EC aims for a 50% reduction in pesticide use by 2030, advocating IPM approaches, including biopesticides

and innovative formulations. Initiatives range from novel chemistries and biopesticides (75, 141) to innovative formulations and application systems (106, 137) and monitoring strategies (114, 148).

### 8.1. Nontarget Impact in Organic Versus Conventional Systems

Organic farming systems, relying on biopesticides and devoid of synthetic chemicals, are often viewed as safer alternatives (42, 101). However, safety depends on the chemical properties of the compound, not its origin. Contrary to common belief, organic systems do not necessarily guarantee lower pesticide residues or superior food quality (56, 127). This challenges the perception of the safety and quality of organic produce, aligning with the EC's approach of disregarding the distinction in regulating pest control agents (Council Directive 91/414/EEC and subsequent EC Regulation 1107/2009).

Despite being perceived as safer, biopesticides and organic systems are not without risks. They can induce nontarget impacts, even when considered less toxic than synthetic pesticides (67, 153). Furthermore, the environmental context of pesticide application—such as faster degradation rates—raises concerns about their sustainability, as more frequent applications may be required (42, 67). Neglecting the indirect impacts on ecosystems, such as effects on food webs, remains a significant gap in assessing organic and biopesticide safety.

### 8.2. Monoculture and Polyculture Systems

Developing safer pesticides with lower environmental persistence is challenging, particularly when increased use compensates for their reduced longevity (42, 101). This issue is particularly evident in monoculture systems, for which extensive single-crop cultivation leads to pest outbreaks and, consequently, more pesticide use (66, 144). Although exceptions exist, global trends favor increasing pesticide use in monocultures (135).

In contrast, polyculture systems, which foster crop diversity and are integral to agroecological approaches (152, 158), can promote sustainable pesticide use by enhancing natural enemy populations and improving pest control (149, 152). However, polyphagous pests, such as the fall armyworm (*Spodoptera frugiperda*), may adapt to diversified landscapes, requiring high-scale, multicropping approaches for sustainable insecticide use, a challenge exacerbated by resistance issues (87).

### 8.3. Seed Coating

Seed coating, an increasingly popular pesticide application modality, introduces challenges to sustainable use based not just on compound selection but also on application methodology (79, 89). Despite historical examples involving compounds such as brine and copper sulfate, modern seed coatings, especially those containing neonicotinoid insecticides, have sparked controversy (59, 79, 125). In seed coating, pesticides carried by the seed are solubilized in the soil, are absorbed by plant roots, and translocate to the canopy, offering control over early insect pests (89). While seed coating is touted as ecologically and economically justifiable, reducing pesticide use and associated risks (33, 79), potential misconceptions must be addressed.

Cost reduction with seed treatments is demonstrable, enabling simultaneous application of multiple compounds during seed sowing. However, claims of lower health risks and nontarget impacts may stem from misperceptions. Pesticides in seed coats can alter the soil biota, cascading to other trophic levels (125, 162) and even extending to passerine birds (120). Their potential persistence raises risks of soil, water, and plant tissue contamination (79, 89). Nonetheless, recent advances in biological and nano-enabled seed treatments hold potential for mitigating these risks,

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**Organic farming system:** farm

cultivating system devoid of use of synthetic compounds, thus relying on natural (input) agents

**Polyculture system:**

farming cultivation system that employs increased crop diversity as an integral component of its practices

**Seed coating:**

technique in which one or more biologically active ingredients are applied to the seed surface by using a carrier

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but more research is needed to assess their long-term ecological impacts, contributing to a more sustainable practice (136).

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**Monophasic dose response:**

conventional toxicological model where toxicity increases linearly with dose or concentration

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#### 8.4. Pesticide Mixtures

Beyond concerns about compound type and application, the use of pesticide combinations poses a significant challenge to sustainability. In contrast to the use of individual pesticides, which seldom occurs (67, 70), environments frequently experience multiple stressors, such as nutrient deficiency and water or heat stress, alongside multipesticide use (66, 67). Seed coating typically involves an insecticide with a fungicide, sometimes even an herbicide, and plant nutrients (79, 89).

Antagonism, potentiation, and synergism arise when pesticides are coapplied or sequentially used. This practice allows residues to overlap, increasing uncertainty and risk (11, 80). In antagonism, one pesticide reduces the effectiveness of the other, while potentiation increases toxicity. Synergism, a particular type of potentiation, occurs when nontoxic components enhance the toxicity of others in the mixture. The mechanisms underlying antagonism and potentiation involve the mode of action of the mixture components and their cross-interference with activation or detoxification processes (80). The cascading risks for nontargeted species, as recorded for pollinators (161), birds, and humans (107, 116), underscore the challenge for their sustainable use, emphasizing the need for a cautious approach to pesticide mixtures to ensure sustainable practices.

### 9. FUTURE OUTLOOK

The history of pesticides is deeply entwined with the evolution of agriculture and scientific progress (43, 48). Despite environmental concerns, megatrends such as rapid urbanization, climate change, and technological advancements suggest continued reliance on pesticides, particularly insecticides, to mitigate disease transmission and secure food production. The challenge now lies not in eliminating pesticides but in refining their selection and application. However, persistent misconceptions in this field call for critical reassessment.

Even the cornerstone principle of toxicology—Paracelsus’s 1538 maxim “the dose makes the poison”—warrants revision. Traditional models emphasize dose and concentration but often overlook exposure duration, a crucial factor. Similarly, the conventional monophasic dose response model does not fully capture sublethal effects, which often follow a biphasic (hormetic) pattern (4). Given that pesticide residues in the environment typically persist at sublethal levels due to degradation, target and nontarget species are chronically exposed, leading to subtle yet significant ecological consequences.

Over the past three decades, societal concerns have driven the development of narrow-spectrum and low-persistence pesticides (140). While these are environmentally favorable, their reduced longevity often necessitates more frequent applications and pesticide mixtures, increasing the likelihood of interactions with other stressors in complex ecological networks. Understanding these interactions is key to advancing sustainable pest management.

Similarly, heightened interest in natural pesticides, particularly biopesticides, reflects societal emphasis on safety. However, the assumption that natural origin equates to safety is flawed. Furthermore, the frequent use of natural compounds at levels exceeding their natural background concentrations imposes environmental stress and may lead to unforeseen and unintended consequences, highlighting the need for rigorous case-by-case evaluations akin to those applied to synthetic pesticides.

The growing diversity of pesticides, evolving application strategies, and intensifying ecological concerns underscore the need for innovative research tools. Approaches such as the stress response

pathway offer promising avenues for realistic risk assessments. By integrating pesticide ecotoxicology with stress ecology, researchers can unravel complex interactions and cascading effects on nontarget organisms. In this quest for clarity and sustainability, the mandate is clear—*fiat lux!*

### SUMMARY POINTS

1. Stress responses are integrated phenomena, originating at the molecular level and escalating to higher biological organizations.
2. Secondary sites and modes of action are crucial, particularly for sublethal responses, not just lethality.
3. Physiological stress responses from molecular interactions are often translated into integrated behavioral outcomes, with dose/concentration and exposure length determining toxicity.
4. Population growth, rather than mortality alone, provides a more comprehensive toxicological end point, although complex dynamics such as metapopulations and chaos can complicate this assessment.
5. The biphasic dose response (hormesis) model is increasingly preferred over the traditional monophasic model, especially when one is considering sublethal effects.
6. Insecticide resistance is influenced by sublethal exposure, and hormesis further complicates this phenomenon, with most studies focusing on primary pest species.
7. Species and stressor interactions are vital to consider in insecticide impact assessments, as no species or stressor exists in isolation.
8. Differential insecticide effects on co-occurring species can shift species dominance, alter community structure, and cause outbreaks, with many studies being short term or focusing on low-level trophic groups.

### FUTURE ISSUES

1. Integrated stress responses within organisms, considering their endosymbionts and microbial fauna, and between interacting organisms need more consideration.
2. Secondary sites and modes of action in both conventional pesticides and biopesticides are critical, as they go beyond mortality and are harder to predict.
3. Integrated behavioral responses should be linked to their underlying physiological stress responses.
4. The interference of insecticide exposure with complex population dynamics is neglected, compromising forecasting and warranting further research.
5. Hormesis and insecticide resistance complicate impact risk assessments, particularly when one is considering nontarget pest species, natural enemies, detritivores, pollinators, and aquatic insects.
6. More attention is needed on multiple species bioassays, as they better reflect species interactions in natural settings.

7. The possibility of community hormesis among arthropods remains unexplored, with unknown environmental consequences requiring investigation.
8. Research on urban and periurban areas is neglected, despite their potential impact on human populations.
9. The co-occurrence of multiple stressors is prevalent, yet insecticide studies often focus on single stressors, necessitating suitable pesticidal combinations and (positive) controls.

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