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REVIEW ARTICLE

An Insight about Possible neuroprotective role of Spirulina platensis

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ABSTRACT

Background: Spirulina platensis (SP) is a blue-green algae of the Cyanobacteria family, recently used as a food supplement or a medication as it is a potent anti-oxidant, anti-inflammatory, and anti-apoptotic natural substance due to its high content of vitamins, minerals, polysaccharides, proteins, essential fatty acids, carbohydrate, phycocyanin, chlorophyll, and carotenes. It has multiple health benefits such as neural, cardiovascular, reproductive, and hepato-renal benefits. This review aims to discuss the possible neuroprotective role of SP on different regions of the nervous system as the cerebrum, cerebellum, hippocampus, and spinal cord, its role in decreasing stress, and its adjuvant role in the treatment of neurodegenerative diseases as Alzheimer's disease (AD), Parkinson's disease (PD) and multiple sclerosis (MS), and acute neural diseases as cerebral ischemia and hemorrhage.
Conclusion: SP has beneficial effects in decreasing stress and anxiety, treatment of neurotoxicity induced by many environmental factors, treatment of neurodegenerative diseases such as MS, PD, and AD, and acute neurological problems such as cerebral ischemia and hemorrhage.
Keywords: Spirulina platensis, Neurodegenerative diseases, Neuroprotection.

INTRODUCTION

Spirulina platensis (SP) is a multicellular, photosynthetic, and filamentous blue-green alga belonging to the Oscillatoriaceae family of Cyanobacterium that can have either a rod or disk shape and can grow in freshwater, certain marine, alkaline environments, and brackish lakes [1]. The nutritional value of SP was determined by its components, which comprised proteins with essential amino acids (55–70%), carbohydrates (15–25%), essential fatty acids (18%), minerals, vitamins, and pigments like chlorophyll, phycocyanin, and carotenes. Phycocyanin, polyunsaturated fatty acids, and phenolic contents are the key components of SP that contribute to its antioxidant effects together with selenium and zinc [2].

Neuroprotective role of SP:

SP has multiple health roles, including prevention of oxidative stress and has positive impacts on neurological system development, as shown in numerous studies [3,4], treatment of diseases precipitated by reactive oxygen species (ROS), and discovery of novel therapies for neurodegenerative disorders including multiple sclerosis (MS), Parkinson's (PD) and Alzheimer's (AD) [5].

SP nutrients are easily absorbed by the body and can quickly restore deficient nutritional status to reach normal physiological levels. SP micronutrients have a high bioavailability, allowing for rapid distribution even within the nervous system. Fatty acids, B vitamins, and magnesium supplemented via SP easily reach the brain through specific carriers, exerting positive neuronal effects [6].

Several neurological and neurodegenerative illnesses are associated with oxidative stress, and could be ameliorated by SP through its constituent; C-Phycocyanin (C-Pc), which acts as an inhibitor of NADPH oxidase [7]. Polysaccharides are also found in SP including numerous functional groups that contribute to the microalgae antioxidant and free radical scavenging capabilities. This antioxidant ability of SP is through boosting the activity of enzymes in the liver, kidneys, and brain that neutralize free radicals, including catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPX), as well as nitric oxide (NO), and reduced glutathione (GSH) [8].

It was demonstrated that fucosterol and other phytosterols found in SP have been studied for their potential therapeutic roles through their antioxidant,anti-inflammatory, immunomodulatory, and cholesterol-lowering

effects, which have led to numerous health benefits such as antidiabetic, anti-obesity, anti-Alzheimer's, anti-aging, anticancer, neuroprotection, and hepatoprotection [9].

SP, as shown by **Pérez-Juárez et al.** [10] can decrease neuronal death in experimental mice by decreasing apoptotic gene expression, promoting the anti-apoptotic pathway, and reducing oxidative stress in the hippocampus, so, it protects against the neurobehavioral damage caused by systemic kainic acid. SP can protect hippocampal neural stem cells from acute systemic inflammation caused by lipopolysaccharide (LPS) [11].

Moreover, the SP has perinatal neuroprotective roles in maternal protein-deprived mothers, where the neuropathological changes in rat offspring including shrunken neuronal cells with pyknotic nuclei, poor dendritic arborization, reduced cerebral cortical thickness, glial activation (astrocytes and microglia), and decreased functionally viable spine count were prevented when the mothers supplemented with SP during pregnancy and lactation [12].

It was reported that in primary neurons subjected to oxygen-glucose deprivation (OGD) to induce neuronal injury; selenium-enriched SP considerably increased neuronal survival (from 57.2% to 94.5%) and prevented apoptosis (from 45.6% to 6.3%), through reducing the buildup of ROS in neurons and decreasing the caspase activity. The selenium-enriched SP successfully reduced OGD-induced DNA damage (from 225.6% to 106.3%) [13].

Ghanbari et al. [14] reported that SP likely reduces oxidative stress, which helps the memory deficit brought on by scopolamine. The inhibition of oxidative stress is achieved by lowering the Malondialdehyde (MDA) level. **Khalil et al.** [15] stated that SP protects cerebral and cerebellar tissues from neurotoxicity induced by lead acetate by increasing SOD, GSH, CAT, and brain anti-inflammatory cytokines such as interleukin–10 (IL-10) while decreasing the immunoreactivity of caspase-3 and heat shock protein 70 (HSP70).

SP modulated the cerebellar damage elicited by methotrexate in adult male albino rats by decreasing myeloperoxidase (MPO), MDA, and acetylcholinesterase (ACHE) activity while raising dopamine, norepinephrine, GSH, serotonin, and SOD levels. In addition, it decreased the gene expression of ionized calcium-binding adapter molecule 1, glial fibrillary acidic protein (GFAP), and neurofilament light chain antibody (NFL) [16].

SP modulated chronic nicotine-induced neurotoxicity by regulating the expression of phospho-Tau (p-Tau) protein and cytochrome P450 enzymes (CYP2B1 and CYP2E1), increasing the antioxidant markers (SOD, CAT, and GSH), and decreasing MDA, the inflammatory markers, and the immunohistochemical reaction of Caspase-3. SP has a potent antioxidant, anti-inflammatory, and anti-apoptotic impact on the nervous system [17].

SP decreased brain damage caused by ionizing radiation by decreasing neurotransmitter levels (γ-aminobutyric acid (GABA) and S100 calcium-binding protein (S100B)) and inflammatory cytokines (interleukin-1β (IL-1β), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNFα)) while increasing level of glutamate and immunohistochemical reaction of brain-derived neurotrophic factor (BDNF) in brain tissue [18].

SP decreased cognitive impairment induced by l-methionine in adult male albino rats by increasing GSH level and decreasing ACHE activity and thiobarbituric acid-reactive substances (TBARS) causing improvement in neurobehavioral and motor tests [19]. SP alleviated motor signs of neuropathy in mice, injected with acrylamide to induce axonal injury. Results of this experimental study showed that SP, at a dose of 400 mg/kg for five weeks, caused improvement in motor assessment by faster restoration of action potential values and shortening of the recovery period [20].

Role of SP in decreasing stress and anxiety:

Restraint stress was applied to adolescent rats (postnatal days 30–40) for 10 days at a rate of 2 hours per day. The rats were then treated with SP (200 mg/kg/day) for 15 days (postnatal days 41–55), thereafter biochemical, molecular, and morphological evaluations of the basolateral amygdala were performed. To prevent stress-related diseases, this study's findings suggest that SP, when taken as a non-pharmacological intervention during adolescence, can protect against neuroanatomical, biochemical, and molecular deficiencies generated by chronic stress in adulthood [21].

SP helps the body deal with stress and mental strain better [22]. SP can reduce anxiety and depression behavior caused by chronic high-fat diet (HFD) in Sprague Dawley male rats by increasing the level of leptin in the hippocampus [23].

Role of SP in cerebral ischemia and hemorrhage:

Reduced lipid peroxidation and increased endogenous non-enzymatic and enzymatic antioxidants may explain the substantial neuroprotective impact of SP in localized cerebral ischemia caused by middle cerebral artery blockage. The SP is an antioxidant cocktail that

may prevent neurons from focal cerebral ischemia-reperfusion injury by coordinating their actions at various points in the free radical production process [24]. SP showed a neuroprotective effect in an experimental rat model with induced cerebral hemorrhage by collagenase as pretreatment with SP showed more viable neurons in the histological section of the brain and improved motor function during the open field assessment when compared to a collagenase-treated group, this is due to its anti-oxidant and anti-inflammatory advantages, thus SP has a positive role in the management of acute neurological problems [25].

Protective role of SP on the spinal cord:

Evidence suggests that SP may have neuroprotective properties that mitigate the consequences of spinal cord injury and enhance functional recovery in rats. In Sprague Dawley rats, supplementation with SP improved the fine ultrastructure of spinal cord gray matter compared to the control group, after a partial crush injury was induced at the level of T12 [26]. **Aziz et al.** [27] reported that SP enhanced the locomotor function of the hind limb and diminished the morphological injury of the spinal cord.

Role of SP in the treatment of MS:

MS seems to be a neurodegenerative and chronic inflammatory disease of the central nervous system with a possible autoimmune cause. Acute inflammatory demyelination and axonal damage are the two pathognomonic events that cause neurological disorders in patients suffering from MS [28]. **Pentón-Rol et al.** [29] explained that C-Phycocyanin (C-PC), main component of SP, increases the expression of genes related to remyelination, axon-glia processes and gliogenesis (Bmpa, Mobp, Mog, Mal, Nkx2-2, Nkx6-2, and transcription factor Olig1) while markedly decreases the expression of genes involved in demyelination, such as PPAR and CD44. C-PC also reduces the levels of MDA and increases SOD, CAT, and GPX levels. **Pentón-Rol et al.** [30] reported that C-PC can aid oligodendrocyte stem cells to recruit, differentiate, and mature in demyelinated lesions by decreasing oxidative stress, pro-inflammatory cytokines, apoptotic gene expression, and Cyclooxygenase-2 (COX-2) level. Moreover, it has a regulatory effect on T-cell activity to modulate immunity.

Role of SP in the treatment of PD:

PD is a chronic, progressive in nature, neurodegenerative disorder with multiple causes. It primarily affects older individuals. Age is thought to be the most important risk factor for PD. Environmental and genetic factors may also aid in predisposing the disease [31].

SP can be used as an adjuvant treatment for PD in rats in which behavioral and neurochemical alterations were partly reversed by SP supplementation for 2 weeks. This is achieved by reduction of the apomorphine-induced rotational behavior, dopamine (DA) and 3,4-Dihydroxyphenylacetic acid (DOPAC) depletions, nitrite level, and TBARS, as well as, decreasing the immunohistochemical reaction of COX-2, Inducible nitric oxide synthase (iNOS), Tyrosine hydroxylase (TH), and dopamine transporter (DAT) in brain tissue [32].

SP exerted a useful therapeutic role in DJ-1βΔ93 flies, a model of PD in Drosophila, by regulating the expression of Jun-N-terminal kinase and HSP70 and increasing the anti-oxidant markers (CAT and SOD) due to its component of C-PC which is potent anti-oxidant thus, SP improved the locomotor activity and life span in flies [33].

Rats given a spirulina-enriched diet for four weeks after receiving an intrastriatal injection of 6-hydroxydopamine (6-OHDA) showed a marked increase in the regenerating of DA terminals into the striatum's Tyrosine Hydroxylase (TH)-negative zone. Major Histocompatibility Class II (MHC) immunohistochemistry revealed that microglia activation had decreased in conjunction with this regeneration. This implies that the advantageous effects of SP are modulated by reductions in microglial activation [34].

SP was found to be neuroprotective in the α-Synuclein experimental rat model of PD. It increased the number of TH+ and NeuN+ cells while reducing the number of activated microglial cells as measured by MHC II expression. The reduction in microglia activation could be attributed in part to the ability of SP to increase expression of the Fractalkine receptor (CX3CR1) on microglia, which has a neuroprotective effect in the 6-hydroxydopamine (6-OHDA) rat model of PD [35].

Role of SP in the treatment of AD:

AD is the most prevalent kind of age-related neurodegeneration. This disease is expected to affect over 100 million people worldwide by 2050. AD is a disease with multiple possible causes, such as chronic inflammation, oxidative stress, disturbed metabolism of insulin, and cholinergic lack [36].

An experimental study examined the effects of phycocyanin on rats that had been intracerebroventricularly inducted with streptozotocin (STZ). The results showed that phycocyanin has great promise in reducing the cognitive deterioration caused by STZ. This

medical research suggests that blue-green algae may have a potential treatment for AD [37].

Zhou et al. [38] reported that SP can be used as a beneficial supplement to treat AD in mice with HFD by improving the cognitive impairment, learning, and memory in Morris water maze and Barnes Maze tests as it reduced the accumulation of amyloid-β (Aβ) and inflammatory cytokines such as IL-1β, TNF-α, and IL-6 in the hippocampus. It also prevented gut microbial dysbiosis and microbial metabolite imbalance that was caused by HFD, indicating that it has a regulating effect on the gut-brain axis.

Tamtaji et al. [39] reported that giving SP to patients with AD can improve cognitive function and memory loss by decreasing serum concentration of C-reactive protein (CRP), fasting glucose, and insulin resistance as well as, increasing the sensitivity of insulin thus, enhancing the metabolic status. Also, Madhavadas & Subramanian [40] stated that a combination of SP and glycyrrhizin can improve cognitive impairment and memory deficits associated with aging and obesity, induced by monosodium glutamate (MSG), by decreasing the activity of AChE in hippocampal tissue and also modulating parameters of glucose homeostasis.

CONCLUSION

SP has the potential to become a valuable food in the future. It is a potent antioxidant, anti-inflammatory, and anti-apoptotic natural substance due to its high content of vitamins, minerals, polysaccharides, proteins, essential fatty acids, carbohydrates, phycocyanin, chlorophyll, and carotenes. It exerts a neuroprotective impact on different regions of the nervous system as the cerebrum, cerebellum, hippocampus, and spinal cord. It has a positive effect in decreasing stress, anxiety, and depression. It can be used as an adjuvant treatment for neurodegenerative diseases such as MS, PD, and AD. It has a protective role in acute neurological disorders such as cerebral hemorrhage and ischemia.

Conflicts of interest: The authors report no conflicts of interest.

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