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Review Article

Role of a Micronutrient Mixture, Probiotics, Collagen Peptides, Omega 3, and CBD in Prevention and Improved Treatment of Parkinson's Disease

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Abstract

Parkinson's Disease (PD) is a slow, progressive, incurable neurological disorder of the central nervous system which exhibits motor symptoms like tremor, rigidity, and bradykinesia (slowed movement) and nonmotor symptoms such as depression, anxiety, constipation, fatigue, cognitive decline, and sleep disorders. The current preventive recommendations like adopting healthy diet and lifestyle and reducing exposure to environmental toxins have failed. Consequently, the incidence of PD continues to increase. The main reason for the failure of preventive recommendations is that it did not attenuate all internal stressors such as increased oxidative stress, neuroinflammation, intestinal dysbiosis, loss of collagen and omega 3 dysfunction. The current drugs and deep brain stimulation therapies have improved motor symptoms without affecting non-motor symptoms, but after some time they aggravate these symptoms. The current treatment which treats symptoms of PD but not the causes of it, the quality of life remains poor. One of the reasons could be that it has not suppressed all internal stressors which causes PD. We propose that a novel prevention plan which include supplementation with a micronutrient mixture which would reduce oxidative stress and neuroinflammation, probiotics with prebiotics which would reverse the harmful effects of intestinal dysbiosis, collagen peptides which would restore the loss of collagen, omega 3 to replace dysfunctional oxidized omega 3. The proposed prevention plan in combination with current recommendations may markedly reduce the incidence of PD and in combination of current treatments may prolong their beneficial effects by protecting dopamine neurons from oxidative damage while reducing their side- effects.

Key words: External and internal stressors; Increased oxidative stress; Chronic inflammation; Intestinal dysbiosis; Loss of Collagen and Omega 3

Introduction

Parkinson's disease (PD) is a slow, progressive, incurable neurological disorder of the central nervous system. It is the second most common neurodegenerative disease. Descriptions of PD-like symptoms have been recorded in many ancient texts from both the Western and Eastern literatures such as the Old Testament of the Bible (2000–440 BC), the Caraka Samhita of Ayurvedic medicine (~1000 BC), and The Yellow Emperor's Classic of Medicine (*Huang Di Nei Jing*, ~425–221 BC) [1]. While earlier literatures provided general description of most of the symptoms of PD, it was not until in 1817, Dr. James Parkinson, a British physician, published an article on "The Shaky Palsy" describing the major symptoms of the disease that would later bear his name. In the mid-1800s, Jean-Martin Charcot separated Parkinson's disease from multiple sclerosis and other neurological disorders characterized by tremor [2]. In 1899, Édouard Brissaud was first to suggest that PD originated from the damaged substantia nigra [3]. In 1912, Frederick Lewy observed that aggregated inclusions were located inside the substantia nigra of PD patients. Later, in 1919, Konstantin Tretiakoff, a Russian neuropathologist, named these inclusion particles as Lewy body [3]. He found that Lewy body is present idiopathic and post-encephalitic Parkinsonism [4,5].

Parkinson's disease is a movement and mood disorder which exhibits motor symptoms like tremor, rigidity, and bradykinesia (slowed movement). In addition, Parkinson's disease symptoms may include rigid muscles, poor posture and balance, loss of automatic movements, speech changes, and writing alterations. The nonmotor symptoms of PD may include depression, anxiety, constipation, fatigue, cognitive decline and sleep disorders. Approximately, 90% of PD are acquired while 10% of them are inherited. Several mutated genes have been identified in familial PD [6]. They include mutated alpha-synuclein (SNCA), parkin, PIEN-induced kinase-1 (PINK-1), DJ-1, leucine-rich repeat kinase-2 (LRRK2) [7-10], and VPS35 [11]. Among familial PD, mutated PARKIN gene accounts for about 50%, PINk1 8-15%, and DJ1 about 1% of cases [12] and are responsible for causing autosomal recessive Parkinsonism. Mutated PD genes cause damage to mitochondria leading to increased oxidative stress, because damaged mitochondrial cannot utilize respired oxygen for generating energy. Accumulation of oxygen in the substantia nigra may oxidize dopamine and L-dopa, a precursor of dopamine, leading to their accelerated loss of function of dopamine neurons that initiate and promote PD at an early age [13,14].

Despite current preventive recommendations, the incidence of PD is increasing in the USA. In 2020, approximately 930,000 people were living with PD; in 2024, the number increased to 1 million, and

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it is estimated that in 2030 the number would increase to 1.2 million. In the USA, 90,000 people are diagnosed each year. In 2020, 9.4 million people were living with PD in the world. Men are 1.5 time more likely to develop PD than women. In 2024, economic cost of management of this disease was 52 billion and it is expected that by 2037, the cost would increase to 80 billion (From Parkinson's Foundation).

Current treatments of PD with drugs and deep brain stimulation surgery primarily focus on managing the symptoms of the disease but not the causes of the progression or degeneration of dopamine neurons. Although drugs like levodopa and dopamine agonists reduce motor symptoms in the early stages of PD, their effectiveness is reduced over time. In addition, new symptoms such as motor fluctuations, dyskinesias and hallucinations appear. The non-motor symptoms such as blood pressure, digestion, mood changes, cognitive decline, sleep problems, and pain are not affected by the treatment. Therefore, a new approach for enhancing the effectiveness of current prevention and treatment of PD is proposed.

This review describes briefly external and internal risk factors which initiate the development and progression of PD; current prevention recommendations for reducing the risk of PD and difficulty in implementing them and current treatment of PD and their limitations. It proposes a novel plan for enhancing current prevention recommendation and prolonging the effectiveness of current treatment of PD.

External and internal risk factors for PD

To develop an effective prevention and improved treatment strategy, it is essential to know about external and internal factors which initiate development and progression of PD and how to attenuate them. Current prevention program has relied only on diet, lifestyle and exposure to environmental toxins and treatment strategy has focused on improving the symptoms of the disease and not its causes.

External risk factors which increase the risk of PD

Poor diet [15] including milk and milk products [16], poor lifestyle such as lack of physical activity, suffering from stress and anxiety [17], traumatic brain injury and bipolar disorder [18], diabetes [19], exposure to pesticides and herbicides, the solvent trichloroethylene, air pollution [20], and certain metals such as lead, mercury, and aluminum [21].

Internal risk factors such as oxidative stress and chronic neuroinflammation that increase the risk of PD

The brain especially substantia nigra is daily exposed to high level of oxidative stress and neuroinflammation; therefore, they play a central role in the development and progression of PD [22,23]. Pathologists and neurologists have repeatedly reported that loss of dopamine (DA) neurons from the substantia nigra region of the brains is primarily responsible for the most motor control abnormalities observed in PD patients, although other cells are also affected in this disease. It is estimated that in normal individuals about 5%–10% of DA neurons are lost every decade [24]; however, in PD patients, the rate of loss is greater than that found in normal individuals. Neuropathological studies of PD brain revealed that about 50–80% of DA neurons are lost by the time the disease becomes detectable [25].

Death of DA neurons occurs due to increased levels of oxidative stress generated by multiple mechanisms which include (a) production of free radicals during use of oxygen by the mitochondria to generate energy, (b) enzymatic oxidation of DA which generates hydrogen peroxide that in the presence of iron in the substantia nigra produces hydroxy radicals [26], (c) non-enzymatic oxidation of DA produces superoxide which interacts with nitric oxide to produce peroxynitrites, (d) formation of 6-hydroxy dopamine, a metabolite of DA which is neurotoxic [27]. Thus, the brain is daily exposed to high levels free radicals. Since the brain has low levels antioxidant system [28,29], continue high levels of oxidative stress damages glia cells, particularly microglia cells and astrocytes. Damaged glia cells produce chronic inflammation products such free radicals and pro-inflammatory cytokines [30,31]. Thus, the brain is daily exposed to high levels of oxidative stress and inflammation.

Intestinal dysbiosis

Growing evidence show that intestinal dysbiosis plays an important role in the pathogenesis and progression of PD by increasing pro-inflammatory cytokines and decreasing the levels of short-chain fatty acids such as butyric acid, acetic acid and propionic acid [32-34]. Stress increases the level of intestinal dysbiosis [35]. It has been suggested that severe stress can trigger preclinical to clinical symptoms of PD [36]. In addition, psychological stress can aggravate motor symptoms and non-motor symptoms such as anxiety and depression [37]. Therefore, reversing the adverse effects of intestinal dysbiosis by probiotics with prebiotics would be helpful in prevention and improved treatment of PD.

Loss of collagen

Collagen is a major structural protein in the brain [38] and it represents 20-30% of total body protein [39]. One the major function of collagen is to maintain structural integrity of all organs in the body. There are 29 different types of collagen and are produced mostly by neurons, and astrocytes and vascular cells [40].Collagen types I, IX, and XVIII are involved in neural maturation, neural circuit formation, axon guidance, and synaptogenesis [41-43]. Collagen types I and III are abundant in the vascular wall and maintain firm structure of the wall. Type I provides resistance to stretching and type III is responsible for maintaining firm structure of vascular wall [44,45]. Loss of collagen occurs because of increased activity of collagenase during aging. Consequences of loss of collagen are visible in the skin but not in other organs such as the brain. It is possible that loss of collagen in the brain could lead shirking of the size causing reduced function. The role of collagen peptides in prevention or improved treatment of PD has not been evaluated. However, loss of collagen could enhance the initiation and progression of PD.

Dysfunction of omega 3 fatty acids

Omega-3 polyunsaturated fatty acids and its components DHA (docosahexaenoic acid) and EPA (Eicosapentaenoic acid) and ALA (alpha-linolenic acid) are essential for health and brain function [46,47]. It has an impact on cognitive function at all stages of life. Approximately, 50-60% of the brain weight are lipids, of which 35% consists of omega3 fatty acids. DHA accounts for more than 40% of total fatty acids in the brain, while EPA represents only 1% of total fatty acids. In the high oxidative environment of aging individuals, omega 3 is easily oxidized and becomes dysfunctional.

Current prevention recommendations for PD

They include adopting healthy diet and lifestyle and reducing exposure to environmental toxins. Dietary suggestions include

consumption of low-fat high fiber diet with plenty of fruits and vegetables, reduced fat intake, drinking in moderate amount of coffee and green tea; changes in lifestyle include moderate to aerobic exercise regularly, reducing stress by meditation or yoga; and decreasing exposure to environmental toxins such as heavy metals, pesticides, herbicides, polluted air and water. These recommendations have failed to reduce the risk of PD. One of the reasons could be that human behaviors with respect to diet and lifestyle are difficult to change. Many aspects of environmental exposure are beyond our control. Other reasons could be that all internal stressors that contribute to the initiation and progression of PD are not attenuated.

Proposed plan to attenuate all internal stressors for prevention of PD

Attenuation of oxidative stress and chronic inflammation: Since one of the internal stressors such as increased oxidative stress and chronic neuroinflammation play a central role in the development and progression of PD, daily oral supplementation with antioxidants which would decrease them and thereby reduce the risk of developing this disease. We propose that simultaneous reduction of oxidative stress and chronic neuroinflammation is essential for reducing the risk of developing of PD. The use of a single antioxidant cannot achieve this goal for the following reasons:

a. Supplemented single antioxidant in the above environment would be oxidized and then acts as a pro-oxidant rather than as an antioxidant. Therefore, a single antioxidant is not expected to reduce oxidative stress and chronic inflammation at the same time.

b. Different antioxidants are distributed in different amounts in various organs as well as in the sub-cellular compartments of the same cell. Administration of a single antioxidant at an arbitrarily selected dose cannot accumulate in all organs and in all parts of the cell in sufficient amounts to reduce oxidative stress and chronic inflammation.

c. Since most antioxidants are either lipophilic or hydrophilic, administration of a single antioxidant cannot protect both the aqueous and lipid compartments of the cell against oxidative damage.

d. The brain uses very high level of oxygen for production of energy. The efficacy of antioxidant varies depending upon the levels of oxygen. For example, vitamin E is more effective scavenger of free radicals in reduced oxygen pressure, whereas beta-carotene and vitamin A are more effective in higher oxygen pressure of the cells [48]. Therefore, administration of one antioxidant may not retard oxidative stress and chronic inflammation in neurons.

e. In order to reduce oxidative stress and chronic neuroinflammation at the same time, simultaneous elevation of antioxidant enzymes, and dietary and endogenous antioxidant compounds is essential [49]. The levels of antioxidant compounds can be enhanced by an oral supplementation; however, enhancing the levels of antioxidant enzymes requires an activation of a nuclear transcriptional factor Nrf2 [49,50].

Proposed Micronutrient mixture to attenuate oxidative stress and chronic inflammation

The proposed micronutrient mixture can enhance the levels of antioxidant enzymes by activating Nrf2 as well as dietary and endogenous antioxidant compounds. This mixture includes vitamin A (retinyl palmitate), vitamin E (both d- alpha-tocopherol acetate and d-alpha-tocopheryl succinate), natural mixed carotenoids, vitamin C (calcium ascorbate), vitamin D3, all B-vitamins, coenzyme Q10, alpha-lipoic acid, N-acetylcysteine (NAC), resveratrol, curcumin, quercetin, green tea extract, and minerals selenium and zinc. This micronutrient mixture has no iron, copper, or manganese. Although these trace minerals in tiny amounts are essential for the growth and survival, slight excess of free iron and copper can increase the risk of chronic diseases, because these trace minerals when combined with vitamin C produce extensive amounts of free radicals and they in the presence of antioxidants are rapidly absorbed. This micronutrient mixture also has no heavy metals such as vanadium, zirconium, and molybdenum, because increased levels of these heavy metals are neurotoxic. There are no methods of elimination of either trace minerals or heavy metals from the body; therefore, taking them with a micronutrient mixture could be harmful after a prolonged consumption. Supplementation with this Micronutrient mixture would simultaneously reduce oxidative stress and chronic inflammation by increasing the levels of antioxidant enzymes via activation of Nrf2 and dietary and endogenous antioxidant compounds [49,50].

Reversing the effects of intestinal dysbiosis

Since intestinal dysbiosis plays an important role in the pathogenesis and progression of PD by increasing pro-inflammatory cytokines and decreasing the levels of short-chain fatty acids such as butyric acid, acetic acid and propionic acid [32-34]. We propose that supplementation with probiotics and prebiotics together may reverse harmful effects of intestinal dysbiosis, and thereby, may help in reducing the risk of PD. It is important to point out that Bifedo and lactobacillus strains of bacteria are very sensitive to acid pH of stomach and bile acid of the small intestine. Therefore, an effective probiotic must contain acid resistance probiotics.

Restoring the levels of collagen

Importance of restoring collagen has not been demonstrated in a clinical study of PD; however, an oral supplementation with 5 g collagen hydrolysate for 4 weeks in adults aged 49-63 improved brain structure and language cognitive function [51]. Collagen hydrolysate may help recovery from brain injury by promoting angiogenesis in mice [52], and that it exerts neuroprotective action by suppressing inflammatory effects and promoted learning and memory in aged mice [53]. These studies suggest that supplementation with collagen 3 may reduce the risk of developing PD by preventing changes in the structure of the brain and its function.

Restoring the function of omega 3

Omega-3 fatty acids reduce oxidative stress and chronic inflammation in the brain [54-56]. A review of several clinical studies suggests that supplementation with omega-3-fatty acids enhances learning, memory, cognitive function, and blood flow in the brain. For maintaining an optimal brain structure and function, life-long consumption of DHA is essential [57]. DHA is more effective than EPA in reducing markers of chronic inflammation [58]. Omega-3 acts as an endocannabinoid ligand such as anandamide and 2-AG which activates endocannabinoid receptors CB1 and CB2 [59]. Plant-derived ALA prevents stroke, reduces post-stroke injury, increases brain-derived growth factor (BDNF) which improves learning ability and memory, reduces formation of plaques in the carotid artery [60,61]. ALA also repairs blood-brain barrier (BBB) damage and improves its function and restores DHA level in the brain [62].

Current treatments of PD with drugs and deep brain stimulation surgery

The current drug treatments of PD are based on restoring dopamine level in the substantia nigra to improve the symptoms of PD rather than on reducing the causes of this disease. In 1960, Oleh Hornykiewicz found reduced dopamine level in the striatum of PD patients [63]. Based on this finding, in 1961, André Barbeau was first to observe that oral administration of L-dopa, a direct precursor of dopamine, was effective in improving PD symptoms [64]. In 1967, George Cotzias in open-label studies established that combined use of L-dopa with a peripheral dopa decarboxylase inhibitor was very effective in improving the symptoms of PD [65,66]. The dopamine receptor agonists bromocriptine and apomorphine were also found to be potent for PD therapy [67]. Levodopa alone increased the level of dopamine. It in combination with inhibitors of monoamine oxidase B which reduces degradation dopamine and catechol-O-methyltransferase which promotes entry of L-dopa by inhibit its degradation maintains increased level of dopamine for a longer period. Deep brain stimulation by surgery also has been useful in improving the symptoms of PD. Levodopa is considered most potent drug in controlling the symptoms of the disease at least for certain period especially those related to bradykinesia (slowness of movements). Treatment with levodopa is associated with motor complications such as fluctuation in dyskinesia (uncontrolled movement). Levodopa/carbidopa is also used in early stage of PD to relieve the motor symptoms carbidopa prevents degradation of DOPA before it reaches the brain. More recent therapy includes infusion-based therapies like Vyalev (levodopa/carbidopa infusion) and Onapgo (apomorphine infusion), and adaptive deep brain stimulation (aDBS) to relieve the symptoms of PD [68,69]. Alpha synuclein antibodies are being investigated as a potential therapy for Parkinson's disease (PD) [70]. These antibodies may neutralize alpha-synuclein aggregates, which are a hallmark of the disease. Despite some beneficial results from the use of these antibodies, variability in their efficacy and discontinuation of trial poses serious challenge. Medications used in the treatment of PD produce adverse side effects [71-74].

Although deep brain stimulation (DBS) therapy produces significant relief for some Parkinson's disease symptoms, particularly motor symptoms, but it has some significant limitations. For example, DBS can improve tremors, stiffness, and slowness, it does not affect other symptoms of PD like speech difficulties, gait freezing, balance issues, or non-motor symptoms like dementia. In addition, DBS may aggravate some symptoms and may develop the new ones [75-78]. Parkinson disease (PD) is the one of the fastest growing neurological disorders in the USA and globally and poses serious challenge in the management of this disease due to progressive disability, emergence of levodopa-resistant symptoms, and treatment-related complications [69].

None of the current treatments of PD protect dopamine neurons from oxidative and inflammatory damage and none of them influence other internal stressors such as intestinal dysbiosis, loss of collagen and omega 3 fucntion causing treatment resistance and enhanced side effects. Combining the current treatment with medications or deep brain stimulation surgery with those agents which attenuate all internal stressors would markedly enhance the current management of PD.

Attenuation oxidative stress and inflammation by antioxidants in PD

Since increased oxidative stress and neuroinflammation play a central role in the initiation and progression of PD; therefore, the use of antioxidants in the management of PD is a rational approach.

Vitamin E alone or in combination of vitamin C

Administration of vitamin E alone did not protect mice against MPTP-induced PD [79-80] and did not affect the risk or symptoms of PD [81,82] however, administration of a combination of vitamin C as ascorbate and vitamin E as α-tocopherol slowed the progression of the patients with early PD [83].

Coenzyme Q_{10}

Supplementation with CoQ_{10} protected against MPTP-induced loss of dopaminergic neurons have been shown in various *animal* models of PD. In mice, CoQ_{10} protected against MPTP-induced loss of dopamine neurons [84]. Short-term oral administration of CoQ_{10} also prevented dopaminergic neurons degeneration after MPTP administration in monkeys [85]. However, no significant benefit in Unified Parkinson's Disease Rating Scale (UPDRS) score was observed in most clinical studies [49,82,86]. Moreover, the adverse trend in the primary outcome, led to the termination of the study [87].

Glutathione and N-acetylcysteine NAC

Reduction in the levels of glutathione was found in the substantia nigra part of the autopsied brain tissue of PD patients [88] Lewy body disease [89] and loss of dopamine neurons occurs in people with advanced age [90]. However, supplementation with glutathione was ineffective in improving the symptoms of PD patients [91]. Oral and intravenous administration of NAC enhance dopamine transporter binding which corelated with improved PD symptoms [92].

Vitamin C

Administration of vitamin C did not improve symptoms of PD [91]. Supplementation with vitamin C modified the levodopa pharmacological kinetics in patients by increasing its absorption and bioavailability [93].

The reasons for the failure of individual antioxidants in improving the symptoms of PD are the same as discussed in the section of "prevention of PD". We propose the same micronutrient mixture as suggested in the section of "Prevention of PD". In combination with current treatments would prolong the beneficial effects of therapies by reducing motor and nonmotor symptoms of PD. Increased oxidative stress causes aggregation of alpha-synuclein which plays an important role in progression of PD [94] and antioxidants prevent it [95,96]. Therefore, proposed micronutrient mixture would prevent aggregation of alpha-synuclein.

In addition to oxidative stress and inflammation, other internal stressors such as intestinal dysbiosis, loss of collagen, and dysfunction of omega 3 contribute to the progression and treatment outcome in PD. These internal stressors have not drawn attention from neurologist when devising treatment plan for PD patients.

Reversal of Intestinal dysbiosis by probiotics with prebiotics in the treatment of PD

Since intestinal dysbiosis is involved in the pathogenesis of PD [97], we propose to consume probiotics with prebiotics alone or in combination with oral medications to improve the motor and non-motor symptoms of PD. Indeed, it has been shown that supplementation with probiotics improved motor and non-motor symptoms in 6-OHDA- or MPTP-induced PD in animals [98,99]. In addition, it improves apomorphine-induced rotational behavior and spatial memory in 6-OHDA-induced animal model of PD, and in humans it may reduce the symptoms of constipation [99]. Administration of PD Drugs (Benserazide and dopamine agonist) in combination with probiotics significantly improved motor symptoms of PD in humans [100] as well as in mouse model of PD [101].

Restoring collagen by collagen peptides in the treatment of PD

Although the role of collagen peptides in maintaining structure and function of the brain during aging has been established, its role by itself or in combination with drugs in the treatment of PD has not been established. To demonstrate importance of collagen in maintaining structure and function of the brain during aging, it was shown that an oral supplementation with 5 g collagen hydrolysate for 4 weeks in adults aged 49-63 improved brain structure and language cognitive function [51]. Collagen hydrolysate may help recovery from brain injury by promoting angiogenesis in mice [52], and that it exerts neuroprotective action by suppressing inflammatory effects and promoted learning and memory in aged mice [53]. Therefore, we suggest that a similar study in combination with medications or deep brain stimulation may prolong their beneficial effects on motor and non-motor symptoms of PD.

Omega 3 in the treatment of PD

The significance of omega 3 in PD patients has been reviewed [102]. A pilot clinical study on PD patients showed that supplementation with fish oil with or without antidepressant medication for a period of 3 months improved depressive symptoms [103]. This indicates that consumption of omega 3 can be used as an adjective therapy in combination with other medications. The observational studies revealed that there is an association between consuming diet rich in omega 3 and a lower risk of PD [104]. Omega 3 in combination with vitamin E improved some symptoms of PD [105]. In high oxidative environment of aging individuals and PD, omega 3 is oxidized and becomes dysfunctional. The consequences of dysfunctional omega 3 in the brain if not replaced by functional omega 3 could adversely affect function of the brain including the symptoms of PD.

Expression of abnormal behaviors

The patients with advanced Parkinson's disease show abnormal behaviors such as psychosis (hallucinations and delusions), apathy, depression, anxiety, agitation, impulse control disorders, a cognitive decline, and stereotypic movements. Some of these can be enhanced after treatment with certain medications or at the advanced stage of the disease [106,107].

Supplementation with CBD (Cannabidiol) may reduce abnormal behaviors associated with advanced PD

Treatment with CBD reduced agitation and anxiety [108]. CBD acts as a partial agonist of dopamine receptor D2 and produced anti-psychotic effect like that produced by a prescription drug aripiprazole [109]. In a mouse model of depression, administration of CBD caused rapid and sustained anti-depression effect by enhancing cortical serotonin receptor [110,111]. CBD stimulated serotonin receptor and inhibited serotonin re-uptake [112,113]. A review has described the role of CBD in reducing oxidative stress and chronic inflammation [114].

Proposed improved treatment plan

We suggest that combining the proposed prevention plan with medications or deep brain stimulation therapy may prolong their beneficial effects on motor and non-motor symptoms of PD, while reducing their potential side effects.

Conclusion

Although some of the symptoms of Parkinson's disease (PD) were observed as early as 2000 BC; pathology of PD was not clarified until in 1817-1919 years. During 1960-1967, it was established that loss of dopamine neurons or their function in the substantia nigra account for the development and progression of PD. This led to the development of drugs that can elevate dopamine level in patient with PD. Current prevention recommendations include adopting of healthy diet and lifestyle and reducing exposure to environmental toxins. This recommendation has failed to reduce the incidence of PD, because it did not attenuated internal stressors such as increased oxidative stress, chronic neuroinflammation, intestinal dysbiosis and loss of collagen and omega 3 function. In addition, human behaviors with respect to diet and lifestyle are difficult to change, and many aspects of environmental toxins are beyond our control. Consequently, the incidence of PD is increasing in the USA. Our proposed prevention plan which includes daily oral supplementation with a well-tested micronutrient mixture which would reduce oxidative stress and chronic inflammation, probiotics with prebiotics to reverse the harmful effects of intestinal dysbiosis, collagen peptides to restore collagen level to maintain structural integrity and function of the brain and omega 3 to replace dysfunctional omega 3 would markedly reduce the incidence of PD. The current PD therapy improved the motor symptoms but not the non-motor symptoms of the disease, but after some time, both type of symptoms become aggrevated. Our proposed plan suggests that combination proposed prevention plan with current drug or deep brain stimulation therapy would markedly prolong the beneficial effects of these therapies and reduce risk of side effects.

Declaration

Conflict

The author is Chief Scientific Officer of Engage Global of Utah. This company sells nutritional products to consumers.

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