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## The gait restorative effects of robotic-assisted gait training for multiple sclerosis, Parkinson's disease, and progressive supranuclear palsy

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**Background:** Neurodegenerative diseases and disorders present with a wide range of clinical and neuropathological symptoms caused by progressive neuronal dysfunction and eventual neuronal death. As individuals with neurodegenerative diseases experience gradual sensory, motor, and cognitive debilitation, the maintenance and recovery of a functional gait holds physiological, psychological and financial importance. Developments in robotically-aid therapies are becoming more commonly used as a therapeutic tool for the improvement of gait characteristics and overall motor function for individuals with various gait impairments. To date, studies examining the effects of robotic-assisted gait training (RAGT) as treatment for neurodegenerative diseases, have only been performed in individuals with multiple sclerosis (MS), Parkinson's disease (PD) and progressive supranuclear palsy (PSP).

**Purpose:** The purpose of this review is to summarize and show trends to the efficacy of RAGT as a gait restorative and preservative modality for individuals with these neurodegenerative diseases including MS, PD, and PSP.

**Results:** The overall trends reported by these reviewed studies show that RAGT may be an effective therapy for producing significant improvements in multiple gait characteristics including balance, walking speed, endurance, leg strength, gait safety, and motor function for individuals with neurodegenerative disease.

**Conclusion:** The studies in this review suggest that RAGT therapies may be an effective substitute for, or addition to, present conventional therapies for individuals with neurodegenerative disease, however the long-term effects of this therapy is still not known for these individuals.

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## Glutathione deficiency as a cause and consequence of Parkinson's disease

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Parkinson's disease (PD) is one of several prevalent neurodegenerative diseases plaguing the aging population. To date, no biological therapies have been shown to slow, stop, or reverse PD progression; the disease is considered irreversible and progressive. The hypothesis that deficiency of reduced glutathione (GSH) contributes to PD degeneration was proposed over thirty years ago. Advances in neuroimaging and pharmaceutical science now permit quantification of brain GSH concentrations and novel methods of delivery, respectively. The goal of this lecture will be to present the data in evaluation of this hypothesis and identify gaps in knowledge. Post mortem brain from individuals with premotor PD shows a deficiency of GSH and it has been hypothesized that deficiency of GSH contributes to PD neurodegeneration. The role of GSH in the healthy brain will be described, and evidence of GSH deficiency in PD will be reviewed. The pros and cons of various augmentation strategies will be discussed, e.g. oral, intravenous and intranasal. All four clinical trials of GSH in PD have demonstrated a mild symptomatic improvement. In a cross-sectional analysis of 58 individuals with PD, low blood GSH was associated with greater disease severity. Taken together, these data support the hypothesis that GSH depletion contributes to PD and that intranasally-administered GSH has therapeutic potential as both a symptomatic treatment and a disease modification strategy.

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