Nutrition 29 (2013) 474–475

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Editorial

Good and bad sides of diet in Parkinson's disease

Parkinson's and Alzheimer's diseases are disorders of unknown etiology, which appear late in life. Genetic and non-genetic causes have been considered in the two diseases, with most studies of causes focused on the adult. Parkinson's disease is the second most common worldwide neurodegenerative disease after Alzheimer's disease. Parkinson's disease is a progressive illness affecting the elderly population. Although there are rare cases of familial forms of the disease, most patients have the sporadic (~90%) form of the illness, as with Alzheimer's disease [1,2]. Parkinson's disease typically affects patients older than 50 y, with a momentous increase in patients older than 65 y [3].

The prevalence of the disorder in the worldwide population older than 65 y is estimated at 1%, with a global incidence of 10 to 14 cases per 100 000 per year. The incidence rate of Parkinson's disease in patients older than 65 y in the USA is approximately 160 cases per 100 000 per year. In the USA, there are approximately 60 000 newly diagnosed cases every year, and it has been estimated that about 1 million people have the disease [4] and more than 20 000 deaths per year are associated with Parkinson's disease [1]. The increased mortality risk in Parkinson's disease is dependent on illness duration and dementia [1,5]. This suggests that part of the decreased life expectancy of patients with Parkinson's disease can be significantly associated with their increased risk of developing dementia [1,5]. The prevalence of dementia in community-based investigations has been estimated at 30% to 40% of patients with Parkinson's disease [5]. In 2005, the diagnosed number of patients with Parkinson's disease worldwide was circa 4.4 million [1]. By 2030, the number of diagnosed cases is expected to double to circa 9 million based on the expected growth of the population 65 y and older [6]. The prevalence, incidence, and mortality are higher in men than in women [1,4].

Parkinson's disease is a chronic illness that is caused mainly by the progressive degeneration of nigrostriatal dopaminnergic pathways. A curved posture, a shuffling gait, a resting tremor, bradykinesia, rigidity, and postural instability are its main symptoms and signs, which, with the long-term side effects of dopaminergic therapy (dyskinesias and motor blocks), lead step by step to a dangerous disability [7]. In addition, not only motor but also non-motor problems and some offered therapies are factors, which substantially change the nutritional condition during the development of the disorder [8]. Thus, the modification of nutritional intake must be taken into consideration at all times. It is widely recognized that a nutritional evaluation should be incorporated in the workup of patients with Parkinson's disease [7]. Nutritional changes may contribute to the amelioration of symptoms and health-related quality of late life. In support of this advice is an article in *Nutrition* by Barichella et al. [9] who examined how changes to the poor nutritional status and dietary habits in a Ghanaian population could positively influence gastrointestinal dysfunctions such as dysphagia and constipation. This suggests that Mother Nature can influence some symptoms and partly improve the quality of life of patients with Parkinson's disease in developing countries in Africa. In addition, a low-protein diet increased the absorption of levodopa from the gut and decreased the adverse effects of therapy. In contrast, the anthropometric data related to cases of Parkinson's disease in Ghana indicated that their body weight and body mass index were significantly lower than in those without Parkinson's disease.

The etiology of sporadic Parkinson's disease has not been identified, but further investigations support the view that environmental risk factors play a key role in its onset and development [10,11]. The major suspect events in early life are stress, infections, poor nutrition, chemicals, and pesticide exposure and risk factors late in life for Parkinson's disease include aging and artherosclerosis [10,11]. Because of the nature of the illness, patients with Parkinson's disease have been found to be at higher risk of malnutrition than other people at the same age [8]. Malnutrition includes an imbalance of protein, energy, and other nutrients that influence the shape, composition, and size of the body and affects function and clinical outcomes [8]. Although the pathways of these neurodegenerative mechanisms remain under examination, the possible contribution of dairy products, including milk, has been vigorously promoted in recent years [12–14]. Milk may occupy an initiating/triggering and/or intermediate position in the chain of neuropathogenesis events in the onset of Parkinson's disease, ending with cognitive failure and dementia [1,5]. Notably, the recent nutritional study performed in Ghana by Barichella et al. [9] has drawn attention to the etiologic relevance of milk in the control of the neuropathogenesis, management, and other effects of disease, suggesting a possible contribution for the increased incidence of Parkinson's disease after the excessive consumption dairy products, especially milk. Other investigators have presented direct evidence that the higher consumption of dairy products other than milk is associated with an increased risk of Parkinson's disease [12–14]. The association was stronger in men and generally

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http://dx.doi.org/10.1016/j.nut.2012.09.013

explained by excessive milk consumption [12,14]. The only possible explanation for this effect is the evidence of the contamination of milk by neurotoxins [13]. High levels of organochlorine residues have been found in milk, and high organochlorine levels in the substantia nigra have been detected in patients with Parkinson's disease [13]. Moreover, the pollution that has been noted in milk is the tetrahydro-isoquinoline component of pesticides, which induces Parkinsonism in primates [13]. Reports of pesticides in milk exist across the globe; the dairy industry should require toxin screenings of milk [15].

These findings are consistent with observations of an association between milk products and Parkinson's disease, and further confirmation is needed, especially in identifying the exact elements of milk that could trigger Parkinson's disease. Whether the monitored effects are provoked by unknown nutrient substances other than calcium or by neurotoxins warrants further study. This requires determining at what stage of life a given exposure has its greatest effect and how such exposures can accumulate during the life span. Future investigations also need to determine ways to improve the survival and quality of life of patients with Parkinson's disease in the later course of the illness. Epidemiologic studies are also necessary to assess the evolution over time of the burden of Parkinson's disease at the community level, to have a better knowledge of the need for resources and its progression over time. The impact of nutritional management on the course of Parkinson's disease is still in its infancy and should be investigated, as strongly suggested by Barichella and colleagues [9] in this issue of *Nutrition*.

Acknowledgments

This work was supported by grants N N312 233938 and N N405 162639 from the Polish Ministry of Science and Higher Education.

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