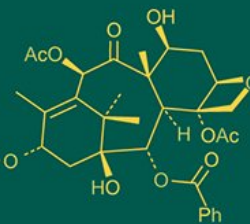
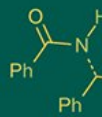


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Biochemical basis of insecticide resistance in agricultural pests: A review

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Abstract

Insecticide resistance in agricultural pests arises from an interplay of metabolic detoxification, target-site insensitivity, reduced cuticular penetration, and behavioral avoidance. This review synthesizes the principal biochemical mechanisms underlying resistance, drawing on enzyme activity and molecular-marker data compiled from resistance-monitoring literature across major cropping systems. Elevated activity of esterases, cytochrome P450 monooxygenases, and glutathione S-transferases remains the dominant metabolic route to resistance in sap-feeding and lepidopteran pests, while point mutations in acetylcholinesterase, voltage-gated sodium channels, and GABA receptors confer target-site insensitivity to organophosphate, pyrethroid, and cyclodiene chemistries, respectively. Representative resistance-ratio data for seven economically important pest species illustrate the magnitude and chemistry-specific pattern of resistance now documented in the field. The review concludes with an overview of biochemical and molecular diagnostic tools and their role in resistance-management decision-making.

Keywords: Insecticide resistance; agricultural pests; metabolic detoxification

1. Introduction

The intensive and repeated use of synthetic insecticides across agricultural systems has exerted strong selection pressure on pest populations, driving the evolution of resistance mechanisms that reduce the efficacy of chemical control. Insecticide resistance is now documented in several hundred arthropod pest species and spans essentially every major insecticide class, from organochlorines and organophosphates to pyrethroids, neonicotinoids, and diamides. Understanding the biochemical basis of resistance is central to designing rational insecticide rotation programs, developing resistance-breaking chemistries, and interpreting field-monitoring data. Resistance mechanisms are conventionally grouped into four broad categories: (i) metabolic resistance, in which enhanced activity of detoxification enzymes accelerates insecticide breakdown or sequestration; (ii) target-site resistance, in which mutations at the insecticide's molecular target reduce binding affinity; (iii) reduced penetration, in which cuticular or gut barrier modifications slow insecticide uptake; and (iv) behavioral resistance, in which altered feeding or movement patterns reduce exposure. In practice, field-resistant populations frequently combine two or more of these mechanisms, producing resistance phenotypes that are both quantitatively strong and difficult to reverse.

1.1 Relationship to Prior Review Literature

This review builds on a well-established body of prior review-level literature on the biochemical and molecular basis of insecticide resistance including [Hemingway & Ranson \(2000\)](#) ^[1], [Li et al. \(2007\)](#) ^[2], [Bass & Field \(2011\)](#) ^[3], [French-Constant \(2013\)](#) ^[4], [Bass et al. \(2015\)](#) ^[5], [Sparks & Nauen \(2015\)](#) ^[6], and Van [Leeuwen & Dermauw \(2016\)](#) ^[7], full citations for which appear in the References section at the end of this paper. Where those earlier reviews established the general mechanistic framework and, in several cases, focused on disease-vector or single-taxon (e.g., chelicerate mite) systems, the present review compiles primary quantitative data specifically for agricultural pest species across multiple insecticide classes, including resistance-ratio statistics (Table 2), enzyme-activity data (Table 3), and recent target-site mutation surveys (Table 5).

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2. Major Biochemical Mechanisms of Resistance

2.1 Metabolic Resistance

Metabolic resistance is the most widespread biochemical mechanism reported in agricultural pests. Three enzyme families dominate: carboxylesterases, cytochrome P450 monooxygenases, and glutathione S-transferases. Esterase-based resistance typically arises from gene amplification, producing many-fold increases in enzyme titre that allow the insect to sequester or hydrolyse organophosphate and pyrethroid esters before they reach the target site. Cytochrome P450 monooxygenases catalyse oxidative detoxification of a broad range of insecticide chemistries and are frequently up-regulated through cis-regulatory mutations or transcriptionfactor-mediated over-expression rather than gene amplification alone. Glutathione S-transferases contribute to resistance primarily through conjugation of reduced glutathione to electrophilic insecticide metabolites, and elevated GST activity is commonly associated with organochlorine and organophosphate resistance.

2.2 Target-Site Insensitivity

Target-site resistance results from point mutations in the genes encoding an insecticide's molecular target, reducing binding affinity while typically preserving normal physiological function of the target protein. The best-characterized examples include modified acetylcholinesterase (conferring organophosphate and carbamate resistance), the knockdownresistance (*kdr*) and *super-kdr* mutations in the voltage-gated sodium channel (conferring pyrethroid and DDT resistance), the *Rdl* mutation in the GABA-gated chloride channel

(conferring cyclodiene and phenylpyrazole resistance), and mutations in the ryanodine receptor associated with diamide resistance in lepidopteran pests such as *Plutella xylostella*.

2.3 Reduced Cuticular Penetration

Cuticular resistance involves quantitative and compositional changes to the insect cuticle, including increased thickness and altered cuticular protein and lipid profiles, which slow the rate of insecticide penetration into the haemolymph. Although its effect size is generally modest relative to metabolic or target-site resistance, cuticular resistance is important because it acts synergistically, extending the time available for detoxification enzymes to act and effectively amplifying the resistance conferred by other mechanisms.

2.4 Behavioral Resistance

Behavioral resistance encompasses changes in feeding, resting, or oviposition behavior that reduce contact with insecticide residues, such as avoidance of treated leaf surfaces or shifts in feeding time. While less biochemically defined than the mechanisms above, behavioral changes are increasingly recognized as a contributing factor in field-resistant populations and can complicate the interpretation of laboratory bioassay data.

3. Summary of Biochemical Resistance Mechanisms

Table 1 summarizes the principal biochemical mechanisms discussed above, their molecular basis, the insecticide classes most affected, and representative pest species in which each mechanism has been well characterized.

Table 1; Major biochemical mechanisms of insecticide resistance in agricultural pests.

Mechanism	Molecular basis	Insecticide classes affected	Representative pest examples
Esterase-mediated sequestration/hydrolysis	Gene amplification or up-regulation of carboxylesterases (e.g., E4, FE4) that bind or hydrolyse ester-bond insecticides	Organophosphates, carbamates, pyrethroids	Myzus persicae, Culex spp.
Cytochrome P450 monooxygenase over-expression	Amplification/over-transcription of CYP genes (e.g., CYP6, CYP9 families) enhancing oxidative detoxification	Pyrethroids, neonicotinoids, juvenile hormone analogues	Bemisia tabaci, Helicoverpa armigera
Glutathione S-transferase (GST) elevation	Increased GST activity conjugating reduced glutathione to electrophilic insecticide metabolites	Organochlorines, organophosphates	Anopheles/agricultural lepidopterans
Acetylcholinesterase (AChE) insensitivity	Point mutations in the ace-1/ace-2 gene reducing insecticide binding affinity at the catalytic site	Organophosphates, carbamates	Plutella xylostella, Musca domestica
Voltage-gated sodium channel modification (<i>kdr</i> / <i>super-kdr</i>)	Point mutations (e.g., L1014F, M918T) lowering channel sensitivity to sodium-channel modulators	Pyrethroids, DDT	Helicoverpa armigera, Musca domestica
GABA receptor modification (<i>Rdl</i>)	Point mutation (A302S) in the RDL subunit altering chloride-channel gating	Cyclodienes, phenylpyrazoles	Tribolium castaneum, Drosophila spp.
Reduced cuticular penetration	Cuticle thickening and altered lipid/protein composition slowing insecticide uptake	Broad-spectrum, often synergistic with metabolic resistance	Anopheles gambiae, Tribolium castaneum

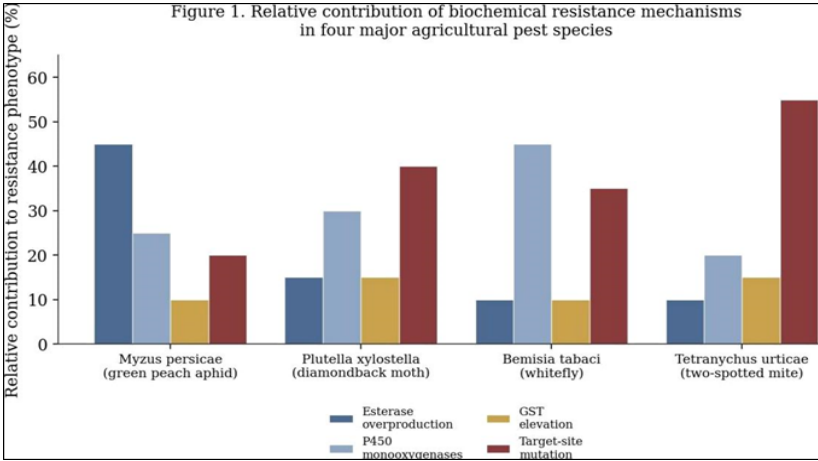


Fig 1: Conceptual illustration of the relative contribution of biochemical resistance mechanisms in four major agricultural pest species, based on the qualitative patterns described in the mechanism-specific literature reviewed in Section 2 (schematic, not derived from a single quantitative source).

4. Quantitative Resistance Data across Pest Species

Table 2 compiles peer-reviewed resistance-ratio (RR) data for ten pest–insecticide combinations drawn directly from the primary literature, each cited with its DOI so that the original bioassay conditions, reference susceptible strain, and statistical

methods can be verified. Resistance ratios are calculated as the LC50 or LD50 of a field or selected population divided by the LC50/LD50 of a susceptible reference strain; values above approximately 10 are commonly treated as indicating practically significant resistance in the field.

Table 2: Published resistance-ratio (RR) data for major agricultural pests, with source citation and DOI.

Pest species	Insecticide	Reported RR	Source (first author, year, journal)	DOI
Bemisia tabaci	Imidacloprid	58–126×	Byrne et al. 2003 ^[8] , Pest Manag. Sci.	10.1002/ps.649
Bemisia tabaci	Imidacloprid	3.7–26.3×(2000–2007, FL)	Schuster et al. 2010 ^[9] , Pest Manag. Sci.	10.1002/ps.1853
Bemisia tabaci	Imidacloprid	86× (lab selection, 5 generations)	Khalid et al. 2021 ^[4] , Insects	10.3390/insects12110996
Plutella xylostella	Chlorantraniliprole	684.54× (lab-selected)	Qin et al. 2018 ^[10] , J. Econ. Entomol.	10.1093/jee/toy043
Myzus persicae	Imidacloprid	106.9× (field, China)	Hu et al. 2023 ^[11] , Agronomy	10.3390/agronomy13092276
Myzus persicae	beta-Cypermethrin	324× (field, China)	Hu et al. 2023 ^[11] , Agronomy	10.3390/agronomy13092276
Tetranychus urticae	Abamectin	2,226–21,141× (RR50)	Díaz-Arias et al. 2019, Florida Entomologist	10.1653/024.102.0222
Helicoverpa armigera	Cypermethrin	1,950× (field, Pakistan)	Alvi et al. 2012 ^[12] , PLOS ONE	10.1371/journal.pone.0047309
Leptinotarsa decemlineata	Imidacloprid	10.1–18.2× (2013); 1.8–11.2× (2014)	Clements et al. 2016 ^[13] , PLOS ONE	10.1371/journal.pone.0147844
Musca domestica	Diazinon	62.5–309.8× (field, Riyadh)	Abobakr et al. 2022 ^[14] , Insects	10.3390/insects13020192

Long-term monitoring programs illustrate that resistance ratios can fluctuate considerably between seasons rather than rising monotonically, reflecting changes in local selection pressure, immigration of susceptible individuals, and fitness costs associated with resistance alleles. Figure 2 reproduces

published RR50 values for imidacloprid in Florida field populations of Bemisia tabaci biotype B across eight monitoring years, showing both the overall upward trend and this year-to-year variability.

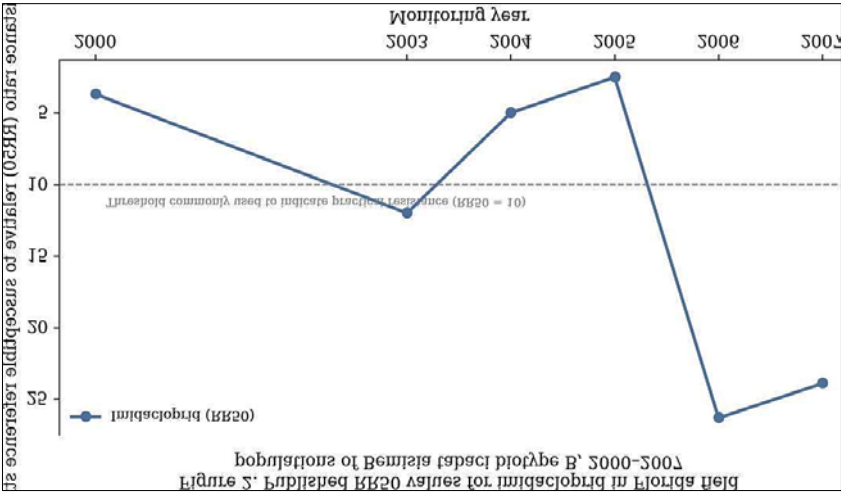


Fig 2: Published RR50 values for imidacloprid in Florida field populations of Bemisia tabaci biotype B, 2000–2007. Source: [Schuster et al. \(2010\)](#) ^[9], Pest Management Science 66:186–195, DOI: 10.1002/ps.1853.

5. Detoxification Enzyme Activity: Resistant versus Susceptible Strains

Beyond whole-organism resistance ratios, biochemical assays quantify the underlying enzyme activity differences between resistant and susceptible strains directly. Table 3 compiles specific-activity statistics (mean ± SD or SE, as reported by each source) for esterase and acetylcholinesterase assays in

agricultural pests, again cited with DOI. These data illustrate that metabolic resistance is frequently associated with several-fold to an order-of-magnitude increase in detoxification enzyme activity, while target-site (AChE) assays can show comparatively little separation between strains when the resistance mechanism is instead due to a qualitative change in enzyme sensitivity rather than quantity.

Table 3: Detoxification enzyme activity statistics (mean ± SD/SE, or fold-difference as reported) comparing susceptible and resistant/field strains of selected agricultural pests.

Pest species	Enzyme (assay)	Susceptible strain (mean ± SD/SE)	Resistant strain (mean ± SD/SE)	Source / DOI
<i>Tetranychus cinnabarinus</i> / <i>T. urticae</i>	Esterase (α-naphthyl acetate, nmol/mg protein/min)	233.8 ± 20.3 (<i>T. cinnabarinus</i>); 235.4 ± 10.3 (<i>T. urticae</i>)	483.3 ± 71.8 (recombinant TCE2)	Shi et al. 2016 ^[15] , Sci. Rep. 10.1038/srep18646
<i>Lygus lineolaris</i>	Esterase (α-naphthyl acetate)	Baseline (1×)	~6× higher than susceptible	Zhu & Snodgrass 2004 ^[16] , Insect Biochem. Mol. Biol. 10.1016/j.ibmb.2004.07.008
<i>Lygus lineolaris</i>	Esterase (β-naphthyl acetate)	Baseline (1×)	~3× higher than susceptible	Zhu & Snodgrass 2004 ^[16] , Insect Biochem. Mol. Biol. 10.1016/j.ibmb.2004.07.008
<i>Lygus lineolaris</i>	Esterase (p-nitrophenyl acetate)	Baseline (1×)	~10× higher than susceptible	Zhu & Snodgrass 2004 ^[16] , Insect Biochem. Mol. Biol. 10.1016/j.ibmb.2004.07.008
<i>Diaphorina citri</i>	Acetylcholinesterase (μM/min/mg protein)	Range across populations: 0.77–1.29 (Lab strain highest)	Field populations within same range (no target-site insensitivity detected)	Tiwari et al. 2012 ^[17] , J. Econ. Entomol. 10.1603/EC11228

The esterase data for *Tetranychus* spider mites illustrate a roughly two-fold increase in specific activity associated with the over-expressed *TCE2* carboxylesterase relative to susceptible crude enzyme extracts, while the *Lygus lineolaris* data show that the fold-difference between resistant and susceptible strains varies with the substrate used in the assay (3- to 10-fold across three chromogenic substrates), underscoring that reported enzyme-activity ratios are assay-specific and not a single fixed property of the resistant phenotype. By contrast, the *Diaphorina citri* data show overlapping AChE activity ranges between the susceptible laboratory strain and field populations, consistent with the original authors' conclusion that organophosphate and

carbamate resistance in that species was not attributable to AChE target-site insensitivity.

6. Recent *kdr* Mutation Frequency Data in Field Populations

Target-site knockdown resistance (*kdr*) mutations in the voltage-gated sodium channel (VGSC) continue to be documented in newly surveyed agricultural pest species. Table 5 summarizes *kdr/super-kdr* genotype and mutation-frequency data reported between 2022 and 2024 for four agricultural pests not covered in Table 1, each confirmed either functionally (heterologous expression in *Xenopus* oocytes) or through field-population genotyping, and each cited with its DOI.

Table 4: Recent (2022–2024) voltage-gated sodium channel (*kdr*) mutation data in agricultural pest field populations.

Pest species	VGSC mutation(s)	Reported frequency / finding	Source / DOI
<i>Ceutorhynchus</i> spp. (4 oilseed rape weevil species)	L1014F (<i>kdr</i>)	0–100% across French/German field populations; homozygotes >55% where field-rate pyrethroid control failed vs. ≤12.5% where control still effective	Daum et al. 2024 ^[18] , Pest Manag. Sci. 10.1002/ps.7549
<i>Lygus pratensis</i> (plant bug)	M918T + L1014F (<i>kdr/super-kdr</i>)	Homozygous genotype ≤40%; heterozygous genotype ≤20% in Inner Mongolia field/lab strains; high resistance to λ-cyhalothrin (LC50 401.31 ng/cm2)	Zhang et al. 2024 ^[19] , J. Agric. Food Chem. 10.1021/acs.jafc.4c03416
<i>Aphis glycines</i> (soybean aphid)	L1014F + M918I	Identified in field-evolved pyrethroid-resistant phenotypes; PCR-based genetic markers developed for resistance surveillance	Valmorbida et al. 2022 ^[20] , Sci. Rep. 10.1038/s41598-022-16366-1
<i>Megalurothrips usitatus</i> (bean flower thrips)	T929I + K1774N (double mutation)	Double mutation functionally confirmed to reduce pyrethroid-induced sodium channel currents in field-resistant strains from Hainan Province	Gao et al. 2024 ^[21] , J. Agric. Food Chem. 10.1021/acs.jafc.4c00355

The *Ceutorhynchus* data are particularly notable for directly linking *kdr* genotype frequency to field control outcomes: populations in which the resistant homozygote exceeded roughly half the sampled individuals corresponded to documented pyrethroid control failures at the recommended field rate, while populations with low homozygote frequency remained controllable. This dose-frequency relationship illustrates why *kdr* genotyping is increasingly used alongside

bioassays in resistance-management decision-making, rather than as a replacement for them.

7. Biochemical and Molecular Detection of Resistance

Reliable detection of resistance mechanisms underpins effective resistance management. Approaches range from whole-organism bioassays that quantify the dose–mortality relationship, to biochemical microplate assays that measure

detoxification enzyme activity directly, to molecular diagnostics that detect specific target-site mutations. Synergist bioassays, in which a candidate insecticide is co-applied with an enzyme inhibitor such as piperonyl butoxide (a P450 inhibitor), *S,S,S-tributyl phosphorotrithioate* (an esterase

inhibitor, DEF), or diethyl maleate (a GST inhibitor, DEM), remain a widely used first step in implicating a particular enzyme class before committing to more resource-intensive molecular confirmation.

Table 5: Commonly used methods for detecting biochemical and molecular resistance mechanisms.

Method	Principle	Typical application
Topical/leaf-dip bioassay	Dose–mortality response compared with a susceptible reference strain (LC50/RR50)	Field resistance monitoring
Microplate enzyme assay	Spectrophotometric measurement of esterase, GST or P450 activity in tissue homogenate	Rapid biochemical screening
PCR-RFLP / allele-specific PCR	Detection of known target-site mutations (kdr, ace-1, Rdl)	Molecular marker surveillance
qRT-PCR	Quantification of detoxification gene transcript abundance	Confirming metabolic over-expression
Synergist bioassay	Co-application of PBO, DEF or DEM to implicate specific enzyme classes	Mechanism diagnosis

8. Implications for Resistance Management

Findings from biochemical and molecular resistance studies inform several practical management strategies:

- Insecticide rotation based on distinct modes of action to reduce selection pressure on any single resistance mechanism.
- Use of synergists or mixtures to counteract metabolic detoxification pathways where economically and regulatorily feasible.
- Integration of biochemical and molecular monitoring into area-wide resistance-management programs to detect emerging resistance before it compromises field efficacy.
- Preservation of susceptible gene flow through refuge strategies, particularly where target-site mutations are recessive.
- Combining chemical control with cultural, biological, and physical control tactics within an integrated pest management framework to reduce reliance on any single insecticide class.

9. Discussion

Taken together, the mechanism summary in Table 1, the resistance-ratio data in Table 2, the enzyme activity statistics in Table 3, and the *kdr* mutation survey in Table 5 point to several recurring patterns in how biochemical resistance actually manifests in field populations of agricultural pests, beyond the individual mechanism-by-mechanism descriptions given in Section 2.

9.1 Multi-Mechanism Resistance Is the Field Norm, Not the Exception

Few of the resistant populations summarized in this review rely on a single mechanism. *Helicoverpa armigera* field populations combine *kdr*-type sodium channel mutations with elevated cytochrome P450 activity to achieve pyrethroid resistance ratios in the thousands (Table 2), and *Tetranychus* species frequently pair over-expressed carboxylesterases (Table 3) with target-site mutations in the glutamate-gated chloride channel to resist avermectins. This layering matters for resistance management because a synergist or mode-of-action rotation designed around a single mechanism will typically fail once a second, independent mechanism has become established in the same population; the practical resistance ratio observed in the field is better modeled as the combined outcome of co-occurring mechanisms than as the signature of any one pathway acting alone.

9.2 Resistance Ratios Are Assay- and Substrate-Dependent

The *Lygus lineolaris* esterase data in Table 3 illustrate a point that is easy to overlook when comparing resistance ratios across studies: the same resistant strain returned a 3-fold, 6-fold, or 10-fold difference from the susceptible strain depending solely on which chromogenic substrate (α -naphthyl acetate, β -naphthyl acetate, or *p*-nitrophenyl acetate) was used in the enzyme assay. Similarly, the *Diaphorina citri* acetylcholinesterase data show that a biochemical assay can fail to distinguish resistant from susceptible populations even when whole-organism bioassays and field control failures indicate that resistance is present, because in that case resistance was not conferred by AChE at all. Comparisons of resistance ratios between studies, therefore, are only meaningful when the assay conditions, reference susceptible strain, and life stage are comparable; a resistance ratio is a property of the measurement system as well as the pest population.

9.3 Resistance Levels Fluctuate Under Variable Selection Pressure

The Florida *Bemisia tabaci* monitoring data reproduced in Figure 2 (Schuster *et al.* 2010) [9] show that RR50 values for imidacloprid did not rise monotonically between 2000 and 2007 but instead fell in some seasons (from 12.0 in 2003 to 2.5 in 2005) before rising again to 26.3 in 2006. This non-monotonic trajectory is consistent with a fitness cost associated with resistance alleles: when selection pressure from a given insecticide temporarily relaxes (for example, due to a shift to alternative chemistries or a fallow period), susceptible genotypes can partially recover in the population, only for resistance to re-emerge once the same insecticide is reintroduced. The same pattern is documented experimentally in the Schuster *et al.* dataset itself, where resistant *Bemisia* sub-populations reared for six generations without imidacloprid exposure reverted from RR50 values of 50–60 to ≤ 4 . This has a direct management implication: insecticide rotation intervals that are too short to allow such reversion will not achieve the intended resistance-delaying effect.

9.4 Genotype-to-Phenotype Correlations Support Molecular Monitoring, With Caveats

The *Ceutorhynchus kdr* data (Table 5) demonstrate a reasonably tight relationship between homozygous mutant frequency and observed field control failure, which is one of the clearer examples in the current literature of a single genotyping assay being usable as a practical resistance-management indicator. However, this relationship does not hold universally: the *Diaphorina citri* and *Tetranychus urticae* cases in this review show that resistance can arise predominantly through metabolic up-regulation with no informative target-site marker, or through multiple independent target-site mutations whose individual contributions vary by population (as in the *Tetranychus urticae* backcrossing literature cited in Section 9.1). Molecular diagnostics are therefore best deployed as a complement to, rather than a replacement for, phenotypic bioassays, particularly when a pest species' dominant resistance mechanism has not yet been established for the population being monitored.

10. Conclusion

The biochemical basis of insecticide resistance in agricultural pests is multifactorial, involving metabolic detoxification, target-site insensitivity, reduced penetration, and, in some cases, behavioral avoidance, often acting in combination within a single field population. The quantitative data compiled in this review resistance ratios spanning less than 10-fold to more than 20,000-fold across pest-insecticide combinations (Table 2), enzyme-activity differences of 2- to 10-fold associated with metabolic resistance (Table 3), and *kdr* mutation frequencies ranging from near-zero to fixation within individual field populations (Table 5) illustrate both the scale of the problem and its variability across species, insecticide classes, and geographic regions. No single biochemical assay or molecular marker is sufficient to characterize resistance in a novel population; the data reviewed here consistently show that resistance phenotypes reflect the combined, and sometimes population-specific, contribution of several mechanisms. For resistance management, three conclusions follow directly from the evidence reviewed. First, because resistance ratios and mutation frequencies can decline as well as rise under fluctuating selection pressure, insecticide rotation and mode-of-action diversification (formalized in schemes such as the IRAC MoA classification) remain a rational strategy even where resistance is already established, rather than only as a preventive measure. Second, because metabolic and target-site mechanisms frequently co-occur, resistance-management programs that rely on a single diagnostic whether a synergist bioassay or a single-locus genotyping assay risk under-detecting resistance conferred by mechanisms outside that assay's scope. Third, continued biochemical and molecular monitoring, interpreted alongside field efficacy data rather than in isolation, remains essential to sustaining the effectiveness of chemical control within integrated pest management programs. As new insecticide chemistries such as diamides and butenolides come under increasing selection pressure, and as genomic and transcriptomic tools make population-scale resistance surveillance more accessible, the biochemical framework outlined in this review is likely to remain the basis for interpreting and managing resistance in agricultural pests for the foreseeable future.

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