

Bioaccumulation and Oxidative Stress in Freshwater Bivalve *Lamellidens marginalis* Exposed to Lambda-Cyhalothrin and Imidacloprid: A Critical Review

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ABSTRACT

Freshwater bivalves can integrate contaminant exposure across water, suspended particles and food, making them useful organisms for investigating pesticide effects. This critical narrative review examines bioaccumulation and oxidative stress in *Lamellidens marginalis* exposed to lambda-cyhalothrin and imidacloprid, while explicitly separating species-specific observations from evidence in other molluscs. Targeted literature searches, completed on 9 October 2026, prioritised primary studies and official testing documents. The two insecticides present contrasting exposure challenges: lambda-cyhalothrin is strongly lipophilic and poorly water-soluble, whereas imidacloprid is substantially more water-soluble and can undergo uptake, metabolism and elimination despite its lower lipid affinity. Available direct studies in *L. marginalis* indicate disturbance of protein and ascorbate pools following lambda-cyhalothrin exposure, and lipid peroxidation and tissue injury following imidacloprid exposure. However, these studies do not establish chemically measured tissue burdens or species-specific bioconcentration factors. An unresolved exposure-concentration discrepancy also limits quantitative interpretation of one imidacloprid study. Research in other

freshwater mussels and marine bivalves supports tissue- and time-dependent antioxidant responses, behavioural disruption and, for imidacloprid in oysters, measurable uptake and depuration. Such findings strengthen biological plausibility but cannot substitute for toxicokinetic measurements in *L. marginalis*. The most defensible future approach combines verified water and tissue concentrations, parent-compound and metabolite analysis, antioxidant and oxidative-damage endpoints, histopathology, functional performance and recovery observations. Binary-mixture effects require explicit concentration-addition or independent-action comparisons rather than inference from larger responses alone. The review concludes that oxidative injury is better supported than quantitative bioaccumulation for the target species-pesticide combinations, and identifies the measurements needed to connect external exposure, internal dose and ecologically meaningful harm.

Keywords: *Lamellidens marginalis*; lambda-cyhalothrin; imidacloprid; bioaccumulation; oxidative stress; lipid peroxidation; freshwater mussels; pesticide mixtures.

INTRODUCTION

Freshwater mussels link the water column with benthic habitats through feeding, respiration and nutrient processing. Their responses to contamination therefore matter both as indicators of organism health and as potential signals of altered ecosystem functioning. Mesocosm experiments by Vaughn et al. (2004) demonstrated relationships between unionid biomass, algal clearance and nutrient dynamics, providing an ecological basis for studying sublethal impairment rather than mortality alone. A surviving mussel that filters less water or redirects energy towards cellular repair may not perform the same ecological functions as an unexposed individual. [1]

Pesticide exposure in aquatic habitats is not adequately represented by a single chemical concentration measured at one time. Runoff can transport insecticides from treated landscapes, while partitioning and degradation redistribute them among dissolved, particulate and sediment-associated fractions. Weston and Lydy (2010) documented aquatic inputs of pyrethroids in California, and Hladik et al. (2014) reported neonicotinoids in streams within an intensively cropped region of the United States. These studies establish plausible exposure pathways, not the occurrence or concentration of the two target insecticides at any particular Indian collection site. Local exposure must be demonstrated by local measurements. [2, 3] Lambda-cyhalothrin and imidacloprid offer a useful contrast for ecotoxicological assessment. The former is a pyrethroid with high lipid affinity and very low aqueous solubility; the latter is a neonicotinoid with appreciably greater water solubility. These properties suggest different sampling priorities, but neither property set alone establishes how much pesticide a freshwater bivalve will retain. Formulation, feeding, valve activity, tissue composition, metabolism and exposure duration can all intervene between the surrounding concentration and the internal dose. [4, 5, 6]

For *Lamellidens marginalis*, the central interpretative challenge is to distinguish evidence of exposure-related injury from evidence of chemical accumulation. Direct reports have examined biochemical responses to lambda-cyhalothrin and oxidative or histological responses to imidacloprid. Their existence justifies a focused review, but their endpoints do not make tissue residues an observed result. Likewise, an antioxidant response is not a quantitative estimate of the concentration reaching a target tissue. [7, 8]

This review addresses three connected questions: what has been demonstrated for the target species, what related-bivalve research can reasonably contribute, and what measurements are still required to connect exposure with adverse outcomes. The emphasis is on bioaccumulation terminology, evidence quality, tissue-specific oxidative responses, mixture interpretation and realistic experimental design. Rather than combining heterogeneous nominal doses into an apparent toxicity ranking, the review develops an evidence-based framework for interpreting existing observations and planning stronger studies.

MATERIALS AND METHODS

Review approach and scope

A critical narrative review was prepared using targeted web-assisted searches of PubMed-indexed records, publisher websites, accessible primary articles and official WHO, OECD and pesticide-information documents. Searches were completed on 9 October 2026. Core search combinations included “*Lamellidens marginalis*” with “lambda-cyhalothrin”, “imidacloprid”, “bioaccumulation”, “lipid peroxidation” and “oxidative stress”. Broader searches substituted “freshwater mussel”, “Unio”, “bivalve”, “oyster”, “uptake” or “depuration”; citation trails were used to identify relevant experimental and analytical-method papers. No claim is made that every database, language or unpublished study was covered.

Evidence was organised by proximity to the review question. The highest relevance was assigned to *L. marginalis* exposed to either target insecticide. Other freshwater unionids were considered next, followed by other freshwater or marine bivalves. Non-molluscan studies were used only for clearly identified mechanistic or mixture hypotheses. Official testing guidance informed terminology and study quality, while original analytical-method papers informed endpoint selection. Findings available only from abstracts were restricted to what those abstracts reported; unavailable experimental detail was not reconstructed. The appraisal considered chemical identity, formulation, active-ingredient basis, exposure duration, nominal versus measured concentration, tissue selection, analytical confirmation, control design and endpoint interpretation. Inconsistent exposure reporting was retained as uncertainty rather than silently corrected. The review was not registered as a systematic review, and formal screening-flow counts were not recorded. Findings were synthesised qualitatively; no meta-analysis was

performed. Differences in species, exposure verification, tissue preparation and outcome units would make a pooled numerical estimate misleading without a larger, harmonised extraction dataset.

LITERATURE REVIEW

Chemical properties and environmental exposure

The strong contrast in physicochemical behaviour provides a starting hypothesis for exposure assessment, not a substitute for toxicokinetics. One manufacturer-specific dataset reproduced in the WHO lambda-cyhalothrin evaluation reports water solubility of 5.4 µg/L at approximately 20 °C and pH 7, with log Kow of 6.61 at approximately 25 °C. These condition-specific measurements indicate that high nominal laboratory doses need careful characterisation of the actual dissolved and dispersed fractions. Imidacloprid has much greater water solubility and lower octanol–water partitioning; its presence in water can therefore remain important even when lipid-driven storage is less favoured. Table 1 summarises the practical contrast. [4, 5]

Table 1. Contrasting properties and their implications for exposure assessment.

Feature	Lambda-cyhalothrin	Imidacloprid
Chemical class	Pyrethroid insecticide. [4]	Neonicotinoid insecticide. [5]
Water solubility	Very low; one WHO dataset reports 5.4 µg/L at about 20 °C and pH 7. [4]	Much greater aqueous solubility than lambda-cyhalothrin. [5]
Partitioning	High lipid affinity; log Kow 6.61 in the specified WHO dataset. [4]	Lower lipid affinity; dissolved exposure remains important. [5]
Sampling priority	Characterise dissolved, dispersed and particle-associated fractions. [9]	Measure water, tissues and relevant metabolites over time. [10]
Main interpretative trap	Nominal dose treated as fully dissolved and bioavailable.	Low lipid affinity treated as proof of no uptake or biological risk.
Required confirmation	Actual exposure fraction and tissue chemistry.	Actual exposure, tissue chemistry and elimination.

Kow denotes the octanol–water partition coefficient. Values are condition- and dataset-specific; they are not bioaccumulation factors or environmental quality criteria.

For lambda-cyhalothrin, measuring total pesticide in an unfiltered sample does not necessarily identify the freely dissolved fraction available at a respiratory surface. Conversely, filtered-water analysis alone can miss exposure associated with particles or ingested material. A filter-feeding organism may encounter several fractions

simultaneously, so a water-only concentration should not be interpreted as a complete exposure inventory. Sorption to vessels, suspended matter and organic material can also change the concentration during laboratory exposure. These are test-design questions that require measurement,

not assumptions about complete mixing. [2, 9]

The imidacloprid problem is different but not simpler. Greater water solubility does not guarantee a constant concentration throughout a renewal interval, and low lipid affinity does not exclude internal exposure. Dissolved inputs may recur, while metabolism and elimination occur concurrently. A short-lived tissue residue can still accompany repeated biological effects when the next pulse arrives before functional recovery. This distinction between chemical residence time and biological recovery time is important when evaluating either repeated runoff events or continuously renewed laboratory exposures. [10, 11]

A nominal concentration expressed as “ppm” is particularly difficult to compare when the article does not specify whether it refers to active ingredient, commercial product or preparation volume. The formulation concentration and dilution calculation should be recorded separately. An active-ingredient concentration in µg/L should be used for cross-study comparisons only when the original information allows an unambiguous conversion. Commercial formulations may contain additional components, making a formulation experiment informative about the tested product but not automatically equivalent to a technical-grade active-ingredient experiment. [9]

Biological context and exposure interfaces

A useful working model for *L. marginalis* is that uptake and injury are distributed processes rather than events confined to one organ. The gills are an obvious interface with surrounding water; the digestive gland

is relevant to ingested material and biotransformation; and the mantle, foot, muscle and haemolymph provide complementary information about tissue distribution or systemic responses. These compartments should be selected to answer distinct questions, not simply to maximise the number of biochemical measurements. Related-bivalve studies demonstrate that the tissue showing the greatest functional or biochemical disturbance need not be the tissue containing the most parent compound. [10, 12, 13]

Valve movement introduces a feedback between exposure and behaviour. Closure may reduce water exchange, but the same response may alter feeding and oxygen conditions. Consequently, two mussels in the same nominal water concentration need not experience identical effective exposure. Szostak et al. (2025), working with *Unio tumidus*, found time-dependent changes in valve activity during imidacloprid exposure. Their results support including continuous behaviour measurements where feasible, without treating a particular valve pattern as a pesticide-specific signature. [14]

The principal implication is methodological: tissue chemistry, oxidative biomarkers and organism performance should be measured over compatible time intervals. A residue sampled immediately after a pulse and an enzyme activity sampled after prolonged recovery describe different biological states. Similarly, gut contents should not be confused with absorbed pesticide, and prolonged pre-analysis depuration can itself change the residue intended for measurement. The sampling decision must therefore specify whether the aim is field-integrated burden, absorbed tissue concentration or elimination kinetics. [6, 10]

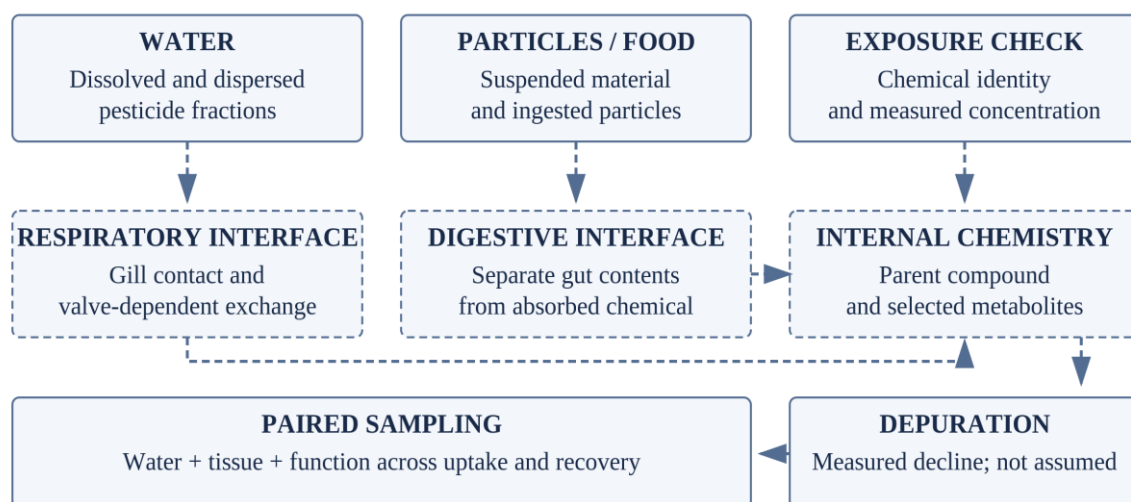


Figure 1. Conceptual exposure-to-internal-dose framework for *L. marginalis*. Water, particle and food routes are hypotheses to be quantified for each pesticide. Sampling should distinguish absorbed tissue residues, gut contents and external material. The framework draws on bioaccumulation principles and comparative bivalve uptake research; it is not an experimentally measured pathway map. [6, 10]

Bioaccumulation, bioconcentration and internal dose

Bioaccumulation describes the net result of uptake from relevant routes and elimination over time. Bioconcentration is more restricted: it concerns uptake directly from water under conditions in which other routes are excluded or controlled. A biomarker response, a nominal exposure concentration or a decline in tissue protein is not a bioaccumulation factor. The distinction matters because the title of a study may refer broadly to “accumulation” even when the experiment measures only physiological stress. The principles below follow established toxicokinetic terminology; OECD Test No. 305 is a fish guideline and should not be presented as a validated *L. marginalis* protocol. [6]

A minimal water-only model can be written as Equation (1). It is a conceptual model proposed here for study planning, not a fit to existing *L. marginalis* data.

$$\frac{dC_t}{dt} = k_u C_w - k_e C_t \quad (1)$$

Here, C_t is the parent-compound concentration in tissue, expressed as $\mu\text{g/kg}$ wet weight; C_w is its water concentration in $\mu\text{g/L}$; k_u is an apparent uptake clearance in $\text{L kg}^{-1} \text{d}^{-1}$; and k_e is an apparent first-order elimination constant in d^{-1} . The model

assumes a reasonably homogeneous compartment, stable physiological behaviour and a specified exposure fraction. With constant C_w , negligible growth dilution and an adequate observation period, the steady-state concentration ratio and the kinetic ratio are related by Equation (2). These assumptions must be checked rather than imposed because the equations are convenient.

$$\text{BCF}_{ss} = C_{t,ss}/C_w; \quad \text{BCF}_k = k_u/k_e \quad (2)$$

Both factors have units of L/kg under the stated concentration units. In a clean-water elimination phase with no renewed uptake, Equation (1) predicts exponential decline and a half-life of $\ln(2)/k_e$. A poor fit would indicate that a single constant-rate compartment is inadequate, possibly because of multiple tissue pools or changing behaviour. Failure to observe steady state does not justify labelling the final sampling ratio a steady-state BCF. Instead, the time-specific ratio and its limitations should be reported explicitly.

Where food contributes to uptake, an additional term $\text{AE} \times \text{IR} \times C_{\text{food}}$ can be included in Equation (1). AE is the dimensionless assimilation efficiency, IR is the food ingestion rate in $\text{kg food kg}^{-1} \text{organism d}^{-1}$, and C_{food} is the pesticide

concentration in the food. This dimensional balance highlights the information needed to separate exposure routes. It also shows why changing the feeding regime during a toxicity experiment can change both chemical intake and nutritional condition. The extended equation is a research hypothesis until its terms are measured for the species and exposure setting.

In field studies, a tissue-to-water ratio is better treated as a time- and context-dependent bioaccumulation metric when dietary and sediment-related routes cannot be excluded. A low water concentration after a runoff pulse may coexist with a measurable tissue residue, producing a large ratio that reflects mismatched sampling times rather than exceptional uptake. Conversely, a high transient water concentration sampled before appreciable uptake can produce a small ratio. Paired time-series measurements are therefore more informative than a single quotient. Detection limits and the treatment of non-detects must accompany any reported ratio.

Direct evidence in *Lamellidens marginalis*

Goswami and Bhalla (2021) exposed *L. marginalis* to nominal lambda-cyhalothrin at 0.75 ppm for 21 days, with and without 50 mg/L L-ascorbate, and examined biochemical recovery. Protein and ascorbate depletion across sampled tissues was less pronounced with supplementation. This is evidence of biochemical disturbance and modulation of antioxidant reserves, not a measurement of pesticide burden: neither tissue-residue kinetics nor a species-specific BCF was established. Antioxidant supplementation also cannot, by itself, identify the initiating molecular lesion or demonstrate environmentally practical remediation. [7]

Ahire and Dethle (2023) reported imidacloprid-associated lipid peroxidation and histological abnormalities in *L. marginalis*. Their paper gives a 96-hour LC₅₀ of 40 ppm, used for histological exposure, but the lipid-peroxidation methods specify 0.7 ppm while the table

caption specifies 0.07 ppm. The discrepancy remains unresolved. Percentage changes described in the text also do not consistently match the displayed means. Accordingly, the study supports a qualitative oxidative-injury signal but is not used here to derive an effect threshold or rank tissue sensitivity quantitatively. It does not provide chemically measured pesticide residues. [8] These studies answer narrower questions than the present review and provide a basis for pursuing chemical verification and more complete endpoint panels. The limitation arises when biochemical depletion or damage is relabelled as bioaccumulation, or when an ambiguous nominal dose becomes a precise environmental threshold. The strongest next step is therefore not another isolated enzyme assay, but a design that measures external exposure and internal chemistry alongside the biological response.

Evidence from other bivalves and its transferability

Yoloğlu (2019) studied *Unio mancus* exposed for 96 hours to an imidacloprid formulation at nominal concentrations of 1–1,000 µg active ingredient/L. Water concentrations were analytically checked and were below the nominal targets. Gill and digestive-gland responses involved cholinesterase, glutathione-associated endpoints and lipid peroxidation, with endpoint-specific concentration patterns. This is stronger exposure characterisation than an unverified nominal-dose experiment, but measured water should not be mistaken for measured tissue. The study supports a multi-endpoint approach while leaving the internal-dose relationship unresolved. [12]

Tessi et al. (2025) exposed *Unio terminalis* to lambda-cyhalothrin at nominal 0.11 and 0.22 mg/L for 48 hours, 7 days and 21 days. Their integrated assessment combined antioxidant, oxidant, haemocyte and histological endpoints. Increased total oxidant status and glutathione peroxidase activity occurred alongside suppression of superoxide dismutase and catalase,

particularly in gills. This mixed response is important: a pesticide challenge need not move all antioxidant enzymes in the same direction. Differences in species and exposure conditions prevent direct assignment of these responses to *L. marginalis*. [13]

In the marine clam *Venus verrucosa*, Fouzai et al. (2020) examined gill responses after six days of lambda-cyhalothrin exposure at 100–500 µg/L. Lipid- and protein-oxidation markers, antioxidant responses and membrane fatty-acid composition were affected. The study links biochemical defence with damage and membrane alteration, but its marine setting and comparatively high nominal exposures restrict environmental and species-specific extrapolation. It provides a mechanistic analogue rather than a freshwater-bivalve safety threshold. [15]

Ewere et al. (2019) supplied a particularly useful toxicokinetic example in the marine oyster *Saccostrea glomerata*: imidacloprid

uptake and depuration were measured analytically, and metabolites were investigated.

Parent-compound concentrations were higher in adductor muscle than in gills or digestive gland; most tissues were below detection after four days of depuration, except the digestive gland. This demonstrates the value of tissue-resolved chemistry, but the elimination pattern cannot be transferred quantitatively to *L. marginalis*. “Below detection” also does not mean that every molecule has been eliminated. [10]

Table 2 identifies what each core study actually contributes. The evidence is deliberately not arranged as a potency ranking: differences in formulation, concentration verification, species and duration make such a ranking unreliable. A study measuring residues answers a different question from one measuring an antioxidant enzyme, even when both are relevant to pesticide stress.

Table 2. Core experimental evidence and its relevance to the review question.

Study / organism	Exposure focus	Evidence contributed	Boundary of inference
Goswami and Bhalla (2021); <i>L. marginalis</i> [7]	Lambda-cyhalothrin	Protein and ascorbate responses.	Direct species evidence; no measured residues.
Ahire and Dethe (2023); <i>L. marginalis</i> [8]	Imidacloprid	Lipid peroxidation and histology.	Direct evidence; unresolved dose reporting.
Yoloğlu (2019); <i>U. mancus</i> [12]	Imidacloprid	Water verification and biochemical panel.	Related unionid; water chemistry is not tissue chemistry.
Tessi et al. (2025); <i>U. terminalis</i> [13]	Lambda-cyhalothrin	Redox, haemocyte and tissue endpoints.	Related unionid; responses vary by tissue and time.
Fouzai et al. (2020); <i>V. verrucosa</i> [15]	Lambda-cyhalothrin	Oxidation markers and membrane lipids.	Marine analogue; not a freshwater threshold.
Ewere et al. (2019); <i>S. glomerata</i> [10]	Imidacloprid	Analytical uptake, depuration and metabolites.	Marine toxicokinetics; not transferable as species-specific rates.
Szostak et al. (2025); <i>U. tumidus</i> [14]	Imidacloprid	Time-dependent valve activity.	Behaviour is not proof of accumulation.
Alvim and Martinez (2019); <i>P. lineatus</i> [16]	Single compounds and mixture	Oxidative and genotoxic interaction evidence.	Fish study; hypothesis-generating for <i>L. marginalis</i> .

All rows summarise published studies, not new observations. Study-specific doses and limitations are discussed in the text. No quantitative potency ranking is implied.

RESULTS OF LITERATURE SYNTHESIS

Oxidative stress: defence, disturbance and damage

The literature supports a response framework in which exposure can alter antioxidant defence, while sustained or insufficiently controlled oxidant pressure can be accompanied by molecular and tissue

injury. These stages are related but not interchangeable. An increase in an antioxidant activity can represent compensation; a decrease can reflect inhibition, substrate limitation, protein damage or reduced physiological activity. Without oxidative-damage or functional endpoints, either direction remains difficult to classify as harmful. The contrasting patterns reported in freshwater and marine bivalves argue against a universal “all enzymes increase” or “all enzymes decrease” model. [12, 13, 15]

Superoxide dismutase converts superoxide to hydrogen peroxide, while catalase removes hydrogen peroxide. This coupling explains why an isolated rise in superoxide dismutase activity does not necessarily indicate that the complete detoxification sequence is adequate. Glutathione-dependent processes add another layer, involving peroxide reduction, thiol buffering and conjugation reactions. The original enzyme-method literature provides the biochemical foundation, but an assay performed in a tissue homogenate measures potential activity under assay conditions, not the instantaneous reaction flux inside the living mussel. [17, 18, 19]

For *L. marginalis*, mitochondria, redox-active metabolism and immune-cell

responses are plausible contributors to oxidant production, but their relative roles under the target pesticide exposures remain hypotheses. Demonstrating a mitochondrial mechanism would require measurements such as respiration, membrane potential or respiratory-complex function alongside exposure confirmation. Demonstrating a particular signalling pathway would require pathway-specific evidence. It is therefore premature to present an unmeasured molecular cascade as an established mechanism merely because it is biologically plausible.

The same caution applies to molecular docking and gene-expression studies. Predicted binding does not establish that a compound reaches the proposed protein at a relevant concentration. Altered transcript abundance does not guarantee altered enzyme activity, and altered enzyme activity does not guarantee damage. A stronger causal sequence is temporal: verified exposure precedes internal chemical detection, defence changes precede or accompany oxidative injury, and injury aligns with impaired function. Figure 2 presents this as a testable framework rather than an experimentally validated pathway for the target species.

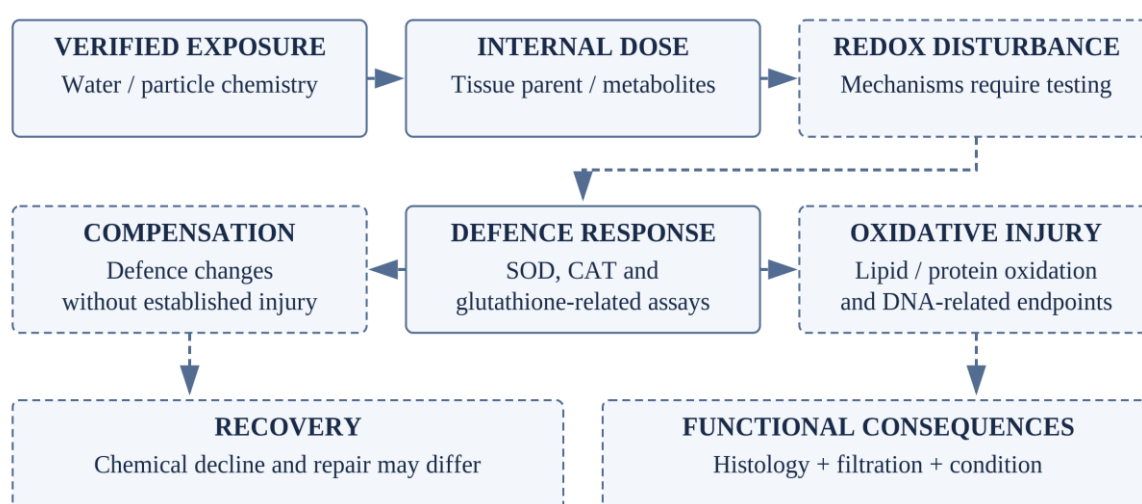


Figure 2. Proposed interpretation of pesticide-related oxidative responses. Defence changes can be compensatory or insufficient; they are not equivalent to damage. Chemical measurements, molecular injury, histology and functional endpoints are needed to test the sequence. Dashed arrows denote hypothesised links requiring validation in *L. marginalis*. [13, 15]

Lipid peroxidation and complementary injury markers

Lipid peroxidation is useful because it addresses damage more directly than an isolated antioxidant activity. However, the commonly used thiobarbituric-acid reaction measures a class of reactive products rather than a uniquely pesticide-specific signal. The original Ohkawa et al. (1979) method provides a widely used analytical basis; in a bivalve study, preparation conditions, blanks, calibration, sample oxidation during processing and the expression of results must still be validated. Reporting “MDA equivalents” is preferable when the assay does not chromatographically establish malondialdehyde identity. [20]

A tissue can show different apparent sensitivity depending on whether results are normalised to wet mass, dry mass, lipid or protein. Protein normalisation is useful, but becomes especially important to interpret when protein itself changes during exposure. The denominator should be reported as a biological measurement rather than hidden inside the final ratio. Parallel reporting of protein concentration and the normalised endpoint, with a consistent protein assay and sample preparation, helps distinguish increased damage from a changed denominator. [21]

Protein carbonyls provide an independent injury measure and can help determine whether a response extends beyond membrane lipids. DNA strand-break measurements provide another dimension, although comet-assay responses should not automatically be called mutations or irreversible genotoxicity. The foundational carbonyl and single-cell gel-electrophoresis methods support these endpoint choices. Their use in a particular bivalve tissue requires viability assessment, appropriate controls and a clear scoring method. [22, 23]

Species-specific feasibility is supported by work outside the two target insecticides. Mundhe et al. (2016) assessed oxidative stress, DNA damage and recovery in *L. marginalis* exposed to monocrotophos. This

establishes that linked oxidative and genotoxic endpoints can be studied in the species, but it does not establish identical responses to imidacloprid or lambda-cyhalothrin. The useful transfer is methodological: pairing damage with recovery is more informative than relying on one terminal observation. [24]

Tissue specificity, immune responses and organism performance

Gill and digestive-gland results should remain separate until a biological reason for combining them is established. Differences may arise from exposure route, tissue turnover, metabolic capacity and baseline composition. A whole-body homogenate can be useful for mass-balance work but may dilute a strong local response. Conversely, selecting only the most responsive organ after inspecting the results can overstate a general effect. An a priori tissue-selection plan, followed by tissue-resolved reporting, is therefore preferable to an assumed universal sensitivity order.

Haemocyte measurements can add an immune dimension to the assessment. Ewere et al. (2020) investigated imidacloprid effects on immune-related endpoints in *S. glomerata*, including cellular responses alongside biochemical measurements. This supports considering haemocyte abundance, viability and phagocytic performance in a broader bivalve panel. Nevertheless, reactive oxygen production by immune cells has physiological functions, so an increased cellular oxidant signal requires interpretation with immune performance and cell viability rather than automatic classification as damage. [25]

Filtration, valve activity, respiration and condition are useful bridges between biochemical responses and organism performance. In a non-bivalve example, Nyman et al. (2013) linked imidacloprid exposure in *Gammarus pulex* with impaired feeding and subsequent adverse outcomes. That result does not establish starvation-mediated toxicity in *L. marginalis*; it

identifies a competing explanation worth testing. Reduced feeding could change energy reserves and antioxidant capacity even without a directly demonstrated mitochondrial toxicant effect. Food consumption and nutritional condition should therefore be monitored when interpreting chronic biochemical responses. [11]

Table 3 groups endpoints by the question they can answer. A balanced panel does not

require every available assay. It requires enough independent information to distinguish exposure, compensation, molecular injury and loss of function. Routine work may reasonably prioritise confirmed chemical exposure, gill and digestive-gland lipid peroxidation, a small antioxidant panel, blinded histology and one functional endpoint before adding exploratory molecular measurements.

Table 3. A question-led measurement panel for future *L. marginalis* studies.

Measurement	Question addressed	Interpretative safeguard
Parent compounds / metabolites	What internal exposure occurred?	Validate each matrix; distinguish gut contents from tissue.
SOD, CAT and glutathione-related endpoints	Is antioxidant defence altered?	Direction alone does not establish injury. [17, 18, 19]
Lipid peroxidation / MDA equivalents	Is membrane-related oxidation increased?	Control processing artefacts and assay specificity. [20]
Protein carbonyls	Is oxidative protein injury present?	Use blanks, appropriate normalisation and an independent endpoint. [22]
Comet assay / cell viability	Are DNA strand-break-related signals altered?	Do not equate a comet response with a fixed mutation. [23]
Histopathology	Does tissue structure show injury?	Blind scoring; sections and fields are subsamples.
Valve activity / filtration / respiration	Does the response affect performance?	Account for baseline behaviour, food and oxygen conditions.
Uptake and recovery time courses	Do chemistry and function recover together?	A non-detect is not proof of complete elimination.

This panel is proposed, not a record of assays completed for the target exposures. SOD: superoxide dismutase; CAT: catalase; MDA: malondialdehyde.

Combined exposure to lambda-cyhalothrin and imidacloprid

The two insecticides can be studied together without assuming that their combined action is necessarily synergistic. Distinct principal insecticidal targets do not ensure independent downstream effects in a non-target organism. They may converge on energy balance, membrane injury or antioxidant demand, while one compound could also change the uptake of the other through altered valve behaviour. These are experimentally separable hypotheses: an internal-dose interaction changes the amount reaching tissues, whereas a toxicodynamic interaction changes the response at a given internal dose.

Alvim and Martinez (2019) examined the exact binary combination in *Prochilodus lineatus* using lambda-cyhalothrin at 0.15

µg/L and imidacloprid at 100 µg/L across exposures of 48–192 hours. They reported oxidative and genotoxic outcomes, including tissue- and time-dependent interaction patterns. This is relevant proof that the combination deserves testing, not proof of synergy in *L. marginalis*. Differences in respiratory physiology, metabolism and tissue distribution make direct transfer of a fish interaction result inappropriate. [16]

Mixture interpretation requires a defined reference expectation. Concentration addition estimates the combination expected from component concentration–response relationships expressed on a common effect scale. Independent action uses the component effects to predict a combined response under a different reference assumption. Jonker et al. (2005)

demonstrated how deviations from these models can depend on concentration level and mixture ratio. Consequently, a larger response in a mixture than in either single treatment is not, by itself, a demonstration of synergy. [26]

For fractional effects between zero and one, the independent-action expectation can be written as Equation (3), where E(A) and E(B) refer to the same adverse endpoint and observation time.

$$E(A,B) = 1 - (1 - E(A))(1 - E(B)) \quad (3)$$

For concentration addition at a specified effect level x , the corresponding isobole is $C(A)/EC_x(A) + C(B)/EC_x(B) = 1$. These equations are not directly applicable to raw enzyme activities that can increase or decrease and have no common bounded effect scale. A proposed *L. marginalis* mixture study should therefore define its primary adverse endpoint in advance, fit single-compound concentration–response curves, and compare mixture observations with reference predictions and uncertainty intervals. Multiple ratios and sampling times would help distinguish a general interaction from a narrow condition-specific deviation.

DISCUSSION

Analytical confirmation as the missing connection

The highest-priority improvement is to measure the chemicals rather than infer their presence from their effects. A defensible study should quantify the parent insecticides in exposure water and relevant tissues, with selected metabolites where standards and validated methods are available. Water sampling should cover the start and end of renewal intervals; tissue sampling should cover uptake and depuration. Matched blank matrices, recovery experiments, calibration range, matrix effects, detection limits and quantification limits are essential to determine whether a reported difference reflects chemistry or analytical bias. [6, 9] Sample preparation should be validated separately for each matrix. A method that works in water or plant material is not

automatically reliable in lipid- and protein-rich bivalve tissue. The QuEChERS extraction approach introduced by Anastassiades et al. (2003) provides a useful starting platform, but its original application to produce is not validation for mussel tissues. Extraction efficiency, cleanup losses and instrumental matrix effects need to be demonstrated for the target analytes and tissue types. Where feasible, suitable internal standards should be added before extraction rather than only before instrument injection. [27]

Parent compounds and metabolites should be reported separately unless a justified residue definition has been established. A summed mass concentration may be misleading when molecular masses and biological potencies differ. The same concern applies to lipid normalisation: it can aid interpretation of a strongly hydrophobic pesticide but is not a universal correction for every compound or tissue. Reporting wet-weight concentration, moisture, lipid content and the chosen normalisation permits the reader to reconstruct the result instead of relying on an unexplained adjusted value.

Non-detects require disciplined interpretation. A concentration below the detection limit is not an observed zero, and replacing every non-detect with zero can bias estimates of depuration. The study should state its handling rule and, where data permit, use methods that acknowledge left-censoring. Clean-water recovery also requires chemical confirmation that the recovery medium remains uncontaminated. Otherwise, an apparent slow elimination rate may represent continuing uptake rather than retention within the organism.

A realistic experimental design for *L. marginalis*

A staged design is preferable to immediately testing a large matrix of doses and biomarkers. The initial stage should establish organism identity, acclimation performance, background contamination and the stability of the dosing system.

Voucher-based identification and a documented collection history would improve reproducibility. Shell dimensions, body condition and reproductive state should be recorded so that strong biological differences are not unintentionally confounded with treatment allocation. Animals with evident disease or poor acclimation performance should be handled according to predefined inclusion criteria rather than removed selectively after observing treatment effects.

Dose selection should distinguish environmental monitoring, chronic sublethal testing and acute hazard characterisation. A fraction of an LC_{50} is a laboratory convention, not proof of environmental relevance. The environmental arm should be anchored to measured site concentrations or a clearly identified monitoring dataset. A separate mechanistic arm may use higher concentrations, provided that dissolved exposure, formulation effects and severe general toxicity are characterised. The study should not describe every dose as environmentally realistic simply because it is lower than a lethal concentration.

The principal treatment structure should include a water control, a carrier control where needed, each insecticide separately and the binary mixture. A formulation comparison requires additional matched treatments rather than replacing active-ingredient controls. Pilot testing should determine an achievable, analytically verified concentration range. Replicate tanks must be allocated independently and placed or blocked to avoid systematic light, temperature or aeration differences. Destructive tissue sampling should use planned cohorts so that repeated removals do not create unequal density or feeding conditions between treatments.

Time points should capture early response, sustained exposure and recovery, with sufficient observations to estimate kinetic parameters when that is an objective. A fixed duration borrowed from a fish protocol should not be called validated for *L. marginalis*. Instead, pilot uptake and

elimination data should determine whether the intended phases are long enough. For repeated-pulse experiments, the interval between pulses should be related to observed chemical elimination and functional recovery, allowing the study to test whether injury accumulates even when the parent compound declines between events.

A small mechanistic module can then be added to address a specific uncertainty. Pair-feeding or carefully monitored food intake can test nutritional confounding. Comparison of technical active ingredient with the commercial formulation can test product-level effects. Measurement of tissue residues in single and mixture treatments can distinguish altered uptake from altered sensitivity. The value of such modules lies in resolving a causal question, not in increasing the number of significant biochemical results.

Biomarker quality control and histological interpretation

Biochemical measurements should use a standardised dissection interval, tissue-to-buffer ratio, processing temperature and storage protocol. Assay conditions should be validated over a linear range of sample amount and reaction time. Each batch should include blanks and a stable quality-control material where feasible. For catalase, GST and acetylcholinesterase, the chosen substrate, wavelength, temperature and calculation should be stated, with appropriate methodological citations. The analytical principle is established, but its performance in a new tissue preparation still requires checking. [18, 19, 28]

Histology should be scored with the observer blinded to treatment. Representative photomicrographs are useful illustrations, but they do not replace an animal-level lesion score or a defined sampling scheme. The number of sections per animal, organ regions examined, lesion criteria and handling of overlapping abnormalities should be specified. Several microscopic fields from one animal are

subsamples, not independent animals. Histological injury also cannot identify the responsible chemical in a field-collected mussel unless alternative stressors and residue evidence are considered.

An integrated biomarker response index can summarise a multivariate pattern. Beliaeff and Burgeot (2002) introduced an integrative approach, and Sanchez et al. (2013) developed a reference-deviation formulation. These tools are useful for comparison within a defined dataset, but their values depend on the selected endpoints, standardisation and reference group. A larger index should not be interpreted as a universal toxicity unit or an independent confirmation of bioaccumulation. Individual biomarker results, direction of change and variability must remain available alongside the integrated score. [29, 30]

Statistical analysis and interpretation

The statistical unit should match the unit of exposure allocation. When several mussels share a tank, the tank is the independently assigned exposure replicate; measurements from individuals and tissues are nested within that structure. Treating all animals from a single exposure tank as independent treatment replicates inflates apparent precision. Technical replicate assays improve measurement reliability but do not increase biological replication. A pilot-based power assessment should therefore use between-tank and between-animal variability relevant to the primary endpoint. A practical analysis would model concentration, time and their interaction, with tank or block represented appropriately and tissue treated as a planned factor or analysed in prespecified models. Repeated non-destructive measurements require correlation within individuals to be accounted for. Residual diagnostics should guide transformation or alternative models, rather than applying a normality test as an automatic decision rule. Reporting effect sizes and confidence intervals is more

informative than describing every result only as significant or non-significant.

Multiple biomarkers create multiple opportunities for false-positive findings. Primary endpoints should be declared before analysis, while exploratory endpoints can be interpreted with a stated multiplicity strategy. Correlations among tissue residues, lipid peroxidation and filtration can strengthen an exposure–effect interpretation, but common dependence on dose or time can also generate correlation. Models that account for these factors, coupled with temporal evidence, are more persuasive than an unadjusted correlation matrix. Mixture analyses additionally require uncertainty in the single-compound reference curves to be carried into the expected combined response.

Environmental relevance and ecological meaning

The field application of *L. marginalis* should combine chemistry with biological context. Water, suspended particles or sediment, and mussel tissues should be sampled on a coordinated schedule. Sampling before, during and after runoff episodes can be more informative than a single seasonal grab sample. Reference sites need comparable habitat and water-quality conditions, not merely geographical separation from agriculture. Temperature, dissolved oxygen, food availability and other contaminants should be recorded as possible explanatory variables rather than ignored because the investigation focuses on two insecticides.

A residue–biomarker association in field animals is not sufficient to establish that one pesticide caused the observed injury. Stronger attribution can come from matched laboratory exposure, compatible tissue-residue ranges and recovery after removal from the contaminated setting. Even then, the conclusion should be scoped to the sampled populations and conditions. In particular, international occurrence studies should not be used as numerical substitutes for Indian freshwater monitoring, and

toxicity in a marine oyster should not be converted into a local freshwater risk threshold.

Ecological interpretation should move beyond the presence of stress markers. A study becomes more informative when it shows that cellular injury coincides with reduced filtration, impaired condition, altered reproductive performance or incomplete recovery. The mesocosm evidence linking mussel abundance with ecosystem processes gives a reason to measure these outcomes, but it does not establish the magnitude of pesticide-driven ecosystem loss. Population and ecosystem consequences require additional demographic or community-level observations rather than extrapolation from a single biochemical endpoint. [1]

The most defensible management implication is therefore to improve exposure characterisation and reduce avoidable pesticide entry into aquatic habitats while evidence develops. The reviewed laboratory studies do not establish a universal safe

concentration for *L. marginalis*, a food-safety threshold or the effectiveness of antioxidant supplementation as an environmental treatment. Such claims would require distinct exposure, residue and risk-assessment evidence beyond the scope of the present synthesis.

Research priorities and limitations

The main research gap is the absence, within the directly relevant studies examined here, of a complete chain linking measured water concentration, tissue burden, oxidative injury and impaired function in *L. marginalis*. The highest-value investigations would establish uptake and depuration for both parent compounds, determine whether metabolites contribute to internal exposure, and relate these measurements to a modest but well-validated biological panel. Table 4 translates these needs into testable questions and clearly separates proposed research from existing findings.

Table 4. Priority evidence gaps and experiments that would resolve them.

Unresolved question	Proposed approach	Decision enabled
How much pesticide reaches tissues?	Matched water–tissue measurements during uptake and depuration.	Estimate internal exposure rather than infer it.
Which route matters most?	Compare verified water-only exposure with controlled food/particle exposure.	Separate bioconcentration from multiple-route accumulation.
Does the parent compound explain injury?	Quantify parent and selected metabolites with biological endpoints.	Identify whether parent-only monitoring is sufficient.
Are oxidative changes adverse?	Combine defence markers with molecular injury, histology and function.	Distinguish compensation from damage.
Does the mixture depart from additivity?	Single-compound curves, multiple mixture ratios and explicit reference models.	Test interaction without presuming synergy.
Is recovery complete between pulses?	Repeated-pulse exposures with chemical and functional recovery tracking.	Identify persistent biological effects despite residue decline.

These are proposed research priorities. No experimental completion, numerical result or established species-specific threshold is implied.

Longer-term work should then address repeated pulses, recovery between exposures, reproductive condition and early life stages. These questions should not be answered by assuming that adult acute responses apply throughout the life cycle. Mixture investigations need both realistic environmental ratios and mechanistically informative combinations, with measured

internal concentrations wherever possible. Comparative studies across bivalve species would be particularly useful when they use the same exposure system, analytical method and endpoint definitions rather than relying on comparisons between unrelated protocols.

This review is limited by the uneven availability of full texts and the small

directly relevant evidence base. It does not prove that no additional *L. marginalis* residue study exists, and it does not provide a meta-analytic effect estimate. Reporting inconsistencies and incomplete exposure verification restrict numerical synthesis. Nevertheless, the evidence is sufficient to support a clear conclusion about research direction: the field needs stronger links between chemical measurement and biological response more urgently than it needs additional unconnected lists of altered biomarkers.

CONCLUSION

Lambda-cyhalothrin and imidacloprid present different physicochemical exposure profiles but share the potential to disturb biological processes in non-target bivalves. For *L. marginalis*, the directly relevant literature supports biochemical disruption and an oxidative-injury signal more strongly than it supports quantitative bioaccumulation. Tissue-residue kinetics and species-specific bioconcentration factors cannot be inferred from protein depletion, antioxidant changes or lipid peroxidation alone.

Evidence from other freshwater mussels and marine bivalves helps identify useful tissues, endpoints and toxicokinetic questions. It also demonstrates why uptake, damage and recovery should not be assumed to follow the same tissue ranking or timescale. Such comparative evidence is valuable when its taxonomic and environmental limits remain explicit. Similarly, mixture effects observed in fish justify targeted testing of the binary combination but do not establish synergy in the freshwater bivalve.

Future investigations should combine analytically verified exposure, parent-compound and metabolite measurements, uptake–depuration time courses, validated antioxidant and oxidative-damage assays, blinded histology and functional endpoints. Independent replication and appropriately scaled statistical models are essential. This integrated approach would allow *L.*

marginalis to contribute more reliably to freshwater pesticide assessment while preventing the common substitution of a biochemical stress response for demonstrated chemical accumulation.

Declaration by Authors

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