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Review Article

A Review of High Glycemic Index (GI) in Relation to Risk of Parkinson's Disease

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Abstract

Objective: The objective was to investigate whether a diet with high glycemic index (GI) is associated with Parkinson's disease (PD)and changes that happen at levels of methylglyoxal (MGO), oligomeric α-synuclein (α-syn), and DJ-1.

Materials and Methods: This reviewed study was carried out by doing search in Scholar Google, PubMed and Elsevier databases using keywords such as Parkinson's disease, high glycemic index (GI), methylglyoxal (MGO), α-synuclein (α-syn), DJ-1. Approximately 40 articles were selected that were fully reviewed.

Conclusions: High GI diets may be risk factor for developing PD by increasing a-synoligomerization and advanced glycated end products (AGEs) formation. Intake of dietary fiber appear to be beneficial in reducing PD.

Keywords: Alpha-synuclein; DJ-1; Glycemic Index; Glycemic Load; Methylglyoxal; Parkinson's Disease

Introduction

Parkinson's disease (PD) is the most common neurodegenerative disorders with a worldwide prevalence in the millions [1,2]. The frequency increases with age, affecting 1% of the population over the age of 60 [3].Genetic, family history of disease and environmental factors such as diet smoking, exposure to solvents and metals, organophosphates are linked to PD [4]. PD was first described by James Parkinson in 1817 [5,6]. Characteristic features of PD include neuronal loss in the substantia nigra [7] and widespread intracellular protein a-synuclein (a-syn)accumulation(8, 9). a-syn may contribute to PD pathogenesis in a number of ways, but it is generally thought that its aberrant soluble oligomers conformations that mediate disruption of cellular homeostasis and neuronal death(10). Studies indicate that glucose metabolism imbalance and subsequent hyperglycaemia lead to biochemicalabnormalities that may associated with PD [11-13]. Researchers about the pathogenesis of age- related noted that glucose rich diet can increase up the generation of AGEs in the brain [14,15]. The major precursor in the formation of AGEs is Methylglyoxal (MGO) [16]. Excessive levels of MGO may directly damage neurons through increasing oxidative stress [17]. DJ-1 is another protein involved in PDpathogenesis and acts as a neuroprotective agent against the toxicity of MGO and also as a major ant glycation in eliminate glycated a-syn. Function of DJ-1 is still not fully understood [18]. In some studieshave shown that mutations DJ-1 caused disease with parkinsonian features [19,20]. However, the cause of PD is not fully known [21,22] and Recently, the management of age-related diseases such as PD has been associated with dietary factors for help to reduce or contrast the symptom of PD and the related pathological mechanisms [23].

Discussion

PD is a neurodegenerative condition which there are currently no fully therapies. The incidence of PD is increasing and expected to double worldwide by 2040 [24,25]. Diet has recently gained importance as a potential therapeutic approach to treat PD and also as a risk factor for develop.higher dietary GI was inversely associated with prevalence of PD.The impact of carbohydrate quality only might be important rather than both quality and quantity. The number of epidemiological studies of the associations between GI and GL and PD is limited. A prospective study by Murakami et al showed that dietary GI or GL carbohydrates might be decrease the risk of PD by an insulin secretion- induced increase in brain dopamine [26]. one pervious study using ELISA reported the presence of significantiy elevated levels of oligomeric forms of a-syn in plasma sample obtained from 34 PD patients compared with 27 controls [27]. Recent studies have suggested that, oligomeric a-syn-induced toxicity causes neuronal death related to PD through mitochondrial dysfunction, altered membrane permeability, and produce high levels of reactive oxygen species [28]. Several previous in vitro studies suggested that DJ-1is an antioxidant protein and free radical scavenger. It can protect against cell dead induced

by oxidative stress [29-32]. Waragai et al. reported high levels of DJ-1 in the CSF and plasma of PD patient [33]. The studies conducted by Maita, et al. did not find any significant difference between the level of DJ-1 in control and patients group and also were not found in correlations of levels DJ-1 with age, level of oxidative stress and clinical severity [34]. Although the interactions between these factors have not been completely elucidated, it seems that excessive levels of MGO cause a condition such as chronic glycative stress. In this condition, the elevated level of MGO leads to the accelerated oligomerization of a-syn. Consequently, an increase in DJ-1 level could be expected a compensatory response to modulating increasing levels of MGO and oligomeric a-syn toxicity. Regarding the correlation between GI and oligomeric a-syn, in 2010, Münch, et al reported association between glycation and glycoxidation and pathogenicity of PD and also showed that there is accumulation of aggregates of an intracellular protein,a-syn and lewy bodies that trigger dopaminergic neurons death [35]. The mechanisms by which dietary GI correlated with DJ-1 may be indirect, via increase glycosylation of a-syn, inflammation, and oxidative stress associated with PD, which can stimulate elevated DJ-1 levels as a protective response [18,36,37]. DJ-1 can play an antiapoptotic and anti-oxidative streesrole in pericytes exposed to high glucose [18]. In a study by Renaud et al, investigated relation between hyperglycemia and nigrostriatal dopaminergic neurodegeneration and they found that elevated levels of glucose lead to the death of dopaminergic neurons in culture through oxidative mechanisms [38].

Conclusion

The results of this study suggest that high GI diets may be risk factor for developing PD by increasing a-synoligomerization and advanced glycated end products (AGEs) formation and elevated levels of DJ-1 for modulating this condition are expected. In addition, intake of dietary fiber appear to be beneficial in reducing PD.

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