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COMMENTARY

MUSCLE STRENGTH IN PARKINSON'S DISEASE. COMMENTARY ON PANG AND MAK

By 2040 neurodegenerative disorders such as Parkinson's disease (PD), which is presently the second most common neurodegenerative disorder among older adults, affecting approximately 0.5–1% of the population of 60–65-year-olds and 1–3% of over 80-year-olds (1, 2), will affect approximately 33% of older adults, becoming the second most common cause of death and disability among older adults (3). Approximately 60,000 individuals are diagnosed with PD annually (in the USA) and the incidence of musculoskeletal impairment, falls, hip fractures, secondary disabling conditions and fall-related nursing home admissions in this population are high.

In this issue Marco Pang and Margaret Mak (4) describe the relationship between lower extremity muscle strength and bone mineral density (BMD) in women with a diagnosis of PD (p. XX–XX). This is a potentially important area for clinical rehabilitation since both factors appear to be amenable by pharmacological and/or exercise intervention. While other studies have linked PD with osteoporosis, few have made the connection with muscle strength. After controlling for several potential confounders, lower extremity muscle weakness accounted for 10% of the variability on a standardized test of BMD. This is noteworthy, since muscle strengthening or exercise are not interventions clinicians would consider when treating a patient with PD. In fact, early practice guidelines on physiotherapy (PT) for PD overlooked muscle as a potential target for exercise intervention, targeting instead the cardinal signs and symptoms of the disease with therapies that were often not supported by published research (5). This practice continues today in many clinical settings. When the late William Koller, a neurologist, and Susan Kase, a senior physical therapist, showed a clear association between upper and lower body muscle weakness and hemi-Parkinson's disease in 1987 and stated that "muscle weakness appears to be a primary symptom of Parkinson's disease which may relate to disturbed motor programming due to basal ganglia dysfunction" (6), their work was largely ignored by the clinical community, perhaps because their statement challenged conventional thought or because it was thought that declines in muscle strength were a part of the normal ageing process. Today experimental evidence suggests that declines in muscle strength among these patients are triggered by central changes secondary to PD (and not normal ageing), including accelerated loss of striatal neural tissue and concomitant depletion of striatal dopaminergic metabolites (for a review see 7).

Pang & Mak (4) suggest that increasing muscle strength through resistance training might improve BMD, mobility and reduce the occurrence of falls and hip fractures. In order to increase strength, the intensity of exercise would, presumably, have to be suitably high, as proposed by Farley and colleagues (8). Current thoughts on PD rehabilitation reflect a much more dynamic interplay between the rehabilitation environment, behavior, brain and rehabilitative outcomes in people with PD (9) (Fig. 1). Indeed, high intensity, task complexity, saliency, novelty and other factors may be necessary to promote structural and metabolic plasticity in the brain and musculoskeletal systems of persons with PD; however, to date, studies are still forthcoming and no agreed exercise guidelines exist. Until recently, intense exercise was feared to worsen the symptoms of PD by perhaps increasing the underlying muscle tone, and so, for these individuals, high intensity exercise was to be avoided. These beliefs still dominate PD rehabilitation research and clinical practice today. Few dare to challenge these "truths", current leading texts on management of PD with exercise still reinforce the notion that high-intensity resistance training has "minimal effects on the symptoms" (9, p. 298) such as postural reflex impairment, and, as a result, relatively little progress has been made in the treatment of these patients.

It will be interesting to see if future studies reinforce the stereotype that people with so-called chronic neurodegenerative conditions such as PD cannot improve under any circumstances or if it is we who cannot advance our own beliefs.

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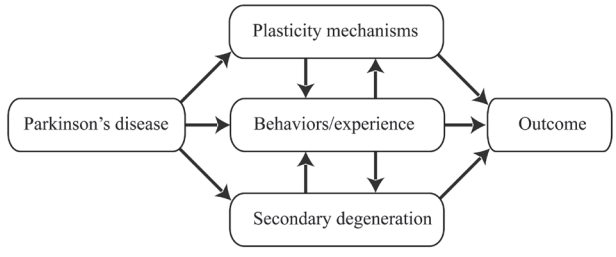

Fig. 1. Relationship between behaviors, brain and Parkinson's disease (10). The figure shows the possible relationships between behaviors/experience and outcomes in Parkinson's disease. Behaviors, such as lack of rehabilitation leading to lack of mobility, falls or low bone mineral density downstream, which may occur if clinicians do not prescribe exercise, and experiences, may trigger secondary degeneration and plasticity mechanisms. Plasticity mechanisms and secondary degeneration affect behavior. The arrows between Behavior/experience, Plasticity mechanisms and Secondary degeneration run both ways. All components affect outcome.

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