Magnesium RESEARCH

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The neuroprotective potential of magnesium in Parkinson's disease

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Abstract

Pathogenic mechanisms implicated in the development of Parkinson disease (PD) are multifaceted and include alpha synuclein aggregation, oxidative stress due to generation of reactive oxygen species (ROS), mitochondrial dysfunction, apoptosis, imbalance of trace elements as well as endoplasmic reticulum stress, and inflammation. Alteration in the homeostasis of bivalent cations, such as iron, magnesium and calcium, has been implicated in the pathogenesis of PD. Low levels of magnesium have been associated with accelerated dopaminergic cell loss in animal PD models, and magnesium has been shown to have a neuroprotective effect in PD models. Evidence of a low magnesium level in the brain of PD individuals, with a low magnesium level in the diet, increasing the risk of PD, further strengthens the role of magnesium deficiency in the pathogenesis of PD. The presence of low-level magnesium in brain tissue and high level in CSF and serum support the possibility of dysfunctional magnesium transporters in PD. Indeed, variants in magnesium transport channels, such as TRPM7 and SLC41A1, have been recently detected in PD individuals. Magnesium, being an NMDA antagonist, could also have a therapeutic role in levodopa-induced dyskinesia. There are no clinical studies indicating a neuroprotective role of magnesium in PD, however, the Mediterranean diet and variants of the diet have been associated with a lower risk of PD, which may be due to the magnesium-rich constituents of the diet. Further clinical trials encompassing therapeutic models to optimize channel function, coupled with a high magnesium diet, may pave the way for promising neuroprotective intervention for PD.

Keywords: MPTP; PD model; dyskinesia; magnesium transporter; neurodegeneration.

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