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Biomarkers of Oxidative Stress in Aquatic Organisms Exposed to Pollutants

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Abstract *— Oxidative stress biomarkers serve as sensitive early warning indicators of pollutant impacts on aquatic biota. This review critically examines the induction of reactive oxygen species by heavy metals, pesticides, polycyclic aromatic hydrocarbons, pharmaceuticals, and emerging contaminants, and the consequent responses of enzymatic antioxidants (superoxide dismutase, catalase, glutathione peroxidase, and glutathione S-transferase), non-enzymatic thiols, and damage products such as malondialdehyde and protein carbonyls. Species, tissue, and exposure regime dependent patterns are compared across fish, molluscs, crustaceans, and other taxa. Limitations of single-marker approaches, advantages of multi-biomarker batteries, and the integration of omics technologies are evaluated. The synthesis underscores the necessity of standardised protocols and contextual interpretation for reliable environmental risk assessment and monitoring.*

Keywords *— **oxidative stress, biomarkers, aquatic organisms, pollutants, antioxidant enzymes***

I. INTRODUCTION

Aquatic ecosystems across the globe are under increasing pressure from a wide range of anthropogenic pollutants that enter water bodies through industrial effluents, agricultural runoff, municipal wastewater discharges, atmospheric deposition, and accidental spills [1][38][39]. These contaminants, ranging from classical heavy metals and persistent organic pollutants to pharmaceuticals, personal care products, microplastics, and engineered nanomaterials, frequently disrupt the delicate redox balance within the cells of aquatic organisms [2][4][5]. The resulting condition, known as oxidative stress, arises when the production of reactive oxygen species (ROS) exceeds the capacity of endogenous antioxidant defence systems to neutralise them, leading to oxidative damage to lipids, proteins, and nucleic acids [1][2][40]. Because ROS are both unavoidable by-products of normal aerobic metabolism and potent signalling molecules, aquatic organisms have evolved sophisticated enzymatic and non-enzymatic antioxidant networks [21][22]. When pollutant-driven ROS generation overwhelms these networks, measurable alterations in biomarker profiles appear well before overt mortality or population-level declines become evident, rendering oxidative stress biomarkers powerful tools for early environmental assessment [3][4][38].

The conceptual and empirical foundation for using oxidative stress markers in aquatic toxicology was laid in the early to mid-2000s. Valavanidis and colleagues provided one of the first comprehensive molecular overviews, detailing how transition metals, polycyclic aromatic hydrocarbons (PAHs), organochlorine and organophosphate pesticides, polychlorinated biphenyls, and dioxins generate ROS and damage cellular macromolecules in aquatic species [4]. Shortly thereafter, Lushchak published a landmark

mechanistic synthesis examining environmentally induced oxidative stress across a broad range of aquatic animals, emphasizing the interactive roles of temperature, dissolved oxygen, salinity, and chemical pollutants [1]. These seminal contributions catalyzed an exponential increase in empirical studies quantifying superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione S-transferase (GST), reduced glutathione (GSH), and lipid peroxidation products, primarily malondialdehyde (MDA) or thiobarbituric acid reactive substances (TBARS), in fish, bivalves, crustaceans, and other taxa under both laboratory and field conditions [5][6][39][40].

Subsequent reviews refined the mechanistic picture and broadened the contaminant repertoire. Lushchak later classified oxidative stress by intensity and temporal dynamics and provided detailed contaminant-specific pathways in fish [5]. Meta-analyses of PAH-exposed fish demonstrated consistent induction of ethoxyresorufin O-deethylase (EROD) and GST, with more variable responses in SOD, CAT, and GPx [6]. Parallel studies on heavy-metal exposure documented tissue-specific and dose-dependent changes in the glutathione system and metallothioneins [7][8]. More recent literature has incorporated pharmaceuticals, microplastics, and nanomaterials, confirming that oxidative stress biomarkers remain responsive across chemically diverse stressors [9][10][11]. Collectively, these studies have established that oxidative stress biomarkers can detect sublethal effects at environmentally realistic concentrations and that their responses often precede histopathological lesions, behavioural alterations, or reproductive impairment [3][15][24].

Despite this substantial progress, several critical limitations and knowledge gaps persist. First, biomarker responses frequently exhibit non-monotonic or biphasic patterns that depend on exposure duration, concentration, species phylogeny, developmental stage, and tissue identity [5][7][18]. Second, laboratory results obtained under controlled, single-contaminant conditions often diverge quantitatively and sometimes qualitatively from field observations, where organisms experience complex chemical mixtures, fluctuating abiotic factors, and concurrent biological stressors such as pathogens or nutritional deficits [6][14][25]. Third, the majority of published studies still rely on a relatively narrow suite of classical enzymatic assays performed on tissue homogenates, while omics-derived markers, redox-sensitive transcription factors (Nrf2–Keap1, HIF 1α), and non-invasive sampling matrices remain underutilised in routine monitoring programmes [3][8][21]. Fourth, the ecological relevance of biomarker alterations, specifically their predictive power for individual fitness, population dynamics, and community structure, requires more rigorous validation through adverse outcome pathway frameworks and integrated field experiments [15][24][38].

This review therefore adopts a deliberately critical and comparative approach. After summarizing the fundamental biochemical mechanisms by which pollutants increase ROS production, it examines the principal classes of oxidative stress biomarkers in detail, presents comparative tables based on published quantitative data, evaluates methodological constraints and sources of variability, and outlines emerging multi-marker and molecular strategies. Emphasis is placed on analytical depth rather than exhaustive cataloguing, with the goal of furnishing environmental scientists, risk assessors, and regulators with a nuanced interpretive framework. The ultimate objective is to clarify both the strengths and the limitations of oxidative stress biomarkers so they can be applied more effectively to protect aquatic ecosystems under intensifying anthropogenic pressure [1][3][5].

II. FUNDAMENTAL MECHANISMS OF POLLUTANT-INDUCED OXIDATIVE STRESS

Reactive oxygen species arise primarily from the incomplete reduction of molecular oxygen within the mitochondrial electron transport chain, from membrane-bound NADPH oxidases, and from the redox cycling of certain xenobiotics [1][2][39]. The superoxide anion is the initial product; spontaneous or SOD-catalysed dismutation yields hydrogen peroxide, which, in the presence of reduced transition metals, participates in Fenton and Haber–Weiss reactions to generate the highly reactive hydroxyl radical [4][5][40]. Organic pollutants, particularly

PAHs and many pesticides, undergo cytochrome P450-mediated monooxygenation, producing superoxide and other radical intermediates as by-products of incomplete catalytic cycles [6][16]. Pharmaceuticals such as diclofenac and certain engineered nanomaterials can impair mitochondrial function or activate inflammatory pathways, further elevating intracellular ROS [8][10][11].

Once formed, ROS attack polyunsaturated fatty acids in membrane phospholipids, initiating a chain-propagating lipid peroxidation cascade that yields a cascade of reactive aldehydes, most notably MDA and 4-hydroxynonenal [4][29][31]. Protein amino acid side chains are oxidised to carbonyl derivatives, while DNA bases, especially guanine, form 8-oxo-7,8-dihydro-2′-deoxyguanosine [30][36]. These lesions compromise membrane fluidity and integrity, inactivate enzymes, and introduce mutations or strand breaks that threaten genomic stability [2][38]. At the organismal level, chronic oxidative damage manifests as reduced growth, impaired reproduction, immunosuppression, and increased susceptibility to secondary stressors [5][15][24].

Aquatic organisms counter these threats through a multi-layered antioxidant network. The enzymatic first line of defence comprises SOD (which converts superoxide to hydrogen peroxide), CAT (which decomposes hydrogen peroxide to water and oxygen), and the selenium-dependent and selenium-independent GPx enzymes (which reduce both hydrogen peroxide and organic hydroperoxides using GSH as a reductant) [1][21][22]. GST conjugates electrophilic xenobiotics and lipid peroxidation products with GSH, facilitating their excretion, while glutathione reductase regenerates GSH from glutathione disulfide at the expense of NADPH [4][9][22]. Non-enzymatic scavengers, such as ascorbate, α-tocopherol, carotenoids, and uric acid, provide additional protection, particularly in membranes and aqueous compartments [19][23][28]. When pollutant pressure exceeds the adaptive capacity of this network, the activities or concentrations of these antioxidants, as well as the levels of oxidative damage products, deviate from baseline values and thereby serve as quantifiable biomarkers of oxidative stress [5][7][18].

III. PRINCIPAL CLASSES OF OXIDATIVE STRESS BIOMARKERS

3.1 Enzymatic Antioxidants

SOD, CAT, GPx, and GST constitute the core enzymatic battery routinely measured in aquatic toxicology [1][4][5]. SOD activity often increases under moderate metal or pesticide exposure as cells attempt to limit superoxide accumulation; under severe or prolonged stress, however, the enzyme itself may undergo oxidative inactivation, producing a biphasic response [7][12][18]. CAT responses are notably variable across studies: induction is common under moderate hydrogen peroxide challenge, yet inhibition can occur when substrate concentrations are high enough to overwhelm the enzyme or when catalytic sites are damaged [6][13][16]. GPx and GST activities commonly increase in parallel with the elevated glutathione demand imposed by Phase II detoxification of both the parent pollutant and its reactive metabolites [6][9][15]. Tissue-specific differences are pronounced; hepatic or hepatopancreatic activities generally show the largest fold changes because of the high metabolic activity and xenobiotic processing capacity of these organs [14][25][28].

3.2 Non-Enzymatic Antioxidants and Cellular Thiols

GSH content and the GSH/GSSG ratio are sensitive indicators of cellular redox status [9][22]. Depletion of GSH often precedes measurable lipid peroxidation and is therefore regarded as an early biomarker of oxidative challenge [4][17][23]. Metallothioneins, cysteine-rich, low-molecular-weight proteins induced primarily by metal ions, also function as ROS scavengers and are frequently co-measured with classical antioxidants [7][14]. Ascorbate and tocopherol levels provide complementary information on non-enzymatic scavenging capacity, although their quantification is less common in routine surveys [19][28].

3.3 Oxidative Damage Products

MDA (or the broader TBARS assay) remains the most widely reported index of lipid peroxidation because of its analytical simplicity and clear mechanistic link to membrane damage [4][29][31][32]. Protein carbonyls offer a more stable and specific marker of protein oxidation, while 8 oxo 7,8 dihydro 2′ deoxyguanosine quantifies oxidative DNA lesions;

both require more specialised instrumentation and are therefore less frequently used in large-scale field programmes [30][36]. Lysosomal membrane stability and mitochondrial membrane potential assays provide functional endpoints that integrate multiple forms of oxidative injury at the organelle level [3][11][34].

IV. COMPARATIVE RESPONSES TO MAJOR POLLUTANT CLASSES

4.1 Heavy Metals

Heavy metal exposure consistently elevates lipid peroxidation and modulates antioxidant enzymes in a tissue, metal, and dose-dependent manner [7][12][14].

Table 1. Selected antioxidant and damage biomarker responses in fish exposed to heavy metals

Species	Metal / Exposure Regime	Tissue	SOD	CAT	GPx	MDA/ TBARS	Key Reference
Oreochromis niloticus	Cd, sublethal acute/chronic	Liver	↑	↑	↑	↑	[12]
Clarias batrachus	Pb, 10–30 % of LC_{50}, 60 d	Whole	↑	↑	–	↑	[13]
Indian major carps	Cu, Ni, Pb, Cd (field polluted)	Liver	↑	↑	Variable	↑	[14]
Multiple teleosts	Various metals (meta analysis)	Liver/ Gill	Generally ↑	Variable	↑	↑	[7]
Cyprinus carpio	Cr(VI), environmentally relevant	Liver	↑ then ↓	↑	↑	↑	[10][18]

Acute high-dose exposures often produce biphasic patterns (initial induction followed by inhibition), whereas chronic low-dose exposures tend towards sustained elevation of the glutathione system and metallothioneins [5][7][12]. Liver tissue almost invariably shows larger responses than muscle or gill tissue, reflecting its central role in metal sequestration and metabolism [14][25].

4.2 Pesticides

Organophosphate, organochlorine, carbamate, and pyrethroid pesticides induce oxidative stress both by directly generating ROS during metabolic activation and by depleting GSH during Phase II conjugation [15][16]. Meta-analytic evidence indicates robust elevation of GST and more variable changes in SOD and CAT [15][24]. Deltamethrin, for example, elevates MDA in the liver, kidney, and gills of catfish while depleting CAT reserves [16][29]. Atrazine and other herbicides commonly increase lipid peroxidation and upregulate SOD, CAT, and glutathione reductase in freshwater fish at sublethal concentrations [16][17].

4.3 Polycyclic Aromatic Hydrocarbons

PAHs induce EROD (a Phase I biotransformation marker) and GST most consistently; SOD and GPx also tend to increase, whereas CAT and GSH responses are frequently nonsignificant or highly variable [6]. Field and laboratory data converge on these directional patterns, although absolute fold changes are typically larger under controlled laboratory conditions than in complex field settings [6][25][26]. Exposure route (waterborne versus dietary) and habitat type (pelagic versus demersal) modulate the magnitude of certain responses, particularly for GST and GPx [6].

4.4 Emerging Contaminants

Pharmaceuticals (e.g., diclofenac, carbamazepine) and microplastics elicit tissue-specific modulation of antioxidants and elevated lipid peroxidation at environmentally realistic concentrations [8][11][27]. Metallic nanoparticles of silver, copper oxide, and titanium dioxide disrupt redox balance, frequently elevating SOD and MDA while depleting GSH and impairing lysosomal membrane stability [10][11]. Mixture exposures, which more closely approximate

real-world conditions, often produce synergistic or antagonistic interactions that cannot be predicted from single contaminant studies alone [11][15][24].

V. TAXONOMIC, TISSUE SPECIFIC, AND ENVIRONMENTAL PATTERNS

Fish remain the most intensively studied group due to their ecological importance, economic value, and relatively well-characterised physiology [5][7][14]. Nevertheless, bivalve molluscs (especially mussels of the genera Mytilus and Perna) and crustaceans display equally informative biomarker profiles and offer practical advantages for active biomonitoring programmes, including ease of transplantation and sessile or limited-mobility lifestyles [3][28][34]. Within an individual organism, the liver (or hepatopancreas in invertebrates) typically exhibits the strongest and most sustained antioxidant responses, reflecting its high metabolic rate and central role in xenobiotic processing [14][25][28]. Gills show rapid but often transient changes associated with direct waterborne exposure and high surface-to-volume ratios [13][29]. Muscle tissue generally responds more modestly and is therefore less useful for early warning, although it remains relevant for assessing the food safety implications of contaminant accumulation [14][32].

Abiotic factors interact strongly with chemical stressors. Temperature elevation increases metabolic rate and mitochondrial ROS production, amplifying pollutant-induced oxidative stress [1][19][20]. Hypoxia–reoxygenation cycles generate bursts of ROS upon reoxygenation [18][20][23]. Salinity fluctuations alter osmoregulatory energy expenditure and can modify metal speciation and bioavailability [19][28]. These interactions underscore the necessity of measuring concurrent physicochemical parameters when interpreting biomarker data from field surveys [1][3][14].

VI. METHODOLOGICAL CONSIDERATIONS, SOURCES OF VARIABILITY, AND LIMITATIONS

Despite decades of application, several methodological inconsistencies continue to limit the comparability of oxidative stress biomarker data across studies [3][6][15]. Units of enzyme activity are variously expressed per milligram of protein, per gram of wet tissue, or per milligram of haemoglobin, complicating quantitative meta-analysis [5][7]. Assay conditions (pH, temperature, substrate concentrations) are not fully standardised [4][40]. Seasonal, reproductive, and nutritional status of organisms can produce baseline fluctuations that rival or exceed contaminant-driven changes [14][25]. Laboratory-to-field extrapolation remains imperfect due to differences in exposure regimes, chemical mixtures, and the presence of additional stressors [6][26].

Single-biomarker approaches carry a high risk of false negatives when compensatory mechanisms mask damage or when the chosen marker is not responsive to the specific contaminant class under investigation [3][8]. Multi-biomarker batteries that combine enzymatic, non-enzymatic, and damage indices, ideally integrated with histopathological, behavioural, and molecular endpoints, provide substantially greater diagnostic power and reduce the likelihood of misinterpretation [3][15][24]. Non-lethal sampling matrices (blood, mucus, scales) are gaining traction but require further validation of their correspondence with internal tissue responses [8][27].

VII. ADVANCES, EMERGING TECHNOLOGIES, AND FUTURE DIRECTIONS

Transcriptomic and proteomic profiling of Nrf2-regulated genes, redox metabolomics, and high-throughput microplate or microfluidic assays promise to expand the biomarker repertoire beyond classical enzymes [3][8][21]. Real-time biosensors capable of detecting ROS or specific damage products in vivo, together with non-invasive imaging techniques, will facilitate repeated measures on the same individuals and reduce the need for destructive sampling [3][11]. Coupling biomarker data with population dynamic models and formal adverse outcome pathways will strengthen the ecological relevance of molecular and cellular observations [15][24][38]. International inter-laboratory calibration exercises, open access databases of baseline values for sentinel species across biogeographic regions, and the development of standardised operating procedures under the auspices of organisations such as the Organisation for Economic Co-operation and Development or the

International Council for the Exploration of the Sea are urgently needed to improve data quality and comparability [3][7].

Mixture toxicity remains a frontier. Most real-world exposures involve complex chemical cocktails whose interactive effects on redox homeostasis cannot be predicted from single-compound studies [11][15][24]. Experimental designs that incorporate environmentally realistic mixtures, pulsed exposures, and multi-stressor scenarios will improve the transferability of laboratory findings to field conditions [6][25][26]. Finally, the continued emergence of novel contaminants, increasingly complex nanomaterials, microplastic-associated additives, and pharmaceutical transformation products will require continuous revalidation of existing markers and the proactive development of new ones tailored to their unique modes of action [8][10][11].

VIII. CONCLUSION

Oxidative stress biomarkers occupy a central and well-justified position in contemporary aquatic toxicology because they capture the earliest cellular consequences of pollutant exposure and thereby provide a mechanistic bridge between chemical contamination and higher-level ecological effects. The extensive body of experimental and observational literature synthesised in this review demonstrates that superoxide dismutase, catalase, glutathione peroxidase, glutathione S-transferase, reduced glutathione, and malondialdehyde respond, often in coordinated and dose-dependent fashion, to heavy metals, pesticides, polycyclic aromatic hydrocarbons, pharmaceuticals, microplastics, and engineered nanomaterials across a wide phylogenetic spectrum of aquatic organisms. Comparative analyses reveal consistent directional trends in the induction of Phase II detoxification enzymes, elevation of lipid peroxidation products, and frequent modulation of first-line antioxidant enzymes yet also underscore substantial quantitative variation attributable to species identity, tissue type, developmental stage, exposure regime, co-occurring abiotic factors, and the chemical complexity of real-world mixtures. These sources of variability do not invalidate the biomarkers; rather, they demand rigorous contextual interpretation, transparent reporting of covariates, and the routine adoption of multi-marker batteries.

Mechanistically, the generation of reactive oxygen species proceeds through well-characterised routes, including redox cycling of transition metals, cytochrome P450-mediated activation of organic xenobiotics, mitochondrial dysfunction, and inflammatory cascades. Antioxidant defences counteract these insults at multiple levels of biological organisation, yet when overwhelmed they themselves become quantitative indicators of stress. The dual nature of many markers protective and inducible under mild challenge, inhibited or depleted under severe challenge explains the biphasic dose-response curves frequently observed and cautions against simplistic linear extrapolations from laboratory concentration–response relationships to field conditions.

Methodological refinement remains an ongoing necessity. Standardisation of analytical protocols, consistent expression of activities relative to protein content, inclusion of appropriate reference sites or laboratory controls, and explicit reporting of concurrent physicochemical and biological covariates will substantially reduce inter-study heterogeneity and improve the reliability of meta-analyses. Integration of classical enzymatic assays with omics-derived endpoints, redox-sensitive transcription factor activity, non-invasive sampling matrices, and functional organelle assays will further enhance both sensitivity and ecological relevance. Ultimately, the predictive value of oxidative stress biomarkers for environmental management will be fully realised only when they are embedded within formal adverse outcome pathway frameworks that explicitly link molecular initiation events to organismal performance, population dynamics, and community structure.

Looking ahead, the continued influx of novel and complex contaminants will require ongoing validation of existing markers and proactive development of new ones. Collaborative, open science initiatives that compile baseline data for sentinel species across geographic regions, together with structured inter-laboratory proficiency testing, will strengthen the reliability of biomarker-based monitoring programmes. Experimental designs that incorporate realistic exposure scenarios, including

pulsed, mixture, and multi-stressor regimes, will improve the transferability of laboratory findings to the field. When these advances are combined with improved mechanistic understanding and rigorous ecological validation, oxidative stress biomarkers will continue to serve as indispensable early warning tools for safeguarding water quality, biodiversity, and the ecosystem services on which human societies depend in an era of intensifying anthropogenic pressure on aquatic environments.

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