Review

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A Potential Role of Natural Bioactive Compounds Found in Food in the Prevention of Idiopathic Parkinson's Disease

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Abstract

The molecular mechanism behind the loss of neuromelanin-containing dopaminergic neurons in the nigrostriatal system in idiopathic Parkinson's disease remains unclear. However, research suggests that aminochrome, an endogenous neurotoxin, may trigger the degeneration of these neurons through a single-neuron degeneration model. In this model, aminochrome selectively destroys individual neurons without spreading to neighboring cells. Aminochrome is produced during neuromelanin synthesis, a process that is normally harmless because protective enzymes like DT-diaphorase and glutathione transferase M2-2 neutralize aminochrome's neurotoxic effects. Increasing the levels of these enzymes could offer neuroprotection. The KEAP1/NRF2 signaling pathway plays a crucial role in regulating antioxidant enzymes, including DT-diaphorase and glutathione transferase M2-2. Notably, certain dietary bioactive compounds can activate the KEAP1/NRF2 pathway, enhancing the production of these protective enzymes. For example, the omega-3 fatty acids eicosapentaenoic acid and docosahexaenoic acid, as well as astaxanthin—found in cold-water fish like salmon—have been shown to boost enzyme expression. This raises an important question: Could dietary interventions help prevent idiopathic Parkinson's disease?

Keywords: neuroprotection; neurodegeneration; KEAP1/NRF2 pathway; aminochrome; dopamine; neuromelanin; natural compounds; Omega-3; DT-diaphorase; glutathione transferase M2-2

1. Parkinson´s Disease

Parkinson's disease is the second most common neurodegenerative disorder, characterized by the loss of dopamine-producing, neuromelanin-containing neurons in the nigrostriatal system. Most patients (70%) are diagnosed with idiopathic Parkinson's disease, which primarily affects individuals aged 55–60 and older. Since 1967, the standard treatment has been L-dopa, which greatly improves motor function and helps patients maintain a near-normal life. However, after 4 to 6 years of treatment, side effects like dyskinesia often develop, significantly reducing patients' quality of life. Despite extensive research, no new drugs have successfully slowed or stopped disease progression. Preclinical studies on compounds such as coenzyme Q, mitoquinone, urate, deferiprone, TCH346, and neurturin have shown promise, but these benefits haven't carried over into clinical trials [1–5]. The failure of these trials has been attributed to flaws in trial design and the lack of reliable biomarkers [6–8].

However, we believe the failure of these clinical trials can be attributed to two key factors:

(i) The use of preclinical models that poorly replicate the disease process. These models rely on exogenous neurotoxins, which induce an extremely rapid and widespread degenerative process [9, 10]. This sudden, aggressive degeneration seen in preclinical neurotoxin models sharply contrasts with the slow progression of idiopathic Parkinson's disease, both before and after motor symptoms

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appear. The most common preclinical models for testing Parkinson's drugs—1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) and 6-hydroxydopamine—produce effects that are inconsistent with the natural course of the disease. For example, MPTP can induce severe parkinsonism in just three days in individuals exposed to contaminated drugs [11], whereas idiopathic Parkinson's develops over many years, with neurodegeneration progressing gradually long before and after motor symptoms emerge. It is unlikely that a drug effective in these rapid, extreme neurotoxin models would translate to patients with idiopathic Parkinson's, where degeneration occurs at a far slower pace.

(ii) The lack of a methodology capable of detecting subtle degenerative changes. Parkinson's disease may follow a single-neuron degeneration model [12], consistent with its gradual progression before and after symptom onset. This raises the question of whether current tools, such as the Unified Parkinson's Disease Rating Scale (UPDRS), are sensitive enough to measure these minute, incremental changes in neurodegeneration over time.

To this day, the exact triggers that initiate the loss of dopamine-producing neurons containing neuromelanin in the nigrostriatal system in idiopathic Parkinson's disease remain unknown. However, scientists widely agree that multiple factors contribute to this process. These include mitochondrial dysfunction, oxidative stress, the formation of toxic alpha-synuclein clumps, disruptions in both proteasomal and lysosomal protein-clearing systems, endoplasmic reticulum stress, and neuroinflammation [13-20].

2. Single-Neuron Degeneration

Research suggests that the neurodegeneration affecting neuromelanin-containing dopaminergic neurons in the nigrostriatal system follows a single-neuron degeneration model [12]. According to this model, the neurotoxin responsible for triggering mechanisms like mitochondrial dysfunction, oxidative stress, neurotoxic alpha-synuclein oligomer formation, impaired proteasomal and lysosomal degradation, endoplasmic reticulum stress, and neuroinflammation originates within the neuron itself and does not spread to nearby cells**.** This model explains why the degenerative process is so slow, taking years before motor symptoms emerge and continuing gradually throughout the disease. Patients typically survive 10 to 20 years after diagnosis before succumbing to the condition. A recent study estimates that the total number of dopaminergic neurons in the substantia nigra (across both hemispheres) ranges from 800,000 to 1,000,000 [21]. For a patient who lives 15 years after motor symptoms begin—by which point 60% of neuromelanin-containing neurons are lost—this translates to a loss of 58 to 73 neurons per day. Such a slow progression can only occur if an endogenous neurotoxin selectively destroys neurons one at a time without affecting neighboring cells. Over time, the cumulative loss of these neurons eventually reaches a threshold where symptoms develop.

3. Endogenous Neurotoxins in Parkinson´s Disease

Potential endogenous neurotoxins in a single-neuron degeneration model include alpha-synuclein, 3,4-dihydroxyphenylacetaldehyde (DOPAL), and aminochrome. Mutations in alpha-synuclein that generate neurotoxic oligomers have been associated with familial Parkinson's disease, accounting for roughly 10% of all Parkinson's cases. However, in idiopathic Parkinson's disease—which makes up 70% of cases—alpha-synuclein's neurotoxicity depends on another neurotoxin to trigger the formation of these harmful oligomers. Studies indicate that aminochrome can induce the creation of such neurotoxic oligomers [20]. Furthermore, alpha-synuclein has been shown to cause widespread neurotoxicity, affecting nearby neurons [22-27]. Because of this, alpha-synuclein cannot be the sole endogenous neurotoxin responsible for initiating single-neuron degeneration.

During the oxidative deamination of dopamine—a reaction catalyzed by monoamine oxidase (MAO)—ammonia, hydrogen peroxide, and 3,4-dihydroxyphenylacetaldehyde (DOPAL) are produced. DOPAL is subsequently converted into 3,4-dihydroxyphenylacetic acid (DOPAC) by the

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enzyme aldehyde dehydrogenase-1 (ALDH1) [28]. Studies analyzing postmortem brain tissue from Parkinson's disease (PD) patients found reduced ALDH1 expression, suggesting that DOPAL accumulation may contribute to the degeneration of the nigrostriatal system [29]. However, postmortem tissue does not exclusively reflect the vulnerable neurons where neurodegeneration occurs. Furthermore, DOPAL exhibits intercellular spread, as it has been shown to transfer from neurons to glial cells [30].

Aminochrome is an endogenous neurotoxin produced during neuromelanin synthesis. The catechol group of dopamine undergoes oxidation, generating three ortho-quinones in a sequential process: dopamine ortho-quinone, aminochrome, and 5,6-indolequinone. Among these, aminochrome is the most stable ortho-quinone and is known to induce mitochondrial dysfunction, neurotoxic oligomer formation, oxidative stress, disruption of proteasomal and lysosomal protein degradation systems, endoplasmic reticulum stress, and neuroinflammation. Although aminochrome is a transient neurotoxin, it remains stable for approximately 40 minutes in in vitro experiments before converting to 5,6-indolequinone [31]. When aminochrome forms in the cytosol of a neuron during neuromelanin synthesis, it is quickly neutralized by flavoenzymes that transfer one or two electrons or form protein adducts. As a result, it cannot be exported to affect nearby neurons, limiting its neurotoxic effects to the neuron where it originates. These properties suggest that aminochrome may drive a single-neuron degeneration model, in which dopaminergic neurons containing neuromelanin are gradually lost over many years.

4. Why Can Aminochrome be Neurotoxic During Neuromelanin Synthesis?

There seems to be a contradiction regarding the neurotoxic effects of aminochrome, which is produced during neuromelanin synthesis. Normally, neuromelanin synthesis is a harmless process—healthy elderly individuals often retain intact, neuromelanin-containing dopaminergic neurons at death [32-34]. This raises a key question: Why don't healthy people experience aminochrome's neurotoxicity during neuromelanin synthesis? The answer lies in two critical enzymes that neutralize aminochrome's harmful effects.

(i) DT-Diaphorase – DT-diaphorase (NAD(P)H:quinone oxidoreductase; NQO1; EC 1.6.99.2) is a distinct flavoenzyme that catalyzes the two-electron reduction of quinones to hydroquinones [35]. Unlike other flavoenzymes—which use NADH or NADPH as electron donors to drive the one-electron reduction of quinones, producing highly reactive semiquinones—DT-diaphorase avoids semiquinone formation entirely. Semiquinones react with oxygen, generating superoxide and contributing to oxidative stress. For instance, NADPH-cytochrome P450 reductase catalyzes the reduction of aminochrome to leukoaminochrome radicals, which are highly reactive with oxygen [36]. In contrast, DT-diaphorase facilitates the two-electron reduction of aminochrome directly to leukoaminochrome [37]. Inhibition of DT-diaphorase via siRNA has been demonstrated to trigger cell death in catecholaminergic cell cultures [38]. DT-diaphorase is expressed in multiple organs, including the brain, with notable activity in the substantia nigra, striatum, hypothalamus, hippocampus, and cerebral cortex. It represents 97% of total quinone reductase activity (which includes other NADH/NADPH-dependent flavoenzymes) and is present in both dopaminergic neurons and astrocytes [39]. DT-diaphorase provides protection against: aminochrome-induced cell death, formation of neurotoxic α-synuclein oligomers, mitochondrial dysfunction, oxidative stress, autophagy and lysosomal dysfunction, disruption of cytoskeletal architecture [38, 40–52].

(ii) Glutathione transferase M2-2 – This enzyme catalyzes the conjugation of aminochrome with glutathione, forming 4-S-glutathionyl-5,6-dihydroxyindoline, a compound resistant to biological oxidizing agents such as superoxide, hydrogen peroxide, and dioxygen [53,54]. Glutathione transferase M2-2 also conjugates dopamine ortho-quinone (a precursor of aminochrome) to produce 5-glutathionyldopamine, which is typically metabolized into 5-cysteinyldopamine [55]. The detection of 5-cysteinyldopamine in human cerebrospinal fluid and neuromelanin suggests it is a stable end product, supporting its potential neuroprotective role.

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Notably, while glutathione transferase M2-2 is predominantly expressed in astrocytes, these cells secrete exosomes containing the enzyme, which then enter dopaminergic neurons and release the enzyme into their cytosol. This mechanism implies that astrocytes contribute to neuroprotection by boosting the defensive capacity of DT-diaphorase in neuromelanin-containing dopaminergic neurons [56-59].

The combined neuroprotective effects of DT-diaphorase and glutathione transferase M2-2 play a key role in preventing aminochrome-induced neurotoxicity during neuromelanin synthesis. However, decreased expression of these enzymes—along with excessive dopamine production and a resulting rise in aminochrome levels that overwhelms their protective capacity—may explain why neuromelanin-containing dopaminergic neurons are lost in Parkinson's disease (Figure 1).

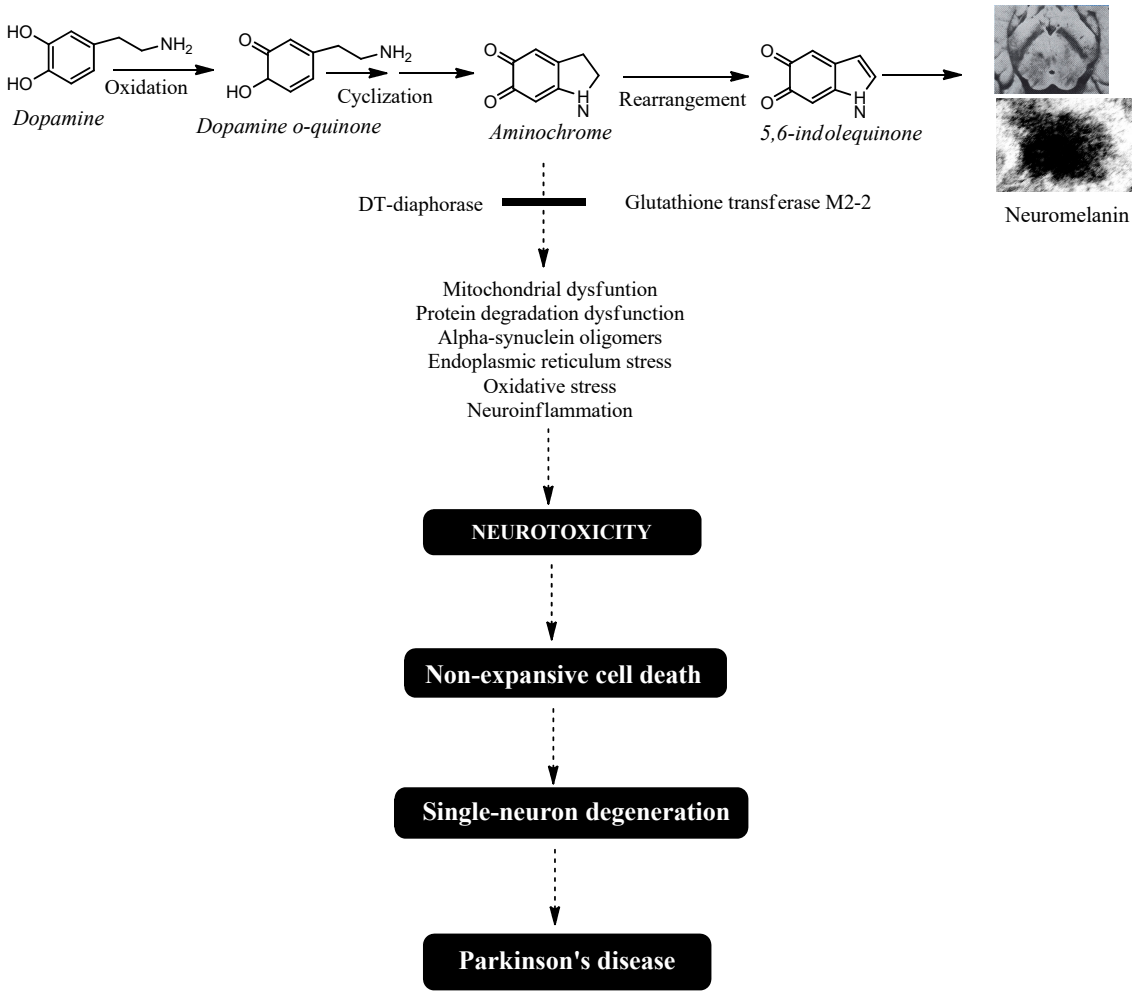

Figure 1. Aminochrome-induced single-neuron degeneration is a key process in Parkinson's disease. The synthesis of neuromelanin within dopaminergic neurons requires the oxidation of dopamine's catechol group. This reaction generates three ortho-quinones: dopamine o-quinone, aminochrome, and 5,6-indolequinone. Among these, aminochrome is the most stable and neurotoxic intermediate. It triggers a cascade of detrimental effects, including mitochondrial dysfunction, impaired protein degradation, the formation of neurotoxic alpha-synuclein oligomers, endoplasmic reticulum stress, oxidative stress, and neuroinflammation. Crucially, aminochrome's neurotoxicity is highly focused, selectively damaging the neuron it forms in while sparing adjacent cells, resulting in single-neuron death. The slow, cumulative loss of these individual neurons over many years is what ultimately initiates the motor symptoms and drives the progression of idiopathic Parkinson's disease.

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5. Natural Bioactive Compounds that Trigger Neuroprotection in Dopaminergic Neurons Containing Neuromelanin

The proposed neuroprotective role of DT-diaphorase and glutathione transferase M2-2 suggests that higher levels of these enzymes may be key in preventing neurotoxic effects on neuromelanin synthesis in idiopathic Parkinson's disease. The increased expression of these antioxidant enzymes is controlled by the KEAP1/NRF2 signaling pathway, which includes DT-diaphorase and glutathione transferase M2-2 [60]. Many natural compounds—such as curcumin, hyperoside, resveratrol, quercetin, sulforaphane, and safranal—have been shown to activate the KEAP1/NRF2 pathway [61]. However, not all studies on KEAP1/NRF2 activation demonstrate increased expression of DT-diaphorase and glutathione transferase M2-2. Since this pathway regulates multiple antioxidant enzymes, researchers often focus on measuring just one or two specific enzymes relevant to their study goals (Figure 2).

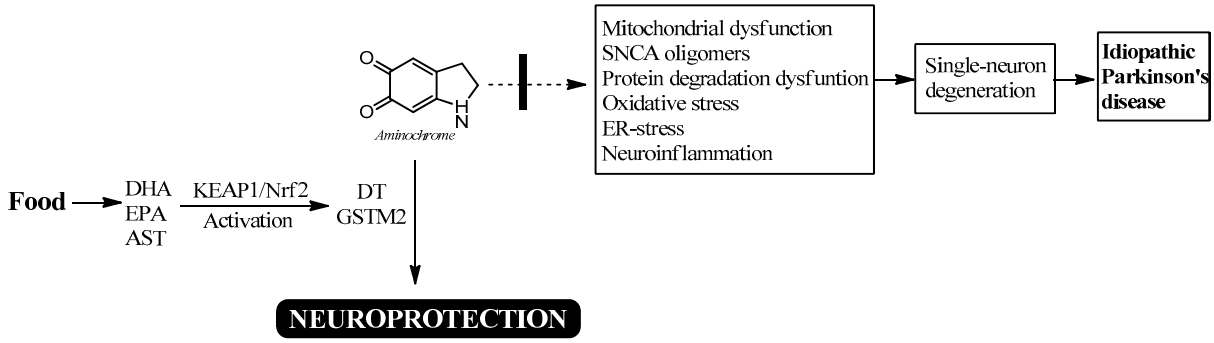

Figure 2. Possible neuroprotective mechanism of docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), and astaxanthin (AST) in idiopathic Parkinson's disease. DHA, EPA, and AST activate the KEAP1/Nrf2 signaling pathway, increasing the expression of DT-diaphorase and glutathione transferase M2-2, which prevent the neurotoxic effects of aminochrome.

6. Bioactive Compounds in Food

Natural foods not only provide proteins, fats, and glucose to produce the energy the body needs to function, but they also contain a variety of essential minerals and vitamins that support critical physiological processes. For example, iron is crucial for oxygen transport, while many enzymes and transporters depend on small molecules like magnesium, selenium, copper, and iron as cofactors. Animal-based foods deliver key nutrients such as vitamin B12, vitamin D, iron, zinc, and calcium, while seafood supplies essential fatty acids like eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). In contrast, vegetables and fruits are rich in vitamins A, B, C, and K, folate, and minerals such as magnesium, potassium, and non-heme iron. They also contain beneficial plant compounds, including phytochemicals, flavonoids, phenolic acids, and carotenoids [62, 63].

Research suggests that plant-derived neuroprotective compounds could help prevent neurodegenerative diseases. For example, nobiletin—a polymethoxylated flavone from *Citrus depressa* peel—has been shown to improve cognitive and motor deficits in preclinical Parkinson's disease models [64]. Similarly, tangeretin, a citrus flavonoid found in the peel and other parts of *Citrus L.* plants, exhibits neuroprotective effects in MPTP- and MPP+-induced Parkinson's models [65]. Other compounds, such as iridoids (geniposide, harpagoside, catalpol, and 10-O-trans-p-coumaroylcatalpol), have demonstrated neuroprotective activity in Parkinson's models by boosting antioxidant enzymes (e.g., glutathione peroxidase and superoxide dismutase) and increasing tyrosine hydroxylase-positive neurons [66]. Polydatin, a natural compound in peanuts, grapes, and red wine, has shown neuroprotective effects by suppressing microglia activation and reducing pro-inflammatory factors in a lipopolysaccharide-induced Parkinson's model [67]. Additionally, caffeic acid—a natural phenol in argan oil—has been found to protect dopaminergic neurons, enhance

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autophagy, and reduce alpha-synuclein aggregation in the substantia nigra of A53T alpha-synuclein transgenic models [68].

Chicoric acid, a polyphenol found in chicory and purple coneflower, has been shown to prevent MPTP-induced motor dysfunction, overactivation of glial cells, and the loss of dopaminergic neurons [69]. Morin, a flavonol present in wine and fruits, has been found to reduce motor dysfunction, protect dopaminergic neurons in the substantia nigra and striatum, and decrease astrocyte activation in an MPTP-induced mouse model. In primary cultures treated with MPP+, Morin demonstrated neuroprotective effects by lowering reactive oxygen species (ROS) production, preserving mitochondrial membrane potential, and inhibiting astroglial activation [70]. Wolfberry (the fruit of *Lycium barbarum* L.) has shown neuroprotective properties in multiple preclinical Parkinson's disease models, including 6-hydroxydopamine-treated rats, MPTP-treated mice, and α-synuclein A53T mice. It helped alleviate motor deficits and prevented dopaminergic neuron loss by regulating iron metabolism [71]. In another study, extracts from *Vicia faba* L. sprouts increased dopamine levels in the striatum, improved motor function, reduced inflammatory markers, and lowered malondialdehyde levels in a rotenone-treated mouse model [72]. Additionally, research using MPTP-treated animal models and cell cultures found that the alkaloid *N*-methylene-(5,7,4-trihydroxy)-isoflavone, derived from *Sophora alopecuroides* L. fruits, reduced motor deficits, oxidative stress, neuroinflammation, and dopaminergic neuron loss in both the striatum and substantia nigra [73].

Hericium erinaceus, a medicinal mushroom, has demonstrated neuroprotective effects in neurodegenerative diseases like Parkinson's. Its benefits are tied to boosting the production of neurotrophic factors [74, 75]. Studies in rats have shown that Cinnamomum osmophloeum Kanehira extract increases dopamine and tyrosine hydroxylase levels while reducing alpha-synuclein buildup in the striatum. In the midbrain, it also enhances antioxidant enzymes like superoxide dismutase, catalase, and glutathione peroxidase [76]. Additionally, two neuroactive β-carbolines in coffee provide neuroprotective, antioxidant, and anti-inflammatory effects, potentially lowering Parkinson's risk [77]. Another compound, nobiletin—a polymethoxylated flavone found in Citrus depressa peel—has been shown in animal models to improve both motor and cognitive deficits linked to Parkinson's [78].

Resveratrol glucoside (also called polydatin), found in red wine, peanuts, and other foods, acts as a neuroprotectant in a preclinical model of lipopolysaccharide-induced Parkinson's disease. It protects dopaminergic neurons from degeneration and improves motor dysfunction. Additionally, polydatin suppresses microglia activation and blocks the release of pro-inflammatory factors [79]. Caffeic acid, present in fruits, vegetables, coffee beans, and other dietary sources, reduces neurotoxicity caused by A53T alpha-synuclein overexpression in SH-SY5Y cells by activating the Nrf2/Bcl-2-mediated autophagy pathway [80]. Studies suggest that phytochemicals can prevent α-synuclein from forming neurotoxic oligomers and may even help break down existing aggregates [81]. Curcumin, a polyphenol in turmeric (Curcuma longa) used as a spice and food coloring, provides neuroprotection by modulating the brain-derived neurotrophic factor (BDNF) and PI3K/Akt signaling pathways [82]. Quercetin, abundant in apples, citrus fruits, onions, tea, and red wine, has been shown to inhibit alpha-synuclein aggregation into toxic oligomers [83]. Finally, L-theanine, found in green and black tea as well as certain mushrooms, exhibits neuroprotective effects in MPTP-treated SH-SY5Y cells. It boosts tyrosine hydroxylase-positive cells while decreasing alpha-synuclein clumping and Lewy body formation [84].

Omega-3 fatty acids, which are highly concentrated in salmon, have demonstrated neuroprotective effects in preclinical models of Parkinson's disease [85-88]. These benefits are linked to multiple mechanisms, including: reducing endoplasmic reticulum stress, inhibiting microglial activation and the release of pro-inflammatory factors, decreasing mitochondrial dysfunction, promoting the expression of neurotrophic factors, maintaining calcium homeostasis and alpha-synuclein proteostasis [89]. In studies using unilaterally 6-hydroxydopamine-lesioned animals, treatment with fish oil for 50 days reduced neuronal loss in the substantia nigra pars compacta and their terminals in the striatum. The neuroprotection from fish oil was associated with fewer iNOS-

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immunoreactive cells and reduced microglial and astrocyte reactivity [90]. Additionally, omega-3 polyunsaturated fatty acids improved motor symptoms in 6-hydroxydopamine-treated animals, further confirming their neuroprotective role [91].

DHA, an omega-3 fatty acid, has been shown to restore tyrosine hydroxylase-positive neurons and decrease lipid peroxidation in rotenone-treated animals. It also boosts the production of antioxidant enzymes like catalase and superoxide dismutase [92]. In a rat model of 6-OHDA-induced Parkinson's disease, DHA exhibited neuroprotective benefits by enhancing tyrosine hydroxylase levels and improving motor function, including gait and posture [93]. Additionally, DHA suppresses microgliosis and astrogliosis in both the substantia nigra and striatum in partial 6-OHDA lesion models, further supporting its neuroprotective effects [94].

The EPA has shown neuroprotective effects in differentiated human SH-SY5Y cells and primary mesencephalic cells exposed to MPP+ by countering the neurotoxin's effects through the suppression of pro-inflammatory factor release [95]. Studies also indicate that EPA may help prevent Parkinson's disease by reducing the neurotoxic effects of 6-hydroxydopamine in vitro. It helps restore mitochondrial function and boosts the expression of glial cell line-derived neurotrophic factor (GDNF) and brain-derived neurotrophic factor (BDNF), both of which are essential for neuronal survival, differentiation, and synapse formation [96]. Additionally, EPA has demonstrated protective effects in an MPTP-probenecid animal model, decreasing pro-inflammatory factor production and improving memory deficits [95]. A systematic review of 39 published studies further confirms the neuroprotective role of omega-3 fatty acids in Parkinson's disease, noting improvements in behavior, pathological markers, and antioxidant, anti-inflammatory, and anti-apoptotic effects, along with higher omega-3 levels in the brain [97].

Astaxanthin (AST), a red dietary carotenoid found in foods such as salmon, krill, shrimp, crayfish, trout, yeast, and algae, has neuroprotective, antioxidant, and anti-inflammatory properties. Its antioxidant effects come from its ability to increase the expression of DT-diaphorase and glutathione transferase M2-2 [98]. Notably, AST—along with DHA and EPA—activates the KEAP1/NRF2 signaling pathway, which boosts the production of these enzymes. These mechanisms are thought to play a key role in AST's potential neuroprotective effects against Parkinson's disease.

7. Conclusions

In our view, the degenerative process in Parkinson's disease follows the single-neuron degeneration model, where degeneration occurs through the loss of one neuron at a time during each degenerative event. The non-expansive nature of this model suggests an exceptionally slow progression of degeneration, unfolding over many years. According to this model, the neurotoxin responsible for triggering the degeneration of neuromelanin-containing dopaminergic neurons is believed to be aminochrome, which forms inside these neurons and lacks an expansive effect. However, the enzymes DT-diaphorase and glutathione transferase M2-2 counteract aminochrome's neurotoxic effects, protecting these neurons from degeneration. Thus, increasing the expression of these enzymes could enhance neuroprotection in dopaminergic neurons when aminochrome is produced. The KEAP1/NRF2 signaling pathway plays a key role by activating the expression of antioxidant enzymes, including DT-diaphorase and glutathione transferase M2-2 [60, 61]. Notably, bioactive compounds in certain foods—such as omega-3 fatty acids found in salmon, herring, pollock, chia oil, and others (Table 1)—activate this pathway. Among these neuroprotective foods, salmon stands out due to its high levels of DHA, EPA, and astaxanthin, all of which enhance the expression of protective enzymes via KEAP1/NRF2 activation. Therefore, increasing dietary intake of these bioactive-rich foods may help strengthen neuroprotection in dopaminergic neurons against aminochrome's toxicity during neuromelanin synthesis.

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Table 1. Natural sources of omega-3 compounds and alpha-linolenic acid.

	Source	Amount of total lipids	Reference
Eicosapentaenoic acid (EPA)	Herring	15 %	[85]
	Wild sardine	13.6 % in muscle	[86]
	Pollock roe	18.8	[85]
	Undaria pinnatifida	13 % of essential oil composition)	[85]
	Rhododendron sochadzeae	2 % of leaf extract	[85]
Docosahexaenoic acid (DHA)	Flyingfish	27.9 %	[85]
	Herring	22.6 %	[85]
	Pollock	22.2 %	[85]
	Salmon roe	17.4 %	[85]
	Cirrhinus mrigata	18.07 g/ 100 g muscle	[85]
	Catla catla	17.98 g/100 g muscle	[85]
	Jackalberry	4.54 g/ 100 g oil	[85]
Alpha-linolenic acid	Chia (*Salvia hispanica* L.) seed	64.04% of seed oil fatty acids	[85]
	Trichosanthes kirilowii	33.77–38.66% of seed oils	[85]
	Paprika Capsicum annuum	29.93% of fresh pericarp fatty acids in the Jaranda variety and 30.27% in the Jariza variety	[85]
	Sardine (Sardina pilchardus)	1.1	[86]
	Linum usitatissimum	1.1 to 65.2 %	[85]
	Rapeseed oil	9.1 %	[88]
	Olive oil	0.76 %	[88]
	Flaxseed oil	53.4 %	[88]
	Soybean oil	6.7 %	[88]
	Corn oil	1.2 %	[88]
	Walnut oil	10.4 %	[88]
	Walnuts seed	9.0 % of the total seed weight	[88]
	Flaxseed seed	22.8 % of the total seed weight	[88]
	Hemp seed	10 % of the total seed weight	[88]

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