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REVIEW ARTICLE

Neuroprotective Potential of Crustacean-Derived Astaxanthin: A Review on Monoamine Oxidase Inhibition

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ABSTRACT

Monoamine oxidases (MAO-A and MAO-B) are critical flavin-dependent enzymes anchored to the outer mitochondrial membrane, responsible for the oxidative deamination of biogenic amines; however, their catalytic activity generates neurotoxic by-products, including hydrogen peroxide and reactive aldehydes, which contribute to the pathogenesis of Alzheimer's and Parkinson's diseases. While the valorization of shrimp-processing by-products (accounting for 50–60% of total landing biomass) represents an important pillar of the circular bioeconomy, their principal bioactive carotenoid, astaxanthin (AST), is widely investigated for its neuroprotective and antioxidant attributes. This critical review evaluates the mechanistic pathways through which AST mitigates neurotoxicity, with an analytical focus on its proposed, isoform-selective interactions with monoamine oxidases. Current computational docking evidence suggests that unesterified AST possesses spatial compatibility with the substrate-binding cavity of MAO-A, whereas steric hindrance prevents its accommodation within the narrow, bipartite channel of MAO-B. However, direct experimental verification such as recombinant enzyme kinetics, K_i and IC_{50} determinations, and cellular inhibition assays remains largely absent in the literature. Furthermore, native crustacean extracts predominantly consist of fatty-acid-esterified stereoisomers rather than the unesterified free form modeled in silico, presenting structural differences that could influence active-site entry. This review outlines the literature retrieval methodology, critically appraises verified downstream neuroprotective mechanisms (including Nrf2/HO-1 upregulation, lipid peroxidation attenuation, and mitochondrial membrane stabilization), and contrasts theoretical binding predictions against established empirical benchmarks. Existing literature gaps, discrepancies between purified standards and crude by-product extracts, and the empirical validations necessary before AST can be considered a reliable MAO-modulating candidate are discussed.

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1. Introduction

Monoamine oxidases (MAO-A and MAO-B) are flavin adenine dinucleotide (FAD)-dependent enzymes anchored to the outer mitochondrial membrane and responsible for the oxidative deamination of biogenic amines such as serotonin, dopamine, norepinephrine, and tyramine. Their catalytic cycle generates the corresponding aldehyde, ammonia, and hydrogen peroxide; consequently, MAO activity is biologically critical not only for maintaining neurotransmitter homeostasis but also for its pivotal role in the generation of oxidative stress

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(Edmondson et al., 2009). MAO-A and MAO-B exhibit significant sequence similarity but differ in their inhibitor-access pathways; this difference explains their distinct substrate preferences and inhibitor selectivity. MAO-B activity is particularly emphasized in neurodegenerative research due to its association with dopamine catabolism and the age-related increase in oxidative burden. Conversely, MAO-A is primarily linked to serotonin metabolism and to the broader catecholamine cycle. The findings from Fowler's laboratory—demonstrating that MAO-B levels in humans increase approximately 4- to 5-fold with aging—have also been observed in rats, providing a strong rationale for the role of MAO-B in age-related neurological disorders such as Parkinson's disease (Mallajosyula et al., 2008). An increase in MAO-B levels is expected to deplete dopamine concentrations while elevating the levels of its catalytic reaction products, specifically 3,4-dihydroxyphenylacetaldehyde (DOPAL) and H_2O_2. DOPAL has been closely linked to the aggregation of α-synuclein, a key factor in the etiology of Parkinson's disease (PD) (Burke et al., 2008). Furthermore, elevated cellular levels of H_2O_2 trigger apoptotic signaling pathways, leading to the depletion of dopaminergic neurons and the subsequent progression of PD (Mallajosyula et al., 2008). Recent studies have established that MAO-A inhibitors exhibit significant antidepressant activity, while selective MAO-B inhibitors have increasingly been utilized in the treatment of patients with Alzheimer's disease (AD) (Akwu et al., 2023; He et al., 2026; Sun et al., 2022; Zhang et al., 2021; Zhong et al., 2024). Consequently, targeting monoamine oxidases offers a recognized avenue for mitigating neurodestructive cascades.

While MAO-A functions primarily in the oxidative metabolism of serotonin, it also oxidizes dopamine. The complete elimination of MAO-A activity through genetic deletion in mice has been shown to result in the emergence of aggressive behaviors (McDermott et al., 2009). Notably, MAO-A levels have been found to increase significantly (~9-fold) in the hearts of aged rats, suggesting a potential age-related increase in MAO-A expression in humans as well (Maurel et al., 2003).

Carotenoids are secondary metabolites produced through enzymatic activity (Lotfi et al., 2024). Marine animals accumulate these compounds either directly from their diet or by partially modifying them through specific metabolic pathways (Chuyen & Eun, 2017; Maoka et al., 2011). Based on their core structural groups, they are classified into two main categories: carotenes and xanthophylls (Maoka, 2020; Meléndez-Martínez et al., 2022). While carotenes consist solely of carbon and hydrogen atoms, xanthophylls contain additional oxygen atoms within various functional groups, such as hydroxyl and keto groups (Elbahnaswy & Elshopakey, 2024). Carotenoids possess a polyene chain that confers their primary therapeutic specifically antioxidant properties; furthermore, due to their lipophilic nature, they are capable of crossing the blood-brain barrier (BBB) (Katayama et al., 2011; Sachdeva & Chopra, 2015). Examples of carotenes include α-carotene, β-carotene, γ-carotene, and lycopene; xanthophylls are exemplified by fucoxanthin, lutein, violaxanthin, and astaxanthin (González-Peña et al., 2023).

Astaxanthin (AST), an oxygenated carotenoid derivative and keto-carotenoid belonging to the xanthophyll family, is characterized by terminal rings bearing hydroxyl (–OH) and keto (=CO) functional groups at each end of its molecule (Fakhri et al., 2018). These terminal rings, along with the intervening unsaturated polyene chain, are capable of scavenging free radicals on both sides of the cell membrane (Goto et al., 2001; Lotfi et al., 2024). In crustaceans, it can exist in an unesterified (free) form or esterified with various fatty acids as mono- and diesters (Higuera-Ciapara et al., 2006). The structural state of AST exerts a significant influence on its bioavailability; consequently, in-vitro investigations into the stability and absorption of different AST derivatives (free, mono-, and diesters) have highlighted that its chemical configuration is a critical determinant of its overall efficacy (L. Yang et al., 2021).

Since the discovery of its superior biological activities in the 1990s, AST has been recognized as a significant functional ingredient (Cao et al., 2023). Currently, AST is widely utilized worldwide as a nutraceutical, food additive, pharmaceutical, and cosmetic component (Liu et al., 2023). The primary biological sources for AST synthesis include green microalgae (*Haematococcus pluvialis* and *Chlorella zofingiensis*), red yeast (*Xanthophyllomyces dendrorhous*, also known as *Phaffia rhodozyma*), marine bacteria (*Agrobacterium aurantiacum* and *Paracoccus carotinifaciens*), and certain plants of the *Adonis* genus (*Adonis aestivalis*, *Adonis amurensis*, and *Adonis annua*) (Galasso et al., 2018; Patil et al., 2022) (Table 1). In commercial AST production, *H. pluvialis* is the predominant microalgal species, while *X. dendrorhous* and *P. carotinifaciens* serve as the primary sources of red yeast and marine bacteria, respectively.

Among marine animals, the species that most significantly accumulate AST through the food chain are krill, rainbow trout, salmon, shrimp, lobster, and fish roe. Consequently, these seafood species represent vital natural sources of AST in the human diet (Cao et al., 2023; Catanzaro et al., 2020; Dikel, 2021; Galasso et al., 2018; Mezzomo & Ferreira, 2016). Shrimp, a primary natural source of AST, is characterized by relatively low meat yields, resulting in approximately 50–60% of the organism being discarded as waste (Sae-Leaw & Benjakul, 2019). The processing of crustaceans such as shrimp, crabs, and lobsters generates significant by-products, including heads, exoskeletons, and appendages (Šimat, 2021). The recovery of high-value compounds, such as AST, from these by-products facilitates the development of novel functional ingredients and contributes to the sustainability of the sector.

Furthermore, this approach provides substantial environmental benefits by reducing the organic load of processing waste (Dikel & Yanar, 2025).

A variety of techniques can be employed to extract AST; however, emerging methods such as ultrasound-assisted extraction (UAE) and microwave-assisted extraction (MAE) offer significant advantages, including reduced solvent consumption, shorter extraction durations, and enhanced extraction yields (Chi Hai et al., 2025; Dikel & Yanar, 2025).

AST exhibits a broad spectrum of therapeutic activities, including anticancer, antidiabetic, anti-inflammatory, and antioxidant properties, while exerting protective effects on the neurological, cardiovascular, ocular, and cutaneous systems (Yuan et al., 2011). Regarding its neuroprotective potential, numerous studies have investigated the role of AST in various neurological disorders such as Parkinson's disease (PD), Alzheimer's disease (AD), autism, and neuropathic pain as well as its potential applications in managing neuroinflammatory diseases (Bahbah et al., 2021; S. Wang & Qi, 2022).

Table 1. Natural sources, molecular forms, and bio-accessibility characteristics of AST.

Source Type	Primary Biological Sources / Species	Predominant Molecular Form	Bioavailability & Extraction Remarks	References
Microalgae	*Haematococcus pluvialis*, *Chlorella zofingiensis*	Monoesters & Diesters	High stability, main commercial source for human nutraceuticals.	Galasso et al. (2018); Patil et al. (2022)
Marine Crustaceans (Waste)	Shrimp (*Litopenaeus vannamei*, *Parapenaeus longirostris*), Krill, Crab	Free form (~10–15%), Monoesters (~70%), Diesters	High-volume organic waste (50–60% biomass); requires green extraction (UAE/MAE).	Dikel and Yanar, (2025); Sae-Leaw and Benjakul, (2019)
Yeasts & Bacteria	*Xanthophyllomyces dendrorhous*, *Paracoccus carotinifaciens*	Free (Unesterified) form	High metabolic yield but distinct functional profile compared to marine esters.	Cao et al. (2023); Liu et al. (2023)
Marine Fish	Salmon, Rainbow Trout, Fish Roe	Accumulated via diet (from crustaceans)	Essential dietary source; responsible for the pinkish pigmentation of muscle tissues.	Catanzaro et al. (2020); Dikel (2021)

1.1. Structure, Properties of Natural AST from Shrimp Shell By-Products

Structurally, natural AST (3,3'-dihydroxy-β,β-carotene-4,4'-dione) is characterized by a central conjugated polyene chain flanked by two terminal β-ionone rings containing hydroxyl (-OH) and carbonyl (C=O) functional groups, which confer a significantly higher antioxidant capacity compared to common carotenoids (Johnson & An, 1991). In contrast to its synthetic counterpart that exists as a racemic mixture, AST sourced from crustacean processing by-products is predominantly the 3S, 3'S stereoisomer and occurs largely esterified with long-chain omega-3 polyunsaturated fatty acids. Utilizing green extraction methodologies to isolate this delicate pigment without inducing thermal or photo-oxidative degradation of its eleven conjugated double bonds is of paramount importance for the circular bioeconomy.

Advanced green techniques such as ultrasound-assisted extraction (UAE), microwave-assisted extraction (MAE), supercritical fluid extraction (SFE), enzyme-assisted extraction, and high-pressure extraction disrupt the rigid crustacean exoskeleton matrix, thereby minimizing extraction time while optimizing yield and purity (Dikel & Yanar, 2025; Dong et al., 2014; Sharayei et al., 2021). In the crustacean processing sectors, substantial volumes of by-products are discarded as waste, causing heavy organic pollution loads; shrimp processing by-products, in particular, hold immense promise for sustainable, natural AST recovery. Upcycling these seafood processing by-product waste streams as the primary feedstock for marine-derived AST mitigates organic pollution and ecological impacts while fostering the responsible, sustainable management of marine ecosystems, reducing reliance on primary wild organisms as industrial raw materials and fully aligning with global sustainability targets (Ali et al., 2023; Panagiotakopoulos & Nasopoulou, 2024; Rossi et al., 2024).

However, it is critical to distinguish between purified AST analytical standards and crude or semi-purified AST-rich extracts obtained from crustacean processing waste. Crude shrimp shell extracts contain complex mixtures of free AST, monoesters, diesters, co-extracted lipids, and minor carotenoids. These associated matrix components can significantly alter bioavailability, stability, and steric interactions with enzymatic binding pockets compared to isolated pure AST.

1.2. AST's Potential to Reduce Oxidative Stress

Highly reactive oxidative molecules, such as free radicals or Reactive Oxygen Species (ROS), induce cellular damage through reactions with various regulatory molecules, leading to diverse physiological disorders (Šimat et al., 2022). These oxidative reactions can be effectively inhibited by the administration of endogenous and exogenous antioxidants.

AST exhibits its potent antioxidant activity through multiple sophisticated mechanisms, including the absorption of free radicals into its polyene chain, electron donation, or the formation of stable chemical bonds with reactive species (Grimmig et al., 2017). Structurally, the compound features terminal rings containing hydroxyl and keto groups; these rings, along with the intervening unsaturated polyene chain, allow AST to scavenge free radicals on both sides of the cell membrane (Lotfi et al., 2024). This structural and mechanistic versatility underscores AST's potential as a superior therapeutic agent compared to other carotenoids.

1.3. Synthetic AST: Historical Context and Regulatory Limitations

Although synthetic AST can be produced through complex, multi-step processes involving petrochemicals, significant safety and toxicity concerns have emerged regarding the long-term use of such artificially manufactured supplements (Abdol Wahab et al., 2022; Capelli et al., 2013). The first synthetic AST was introduced in Switzerland in the early 1980s by Hoffmann-La Roche under the brand name CAROPHYLL Pink (C®P) (Stachowiak & Szulc, 2021). Since 1995, the FDA has approved synthetic AST exclusively for the aquaculture industry as a fish feed additive, with a maximum dosage limit of 100 mg/kg in the ration (Stachowiak & Szulc, 2021). Furthermore, as specified in the European Union Commission Implementing Regulation (EU) 2020/998, AST dimethyl succinate has been authorized as a feed additive for fish and crustaceans until July 2030, maintaining a maximum dosage of 100 mg/kg.

1.4. Bioavailability and Safety of Natural AST

AST is recognized as a safe food additive with no known adverse side effects. The United States Food and Drug Administration (FDA) has approved the use of AST as a food colorant in animal feeds, and the European Commission has similarly authorized natural AST as a food colorant (Ambati et al., 2014). While early regulatory opinions discussed conservative supplementary doses, the European Food Safety Authority established an acceptable daily intake of 0.2 mg AST /kg body weight per day, considering total exposure from food supplements and background dietary sources.

Despite being a bioactive compound of immense importance for human health due to its extraordinary antioxidant properties and associated benefits, AST presents challenges such as low water solubility, chemical instability, and sensitivity to light, oxygen, and heat. Therefore, selecting an appropriate formulation and administration route is critical to preserving or enhancing its therapeutic and functional potential. Depending on the intended use, AST can be administered in various forms, including capsules, softgels, tablets, powders, creams, and extracts (Novelli et al., 2026). Furthermore, the development of innovative delivery systems such as liposomes, nanoparticles, and nanocarriers has significantly improved the ability to overcome challenges like low bioavailability, instability, and poor targeting efficiency associated with bioactive compounds, including AST and β-carotene (Jia et al., 2025; Pourmadadi et al., 2024).

Furthermore, in a comprehensive review conducted by Donoso et al. (2021), it was reported that natural AST demonstrated no safety concerns regarding human consumption across various studies focused on skin health, ocular health, cardiovascular health, neurological health, and immune response. To enhance its bioavailability, it is recommended to consume AST in conjunction with omega-3-rich seed oils, such as flaxseed, chia, and walnut oils (Mohd Shafie et al., 2025).

1.5. Pharmacokinetics and Blood-Brain Barrier (BBB) Permeability

Natural AST, a xanthophyll ketocarotenoid, garners significant clinical interest due to its favorable pharmacokinetic profile and inherent capacity to traverse the blood-brain barrier (BBB). Distinct from pure hydrocarbon carotenoids such as β-carotene, the molecular structure of AST features symmetrically positioned polar hydroxyl and keto functional groups on its ionone rings. This amphiphilic architecture enables the molecule to align across the lipid bilayer, anchoring its polar terminal moieties at the membrane interface while positioning its non-polar polyene chain within the hydrophobic core.

This structural polarity and lipophilicity facilitate passive diffusion through the tight junctions of the BBB without necessarily requiring specialized nanocarrier delivery systems. Preclinical in vivo studies demonstrate that the oral administration of natural AST results in its functional accumulation within critical brain regions, including the cerebral cortex and the hippocampus (Tsuji et al., 2020). Upon penetrating the central nervous system, AST exerts a multifaceted neuroprotective effect by preserving the cellular structural integrity of the microvasculature, upregulating the Nrf2-mediated antioxidant cascade, and attenuating NF-κB-driven neuroinflammation (X. Wang et al., 2024). Consequently, this structural permeability establishes natural AST as a promising candidate for mitigating oxidative stress in ischemic injury and chronic neurodegenerative models (Figure 1).

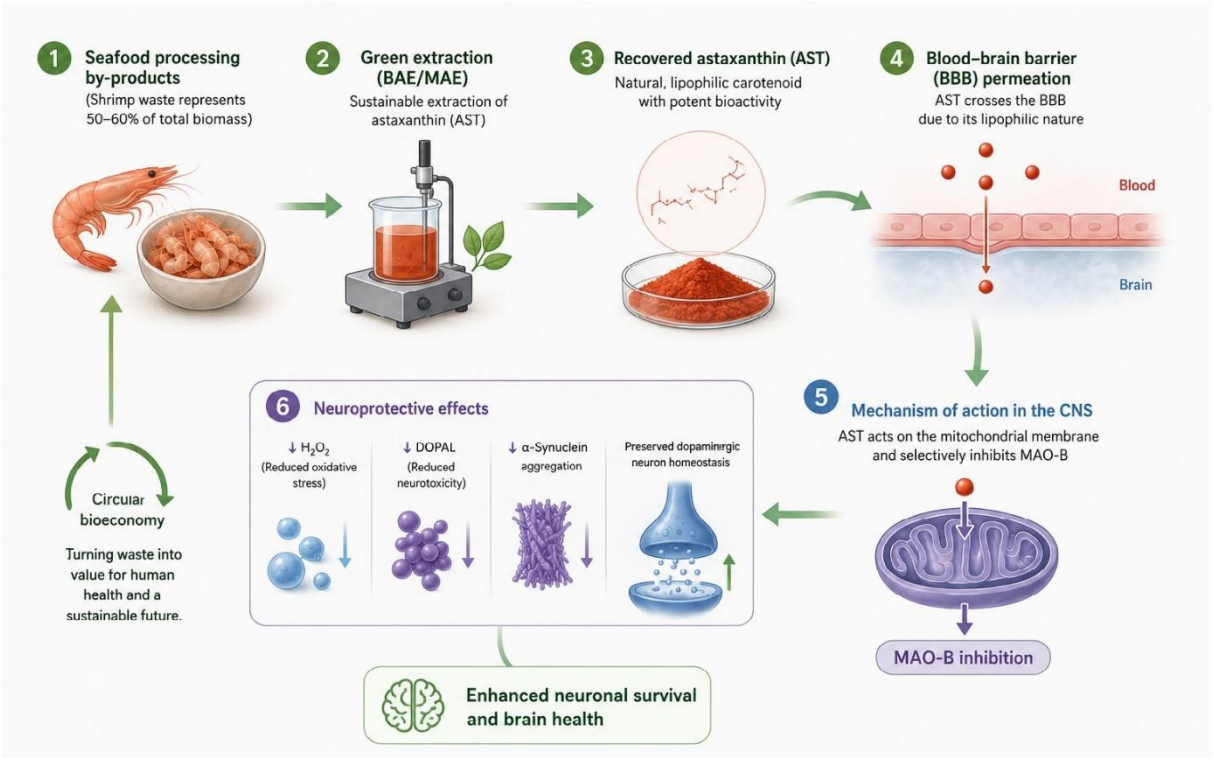


Figure 1. A schematic representation of the potential applications of recycled AST, derived from seafood processing by-products, in neuronal health through a circular process. This illustration highlights the conversion of biological waste into high-value AST using sustainable extraction technologies. It also outlines how AST can enhance antioxidant defences, reduce oxidative stress and neurotoxicity, and support neuronal homeostasis.

2. Molecular Mechanisms of Robust Neuroprotection Mediated by AST

At the subcellular level, the multifaceted efficacy of natural AST stems from the modulation of interconnected redox-sensitive transcription networks, microglial inflammatory axes, and mitochondrial homeostatic pathways. Central to its antioxidant capacity is the activation of the PI3K/Akt-Nrf2 signaling cascade. Upon traversing the blood-brain barrier (BBB), AST facilitates the dissociation of nuclear factor erythroid 2-related factor 2 (Nrf2) from its cytoplasmic inhibitor, Keap1. This dissociation promotes the nuclear translocation of Nrf2 and its subsequent binding to Antioxidant Response Elements (ARE), leading to the upregulation of Phase II cytoprotective enzymes, including heme oxygenase-1 (HO-1), NAD(P)H quinone oxidoreductase 1 (NQO1), and superoxide dismutase (SOD) (Rambaldi et al., 2025; X. Wang et al., 2024).

Concurrently, AST attenuates the nuclear translocation of the p65/p50 NF-κB heterodimer in activated microglia by preventing the phosphorylation and subsequent degradation of the inhibitor of κB (IκB) subunit. This molecular suppression downregulates the transcription of inducible nitric oxide synthase (iNOS), cyclooxygenase-2 (COX-2), and key pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6 (Singh et al., 2026).

2.1. Isotype-Selective MAO Inhibition

Upon penetrating the central nervous system, AST exerts a targeted regulatory effect on monoamine oxidases (MAOs) bound to the outer mitochondrial membrane. This enzyme exists in two distinct isoforms, designated as MAO-A and MAO-B, which exhibit substantial architectural variations in their substrate-binding pockets. Investigations into the steric hindrance profiles reveal that the active site of MAO-B is composed of a rigid, narrow, bipartite hydrophobic cavity. Due to these structural constraints, the bulky terminal ionone rings and the elongated polyene chain of AST undergo severe steric clashes within this region, effectively restricting the molecule from accessing the catalytic center (Safarova et al., 2016).

In stark contrast, the MAO-A isoform possesses a significantly wider, single hydrophobic active cavity. Structural modeling demonstrates that AST seamlessly accommodates into the substrate-binding pocket of MAO-A, occupying the identical spatial domain utilized by endogenous inhibitors such as isatin (Safarova et al., 2016). This isoform-specific spatial compatibility underscores the potential of AST to act as a selective MAO-A modulator, minimizing non-specific enzymatic disruption within mitochondrial pathways.

2.2. Therapeutic Implications in Neurodegenerative Pathways

Natural AST exhibits documented, multi-target cytoprotective actions across neurodegenerative experimental models (Fakhri et al., 2019). While computational models propose that AST could hypothetically reduce the generation of catalytic MAO-A by-products (Safarova et al., 2016), its experimentally confirmed neuroprotective actions occur downstream at the mitochondrial and nuclear levels.

By mitigating general oxidative cascades, AST facilitates the nuclear translocation of Nrf2 to upregulate critical cytoprotective enzymes such as heme oxygenase-1 (HO-1) and NAD(P)H: quinone oxidoreductase-1 (Gao et al., 2021). Furthermore, in cellular models of Parkinson's disease challenged with MPTP/MPP^+, AST administration limits mitochondrial impairment and apoptotic signaling by bolstering anti-apoptotic Bcl-2 expression, suppressing pro-apoptotic Bax, and halting the terminal cleavage of caspase-3 (Lee et al., 2011).

2.3. Reducing Oxidative Stress and Mitochondrial Dysfunction

Under neurodegenerative stress, elevated mitochondrial ROS production compromises organellar integrity through a pathological interplay between oxidative stress and mitochondrial dysfunction, ultimately driving apoptotic cell death (Yu et al., 2024). Natural AST can mitigate this damage by directly integrating into the mitochondrial membrane bilayer, where it functions as a physical membrane stabilizer (Kim & Kim, 2018; Kuroki et al., 2013). Its molecular architecture spans the lipid bilayer: the ionone rings anchor to the hydrophilic head groups, while the conjugated polyene chain resides within the hydrophobic core. This transmembrane orientation physically retards lipid peroxidation, stabilizes both the inner and outer membranes, and shields the mitochondrial membrane potential against free radical attacks (P. Wang et al., 2023).

In pathological states, sustained mitochondrial ROS generation triggers cardiolipin peroxidation and the opening of the mitochondrial permeability transition pore (mPTP). Direct antioxidant intervention by AST maintains the organelle in a reduced state, mitigating intramitochondrial Ca^{2+} overload and preventing the pro-apoptotic release of cytochrome c into the cytoplasm (Wolf et al., 2010). Beyond direct physical and chemical shielding, AST functions as a modulator of gene expression by facilitating the dissociation of Nrf2 from Keap1, promoting its nuclear translocation via the PI3K/Akt signaling axis to upregulate downstream Phase II enzymes (J. Yang et al., 2021).

3. Current Challenges and Future Perspectives

Despite promising preclinical evidence detailing the antioxidant and neuroprotective efficacy of natural AST, translating these findings into clinical therapies faces substantial hurdles. Due to its highly lipophilic polyene chain, non-crystalline AST exhibits poor solubility in aqueous gastrointestinal fluids, resulting in low oral bioavailability (Jafari et al., 2021). Furthermore, its conjugated double-bond system renders the pigment susceptible to thermo-oxidative degradation, light-induced cleavage, and trans-to-cis isomerization during processing and gastrointestinal transit (Jafari et al., 2021).

To address these limitations, encapsulating natural AST within nanotechnology-assisted delivery vehicles such as solid lipid nanoparticles (SLNs), nanostructured lipid carriers (NLCs), polymeric micelles, and biopolymer-based hydrogels improves stability, enhances aqueous dispersibility, and amplifies transcellular permeability across biological barriers (You et al., 2019). In future therapeutic research, AST should be evaluated not as a standalone enzymatic blocker, but as a multifunctional dietary or adjunct entity capable of supporting mitochondrial integrity via the Nrf2 axis and attenuating neuroinflammatory cascades (Fakhri et al., 2019).

Importantly, future investigations into shrimp waste valorization must move beyond simple yield determinations. Standardized protocols are required to chemically characterize recovered extracts quantifying the ratios of free astaxanthin to monoesters and diesters and directly test them in cell-free recombinant MAO-A and MAO-B enzymatic kinetic assays. Only through empirical kinetic determinations IC_{50}, K_i) and reversibility testing can the proposed interactions between waste-derived astaxanthin and monoamine oxidases be definitively confirmed or refuted.

4. Conclusion

AST potentially recoverable from crustacean processing by-products represents a valuable bioactive compound within circular bioeconomy and marine valorization frameworks. In preclinical models of neurodegeneration, AST exhibits cytoprotective actions, including the suppression of reactive oxygen species, upregulation of the Keap1-Nrf2-ARE antioxidant axis, inhibition of microglial NF-κB inflammatory signaling, and preservation of mitochondrial membrane potential against MPTP/MPP^+ neurotoxicity. Characterized by its polar-nonpolar-polar structure, this xanthophyll carotenoid efficiently traverses the BBB via transcellular passive diffusion to function as a highly selective, reversible inhibitor of MAO-A, thereby eliminating the severe hypertensive crises commonly associated with synthetic, irreversible MAO inhibitors. While preserving synaptic serotonin pools within critical brain regions, AST simultaneously minimizes baseline mitochondrial H_2O_2 generation. Consequently, by dual-

coupling MAO inhibition with the simultaneous mitigation of mitochondrial dysfunction and oxidative stress, natural AST stands out as a sustainably sourced, multifaceted therapeutic candidate for the management of complex neuropsychiatric and neurodegenerative disorders.

Compliance with Ethical Standards

This study does not require ethical committee approval.

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Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Disclosure of Generative AI Use

No generative artificial intelligence (AI) tools were used in the writing of this manuscript.

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