Brain and Cognition 122 (2018) 1–8

Contents lists available at ScienceDirect

Brain and Cognition

journal homepage: www.elsevier.com/locate/b&c

Aerobic exercise is more effective than goal-based exercise for the treatment of cognition in Parkinson's disease

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ARTICLE INFO

Keywords:
Parkinson's disease
Aerobic exercise
Goal-based exercise
Cognition

ABSTRACT

Background: Little is known about how different exercise modalities influence cognition in Parkinson's disease (PD). Moreover, the focus of previous investigations on examining the effects of exercise mainly on executive functions and the exclusion of individuals with cognitive impairment may limit the potential to define exercise as a treatment for cognitive decline in PD.
Objective: The aim of this study was to compare the effects of aerobic and goal-based exercise on five cognitive domains in cognitively normal and impaired individuals with PD.
Methods: Seventy-six individuals with PD were randomly allocated into three groups: Aerobic, Goal-based, and Control. Participants in the exercise groups attended 1-h sessions 3x/week for 12 weeks, while those in the Control group carried on with their regular activities. Changes in cognitive domains were assessed using paper-based neuropsychological tests.
Results: Inhibitory control improved only in the Aerobic group ($p = .04$), irrespective of participants cognitive status at baseline. Moreover, participants with cognitive impairment in Aerobic group maintained their set-shifting ability, whereas those in the Control group were worse at post-test ($p = .014$).
Conclusion: This is the first study to show that aerobic exercise is more effective than goal-based exercise for the treatment of cognition in PD with and without cognitive impairment.

1. Introduction

In addition to dopaminergic dysfunction, Parkinson's disease (PD) affects multiple systems in the brain including the noradrenergic, serotonergic, and cholinergic (Kehagia, Barker, & Robbins, 2010). Impaired function in these systems has been linked to the onset and progression of non-motor symptoms in PD, including cognitive decline which has been rated one of the main contributors to decreased quality of life among people with PD (Barone et al., 2009). According to Aarsland and colleagues (Aarsland et al., 2010), approximately 26% of non-demented PD patients have some form of mild cognitive impairment, with deficits primarily in the attention/executive functioning, visuospatial, and memory domains. Moreover, findings from a longitudinal study demonstrated that 46% of individuals with PD developed dementia by the 10 year-mark from diagnosis (Williams-Gray et al., 2013). Hence, the development of treatments for cognitive deficits in PD is critical.

The treatment of cognition in PD is an enormous challenge due to its multifactorial underlying mechanisms (Kehagia et al., 2010). Studies have shown that impaired connectivity in the frontostriatal circuitries (Lewis, Dove, Robbins, Barker, & Owen, 2003; Owen, Doyon, Dagher, Sadikot, & Evans, 1998), structural atrophy in multiple brain areas (Hanganu et al., 2014; Hwang et al., 2013; Kandiah et al., 2014), reduced levels of circulating neurotrophic factors important for brain plasticity (Leverenz et al., 2011), and dysfunction in multiple neurotransmitter systems (Bohnen et al., 2003, 2006; Riekkinen, Kejonen, Jakala, Soininen, & Riekkinen, 1998) contribute to impaired cognition in PD. Thus, in order to overcome this challenge, a combination of pharmacological and non-pharmacological therapies to treat cognitive function in PD has been encouraged. Among non-pharmacological therapies, physical exercise may be a promising approach.

Research in healthy older adults has demonstrated that exercise (primarily aerobic) led to improvements in multiple cognitive domains (Colcombe & Kramer, 2003), decreased risk of dementia (Hamer & Chida, 2009), modulated task-related activation in frontal and parietal areas involved in inhibitory control (Colcombe et al., 2004), and increased brain volume in the hippocampus (Erickson et al., 2011) and pre-frontal cortex (Colcombe et al., 2006). Although it remains

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https://doi.org/10.1016/j.bandc.2018.01.002
Received 23 August 2017; Received in revised form 11 December 2017; Accepted 2 January 2018

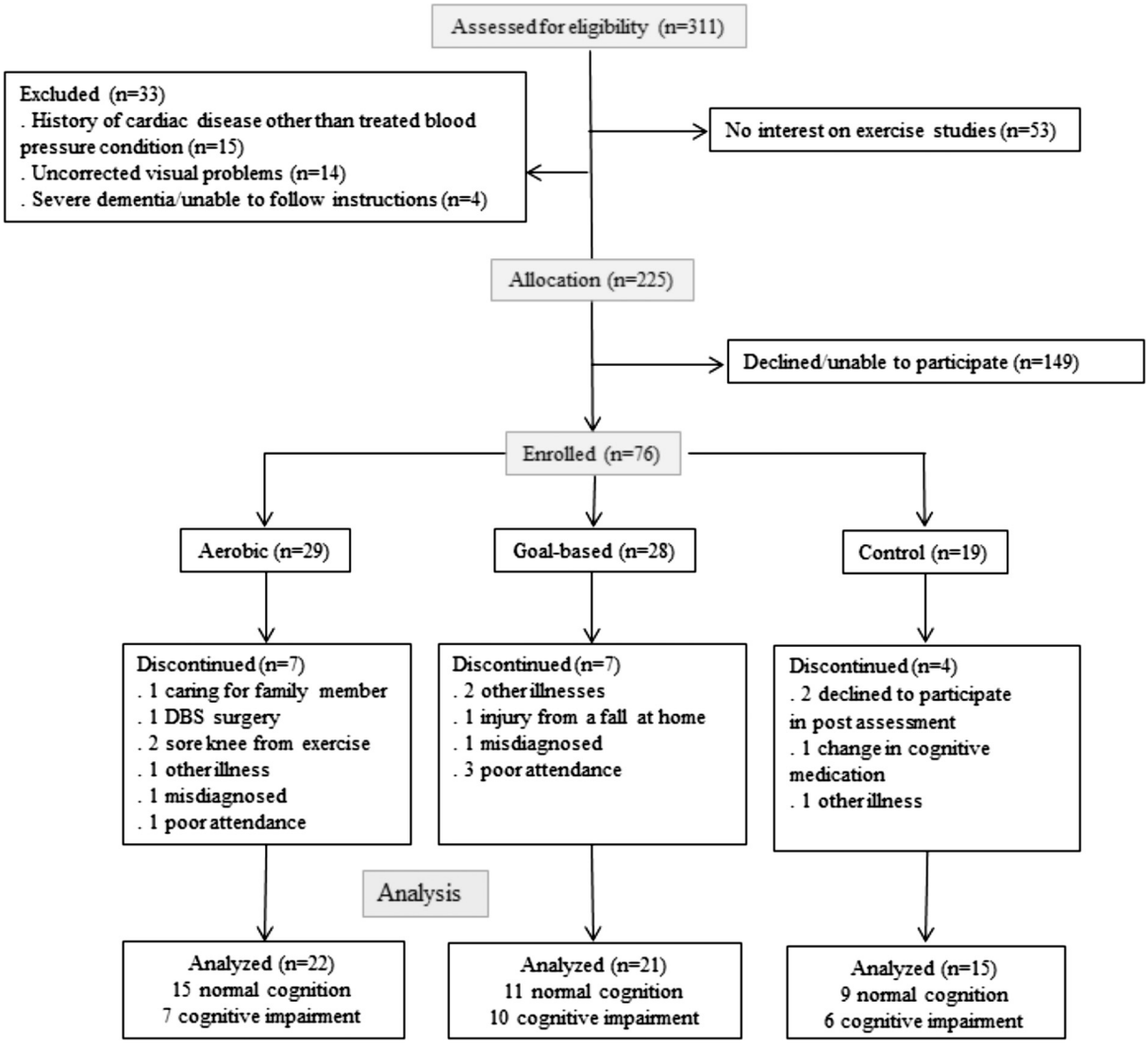

Fig. 1. Flow of participants throughout the study.

unknown how exercise may influence brain function and consequently lead to improvements in cognition in PD, studies have shown that exercise (primarily aerobic) can increase functional activity in cortical and basal ganglia areas affected by PD (Alberts et al., 2016), promote changes in cortical excitability (Fisher et al., 2008), increase levels of serum brain-derived neurotrophic factor (Frazzitta et al., 2014; Marusiak et al., 2015), and increase dopamine transporter D2 expression (Fisher et al., 2013). Taken together, evidence from research in healthy older adults and individuals with PD supports the notion that exercise may counteract neurophysiological changes closely linked to cognitive decline in PD by increasing functional activity in frontal and basal ganglia regions, promoting increase in brain volume and consequently hindering PD-related brain atrophy, driving rise in levels of neurotrophic factors known to be deficient in PD, and contributing to enhanced dopaminergic transmission. Studies investigating the effects of exercise on cognition in PD have revealed positive results (Cruise et al., 2011; David et al., 2015; Duchesne et al., 2015; Mckee & Hackney, 2013; Tanaka et al., 2009; Uc et al., 2014). However, there are critical factors in previous investigations that need to be addressed in order to improve the current knowledge of how exercise influences cognition in PD.

The first factor to be considered is the use of multimodal exercise protocols to examine the effects of exercise on cognition. Although previous investigations have attributed positive changes in cognition to improvements in aerobic capacity, only two studies to date (Duchesne et al., 2015; Uc et al., 2014) have stringently evaluated the effects of aerobic exercise alone in PD. However, even these studies did not compare their effects to other exercise modalities or a control group composed of individuals with PD. A review by Petzinger et al. (2013) suggested that aerobic as well as goal-based exercise may act upon motor and cognitive pathways that are affected in PD, thus promoting neural plasticity. Since previous exercise programs (e.g. multimodal and adapted Tango) involved both aerobic and goal-based components, it remains unknown which one was critical to the improvements in cognitive function found in these studies. In order to address this gap, the effects of aerobic and goal-based exercise were directly compared in the present study.

It is also important to note that the focus of previous investigations on assessing executive functions in isolation is a limitation in determining the potential of exercise as a therapy to prevent and/or treat cognitive decline in PD. Research has shown that despite deficits in executive functions being highly prevalent in PD, deficits in other cognitive domains such as memory, language, and visuospatial function are stronger predictors of dementia in PD than deficits in executive functions (Williams-Gray et al., 2009). Thus, in order to determine whether exercise may be useful to prevent or postpone the onset of