



The Effects of Whole Body Periodic Acceleration on Non-Motor Symptoms in People with Mild to Moderate Parkinson's Disease

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Authors' contributions

This work was carried out in collaboration between all authors. Authors VS and JD designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Author VS managed the analyses of the study. Authors JB, ML and MM managed the literature searches and conducted the intervention and assisted with data collection. All authors read and approved the final manuscript.

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ABSTRACT

Aims This study assessed whether Whole Body Periodic Acceleration (WBPA) would improve non-motor symptoms in persons with Parkinson's disease.

Study Design: Quasi experimental design using repeated measures.

Place and Duration of Study: Academic Health Care Center, NYIT College of Osteopathic Medicine from 2016 – 2018.

Methodology: There were 13 participants with PD, 11 men and 2 women, with a mean age of 70.8 years. The participants were instructed to wear the activity trackers 7 days prior to, during, and 7 days after the intervention, to monitor sleep, awakenings, and step counts for 24 hours per day over 6 weeks. Additionally, the Pittsburgh Sleep Quality Index was used to assess sleep. The Patient Health Questionnaire for depressive symptoms and Parkinson's Disease Quality of Life were used to assess quality of life. Blood pressure was also monitored before and after each session.

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Results: An insignificant effect between pairs of sleep in hours ($P= 0.84$), awakenings ($P=0.10$), and activity ($P=0.37$), was found for the activity trackers. Significant differences were found in the results of these self-reports, Pittsburgh Sleep Quality Index ($P=0.04$), Parkinson's Disease Quality of Life ($P=0.01$), and Patient Health Questionnaire ($P=0.02$). Highly significant results were seen in the assessment of pre-vs post systolic BP ($P=0$).

Conclusion: Meaningful non motor symptom improvements were found. Systolic blood pressure was also seen to significantly decrease after the intervention. Interventions such as this may have a significant role in PD management of non-motor symptoms. Future studies to assess optimal dosing of WBPA for maximal benefit are warranted.

Keywords: Whole body periodic acceleration; Parkinson disease; non-motor symptoms; quality of life.

1. INTRODUCTION

Parkinson's disease (PD) is a neurodegenerative disease that is characterized by a reduction of the neurotransmitter dopamine within the basal ganglia, more specifically the substantia nigra [1,2]. PD has become increasingly more prevalent, affecting 1% of people over the age of 60 [3,4]. The hallmark signs of PD are the presence of various motor symptoms: resting tremors, gait abnormalities such as shuffling gait, festinating gait and freezing gait, dysarthria, hypophonia, bradykinesia and rigidity [2,5]. In addition, PD is accompanied by numerous non-motor symptoms that for the most part, have not been heavily addressed. These include, neuropsychiatric symptoms, sleep disorders and daytime sleepiness, fatigue, gastrointestinal disturbances, depression and cognitive dysfunctions [5,6]. The presence of sleep disturbances have been reported in approximately 98% of patients with PD [7]. Patients will often report complaints of disturbed sleep, excessive daytime sleepiness (EDS), cognitive decline, depression and overall reduction in health-related quality of life due to their sleep-related disturbances [8]. In addition to EDS, nocturnal/early morning tremors, restless legs syndrome (RLS) and rapid eye movement behavioral disorder (RBD) are common sleep-related disorders seen with PD [7]. EDS is characterized by an increased desire to sleep during the day and/or episodes of "sleep attacks" which are sudden and irresistible feelings of sleepiness [9,10]. It is unclear the exact cause of EDS, but it's thought to be related to side-effects of certain dopamine agonist medications such as ropinirole or pramipexole [11].

Muller et al., found the presence of nonmotor symptoms to be equally as debilitating as the traditional motor symptoms, placing a significant impact on health-related quality of life [5].

Sleep is an integral part of life and is essential for survival and longevity. Adequate sleep has added benefits for optimal brain function, including learning and creating new memories, neuronal communication and removal of toxins [12]. Healthy sleeping habits have also been shown to help promote neuroplasticity, thereby improving learning and memory [13]. Sleep consists of two stages, non-rapid eye movement (NREM) and rapid eye movement (REM). REM sleep is described as a dream-like state, characterized by mixed brain wave activity, muscle atonia and rapid eye movements [14,15]. Atonia prevents movement during the dreaming phases of sleep [14]. It has been proposed that REM sleep plays a role in brain development, memory formation and consolidation, neuronal plasticity and excitability, processing emotional information and to serves as a way of activating the brain without disturbing the sleep cycle [14,16]. Additionally, during REM, nitric oxide is produced and plays a vital role in maintaining homeostatic sleep [17]. Nitric oxide (NO), when released by vascular endothelial cells, also acts as a vasodilator to help regulate blood pressure and blood flow [18].

Whole body periodic acceleration (WBPA) is a non-invasive and passive exercise that produces low-frequency and low-intensity horizontal oscillations at a set velocity and frequency. The individual lays supine with feet secured for a predetermined duration [19,20]. The most profound and vastly investigated effect of WBPA is the increase in pulsatile shear stress on the vascular endothelium. This increase in pulsatile shear stress activates the production of endothelial-derived nitric oxide synthase (eNOS), which causes the subsequent release of NO into circulation [21]. NO is produced from the amino acid L-arginine via three different NO synthases; eNOS, neuronal NOS (nNOS) and inducible NOS (iNOS). The NO produced via eNOS has been shown to reduce apoptosis of neuronal

cells as well as protect against the risk of stroke, traumatic brain injury, neurodegenerative and cognitive disorders [22]. In addition to NO, WBPA has been shown to increase the production of Brain Derived Neurotrophic Factor (BDNF) and Glial Derived Neurotrophic Factor (GDNF). Both BDNF and GDNF are neurotrophins found within the substantia nigra and are involved with neuronal development and function, memory formation and dopaminergic neuron maintenance [22]. WBPA has increased in popularity in research and clinical practice as an alternative means of exercise, especially for individuals who are incapable of performing active exercise, such as those with PD or other neurodegenerative disorders and mobility dysfunctions. Sakaguchi et al., concluded that WBPA can serve as a viable alternative to active exercise, with improvements noted in coronary microcirculation, improved endothelial function of peripheral arteries and improved glucose tolerance for patients with Type-2 diabetes [23]. Fukunda et al., also demonstrated the effects of WBPA on coronary microcirculation and coronary flow reserve by the release of NO into the peripheral vasculature, thereby helping to reduce vascular resistance. The authors concluded that the use of WBPA can be beneficial for the treatment of patients who are at risk for developing coronary artery disease [24].

The purpose of this current study was to investigate the effects of WBPA on quality of life, depressive symptoms, sleep disturbances and daily activity. We hypothesized that WBPA would decrease systolic blood pressure, decrease the severity of nonmotor symptoms of sleep and depressive symptoms and subsequently improving activity levels/steps in patients with Parkinson disease.

2. MATERIALS AND METHODS

This study was approved by the New York Institute of Technology IRB board and registered on ClinicalTrials.gov (Identifier: NCT02874261). The participants were recruited from the NYIT Academic Health Care Center.

Thirteen participants participated in the current study (11 males and 2 females), with a mean age of 70.8 years. The inclusion criteria for participation included: diagnosis of PD Hoehn & Yahr (H&Y) stages 1-3, complaints of sleep disturbances and have a smartphone (Apple® or Android®) readily accessible or willingness to use an Apple® Ipad2 provided by the

researchers. The exclusion criteria included: no complaints of sleep disturbances, inability to commit to the specific time needed to complete the study protocol, currently undergoing medication changes/modifications, difficulty with or unable to tolerate the supine position and/or complaints of pain that interferes with sleep that stems from an orthopedic condition or other factors unrelated to the symptoms of PD. Upon participant's consent, demographic information including age, sex, height, weight and United Parkinson Disease Rating Scale (UPDRS), were obtained.

Table 1. Demographics

	Age	BMI	UPDRS III*
Mean/ Std. deviation	70.8/9.40	26.66/3.55	NA
Median	68.5000	25.5400	33.0000
Minimum	55.00	21.46	14.00
Maximum	89.00	33.50	62.00

*United Parkinson Disease Rating Scale Section III

Outcome Measures included three self-report questionnaires administered to all participants' pre and post intervention: Pittsburgh Sleep Quality Index (PSQI), Patient Health Questionnaire (PHQ-9), Parkinson's Disease Questionnaire (PDQ-8).

The Pittsburgh Sleep Quality Index (PSQI) is a self-report questionnaire that assesses sleep quality over a one-month period. It has 19 questions broken up into seven categories relating to the duration of sleep, presence of sleep disturbances, sleep latency, habitual sleep efficiency, use of sleep medication, daytime dysfunction due to sleepiness and overall sleep quality. Each category is graded from 0-3, yielding a total score of 21, with a score of 3 indicating severe dysfunction. People who obtain a score > 5 are categorized as poor sleepers, while a score ≤ 5 are categorized as good sleepers. Gelaye et al., concluded that the PSQI has good internal consistency, concurrent validity and discriminative validity [25]. The minimal clinically important difference (MCID) for the PSQI is 3 points [26].

The Patient Health Questionnaire-9 (PHQ-9) is a self-report version of the PRIME-MD diagnostic tool, called the Patient Health Questionnaire (PHQ) [27]. The PHQ-9 is a 9-item depressive symptom specific scale, with each item scored from "0" (not at all bothered) to "3" (bothered



Fig. 1. WBPA bed

nearly every day) for a total score of 27. A score of 5, 10, 15 and 20 serve as valid and useful cutoff points for the lower limits of mild, moderate, moderately severe and severe depression, respectively. Kroenke et al. concluded that that PHQ-9 is a reliable and valid outcome measure to assess the severity of depression/depressive symptoms [28]. The MCID for the PHQ-9 is 5 points [29].

The Parkinson's Disease Questionnaire-8 (PDQ-8) is a short form version of the PDQ-39, which is used to assess self-perceived health and health related quality of life in patients with PD. It contains 8 items, directly from the PDQ-39, that demonstrates the strongest correlation from within the domain associated with PD [30]. The MCID for the PDQ-8 is a change of >5.78 points [31].

Each participant was issued an activity tracker, a JawboneUP3®, which was properly synchronized with the UP® phone application. The participants were instructed on the correct use, care and synchronization of the JawboneUP3®. Fitness trackers were worn 1-week prior to intervention to obtain a baseline measurement, during the 4-week intervention, and 1-week post WBPA intervention, for a total of 6 weeks. The JawboneUP3® assessed sleep duration, sleep awakenings and activity levels throughout the duration of the study. Sleep time

was defined as the total number of hours spent asleep per night. Sleep awakening was defined as the number of times one wakes up during the night. Activity levels were measured as the number of steps taken daily [32]. Each session, the researchers synchronized the JawboneUP3® to the UP® application to obtain the recorded data.

The WBPA intervention was performed for 45 minutes per session, 3 times weekly, for 4 weeks. The parameters for the WBPA intervention were as follows: frequency of 120 Hz and oscillation of 16cm. Participants were positioned in a supine posture with a pillow underneath their head and knees, with feet strapped onto the foot plate. Blood pressure was measured before and after each session. See Fig. 1.

3. RESULTS AND DISCUSSION

Data were analyzed using a repeated measures ANOVA and indicated no significant main effect within groups or significant interactions using the activity trackers between groups of the dependent variables; sleep ($P=0.84$), awakening ($P=0.39$), and activity ($P=0.72$). Data obtained from the self-administered questionnaires were analyzed. The PHQ-9, yielded a significant change following treatment (Table 2). The pre-intervention scores obtained were indicative of

Table 2. Self report results for sleep, quality of life and depression

Pair	Pre intervention mean	Post intervention mean	Mean difference	t	Significance*	CI 95% for diff lower bound	CI 95% for diff upper bound
Pittsburg sleep quality index	7.15	4.61	2.53	2.24	.04*	.08	5.00
PDQoL-8	11.92	5.00	6.97	2.97	.01*	1.8	12.00
PHQ-9	5.69	3.84	1.84	2.61	.02*	.31	3.38

*<P.05

mild depressive symptoms, but participants reported decreased depressive symptoms post-intervention. The PDQ-8 indicated a mean improvement of 6.97 points. Each self-administered questionnaire was found to demonstrate a statistically significant difference between pre-intervention and post-intervention assessments. (Table 2). The PSQI produced a mean improvement of 2.53 points following intervention, a quantifiably significant difference.

Systolic BP was analyzed using a paired t test. On average, systolic BP decreased 6.10 mmHg following intervention. These data generated a $t=5.75$ and $P=0$.

Similar results were found in a pilot study conducted by Southard et al, which compared participants with PD to a healthy control group (n=10). Assessing the same parameters as the current study, there was a decrease in depressive symptoms as per the PDQ-9 [33].

The present study explored WBPA as a possible non-pharmacological complementary treatment to treat depressive symptoms, sleep dysfunctions, and improve quality of life and physical activity in PD. Pharmacological treatment and therapeutic interventions are effective in treating motor impairments, but often do not adequately manage the non-motor symptoms. Reduced levels of physical activity, sleep disturbances, fatigue, anxiety, depression, and reduced cognition result in decreased quality of life and ability to participate in activities of daily living in individuals with mild to moderate PD [34].

Post test results of the PDQ-8 met the requirements for the MCID for quality of life. Statistically significant reductions in the PDQ-9 and PSQI were found, indicating depressive symptoms and sleep were measurably improved following WBPA intervention.

For all participants, mean systolic BP values had decreased post-intervention when compared to pre-intervention. After sessions on the WBPA unit, it was observed most participants had flushing of the face and chest. The clinical observations of vasodilation on the participants after a WBPA unit session coincides with the blood pressure results found. These results further support others studies in which WBPA has been shown to induce vasodilation [22,35]. Other studies that found an increase of NO also found an increase in BDNF and GDNF. We posit, based on the results of our self-reported measures, it can be inferred there was an increase after WBPA as well [22]. PD suppresses the neurotransmitter catecholamine norepinephrine (NE) and denervates the sympathetic nervous system (SNS). Levodopa and dopamine agonists, common treatments of PD, cause further inhibition of the SNS, which in turn reduces NE levels and BP [34]. This inhibition of the SNS impacts positional BP responses as well as acute responses to external stress. In a typical response, NE increases during exercise, which increases heart rate (HR) and BP to meet the demands of the body [36]. Conversely, individuals with PD exhibit the suppression of the SNS and NE causes BP and HR responses to be suppressed during exercise stress [37].

No significant differences were found when comparing pre-intervention and post-intervention activity tracker data. Participants increased their daily activity an average of 258 steps following WBPA intervention. In a review of literature, Evanson et al. found a common trend of underestimation of activity. When errors were high, the trend found was that there was an under-estimation of steps taken, when compared to the criterion. These errors are more prominent at decreased cadences. There was no option to set stride length using the Jawbone activity tracker [32]. Thus, the tracker used a default setting based on a healthy population. It is

possible decreased cadence, a hallmark symptom of PD, caused the Jawbone® underestimate daily activity.

Following intervention, sleep time was found to slightly increase while number of nightly awakenings slightly decreased. The Jawbone UP3® has multiple settings, sleep and normal modes. Evanson, et al., found an overestimation of sleep time in activity trackers, including the Jawbone UP3® [32]. When using the normal mode setting, DeZambotti et al., found that total sleep time and sleep efficiency were again overestimated by the Jawbone® when compared to metrics from polysomnography (PSG) [38]. In assessing sleep, the Jawbone® was found to have a high sensitivity in detecting sleep, and low specificity in detecting wake states [32]. The Jawbone® was found to overestimate total sleep time and sleep onset, while wake after sleep onset was found to be underestimated when in comparison with PSG findings. On nights with more disruptive sleep, greater discrepancies were found. Overall, however, the Jawbone® showed good agreement with PSG. DeZambotti et al., found only two participants fell outside of the Bland-Altman plot agreement limits and differences found did not meet clinically meaningful cutoffs for total sleep time and sleep efficiency [38]. As sleep disturbances were a significant complaint amongst the participants, perhaps the Jawbone® may have overestimated total sleep time, and underestimated waking's.

These results correlate with other studies that found an increase in NO after periodic acceleration. The link between NO and sleep has been demonstrated, and multiple studies have sought to establish a method to increase the release of NO into circulation in an effort to improve sleep [12]. When WBPA and two different types of cycling were studied to determine if activity releases NO into circulation, results indicated heavy to moderate exercise and the use of WBPA increased circulation of NO [39]. WBPA has been accepted as a form of passive exercise that increases the release of NO due to the stimulation of eNOS, and improves peripheral endothelial function [40]. As the intensity of exercise needed to increase NO circulation cannot be achieved by all individuals, the results of this study suggest that WBPA may be an effective method of passive exercise to increase secretion of NO.

A limitation in this study was it utilized a small sample of convenience, resulting in a quasi-

experimental design, and neither the researchers nor participants were blinded. This study only included participants with mild to moderate PD. Further studies should be conducted to study the effects of WBPA on all severities of PD and assess optimal parameters for WBPA. Long term effects of WBPA and potential applications for home use for this patient population should be further investigated. In future studies with larger sample sizes, attempts at measuring NO directly and correlating that information with improvements found in non-motor symptoms should be attempted to determine clinical guidelines for treatment.

4. CONCLUSION

This study assessed the effects of WBPA on the non-motor symptoms of PD, utilizing fitness trackers self-assessment questionnaires, and blood pressure measurements to monitor motor and non-motor symptoms of PD [33]. Following WBPA intervention, individuals with PD reported a significant decrease in depressive symptoms, and presented with a decrease in systolic BP. WBPA was found to be a well-tolerated and safe intervention for treating non-motor symptoms associated with mild to moderate PD. Further research is warranted to explore the effects of WBPA as a possible intervention to manage non-motor symptoms associated with mild to moderate PD.

CONSENT

A copy of the written consent is available for review by the Editorial office/Chief Editor/Editorial Board members of this journal.

ETHICAL APPROVAL

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Bergman MDH. Pathophysiology of parkinson's disease: From clinical neuro-

- logy to basic neuroscience and back. *Movement Disorders*. 2002;17(3):S28–S40.
2. Bartels AL. Parkinson's disease: The syndrome, pathogenesis and pathophysiology. *Cortex: A Journal Devoted to the Study of the Nervous System and Behavior*. 2009;45:915–921.
 3. Tysnes OB, Storstein A. *J Neural Transm*. 2017;124:901. Available:<https://doi.org/10.1007/s00702-017-1686>
 4. Muangpaisan MDW. A systematic review of the worldwide prevalence and incidence of parkinson's disease. *J Med Assoc Thai* . 2011;94(6):749-755.
 5. Müller B. Importance of motor vs. non-motor symptoms for health-related quality of life in early Parkinson's disease. *Parkinsonism and Related Disorders*. 2013;19:1027–1032.
 6. Kadastik-Eerme L, Muldmaa M, Lilles S, Rosenthal M, Taba N, Taba P. Nonmotor features in parkinson's disease: What are the most important associated factors? *Parkinson's Disease*. 2016;2016:4370674. DOI: 10.1155/2016/4370674
 7. Ferreira T, Prabhakar S, Kharbanda PS. Sleep disturbances in drug naïve Parkinson's disease (PD) patients and effect of levodopa on sleep. *Annals of Indian Academy of Neurology*. 2014; 17(4):416-419. DOI: 10.4103/0972-2327.144016
 8. Shinde UA, Mehta AA, Goyal RK. Nitric oxide: A molecule of the millennium. *Indian J Exp=0. Biol*. 2000;38(3):201-210.
 9. Neikrug AB, Maglione JE, Liu L, et al. Effects of sleep disorders on the non-motor symptoms of parkinson disease. *Journal of Clinical Sleep Medicine : JCSM : Official Publication of the American Academy of Sleep Medicine*. 2013;9(11): 1119-1129. DOI: 10.5664/jcsm.3148
 10. Knie B. Excessive daytime sleepiness in patients with parkinson's disease. *CNS Drugs*. 2011;25(3):203-212.
 11. Loddo G, Calandra-Buonaura G, Sambati L, et al. The treatment of sleep disorders in parkinson's disease: From research to clinical practice. *Frontiers in Neurology*. 2017;8:42. DOI: 10.3389/fneur.2017.00042
 12. Brain Basis. *Understanding Sleep Disorders*. Available:<https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Understanding-Sleep> (Published May 22, 2017) (Accessed April 11, 2018)
 13. Duss S, Seiler A. The role of sleep in recovery following ischemic stroke: A review of human and animal data. *Neurobiology of Sleep and Circadian Rhythms*. 2017;2:94-105.
 14. Institute of Medicine (US) Committee on Sleep Medicine and Research; Colten HR, Altevogt BM, editors. *Sleep disorders and sleep deprivation: An unmet public health problem*. Washington (DC): National Academies Press (US). *Sleep Physiology*. 2006;2.
 15. Lu J, Sherman D, Devor M, Saper CB. A putative flip-flop switch for control of REM sleep. *Nature*. 2006;441(7093):589-94. Available:<http://dx.doi.org/10.1038/nature04767>
 16. Vyazovsky V, Delogu A. NREM, REM sleep: Complementary roles in recovery after wakefulness. *The Neuroscientist*. 2014;20(3):203-219.
 17. Cespeglio R, Amrouni D. Nitric oxide in the regulation of the sleep-wake states. *Sleep Medicine Reviews*. 2012;16:265-279.
 18. Suzuki K, Miyamoto M, Miyamoto T, Iwanami M, Hirata K. Sleep disturbances associated with parkinson's disease. *Parkinson's Disease*. 2011;2011:219056. DOI: 10.4061/2011/219056
 19. Serravite D, Perry A, Jacobs K, Adams J, Harriell K, Signorile J. Effects of whole-body periodic acceleration on exercise-induced muscle damage after eccentric exercise. *International Journal of Sports Physiology & Performance*. 2014;9(6):985-992.
 20. Sackner MA, Gummels E, Adams JA. Nitric oxide is released into circulation with whole-body, periodic acceleration. *Chest*. 2005;127(1):30-9. Available:<http://arktos.nyit.edu/login?url=https://search-proquest-com.arktos.nyit.edu/docview/200452810?accountid=12917>
 21. Uryash A, Bassuk J, Kurlansky P, Altamirano F, Lopez JR, Adams JA. Non-invasive technology that improves cardiac function after experimental myocardial infarction: Whole body periodic acceleration (pGz). *PLoS One*. 2015;10(3).
 22. Adams J, Uryash A, Bassuk J, Sackner M, Kurlansky P. Biological Basis of Neuroprotection and Neurotherapeutic effects of

- Whole Body Periodic Acceleration (pGz). Medical Hypotheses. 2014;82:681-687.
23. Sakaguchi M, Fukuda S, Shimada K. Preliminary observations of passive exercise using whole body periodic acceleration on coronary microcirculation and glucose tolerance in patients with type 2 diabetes. *Journal of Cardiology*. 2012; 60:283-287
 24. Fukuda S, Shimada K, Kawasaki T, et al. "Passive exercise" using whole body periodic acceleration: Effects on coronary microcirculation. *The American Heart Journal*. 2010;159(4):620-626.
Available:<http://arktos.nyit.edu/login?url=https://search-proquest-com.arktos.nyit.edu/docview/1504555367?accountid=12917>
DOI. 10.1016/j.ahj.2009.12.034
 25. Gelaye B, Lohsoonthorn V, Lertmeharit S, et al. Construct validity and factor structure of the pittsburgh sleep quality index and epworth sleepiness scale in a multinational study of African, South East Asian and South American college students. Li S, ed. *PLoS ONE*. 2014;9(12):e116383.
DOI: 10.1371/journal.pone.0116383
 26. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;28(2):193-213.
 27. Huang FY, Chung H, Kroenke K, Delucchi KL, Spitzer RL. Using the patient health questionnaire-9 to measure depression among racially and ethnically diverse primary care patients. *Journal of General Internal Medicine*. 2006;21(6):547-552.
DOI: 10.1111/j.1525-1497.2006.00409.x
 28. Kroenke K, Spitzer RL, Williams JBW. The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*. 2001;16(9):606-613.
DOI: 10.1046/j.1525-1497.2001. 016009 606.x.
 29. Lowe B, Unutzer J, Callahan CM, Perkins AJ, Kroenke K. Monitoring depression treatment outcomes with the patient health questionnaire-9. *Med Care*. 2004;42(12):1194-1201.
 30. Jenkinson C, Fitzpatrick R. Cross-cultural evaluation of the short form 8-item parkinson's disease questionnaire (PDQ-8): Results from America, Canada, Japan, Italy and Spain. *Parkinsonism & Related Disorders*. 2013;(13):22-28.
 31. Tan LC, Luo N, Nazri M, Li SC, Thumboo J. Validity and reliability of the PDQ-39 and the PDQ-8 in english-speaking parkinson's disease patients in singapore. *Parkinsonism Relat Disord*. 2004;10(8):493-499.
 32. Evenson KR, Goto MM, Furberg RD. Systematic review of the validity and reliability of consumer-wearable activity trackers. *The International Journal of Behavioral Nutrition and Physical Activity*. 2015;12:159.
DOI: 10.1186/s12966-015-0314-1
 33. Southard V, Rumba S, Schwartz I, Sparacino N, Weddingfeld K, Donoghue J. The effects of whole body periodic acceleration on non-motor symptoms in persons with parkinson's disease: A pilot study. *J Nov Physiother Phys Rehabil*. 2017;4(3):077-082.
DOI. 10.17352/2455-5487.000052
 34. Claassen DO, Kutscher SJ. Sleep disturbances in parkinson's disease patients and management options. *Nature and Science of Sleep*. 2011;3:125-133.
DOI: 10.2147/NSS.S18897
 35. Sackner M, Gummels E, Adams J. Effect of moderate-intensity exercise, whole-body periodic acceleration, and passive cycling on nitric oxide release into circulation. *Chest*. 2005;128(4):2794-803.
 36. Goldstein AN, Walker MP. The role of sleep in emotional brain function. *Annu Rev Clin Psychol*. 2014;441(7093):589-594.
 37. Barone P, Amboni M, Vitale C, Bonavita V. Treatment of nocturnal disturbances and excessive daytime sleepiness in parkinson's disease. *Neurology*. 2004;63 (8 Suppl 3):S35-8.
 38. De Zambotti M, Claudatos S, Inkelis S, Colrain IM, Baker FC. Evaluation of a consumer fitness-tracking device to assess sleep in adults: Evaluation of wearable technology to assess sleep. *Chronobiology International*. 2015;32(7):1024-1028.
DOI: 10.3109/07420528.2015.1054395
 39. Sakaguchi M, Fukuda S, Shimada K. Preliminary observations of passive exercise using whole body periodic acceleration on coronary microcirculation and glucose tolerance in patients with type

- 2 diabetes. Journal of Cardiology. 2012; 60:283-287.
40. Fukuda S, Shimada K, Kawasaki T, et al. "Passive exercise" using whole body periodic acceleration: Effects on coronary microcirculation. The American Heart Journal. 2010;159(4):620-626.
- Available:<http://arktos.nyit.edu/login?url=https://search-proquest-com.arktos.nyit.edu/docview/1504555367?accountid=12917>
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