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BIPHASIC DOSE RESPONSES IN INSECTS: MECHANISMS AND IMPLICATIONS FOR INTEGRATED PEST MANAGEMENT

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ABSTRACT

Biphasic dose responses, where low doses of a substance stimulate while higher doses inhibit or cause lethality, are common yet often overlooked in insect toxicology. Known collectively as hormesis, hormoligosis, or pesticide-mediated homeostatic modulation, these non-linear responses challenge traditional toxicological models such as threshold and linear no-threshold frameworks. Insects exposed to sublethal levels of pesticides, ionizing radiation, heavy metals, or temperature stress may exhibit enhanced reproduction, faster development, extended lifespan, and increased expression of detoxification genes, all of which impact pest management outcomes. Studies on species like *Myzus persicae*, *Spodoptera litura*, *Spodoptera frugiperda*, and *Phormia regina* reveal that multigenerational exposure to sublethal insecticide doses can simultaneously boost population growth and hasten resistance development. This review explores the conceptual basis, mechanisms, triggers, and hormetic effects across major insecticide classes, emphasizing the significance for integrated pest management (IPM) and the development of more effective, sustainable control strategies.

***Key words*:** Biphasic dose–response, hormesis, hormoligosis, sublethal effects, insecticide resistance, integrated pest management, *Myzus persicae*, *Spodoptera litura*, neonicotinoids, pyrethroids, organophosphates

Introduction

Traditionally, the evaluation of pesticide effects on arthropods focuses primarily on lethality of the toxicant. Standard pesticide bioassays are typically designed to assess acute toxicity by examining individuals at specific developmental stages, ages, and sizes, with the main goal of quantifying mortality through dose-response experiments using binary outcomes (alive versus dead). While these tests are essential for pest control and regulatory compliance, they often overlook sublethal effects, which provide a subtler and ecologically relevant measure of toxicity (Forbes and Calow, 1999; Stark and Banks, 2003). Acute toxicity assessments generally involve high pesticide doses, thereby constraining insights into the effects of lower and sublethal exposures. This emphasis on high-dose testing has faced growing criticism over recent decades (Calabrese and Baldwin, 2001a; Calabrese and Baldwin, 2001b; Murado and Vázquez, 2007).

Stimulatory effects induced by low doses of substances that are otherwise toxic at higher concentrations, particularly pesticides have been extensively documented and are now recognized as a widespread toxicological phenomenon (Morse, 1998; Calabrese and Baldwin, 2001b; Calabrese and Baldwin, 2003a; Calabrese, 2004). Such responses have been observed in arthropod pest species exposed to pesticides and are considered a potential factor contributing to pest resurgence and secondary pest outbreaks (Dittrich *et al*., 1974). The biphasic dose–response model in insects characterizes a scenario where a compound exerts stimulatory effects at low doses but inhibitory or toxic effects at higher doses.

This review highlights the evidence of biphasic dose

responses in insects and delineates the concepts of hormesis, hormoligosis and homeostatic modulation through pesticides. The review discusses the implications of these phenomena to pest management models including integrated pest management (IPM) and mechanistic evidence and its recent developments with studies from Kerala Agricultural University, India. Conclusions and recommendations are made for gaining from hormesis phenomena while avoiding unwanted hormetic stimulation during pest management interventions in managed and natural ecosystems. Using both classical and recent empirical studies, the review provides insights into a biological phenomenon that has important practical implications for the sustainable control of pest populations in the future.

Conceptual framework of biphasic dose responses in insects

Three terms are commonly used to describe biphasic dose–response phenomena in insects: hormesis, hormoligosis, and pesticide-mediated homeostatic modulation (Calabrese, 2002). Although related, these concepts differ in scope and mechanistic implication.

Hormesis:

Hormesis describes a biphasic dose–response relationship commonly depicted as a J-shaped or inverted U-shaped curve, where low doses of a toxic substance stimulate or enhance biological functions, while higher doses inhibit or cause mortality. Although the concept was initially proposed by Schultz (1887), the term "hormesis" was coined by Southam and Ehrlich (1947). The term originated from the Greek word *hormo*, meaning "to excite" (Calabrese and Baldwin, 2001b). Hormesis is now broadly recognized in toxicology, pharmacology, and biology as a fundamental adaptive response. In organisms, hormetic effects may include increased reproduction, accelerated growth, prolonged lifespan, or improved tolerance to stress following sublethal exposure to pesticides (Calabrese, 2004).

Hormoligosis:

Hormoligosis is a more specific term introduced by Luckey (1968) to describe low-dose stimulation observed specifically in arthropod pests exposed to insecticides. The term was derived from the Greek words - *hormo* (to excite) and *oligo* (minute). Luckey defined hormoligosis as the stimulation caused by very small amounts of stressors in organisms that are already experiencing stress. In contrast to the broader concept of hormesis, hormoligosis implies that the stimulatory effect occurs only when an additional minor stressor is applied to an organism which is already under stress. Consequently, hormoligosis is considered a subset of hormesis and holds particular significance in pest management, where insecticide-induced stimulation may increase pest survival and reproductive capacity (Cutler, 2013).

A recent study by Silva *et al*., (2024) presents compelling evidence of hormoligosis in insects. The research focused on the green peach aphid, *Myzus persicae* Sulzer, exposed to two insecticides, chlorpyrifos (an organophosphate) and deltamethrin (a pyrethroid), across four temperature regimes (15, 20, 25, and 28°C). A temperature-dependent decrease in LC_{50} values was reported, indicating an increased insecticide toxicity with rising temperatures. Chlorpyrifos toxicity was four to five-fold greater between 15 and 25°C, while deltamethrin exhibited peak toxicity at 28°C. Notably, sublethal exposure to both insecticides, combined with varying temperature conditions, significantly influenced fecundity and survival in *M. persicae* females. The presence and extent of stimulatory hormoligotic responses were dependent on the specific sublethal concentrations and varied with temperature (Silva *et al*., 2024).

Pesticide-Mediated Homeostatic Modulation:

The concept of "pesticide-mediated homeostatic modulation" was introduced by Cohen (2006) to describe stimulatory effects observed in non-target arthropods exposed to sublethal pesticide doses. Unlike traditional hormesis, these responses do not necessarily exhibit a characteristic J- or U-shaped dose-response curve. A notable example is the increased reproduction of ticks following exposure to chemicals such as carbaryl, DDT, pyrethroids, or imidacloprid. Although these compounds are not primarily intended as acaricides, they have been shown to promote tick population growth when present in shared habitats (Cohen, 2006).

Dose–response models

Toxicity evaluations using bioassays often generate dose-response data that are then fitted either to threshold-type models (to derive NOEL/NOAEL/ZEP or benchmark doses) or to linear no-threshold (LNT) models, especially for genotoxic carcinogens where any nonzero response is treated as carrying some risk. In contrast, many cellular or mechanistic bioassays show biphasic (J- or U-shaped) curves consistent with hormesis, where low doses produce stimulation and higher doses inhibition, so the fitted model must allow a non-monotonic response and explicitly estimate the Zero Equivalent Point (ZEP) at the transition between these phases.

Toxicological evaluations have traditionally been

Table 1: Developmental success of cadmium-fed larvae of *Phormia regina* Meigen (Nascarella *et al*., 2003).

Treatment group	Cadmium concentration (ppm)	Pupation (%)	Emergence (%)
A	200.0002	0.0	0.0
B	2.0002	70.9	20.8
C	0.2002	80.7	54.5
D	0.0202	86.7	57.8
E	0.00022	88.1	67.7
F (Control)	>0.0002	74.4	79.2

based on two primary models: the threshold model and the linear no-threshold (LNT) model. The threshold model assumes a dose below which no harmful effects occur, thereby establishing the basis for defining safe exposure limits. Key parameters derived from this model include the no observed effect level (NOEL), the no observed adverse effect level (NOAEL), and the zero-equivalent point (ZEP), which collectively delineate the safety boundary (Cox, 1987; Hoffmann, 2009). Conversely, the LNT model asserts that any level of exposure, no matter how small, entails some risk, with adverse effects decreasing linearly as the dose is reduced. Both models are monotonic, predicting a consistent increase or decrease in effect throughout the dose range.

Hormesis diverges from the traditional monotonic models by exhibiting a non-monotonic, biphasic dose-response relationship. The characteristic J-shaped or U-shaped curve of hormesis illustrates that low doses enhance biological activity, while higher doses exert inhibitory or lethal effects (Calabrese, 2008). The zero-equivalent point (ZEP) is a key feature of hormesis, representing the dose at which the response shifts from stimulation to inhibition. This dual response pattern highlights hormesis as both a conceptually significant and practically valuable model for interpreting toxicological effects in biological systems.

Hormesis in insects:

Hormesis challenges the conventional belief that all exposure to toxins is inherently harmful. Hormetic effects in insects can be observed as improved resistance to stress, increased lifespan, accelerated growth and development, or enhanced reproductive capacity. These effects are typically the consequence of physiological modifications, such as the activation of stress-response genes, the upregulation of detoxification enzymes, or behavioural modifications that facilitate survival. For example, research has demonstrated that exposure to low doses of DDT or carbaryl can enhance reproductive output in a variety of arthropod species (Hoffmann, 2009).

Stebbing (1998) underscores the importance of interpreting hormesis in the context of dose-time relationships. Homeostasis may be initially disrupted by exposure to low doses of toxic substances, which may then induce an over compensatory response that results in stimulation. In certain circumstances, stimulation may occur immediately without a preceding phase of disruption. Insect populations are persistently exposed to a variety of pesticides in agricultural and forestry ecosystems, with sublethal exposures frequently extending over multiple generations. These exposures, which can affect offspring through ingestion, contact, or residual deposits, are primarily determined by the dose received (Croft and Brown, 1975; Stark and Banks, 2003).

Factors for hormesis induction in insects:

The induction of hormesis in insects is dependent on exposure to low doses of stressors that are typically harmful at elevated concentrations. Although sublethal doses of insecticides and pesticides are the most extensively studied inducers, a variety of physical, chemical, and environmental factors have also been reported to induce hormetic responses in insects.

Temperature stress:

Insects, as ectothermic organisms, are highly susceptible to abiotic environmental changes (Neven, 2000). Temperature significantly influences physiological processes such as respiration, immunity, metabolism, growth, and reproduction (Neven, 2000). Besides, temperature affects the toxicity and efficacy of insecticides in agricultural settings by modulating the physiological mechanisms involved in detoxification and excretion of chemical compounds (Dong, 2022; Noyes *et al*., 2009). For example, the codling moth, *Cydia pomonella*, exhibited a clear hormetic response to mild thermal stress, with improved survival at 43°C following a pretreatment at 37°C (Chidawanyika and Terblanche, 2011). Additionally, Silva *et al*., (2024) confirmed the interaction between temperature and insecticide-induced hormesis by demonstrating significant increases in longevity and fecundity in *Myzus persicae* after sublethal exposure to chlorpyrifos and deltamethrin at lower temperatures compared to unexposed controls.

Low-Dose Ionising Radiation:

Vimal *et al*., (2022) demonstrated that ionizing radiation doses ranging from 0.75 to 1.0 Gy can be used as hormetic treatments for eggs, larvae, and pupae of *Spodoptera litura* Fabricius. Various developmental stages were exposed to low doses (0.25-1.25 Gy) to evaluate the stimulatory effects of gamma irradiation on growth and viability. Application of doses between 0.75 and 1.0 Gy to eggs significantly increased pupal weight,

Table 2: Hormetic responses across insecticide groups and their underlying mechanisms (Roy and Mukhopadhyay, 2025).

Insecticide group	Shows hormesis	Mechanism influencing hormesis
Neonicotinoids	Yes	Targets nAChRs; triggers stress response; enhances survival, reproduction, feeding and movement;upregulates detoxification pathways (Cytochrome P450)
Pyrethroids	Yes	Prolonged opening of sodium channels; increased nerve firing; enhanced stress response; altered insect behaviour
Organophosphates	Yes	Accumulation of acetylcholinesterase (AChE); overstimulation; enhanced stress response; upregulationof detoxifying pathways
Insect Growth Regulators (IGRs)	Yes	Enhancement of moulting and metamorphosis showing hormetic effects on developmental parameters
Spinosyns	Yes	Activation of nAChRs and GABA receptors; increased neuronal activity; increased parasitism efficiency in parasitoids
Diamides	Yes	Target ryanodine receptors; increased muscle contractionand locomotion; increased exposure to insecticides
Benzoylureas	Yes	Stimulation of chitin synthesis; increased growth and development
Anthranilic Diamides	Yes	Selective ryanodine receptor activation
Phenylpyrazoles	No	Mechanism does not induce notable hormetic effects;causes direct and linear biological damage
Tetramic Acid Derivatives	No	Linear dose–response without stimulatory phase at low doses
Carbamates	No	No notable hormetic effects documented at sublethalconcentrations

adult emergence, and growth index, while also shortening the developmental period. Enhanced adult longevity from eggs treated at these doses, along with improved pupation and adult emergence in larvae and pupae treated at 1.0 Gy, provided clear evidence of hormesis. The use of low-dose irradiation (0.75-1 Gy) on preimaginal stages improved moth rearing quality, confirming the hormetic effect induced by low-dose irradiation (Vimal *et al*., 2022).

Heavy Metal Exposure

The first evidence of a hormetic-like biphasic response induced by a heavy metal on insects was provided by Nascarella *et al*., in 2003 on the early developmental success in the larvae of Queen blowfly, *Phormia regina* Meigen. Their study demonstrated increased pupation success at low cadmium concentrations, with a decline observed at higher concentrations. Larvae exposed to low levels of cadmium exhibited mean pupation rates exceeding those of the control group, thereby establishing the initial documentation of a heavy metal-induced biphasic dose–response in an insect model.

Chemical stress (Insecticides and Pesticides)

Sublethal doses of insecticides from diverse chemical classes, including neonicotinoids, pyrethroids, organophosphates, spinosyns, diamides, and insect growth regulators, have been reported to induce hormetic responses in insect pests. The specific hormetic effect depends on the particular chemical or stressor, as not all insecticides or heavy metals elicit hormesis at low doses, and responses may vary even among structurally similar compounds. Additionally, the developmental stage of the insect, timing and duration of exposure, and the genetic and physiological background of the population, including detoxification mechanisms and resistance significantly influence the hormetic response (Cutler, 2013; Sial *et al*., 2018; Tang *et al*., 2019).

Mechanisms of hormesis

Two principal hypotheses currently prevail as mechanistic explanations for hormesis: the growth hormesis theory of Stebbing (2000) and the principle of physiological resource allocation as applied to hormesis (Guedes and Cutler, 2014).

Growth Hormesis Theory (Overcompensation Hypothesis):

Stebbing's (2000) growth theory proposes that hormesis arises from an over compensatory response in homeostasis. Upon exposure to a low dose of a harmful agent, an organism's internal regulatory systems aim to restore homeostatic balance and may overcorrect. This overcompensation can lead to a temporary or sustained enhancement in growth or physiological function at low doses, rather than causing harm. Within this framework, hormesis is primarily viewed as a life-history trait associated with growth-related processes. The broader molecular mechanisms underlying this over compensatory response remain a key area of ongoing investigation.

Principle of resource allocation:

The principle of resource allocation posits that

organisms possess finite energetic resources that must be allocated among a variety of biological functions (Guedes and Cutler, 2014). Jager *et al*., (2013) identified three primary strategies by which organisms manage energy and materials in response to toxic exposure: acquisition, whereby they increase their resource intake from the environment; medication, whereby they deploy resources to mitigate toxicant effects; and allocation, whereby they redistribute resources among competing physiological demands.

Hormesis through acquisition refers to the enhanced performance of an organism under stress, resulting from an increased rate of energy intake that promotes growth and potentially maximizes size, thereby improving reproduction and population growth. Hormesis by medication occurs when the toxicant either contains or is an essential element that the organism is deficient in. The most traditional and widely accepted form, hormesis by resource allocation, involves the redistribution of resources that shifts the balance between competing physiological trade-offs, favoring one function (e.g., reproduction) over another (e.g., longevity) (Jager *et al*., 2013).

Receptor-level mechanistic assessment

Szabadi (1977) posited that a single agonist can induce a biphasic response at the receptor level, characteristic of hormesis, by interacting with two distinct receptor subtypes that mediate opposing effects. At low concentrations, the agonist preferentially binds to a limited number of high-affinity receptors that are readily activated, initiating a stimulatory pathway. At higher concentrations, the agonist also engages more abundant but less readily activated low-affinity receptors, triggering an inhibitory pathway. This dual receptor interaction produces a biphasic dose-response curve, characterized by stimulation at low doses and inhibition at high doses.

Two mechanistic models have been proposed to explain receptor-based hormesis. In the first model, a receptor antagonist blocks both the low-dose stimulation and high-dose inhibition induced by an agonist, indicating that both effects are mediated by the same receptor. The antagonist's blockade of the receptor abolishes both phases, supporting the concept of hormetic modulation through a single receptor pathway (Calabrese, 2013). In the second model, the antagonist selectively blocks the low-dose stimulation but not the high-dose inhibition, suggesting that the latter is mediated by a distinct receptor or molecular pathway. Thus, low-dose stimulation and high-dose inhibition represent separate mechanisms involving different receptors (Calabrese, 2013).

Hormetic responses across insecticide groups

Hormetic responses in insects are demonstrated through a variety of physiological and life-history traits, including increased longevity, accelerated growth, enhanced reproductive capacity, improved stress resistance, and augmented detoxification activity (Cutler, 2013). The subsequent section summarizes the hormetic effects that have been observed in the major insecticide classes and emphasizes the most significant empirical findings.

Insecticides that do not exhibit hormesis adhere to a linear dose–response model, whereby the effect increases proportionally with the dose. These insecticides generally operate through mechanisms that cause direct and proportional biological damage, lacking the over compensatory adaptive responses characteristic of hormesis (Roy and Mukhopadhyay, 2025).

Molecular and biochemical responses to hormesis in insects

A number of molecular mechanisms seem to be involved in coordinating hormetic responses, such as the ERK1/2, p38, and JNK pathways involved in processes such as cell proliferation and development, and directing cellular responses to oxidative, heat, and radiation stress, and inflammatory responses and apoptosis (Calabrese, 2013b; Cargnello, 2011). A general mechanism has also been proposed to account for hormetic responses based upon the activation of the Nrf2 transcription factor, which regulates antioxidant and anti-inflammatory responses (Calabrese and Kozumbo, 2021a).

The ubiquitous nature of hormesis, occurring irrespective of organism or stressor, suggests it should be moderated by primordial, highly conserved stress response pathways. The heat shock response, oxidative stress response, and detoxification response have all been implicated in the modulation of hormetic responses (Berry and Lopez-Martinez, 2020). Temperature, osmotic, nutritional, chemical, and oxidative stress, frequently experienced by insects, all induce the transcription of heat shock proteins (HSP), which act as molecular chaperones, repairing damaged and improperly folded proteins (King and MacRae, 2015; Rattan *et al.*, 2004). Stress also causes the buildup of reactive oxygen species (ROS), resulting in production of antioxidant enzymes, such as superoxide dismutase (SOD), catalase (CAT), and peroxidase (POD). These enzymes work in-tandem to neutralize ROS, which cause damage to DNA, proteins, and lipids (Garcia-Caparros *et al*., 2020).

Increased HSP and antioxidant gene expression or activity can protect insects against cellular damage under

stressful conditions (King and MacRae, 2015; Krishnan and Kodrik, 2006; Micheal and Subramanyam, 2013), including low dose or hormetic stress (Calabrese, 2013c; Hercus *et al.*, 2003) and may be associated with enhanced longevity, reproduction, and stress tolerance (Arking *et al.*, 2000). Genes involved in chemical detoxification are thought to play a role during insecticide-induced hormesis (Cutler, 2013).

Low doses of insecticides and other chemicals can induce expression of genes encoding detoxification enzymes, such as cytochrome P450s (CYP), glutathione-s-transferases (GST), and esterases (EST) (Sial *et al.*, 2018). Sublethal exposure to deltamethrin and lambda-cyhalothrin (pyrethroids), and acetamiprid and imidacloprid (neonicotinoids) insecticides, on the green peach aphid, *Myzus persicae* Sulzer significantly increased reproductive output across multiple generations. Female longevity, daily fecundity, and total fecundity were markedly elevated in the F□ and F,, generations exposed to LC_{20} doses compared to unexposed controls. Prolonged exposure also extended developmental duration, likely representing a compensatory mechanism to enhance survival and reproductive success under chemical stress. Notably, hormetic exposure resulted in significant upregulation of the detoxification genes CYP6CY3 and E4/FE4, establishing a direct association between hormoligosis and the molecular pathways involved in resistance development. This study provides the first evidence that multigenerational low-dose insecticide exposure can concurrently stimulate population growth and increase metabolic resistance gene expression in *Myzus persicae*, underscoring the dual role of hormesis in enhancing both demographic fitness and resistance evolution (Sial *et al.*, 2018).

Impact of hormesis on integrated pest management

Integrated pest management (IPM) depends on the careful integration of chemical, biological, and cultural control methods to sustainably regulate pest populations. However, pesticide-induced hormesis presents challenges to these approaches, notably by contributing to pest resurgence, resistance development, and secondary pest outbreaks, which are among the most critical impacts (Morse, 1998; Cohen, 2006; Guedes and Cutler, 2014; Roy and Mukhopadhyay, 2025).

Campos *et al.*, (2019) examined hormetic responses to the Bt toxin Cry1Fa in Spodoptera frugiperda using one long-term susceptible strain and one strain selected for resistance. Resistance was developed by repeated bioassays with purified Cry1Fa in artificial diet and with leaves from Cry1Fa-expressing maize (hybrid 30F35H) and its non-Bt isoline. Resistant larvae tolerated Cry1Fa concentrations roughly 100,000-fold higher than susceptible larvae, showing no detectable costs to weight or reproduction. At very low Cry1Fa doses, both susceptible and resistant insects exhibited a small but significant increase in reproductive output, consistent with hormesis. Resistant insects performed equally well on Bt and non-Bt maize, indicating that field-level Cry1Fa exposures were inadequate to control them. These results highlight a major concern for integrated pest management: sublethal Bt exposures can enhance fitness across populations, potentially speeding resistance evolution and promoting pest outbreaks rather than suppressing them.

Sublethal pesticide exposure can paradoxically enhance pest fitness by stimulating reproductive output, hastening developmental rates, and promoting population expansion outcomes that contradict the intended suppressive effect of chemical intervention. This phenomenon, commonly associated with pesticide-induced resurgence, can precipitate successive treatment cycles that compound environmental pesticide loads, a pattern often characterized as the "pesticide treadmill." In addition to that, hormetic stimulation of detoxification pathways within pest populations may accelerate the selection of resistant genotypes, progressively compromising the efficacy of targeted insecticides. Beyond the primary pest species, analogous hormetic responses in non-target arthropods may drive secondary population irruptions, introduce additional vectors of pest management failure and further undermining the sustainability of chemical control strategies.

Conversely, a comprehensive understanding of hormetic mechanisms presents strategic opportunities for integration within IPM frameworks. Precise dose determination, calibrated to achieve effective pest suppression while precluding the inadvertent induction of hormetic stimulation is fundamental to the development of sustainable chemical management protocols. Accordingly, IPM programmes should incorporate systematic monitoring of pest population responses following pesticide applications to enable the timely identification and remediation of hormesis-mediated effects. The judicious integration of biological control agents and cultural management practices alongside carefully deployed chemical inputs is advocated as a means of minimising the risk of hormesis-driven pest resurgence.

Cosme (2008) examined the stimulatory sublethal effects of permethrin on the generalist predator *Podisus distinctus* Stål, utilizing fertility table analyses to assess

changes in demographic fitness. Technical grade permethrin was topically applied to third-instar nymphs across a wide dose range (0.02, 0.17, 1.72, 17.20, and 172.00 ppb). At low doses (0.02-0.20 ppb), permethrin induced a hormetic response in *Podius distinctus*, characterized by increased mean survival time, enhanced reproductive capacity, and overall population growth compared to untreated controls. Higher doses (>1.72 ppb) did not elicit these effects, confirming the biphasic nature of the response (Cosme, 2008). This study highlights the potential benefits of hormetic responses in beneficial insects, particularly natural enemies used in biological control, suggesting that sublethal exposures at carefully calibrated doses may improve predator fitness, longevity, and reproductive output, thereby reinforcing their role in integrated pest management programs.

A study conducted at Kerala Agricultural University examined the cross-generational effects of insecticides on the fitness traits of two natural enemies, the larval parasitoid *Bracon hebetor* Say and the lacewing predator *Chrysoperla zastrowi sillemi* (Esben-Peterson) and demonstrated that sublethal doses of certain insecticides can induce biphasic dose-response effects (Harshiya, 2025). Chlorantraniliprole 18.5% SC and flubendiamide 39.35% SC significantly enhanced fecundity, egg laying, and net reproductive rate at LC_{50} levels in both species. Fenvalerate 20% EC increased fecundity, egg emergence, intrinsic rate of increase, and gross reproductive rate at LC_{50} , while lambda-cyhalothrin 5% EC improved fecundity, egg emergence, and gross reproductive rate at sublethal concentrations. Collectively, these results demonstrate hormetic effects, including enhanced reproductive capacity, accelerated growth potential, and increased population growth rates in beneficial insects exposed to sublethal insecticide doses, highlighting the ecological importance of hormesis in pest management (Harshiya, 2025). As evidenced by the findings of Cosme (2008) on Podisus distinctus and the investigations conducted under the KAU studies (Harshiya, 2025), hormetic responses may be deliberately harnessed to augment the functional performance of natural enemies within agroecosystems, provided that pesticide exposure levels are rigorously optimised and maintained within biostimulatory thresholds.

Conclusion

Biphasic dose-response phenomena encompassing hormesis, hormoligosis, and pesticide-mediated homeostatic modulation constitute a toxicologically significant and phylogenetically widespread class of biological responses among insect taxa. By fundamentally contesting the conventional assumption that toxicant exposure invariably produces deleterious outcomes, these phenomena necessitate a comprehensive re-examination of prevailing frameworks in pesticide risk assessment and integrated pest management strategy. Empirical evidence collated across diverse insect species and insecticide classes consistently demonstrates that sublethal exposure regimens are capable of activating stress-response cascades, potentiating detoxification enzyme activity, upregulating resistance-associated gene expression, and redirecting metabolic resource allocation toward enhanced reproductive output. When left unaddressed, the cumulative manifestation of these physiological adjustments carries significant applied consequences, including the escalation of pest resurgence events, the emergence of secondary arthropod outbreaks, and the expedited development of insecticide resistance within target populations.

At the mechanistic level, hormetic responses may be mediated through homeostatic over compensation, the physiological reallocation of metabolic resources, or differential modulation at the receptor level. The expression and magnitude of these mechanisms are subject to the influence of multiple interacting variables, including the physicochemical nature of the stressor, the temporal parameters of exposure, the ontogenetic stage of the insect at the time of contact, and the prevailing genetic constitution of the exposed population. From an applied perspective, the effective management of hormesis necessitates rigorous dose calibration, empirical field-based validation of sublethal exposure thresholds, sustained population monitoring, and the systematic incorporation of non-chemical control interventions within comprehensive IPM frameworks. Consequently, a precise and mechanistically informed understanding of dose-response relationships transcends purely theoretical inquiry, emerging instead as an operational imperative for the rational design of pest management systems that are simultaneously sustainable, functionally resilient, and ecologically responsible.

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