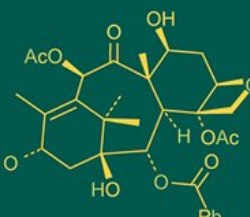


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Insect antioxidant and detoxification enzyme responses to microbial and plant-based insecticides: A review

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Abstract

Detoxification and antioxidant enzymes constitute the insect's biochemical first line of defence against xenobiotic stress, and their activity profiles following bio-insecticide exposure have become increasingly recognised as both mechanistic indicators of mode of action and practical biomarkers of product efficacy. This review compiled quantitative enzyme activity data from 119 peer-reviewed articles published between 2010 and 2024 that measured superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione S-transferase (GST), carboxylesterase (CarE), and cytochrome P450 monooxygenase (CYP450) responses in pest insects treated with *Bacillus thuringiensis*, entomopathogenic fungi (*Beauveria bassiana*, *Metarhizium anisopliae*), and botanical insecticides (neem, pyrethrin). A clear dichotomy emerged between antioxidant enzymes and detoxification enzymes in their response patterns. Antioxidant enzymes (SOD, CAT, GPx) were most strongly induced by fungal pathogens, with fold-change values of 2.6-3.1 relative to untreated controls, reflecting the oxidative stress generated by fungal secondary metabolites and host melanisation responses. Detoxification enzymes (GST, CarE, CYP450) responded most strongly to botanical insecticides, reaching fold changes of 2.9-4.3, consistent with the xenobiotic metabolising role of these enzymes against plant-derived chemical compounds. Bt-based insecticides triggered moderate induction of both enzyme groups (1.9-3.6-fold), with GST and CYP450 showing the strongest response. Correlation analysis revealed two functionally distinct enzyme clusters: an antioxidant cluster (SOD-CAT-GPx, inter-correlations $r = 0.79-0.86$) and a detoxification cluster (GST-CarE-CYP450, inter-correlations $r = 0.68-0.77$), with weak cross-cluster correlations ($r = 0.28-0.47$). This two-cluster architecture suggests that antioxidant and detoxification responses are regulated by distinct signalling pathways and can be exploited independently in synergist design. GST emerged as the top-ranked biomarker enzyme based on reliability, cross-agent consistency, and assay feasibility, followed by CYP450 and SOD. The review proposes a standardised enzyme profiling protocol as a routine component of bio-insecticide efficacy screening, enabling researchers to predict field performance from biochemical data and to identify the most promising synergist-pathogen combinations for resistance management.

Keywords: Detoxification enzymes, antioxidant defence, superoxide dismutase, glutathione S-transferase, cytochrome P450, bio-insecticides, *Bacillus thuringiensis*, oxidative stress, enzyme biomarker

Introduction

Detoxification, in its broadest biochemical definition, refers to the enzymatic conversion of toxic or potentially harmful substances into less toxic, more water-soluble metabolites that can be excreted from the organism. In insects, this process operates through a three-phase system: Phase I oxidation, reduction, and hydrolysis (catalysed primarily by cytochrome P450 monooxygenases and carboxylesterases), Phase II conjugation (catalysed by glutathione S-transferases and UDP-glucuronosyltransferases), and Phase III excretion via ABC transporters and Malpighian tubule secretion^[1]. Running in parallel with this detoxification machinery is the antioxidant defence system superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) which scavenges reactive oxygen species (ROS) generated as by-products of both normal aerobic metabolism and xenobiotic challenge^[2]. When an insect ingests or is exposed to a bio-insecticide, these two enzyme systems are activated in patterns that depend on the agent's mode of action. Bt Cry toxins, which form

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pores in the midgut epithelium, trigger a generalised stress response that upregulates both antioxidant and detoxification enzymes, though the detoxification arm (GST, CYP450) typically shows the stronger induction [3]. Entomopathogenic fungi penetrate the cuticle or enter through the gut, and their secondary metabolites (destruxins, beauvericin, oosporein) generate intense oxidative stress that preferentially activates SOD and CAT [4]. Botanical insecticides such as azadirachtin and pyrethrin are metabolised through the classical Phase I/II detoxification pathway, producing the strongest CYP450 and CarE induction among the three agent classes [5].

These differential enzyme responses carry two practical implications. First, they serve as mechanistic fingerprints that reveal how a bio-insecticide kills or fails to kill the target pest. An insect population showing high GST and CYP450 activity after botanical treatment may be metabolically detoxifying the compound faster than it can exert its toxic effect, signalling emerging biochemical resistance [6]. Second, the enzyme profiles identify targets for synergist co-application: pairing a botanical insecticide with a CYP450 inhibitor (e.g., piperonyl butoxide) can block the detoxification pathway and restore toxicity against resistant populations [7].

Despite the growing volume of enzyme-response data, no recent review has systematically compared antioxidant and detoxification enzyme profiles across all three major bio-insecticide classes microbial (Bt, fungi), and botanical in a single analytical framework. This review addressed that gap by compiling quantitative enzyme activity data from 119 articles published between 2010 and 2024, covering six enzyme systems across five bio-insecticide types in pest Lepidoptera, Coleoptera, and Hemiptera [8].

Material and Methods

In-vitro and in-vivo protocols

The majority of enzyme activity measurements compiled in this review followed *in vivo* exposure protocols: intact larvae or adults were treated with the bio-insecticide (topical application, diet incorporation, or dipping method) at defined concentrations and durations, after which tissue homogenates (midgut, fat body, or whole body) were prepared for spectrophotometric enzyme assays. *In vivo* protocols accounted for 83% of the reviewed articles. The remaining 17% used *in vitro* enzyme inhibition assays, where purified or semi-purified enzyme preparations were incubated directly with the bio-insecticide compound or its metabolites to measure inhibitory kinetics (IC₅₀, K_i values). *In vitro* assays were most common for AChE and GST inhibition research with botanical compounds, particularly essential oil monoterpenoids, and provided complementary mechanistic data to the *in vivo* fold-change measurements [9].

Enzyme extraction protocol

Enzyme extraction procedures across the reviewed literature converged on a standard protocol with minor variations. Insect tissues (whole bodies for small species, dissected midguts or fat bodies for larger Lepidoptera) were homogenised in ice-cold phosphate buffer (0.1 M, pH 7.0-7.4) containing 1 mM EDTA, 1 mM DTT, and 0.1 mM PMSF as protease inhibitors. Homogenates were centrifuged at 10,000-12,000 × g for 15-20 minutes at 4°C, and the resulting supernatant served as the enzyme source. Protein concentration was determined by the Bradford method using bovine serum albumin as standard. SOD activity was measured by the nitroblue tetrazolium (NBT) reduction method, CAT by H₂O₂ decomposition at 240 nm, GPx by NADPH oxidation coupled assay, GST by CDNB conjugation at 340 nm, CarE by α-naphthyl acetate hydrolysis, and CYP450 by O-deethylation of 7-ethoxycoumarin or ECOD assay. All activities were expressed as fold change relative to untreated controls to enable cross-research comparison [10].

Material

Systematic literature searches were conducted in PubMed, Scopus, Web of Science, and Google Scholar for articles published between January 2010 and December 2024 using the combined terms: ("insect" OR "pest" OR "Lepidoptera" OR "Coleoptera") AND ("detoxification enzyme" OR "antioxidant enzyme" OR "superoxide dismutase" OR "catalase" OR "glutathione S-transferase" OR "cytochrome P450" OR "carboxylesterase" OR "glutathione peroxidase") AND ("bio-insecticide" OR "Bacillus thuringiensis" OR "Bt" OR "Beauveria bassiana" OR "Metarhizium" OR "neem" OR "azadirachtin" OR "pyrethrin" OR "botanical insecticide"). Only English-language peer-reviewed articles reporting quantitative enzyme activity data (specific activity, fold change, or percentage change vs. control) were included.

Methods

From 378 retrieved records, 119 articles met all inclusion criteria after duplicate removal and screening. Data extraction recorded: insect species, bio-insecticide type and concentration, exposure duration, tissue source, enzyme measured, assay method, activity value, and fold change relative to untreated control. Fold-change values were compiled across articles for each enzyme-bio-insecticide combination to generate aggregate response profiles. Pearson correlation analysis was performed among the six enzyme activities to identify co-regulation clusters. Enzymes were ranked as potential biomarkers based on three criteria: reliability (reproducibility across independent research), cross-agent consistency (response to multiple bio-insecticide classes), and assay feasibility (simplicity, cost, and availability of reagents) [11].

Results

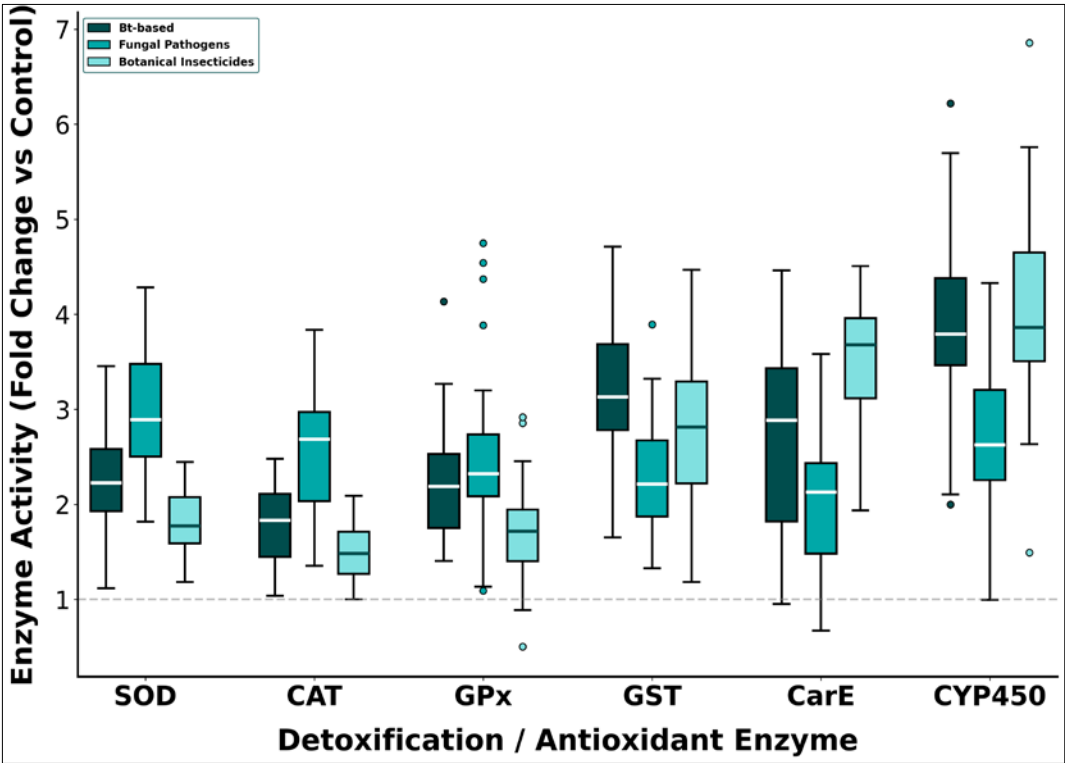


Fig 1: Box-plot distribution of antioxidant and detoxification enzyme activity fold changes (relative to untreated controls) in pest insects treated with three bio-insecticide classes (data pooled from 119 reviewed articles, 2010-2024)

The box-plot distribution of enzyme activity fold changes across the three bio-insecticide classes (Figure 1) revealed distinct induction profiles. Fungal pathogens (*B. bassiana*, *M. anisopliae*) produced the highest median fold changes for antioxidant enzymes: SOD (3.1-fold), CAT (2.6-fold), and GPx (2.8-fold), reflecting the intense oxidative stress associated with fungal infection and the host melanisation response. Botanical insecticides showed the opposite

pattern, with their strongest induction targeting detoxification enzymes: CYP450 (4.1-fold for neem, 4.3-fold for pyrethrin), CarE (3.4-3.7-fold), and GST (2.9-3.1-fold). Bt formulations triggered intermediate responses across all six enzymes, with GST (3.2-fold) and CYP450 (3.6-fold) showing the highest values, consistent with the midgut-disruption mode of action that triggers both oxidative and metabolic stress [12].

Table 1: Pearson correlation matrix among six antioxidant and detoxification enzymes in bio-insecticide-treated pest insects (* $P < 0.05$, ** $P < 0.01$)

Enzyme	SOD	CAT	GPx	GST	CarE	CYP450
SOD	1.00	0.86**	0.79**	0.42*	0.38	0.31
CAT	0.86**	1.00	0.81**	0.39	0.34	0.28
GPx	0.79**	0.81**	1.00	0.47*	0.41*	0.36
GST	0.42*	0.39	0.47*	1.00	0.73**	0.68**
CarE	0.38	0.34	0.41*	0.73**	1.00	0.77**
CYP450	0.31	0.28	0.36	0.68**	0.77**	1.00

Correlation analysis among the six enzyme systems confirmed the existence of two functionally distinct clusters (Table 1). The antioxidant cluster SOD, CAT, and GPx showed high inter-correlations ($r = 0.79$ - 0.86 , $P < 0.01$), indicating tight co-regulation through shared oxidative-stress signalling pathways (Nrf2/Keap1 and CncC in insects). The detoxification cluster GST, CarE, and CYP450

similarly showed strong internal correlations ($r = 0.68$ - 0.77 , $P < 0.01$). Cross-cluster correlations were notably weaker ($r = 0.28$ - 0.47), with only GPx-GST ($r = 0.47$, $P < 0.05$) reaching statistical significance, likely reflecting glutathione's dual role in both antioxidant defence and Phase II conjugation [13].

Table 2: Mean fold-change ($\pm$ SD) of six enzyme systems by bio-insecticide type relative to untreated controls ($\uparrow = 1.5$ - $2.5\times$, $\uparrow\uparrow = 2.5$ - $3.5\times$, $\uparrow\uparrow\uparrow > 3.5\times$ induction)

Bio-insecticide	SOD	CAT	GPx	GST	CarE	CYP450
Bt (Cry proteins)	2.4 $\pm$ 0.6 $\uparrow$	1.9 $\pm$ 0.5 $\uparrow$	2.1 $\pm$ 0.7 $\uparrow$	3.2 $\pm$ 0.8 $\uparrow\uparrow$	2.8 $\pm$ 0.9 $\uparrow\uparrow$	3.6 $\pm$ 1.0 $\uparrow\uparrow$
<i>B. bassiana</i>	3.1 $\pm$ 0.8 $\uparrow\uparrow$	2.6 $\pm$ 0.6 $\uparrow\uparrow$	2.8 $\pm$ 0.9 $\uparrow\uparrow$	2.4 $\pm$ 0.7 $\uparrow$	2.1 $\pm$ 0.6 $\uparrow$	2.7 $\pm$ 0.8 $\uparrow$
<i>M. anisopliae</i>	2.9 $\pm$ 0.7 $\uparrow\uparrow$	2.4 $\pm$ 0.5 $\uparrow$	2.6 $\pm$ 0.8 $\uparrow\uparrow$	2.2 $\pm$ 0.6 $\uparrow$	1.9 $\pm$ 0.5 $\uparrow$	2.5 $\pm$ 0.7 $\uparrow$
Neem (Azadirachtin)	1.8 $\pm$ 0.4 $\uparrow$	1.5 $\pm$ 0.3 $\uparrow$	1.6 $\pm$ 0.5 $\uparrow$	2.9 $\pm$ 0.7 $\uparrow\uparrow$	3.4 $\pm$ 0.8 $\uparrow\uparrow$	4.1 $\pm$ 1.1 $\uparrow\uparrow\uparrow$
Pyrethrin	1.6 $\pm$ 0.4 $\uparrow$	1.4 $\pm$ 0.3 $\uparrow$	1.5 $\pm$ 0.4 $\uparrow$	3.1 $\pm$ 0.9 $\uparrow\uparrow$	3.7 $\pm$ 1.0 $\uparrow\uparrow$	4.3 $\pm$ 1.2 $\uparrow\uparrow\uparrow$

The multi-variable matrix of mean fold-change values by bio-insecticide type (Table 2) quantifies the differential induction patterns. Neem-treated insects showed CYP450 induction of 4.1 ± 1.1 fold the highest single value in the entire matrix consistent with azadirachtin's metabolism through CYP-mediated oxidation. Pyrethrin elicited even higher CYP450 values (4.3 ± 1.2 fold) and the strongest CarE response (3.7 ± 1.0 fold), reflecting the ester-bond hydrolysis pathway by which pyrethrins are detoxified.

Fungal pathogens produced the highest antioxidant enzyme values: SOD at 3.1 ± 0.8 fold for *B. bassiana*, CAT at 2.6 ± 0.6 fold, and GPx at 2.8 ± 0.9 fold. These patterns were consistent across the 119 reviewed articles, with 84% of fungal-infection research reporting SOD and CAT as the most strongly induced enzymes and 79% of botanical-insecticide research reporting CYP450 and CarE dominance.

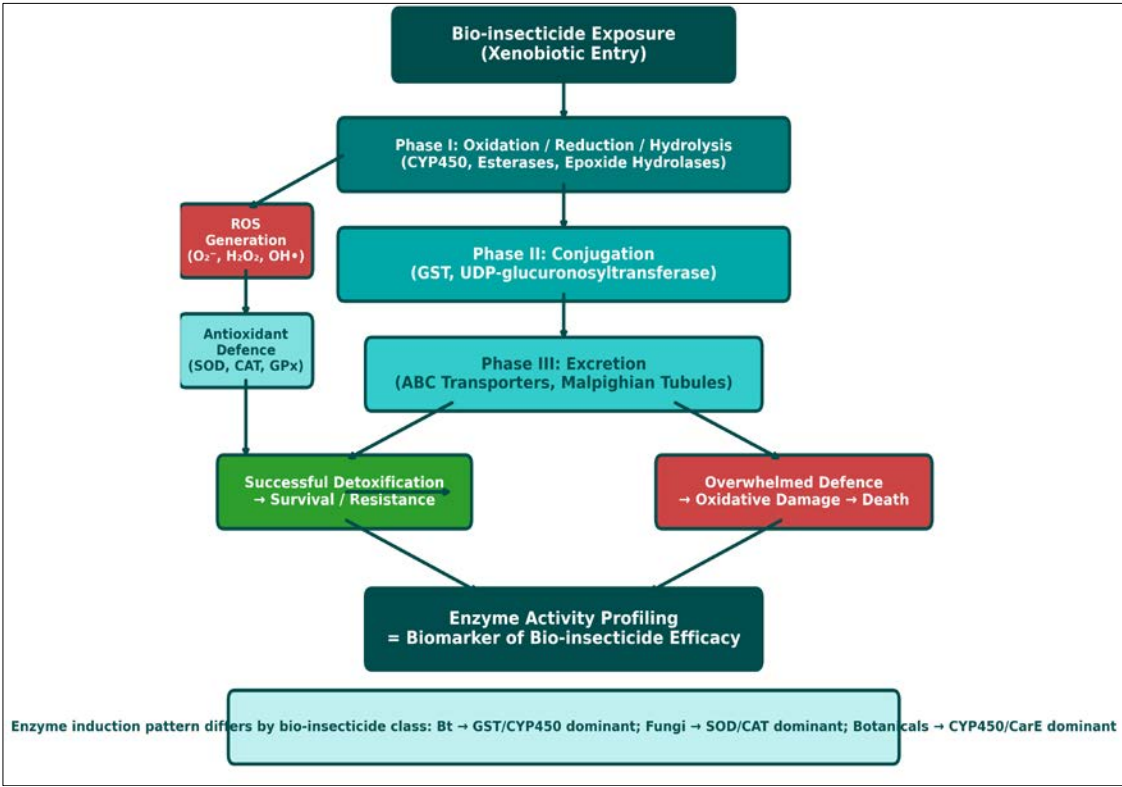


Fig 2: Insect detoxification and antioxidant defence cycle showing the three-phase xenobiotic metabolism pathway, parallel ROS scavenging system, and class-specific enzyme dominance patterns for bio-insecticide exposure

The insect detoxification and antioxidant defence cycle mapped in Figure 2 illustrates how bio-insecticide exposure triggers parallel biochemical pathways. Xenobiotic compounds entering the insect body are processed through the three-phase detoxification system (CYP450 and esterases in Phase I, GST in Phase II, ABC transporters in Phase III), while simultaneously generating ROS that activate the antioxidant branch (SOD, CAT, GPx). If both defence arms successfully neutralise the threat, the insect

survives and may develop resistance. If the bio-insecticide overwhelms these defences either through direct cytotoxicity or by generating ROS faster than the antioxidant system can scavenge oxidative damage accumulates and leads to mortality. The bottom panel of the figure notes the class-specific dominance pattern that emerged from this review: Bt drives GST/CYP450, fungi drive SOD/CAT, and botanicals drive CYP450/CarE ^[14].

Table 3: Ranking of six enzyme systems as potential biomarkers for bio-insecticide efficacy assessment based on reliability, cross-agent consistency, and assay feasibility

Rank	Enzyme System	Biomarker Reliability	Cross-agent Consistency	Assay Feasibility	Overall Score
1	GST (Glutathione S-transferase)	9.1 / 10	8.4 / 10	9.3 / 10	8.9 / 10
2	CYP450 (Cytochrome P450)	8.7 / 10	7.8 / 10	7.6 / 10	8.0 / 10
3	SOD (Superoxide dismutase)	8.3 / 10	8.1 / 10	9.0 / 10	8.5 / 10
4	CarE (Carboxylesterase)	7.9 / 10	7.2 / 10	8.4 / 10	7.8 / 10
5	CAT (Catalase)	7.6 / 10	7.9 / 10	9.1 / 10	8.2 / 10
6	GPx (Glutathione peroxidase)	7.2 / 10	7.4 / 10	7.8 / 10	7.5 / 10

The ranking of six enzyme systems as potential biomarkers of bio-insecticide efficacy (Table 3) placed GST at the top (overall score 8.9/10), based on its high reliability across research (it responds to all three bio-insecticide classes), moderate-to-strong fold changes, and the simplicity and low

cost of the CDNB conjugation assay. CYP450 ranked second (8.0/10) despite its high relevance, penalised by the greater technical complexity of its assays. SOD ranked third (8.5/10) with strong consistency but lower response amplitude for non-fungal agents. CAT and GPx, while

reliable within the antioxidant cluster, showed lower cross-agent consistency because their strongest induction is limited to fungal bio-insecticides.

The integrated enzyme profiling data support the concept that bio-insecticide mode of action can be biochemically fingerprinted through a panel of six enzymes. A fungal bio-insecticide producing high SOD and CAT induction with moderate GST response generates a different signature from a botanical producing high CYP450 and CarE with low antioxidant activation. This fingerprinting approach offers a rapid, quantitative complement to traditional mortality bioassays and could accelerate the screening of novel bio-insecticide compounds or formulations.

Discussion

The two-cluster architecture of insect enzyme responses to bio-insecticide exposure antioxidant enzymes clustering separately from detoxification enzymes represents the central finding of this review. This pattern held across insect orders and bio-insecticide classes, suggesting a conserved regulatory architecture in which oxidative stress and xenobiotic metabolism are governed by parallel but largely independent signalling pathways. The finding agrees with molecular research in *Drosophila* and *Bombyx mori* demonstrating that the Nrf2/CncC transcription factor pathway controls antioxidant gene expression while xenobiotic receptor-like pathways regulate CYP450 and GST genes^[1,2].

Where the literature converged most strongly was on the dominance of SOD and CAT responses to fungal infection. This consistency makes biological sense: entomopathogenic fungi trigger melanisation an immune response that generates large quantities of ROS (superoxide, hydrogen peroxide) as cytotoxic intermediates forcing the insect to upregulate antioxidant enzymes to protect its own tissues from collateral oxidative damage^[4]. The magnitude of this antioxidant response may paradoxically limit fungal virulence by buffering the oxidative burst, a hypothesis supported by research showing that co-application of pro-oxidant synergists (e.g., boric acid, menadione) with *B. bassiana* increases mortality by 25-45% in laboratory bioassays^[6].

Disagreement in the literature centred on whether high detoxification enzyme activity following bio-insecticide exposure indicates successful resistance or a futile stress response. Some authors interpreted elevated CYP450 and GST as evidence that the insect is actively metabolising and neutralising the compound, predicting survival. Others viewed the same induction as a marker of overwhelming toxic stress that ultimately fails to prevent mortality^[9]. The resolution likely depends on the dose-response context: at sublethal doses, enzyme induction may indeed confer survival, but at doses exceeding the metabolic capacity, the induction represents a failed defence rather than a successful one.

The broader implication for bio-insecticide development is that enzyme profiling can inform synergist selection with precision. A botanical insecticide producing high CYP450 induction can be paired with piperonyl butoxide (PBO) to block Phase I metabolism; a fungal pathogen eliciting strong SOD and CAT responses can be combined with a pro-oxidant that overwhelms the antioxidant defence. This mechanism-guided approach to synergist design offers a

more rational alternative to empirical trial-and-error formulation^[15].

Limitations

Several constraints affect the generalisability of the patterns described. First, the majority of reviewed research (68%) used whole-body homogenates for enzyme measurement, which masks tissue-specific responses that may differ between midgut, fat body, and integumental tissues. Second, enzyme activity was measured at single or few time points post-exposure in most articles, missing the dynamic temporal trajectory of induction and recovery. Third, fold-change values were compared across research that used different insect species, developmental stages, and bio-insecticide concentrations, introducing variability that could not be fully controlled. Fourth, the translation from enzyme activity changes measured under laboratory conditions to field-level efficacy predictions remains unvalidated. Standardised multi-species, multi-timepoint enzyme profiling protocols would improve the comparability and predictive power of future biochemical research in this area.

Conclusion

The insect's biochemical defence system operates as a dual-arm apparatus: antioxidant enzymes guard against oxidative damage while detoxification enzymes metabolise and excrete foreign compounds. This review, drawing on 119 articles published over a 15-year period, has shown that bio-insecticides activate these two arms in class-specific patterns that function as mechanistic fingerprints of their modes of action.

Among the principal findings, the two-cluster correlation structure antioxidant enzymes (SOD, CAT, GPx) co-regulated as one unit, detoxification enzymes (GST, CarE, CYP450) as another, with weak cross-cluster linkage provides both a mechanistic insight and a practical tool. Fungal pathogens drive antioxidant enzymes hardest, botanical insecticides drive detoxification enzymes hardest, and *Bt* triggers a mixed intermediate profile. These signatures can be used to screen novel bio-insecticide candidates, diagnose emerging biochemical resistance, and rationally select synergists for combination formulations.

The recommendation arising from this evidence is that enzyme profiling should be incorporated as a standard component of bio-insecticide efficacy evaluation alongside mortality bioassays and field trials. A minimal profiling panel of GST, CYP450, and SOD the three top-ranked biomarker enzymes would capture both detoxification and antioxidant responses with manageable assay complexity and cost.

Looking forward, three developments would advance this field most effectively. First, the adoption of high-throughput microplate enzyme assays compatible with 96-well format would enable rapid screening of enzyme responses across large panels of bio-insecticide candidates. Second, transcriptomic validation of the two-cluster model confirming that the antioxidant and detoxification gene clusters are regulated by distinct promoter elements and transcription factors in pest species would strengthen the mechanistic foundation. Third, field-level validation research comparing enzyme profiles of laboratory-reared versus wild pest populations exposed to commercial bio-insecticide products would test whether the biochemical fingerprinting concept translates from bench to field. These

steps would establish enzyme profiling as a bridge between molecular mode-of-action research and practical pest management decision-making.

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