Emerging Antioxidants against Ferroptosis and their Catalytic Platforms in Redox Biology Network

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Abstract
This narrative review examines emerging antioxidant science through the framework of network redox biology. Rather than treating antioxidants as interchangeable radical scavengers, it emphasizes localization, regeneration, membrane access, metabolic support and disease context as determinants of biological relevance. The article focuses on ferroptosis as a turning point in antioxidant research because it links iron-dependent lipid peroxidation with membrane failure and highlights defense nodes such as GPX4-glutathione, FSP1-CoQ-NAD(P)H, GCH1-BH4 and sterol-related radical trapping. It also discusses melatonin-related antioxidant cascades, molecular hydrogen, mitochondrial and compartment-aware protection, catalytic nanozymes, mechanism-matched biomarkers and disease-oriented translation. Overall, the review argues that antioxidant value should be assessed by fit within a defined redox circuit rather than by a single in vitro assay value.

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1. Introduction

The classical definition of an antioxidant as a molecule that prevents oxidative damage remains useful, but it no longer captures the complexity of contemporary redox biology. Reactive oxygen and nitrogen species are not simply toxic by-products: hydrogen peroxide, nitric oxide and related species also operate as signals that regulate metabolism, proliferation, inflammation and adaptation [1-4]. Consequently, antioxidant intervention is not a matter of eliminating oxidation. It is a matter of preserving redox homeostasis while interrupting pathological chemistry. This distinction helps explain why many older supplementation strategies produced inconsistent clinical outcomes. Compounds with high activity in colorimetric assays may fail in tissues because they are not absorbed, do not reach the relevant compartment, are rapidly metabolized or suppress adaptive signaling [5, 6]. At the same time, new discoveries have renewed the field. Ferroptosis, organelle targeting, metabolite-centered defenses, molecular hydrogen and nanozyme catalysis have made antioxidant research more precise and more disease-oriented than before [2, 7-19]. The aim of this review was to analyze emerging antioxidants through the framework of redox biology network and to clarify how antioxidant relevance is determined by localization, regeneration, membrane access, metabolic support and disease context. The study further aims to highlight ferroptosis as a model mechanism for evaluating antioxidant defense, with particular attention to lipid peroxidation, GPX4-glutathione, FSP1-CoQ-NAD(P)H, GCH1-BH4, sterol-related radical trapping, melatonin-related antioxidant cascades, molecular hydrogen and catalytic nanozyme platforms. The author's studies on immune-support topics also show that common dietary factors may have measurable biochemical relevance [20-23].

2. Methods

The article review based on recent developments in antioxidant research, with emphasis on network redox biology. Relevant peer-reviewed literature was selected to represent both foundational concepts and recent advances in the field. The literature was evaluated according to mechanistic relevance rather than only chronological order. Foundational papers were included when they established key concepts such as oxidative stress, redox signaling, lipid peroxidation or ferroptosis. Experimental and review articles were considered together when they contributed to the interpretation of antioxidant function within defined redox circuits. The synthesis was organized around several thematic categories: classical antioxidant biology, ferroptosis-centered lipid peroxidation, metabolite and membrane-based antioxidant defenses, diffusible or cascade-based redox modulators, catalytic nanozyme systems, assay limitations and disease-oriented translation.

3. Practical implication of antioxidants

A practical implication is that antioxidant research should begin with the injury mechanism. If the dominant process is lipid-chain propagation, a membrane radical trap is logical; if the problem is peroxide signaling imbalance, enzyme systems and peroxiredoxin biology are more relevant; if the injury is acute and localized, targeted or catalytic platforms may be required. This system view first requires attention to the different reactive species and to the compartments in which they act. Redox biology is governed by kinetics and location. Superoxide, hydrogen peroxide, hydroxyl radical, lipid alkoxyl radicals, lipid peroxyl radicals, hypochlorous acid, nitric oxide and peroxynitrite differ greatly in half-life, diffusion radius and target preference [3-6]. A cytosolic reducing molecule may not protect a phospholipid bilayer, while a lipophilic radical trap may have limited influence on peroxide-mediated transcriptional signaling. This means that antioxidant classification must include compartment access. Lipid peroxidation is especially important because it links chemistry to membrane failure, inflammatory amplification and regulated cell death. Polyunsaturated phospholipids can undergo hydrogen abstraction, form lipid radicals and propagate chain reactions after reaction with oxygen. Once lipid hydroperoxides accumulate, downstream aldehydes and oxidized phospholipids may alter proteins, mitochondrial function and immune signaling. Therefore, the most relevant antioxidant is often the one placed at the vulnerable lipid interface rather than the one with the highest generalized reducing power [2, 24, 7]. This chemistry also explains why dose is not a simple variable. Low-level reactive species may promote adaptation, whereas excessive or misplaced oxidants can damage DNA, proteins and lipids. A useful antioxidant must therefore reduce pathological flux without flattening physiological signaling.

Against this mechanistic background, established enzymatic and small-molecule antioxidants provide the benchmark for evaluating newer strategies. Endogenous antioxidant enzymes remain the benchmark for judging newer systems. Superoxide dismutases convert superoxide into hydrogen peroxide; catalase, glutathione peroxidases and peroxiredoxins remove peroxides; and glutathione and thioredoxin systems provide reducing equivalents for detoxification and repair [3-6]. These systems illustrate two principles. First, antioxidant protection is usually catalytic or regenerable, not merely sacrificial. Second, the biological effect depends on substrate specificity and compartmental expression. Vitamin E, vitamin C and coenzyme Q also remain instructive. Tocopherols intercept lipid peroxyl radicals in membranes, ascorbate contributes to aqueous redox buffering and coenzyme Q participates in both bioenergetics and membrane antioxidant defense [24, 7]. What is new is not the existence of these molecules, but their reclassification within more detailed redox maps. For example, coenzyme Q is now viewed not only as an electron transport component but also as a substrate for GPX4-independent ferroptosis suppression through FSP1 [9, 10]. Classical antioxidants also provide a warning. Their historical reputation was built partly on elegant chemistry, yet clinical translation depends on pharmacokinetics and disease biology. Newer antioxidants should be held to the same standard rather than accepted because they are technologically novel.

Among recent developments, ferroptosis has most clearly shown why membrane chemistry must guide antioxidant design. Ferroptosis is an iron-dependent form of regulated cell death driven by lipid peroxidation [7, 8]. It has transformed antioxidant thinking because it identifies a specific chemical vulnerability: lethal propagation of oxidized polyunsaturated phospholipids. In this setting, antioxidants are not judged by a generic ability to reduce an artificial radical, but by their capacity to prevent membrane radical propagation, detoxify lipid hydroperoxides or remodel susceptible lipids. Ferroptosis-sensitive cells may be protected by several partially redundant nodes: GPX4 and glutathione, FSP1 and reduced coenzyme Q, GCH1-derived tetrahydrobiopterin (BH4), iron management, lipid remodeling and sterol-related radical trapping [7-13].

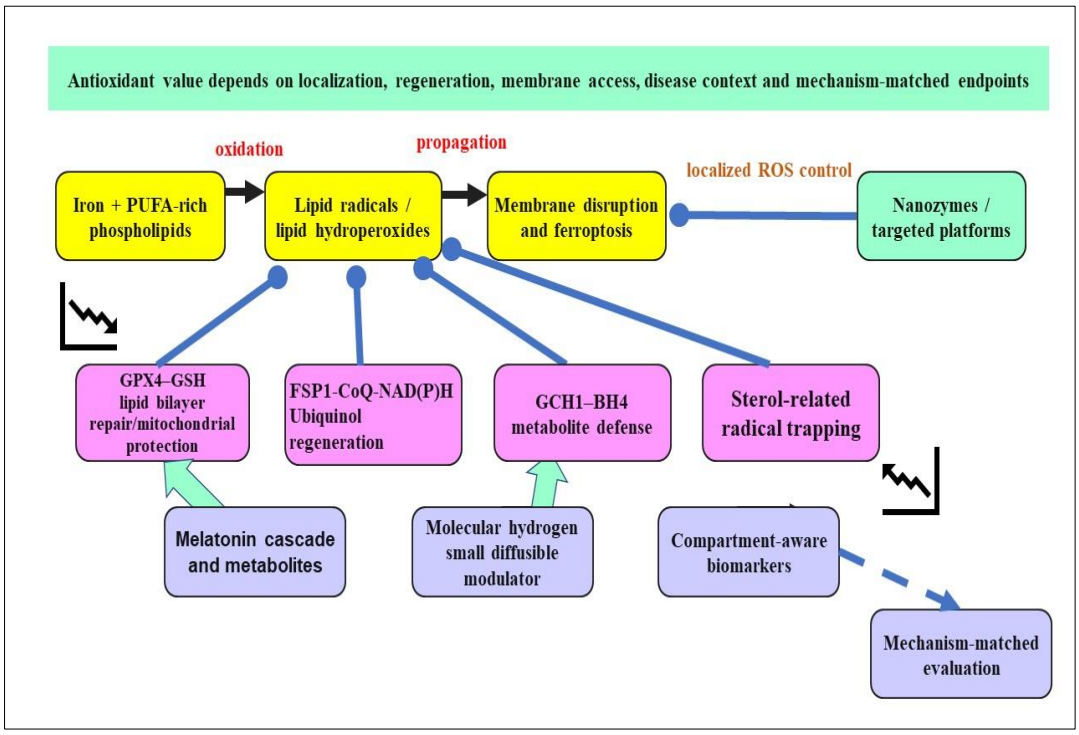

Fig 1: Network antioxidant defenses against ferroptosis-centered lipid peroxidation.

Ferroptosis is driven by iron-dependent oxidation of PUFA-rich phospholipids, leading to lipid radical formation, lipid hydroperoxide accumulation and membrane failure. Protective antioxidant defense is distributed across regenerable and compartment-specific nodes, including GPX4-glutathione lipid peroxide repair, FSP1–CoQ–NAD(P)H ubiquinol regeneration, GCH1-BH4 metabolite defense, sterol-related radical trapping, melatonin-related cascades, molecular hydrogen and catalytic nanozyme platforms. The scheme emphasizes that antioxidant value depends on localization, regeneration, membrane access, disease context and mechanism-matched biomarkers.

This architecture suggests that antioxidant defense is distributed across metabolism, membrane composition and enzyme activity. It also explains why inhibition of one pathway may be insufficient when another compensatory pathway remains active. Because ferroptosis is centered on phospholipid oxidation, it gives investigators a clearer experimental endpoint than broad oxidative stress. It encourages measurement of lipid hydroperoxides, oxidized phospholipid species and rescue by pathway-specific inhibitors rather than relying only on total antioxidant capacity. Within this ferroptosis framework, the FSP1-CoQ-NAD(P)H pathway illustrates how reductive metabolism is linked to membrane defense.

The discovery that FSP1 suppresses ferroptosis independently of GPX4 was a key advance [9, 10]. FSP1 uses NAD(P)H to reduce coenzyme Q, generating ubiquinol that can intercept lipid peroxyl radicals in membranes. The pathway connects cellular reductive metabolism to lipid-phase protection and provides a mechanistic explanation for GPX4-independent survival in some contexts. This axis is important translationally because it may be protective in degenerative or ischemic injury while also supporting resistance in certain cancers. A pathway that protects neurons, kidney cells or cardiomyocytes from lipid peroxidation may protect tumor cells from therapeutic ferroptosis induction. The FSP1-CoQ system therefore should be viewed as a context-dependent antioxidant defense rather than an unconditionally beneficial target [11, 13]. The dependence on NAD(P)H also links antioxidant defense to metabolism. Cells with adequate reductive capacity can regenerate protective ubiquinol more effectively, whereas metabolic exhaustion may convert a potentially protective membrane system into a vulnerable one. The same logic has also led to the rediscovery of metabolites and membrane components as conditional antioxidants. Tetrahydrobiopterin, traditionally known as a cofactor in aromatic amino acid hydroxylation and nitric oxide synthase biology, has also emerged as an anti-ferroptotic metabolite [11]. The GCH1-BH4 pathway can influence lipid remodeling and membrane resistance to oxidation. This is an example of metabolite rediscovery: a familiar biochemical molecule gains a new role when examined within a different disease mechanism. Sterol biology provides another example. 7-Dehydrocholesterol has been shown to affect ferroptosis sensitivity by functioning as a membrane radical-trapping component [12]. This finding reframes membrane composition itself as an antioxidant variable. Rather than asking only which antioxidant molecule should be administered, investigators must ask how lipid saturation, sterol intermediates, iron availability and repair systems jointly determine oxidative vulnerability. These examples support a broader principle: endogenous metabolites may act as conditional antioxidants when placed in the correct membrane or disease context. Their importance can be missed when antioxidant screening is restricted to isolated molecules in aqueous assays. Author studies on inflammation and corrective approaches further support the value of linking redox mechanisms with routine biochemical assessment [25, 26].

Beyond membrane-centered pathways, some emerging modalities act through metabolite relay or physicochemical diffusion. Melatonin is increasingly described as a redox-modulating system rather than only a pineal hormone. It can act through direct radical scavenging, mitochondrial

stabilization, regulation of antioxidant enzymes and formation of downstream metabolites such as cyclic 3-hydroxymelatonin, AFMK and AMK [14, 15]. The cascade concept is valuable because antioxidant capacity may continue through metabolite relay rather than being exhausted after one reaction. Molecular hydrogen occupies a different category. It is a small diffusible gas explored in formats such as inhalation and hydrogen-rich water. Reviews describe it as a selective redox modulator with possible activity against highly damaging oxidants and secondary effects on inflammation and mitochondrial function [16]. Its promise lies in diffusion and apparent safety, but interpretation remains challenging because protocols, endpoints and proposed mechanisms vary widely. Both examples demonstrate that emerging antioxidants do not always resemble conventional nutrients. Melatonin functions through a family of downstream molecules, whereas hydrogen is a physicochemical modality. Their evaluation requires mechanistic endpoints suited to their behavior. A further step beyond molecule-based supplementation is represented by catalytic materials designed to remove reactive species repeatedly.

Nanozymes represent one of the clearest departures from classical antioxidant supplementation. These engineered materials can mimic superoxide dismutase-, catalase-, peroxidase- or glutathione peroxidase-like activity and may repeatedly remove reactive species rather than being consumed stoichiometrically [17, 19]. Cerium oxide, platinum-based systems, manganese oxides, single-atom catalysts and hybrid biomimetic particles are examples of this design space. The appeal of nanozymes is threefold: catalytic turnover, tunable localization and multifunctionality. Surface chemistry and size can be engineered for tissue or microenvironment responsiveness, while one platform may combine antioxidant activity with drug delivery or imaging. However, translation requires attention to biodistribution, biodegradation, protein corona formation, immune interaction, manufacturing reproducibility and regulatory classification [17, 19].

For this reason, nanozyme papers should report not only ROS-removal activity but also persistence, clearance, active-site stability and interaction with biological fluids. Catalytic power without safety and reproducibility is insufficient for translation. These mechanistic distinctions make assay choice a central issue in evaluating antioxidant claims.

Assay selection is a frequent weakness in antioxidant research. DPPH (2,2-diphenyl-1-picrylhydrazyl), ABTS (2,2′-azino-bis(3-ethylbenzothiazoline-6-sulfonic acid)), FRAP (ferric reducing antioxidant power), ORAC (oxygen radical absorbance capacity) and related tests provide useful chemical comparisons but do not predict bioavailability, localization or disease effect [6]. A compound can be weak in a simple aqueous assay yet protective in a membrane model; conversely, a strong reducing agent can be irrelevant *in vivo* if it never reaches the target compartment. Mechanism-matched biomarkers are therefore essential. Ferroptosis-oriented studies should measure lipid hydroperoxides, oxidized phospholipids, 4-hydroxynonenal adducts, iron-related markers and cell death endpoints. Mitochondrial antioxidants require bioenergetic and compartmental readouts. Nanozymes require catalytic kinetics, stability and tissue distribution. Without such alignment, antioxidant claims remain vulnerable to overstatement.

A useful hierarchy is to combine chemical assays for initial screening, cell-based assays for biological plausibility, animal or organoid models for functional relevance and clinical biomarkers for translation. No single antioxidant number can replace this layered evidence. A mechanism-oriented summary of the major antioxidant strategies, biological contexts and recommended endpoints is presented in Table 1.

Table 1: Emerging antioxidant strategies and evaluation logic in ferroptosis-centered redox biology

Antioxidant strategy / platform	Main protective mechanism	Most relevant biological context	Suggested mechanism-matched endpoints
GPX4–glutathione system	Detoxifies lipid hydroperoxides and limits lethal membrane oxidation	Core anti-ferroptotic defense in cells vulnerable to lipid peroxidation	GPX4 activity, GSH/GSSG ratio, lipid hydroperoxides, ferroptosis rescue assays
FSP1–CoQ–NAD(P)H axis	Regenerates reduced coenzyme Q/ubiquinol to trap lipid peroxyl radicals independently of GPX4	GPX4-independent ferroptosis resistance; degenerative injury or cancer resistance	CoQ/$CoQH_2$ status, NAD(P)H availability, lipid ROS, sensitivity to FSP1 inhibition
GCH1–BH4 pathway	Supports tetrahydrobiopterin (BH4)-mediated antioxidant defense and lipid remodeling	Metabolite-based ferroptosis suppression	BH4 levels, GCH1 expression, lipid remodeling profile, oxidized phospholipid burden
Sterol-related radical trapping	Membrane sterol intermediates reduce ferroptosis sensitivity by radical-trapping activity	Membrane-composition-dependent oxidative vulnerability	Sterol profile, PUFA-phospholipid oxidation, lipidomics, ferroptosis sensitivity assays
Melatonin and metabolites	Direct scavenging, mitochondrial stabilization, antioxidant enzyme regulation and metabolite relay	Chronic mitochondrial stress, neurodegeneration, systemic redox modulation	Mitochondrial function, antioxidant enzyme activity, AFMK/AMK metabolites, oxidative damage markers
Molecular hydrogen	Small diffusible redox modulator with proposed activity against damaging oxidants and inflammation	Acute or compartmentally difficult oxidative injury; inflammatory and mitochondrial settings	ROS-linked biomarkers, inflammatory markers, mitochondrial readouts, standardized exposure protocol
Antioxidant nanozymes	Catalytic ROS removal through SOD-, catalase-, peroxidase- or GPX-like activity	Localized or repeated ROS excess; ischemia-reperfusion, inflammation, tissue injury	Catalytic kinetics, biodistribution, clearance, protein corona effects, tissue ROS markers
Mechanism-matched biomarker strategy	Aligns assay choice with injury chemistry instead of relying only on total antioxidant capacity	All translational antioxidant studies	Lipid hydroperoxides, oxidized phospholipids, 4-HNE adducts, iron markers, organelle-specific readouts

Mechanism-matched evaluation also determines how antioxidants should be interpreted across different disease settings. The modern field increasingly avoids the phrase oxidative stress as a universal therapeutic target. In neurodegeneration, antioxidant strategies must address mitochondrial dysfunction, lipid peroxidation, inflammation and protein aggregation without suppressing essential signaling [15]. In ischemia-reperfusion injury, timing is crucial because oxidant production can occur abruptly during reoxygenation. Catalytic materials, molecular hydrogen and anti-ferroptotic pathways may be more rational than slow nutritional interventions in that setting [16, 19]. Oncology is a more complicated field for antioxidants application, since antioxidant systems may protect normal tissues, but they may also protect malignant cells from oxidative killing. FSP1-CoQ, BH4-related defenses and sterol-mediated ferroptosis resistance can become therapeutic barriers when the goal is to induce tumor cell death. Thus, the same antioxidant pathway may be a protective target in degenerative disease and an inhibitory target in cancer. Disease-oriented thinking also prevents therapeutic overgeneralization. Oxidative stress in a retina, intestinal epithelium, ischemic myocardium or tumor microenvironment may involve different oxidant sources, lipid substrates and repair pathways, even when similar terminology is used. Taken together, these examples show why future work must treat antioxidant intervention as context-dependent rather than universally beneficial. Related clinical and laboratory work also shows that redox-linked interpretation should remain connected with practical disease endpoints [27, 31].

Three limitations should guide future work. First, antioxidant terminology remains too broad. A radical-trapping antioxidant, a Nrf2 activator, a hydrogen therapy, a nanozyme and a membrane remodeling pathway should not be ranked on a single scale. Second, novelty is sometimes overstated when in vitro scavenging is reported without absorption, metabolism or disease relevance. Third, indiscriminate antioxidant use can interfere with adaptive redox signaling [3, 6]. Future studies should integrate lipidomics, redox proteomics, compartment-specific probes and disease-specific functional endpoints. The strongest candidates will be those that match the dominant chemistry of a defined pathology: membrane chain-breaking activity for ferroptosis, fast localized activity for reperfusion injury, catalytic turnover for inflammatory ROS excess and metabolite relay for chronic mitochondrial stress. In conclusion, modern antioxidant science should move from asking which compound is strongest to asking where, when and in which redox circuit an intervention can safely restore homeostasis. The next stage of the field should integrate chemistry with systems biology. Uptake, localization, regeneration, sacrificial oxidation, feedback signaling and repair should be mapped together so that antioxidant interventions are designed as circuit-level corrections rather than isolated supplements.

4. Conclusion

Ferroptosis, lipid peroxidation, metabolite-based defenses, melatonin, molecular hydrogen and nanozyme platforms show that antioxidant relevance depends on localization, regeneration, membrane access, disease context and mechanism-matched biomarkers. Modern antioxidant science should evaluate compounds according to their place in specific redox circuits rather than by general radical-scavenging strength. Future research should focus on targeted, context-dependent interventions that restore redox balance without suppressing physiological signaling. This approach may improve translation from antioxidant chemistry to practical disease prevention and therapy.

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