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The Effect of Short Term Beta Alanine Supplementation on Physical Performance and Quality of Life in Parkinson's Disease: A Pilot Study

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ABSTRACT

Allman BR, Biwer A, Maitland CG, DiFabio B, Coughlin E, Smith-Ryan AE, Ormsbee MJ. The Effect of Short Term Beta Alanine Supplementation on Physical Performance and Quality of Life in Parkinson's Disease: A Pilot Study. **JEPonline** 2018;21(1):1-13. Individuals with Parkinson's Disease (PD) experience impairments in muscular fatigue and performance, and consequently low quality of life (QOL). Beta-alanine (BA) supplementation has been shown to attenuate similar motor impairments and improve performance in athletic and aging populations; however, the effect of BA in PD is unknown. Therefore, the purpose of this study was to examine the effects of BA supplementation on performance and QOL in PD. Nineteen participants with PD were stratified by leg strength and randomly assigned to consume 4.8 $g \cdot d^{-1}$ of either slow-release CarnoSyn® BA (BA; n=9) or a maltodextrin placebo (PL; n=10) for 4 wks. Pre- and post-measures of maximal and anaerobic power, functional exercise capacity, muscular performance, and QOL were made. There were no significant differences between groups in any of the variables. Thus, 4 wks of BA supplementation did not improve physical performance or QOL measures in patients with PD.

Key Words: Muscle, Neurological, Performance, Supplement

INTRODUCTION

Parkinson's disease (PD) is a chronic, progressive, neurodegenerative movement disorder characterized by destruction of the dopaminergic neurons and consequent decreases in concentration of the neurotransmitter, dopamine (14). The decrease in dopamine compromises communication in the central nervous system, and leads to motor impairments such as increased muscular fatigue and decreased strength and power, which severely affects quality of life (QOL).

Typical treatment goals include attenuation of related motor impairments enough that functionality can be maintained, while minimizing treatment-related side effects such as nausea, vomiting, drowsiness, hallucinations, and initial exacerbation of existing dyskinesias. Unfortunately, some pharmacological treatments such as Olanzapine for the treatment of psychosis cause secondary side effects such as severe drowsiness (1), and furthermore, other PD drugs such as Piribedil, Pramipexole, and Ropiniole only modestly or negligibly improve motor impairments (13). Thus, natural supplementation strategies that have extensively been shown to improve physical performance and QOL while producing minimal side effects, such as supplementation with beta-alanine (BA), may be of interest to this population (13).

Harris et al. (16) pioneered supplementation with BA, a non-essential amino acid, to endogenously synthesize muscle carnosine. Carnosine acts as a hydrogen ion buffer in active muscle, attenuating the decrease in muscle pH due to hydrogen ion accumulation with strenuous movement, thereby reducing fatigue (7). In a recent position statement, it was determined that 4 wks of BA supplementation (4 to 6 $g \cdot d^{-1}$) is able to significantly increase intramuscular carnosine concentration in healthy individuals, and thus is available to act as and intracellular buffer, particularly in tasks that last 1 to 4 min (31). Although the effects of BA on intramuscular carnosine concentrations in PD are unknown, 12 wks of BA supplementation at 3.2 $g \cdot d^{-1}$ in older adults (60 to 80 yrs old) significantly increased muscle carnosine concentrations (11). However, it is noteworthy that there are significant increases in intramuscular carnosine concentration with BA supplementation in as little as 4 wks in healthy populations (20).

Mechanistically, the improvements in physical performance may be mediated by BA supplementation in a variety of ways. As an example, BA supplementation independent of an exercise regimen has been shown to improve physical work capacity, neuromuscular fatigue, and exercise tolerance in elderly individuals (11,21,30,31). It has also been suggested that BA may improve calcium kinetics (15), and may have an impact on central fatigue (15,17). Thus, it is plausible to hypothesize that BA supplementation may also improve these outcomes in PD patients. Although the etiology of fatigue in PD remains unclear, this population experiences increased fatigue during motor performance with a strong central fatigue component (33). However, the mechanisms of fatigue at the level of the muscle in this population have been sparsely explored. It is unknown whether patients with PD have a dampened ability to buffer hydrogen ion accumulation associated with repetitive movement. Due to the positive effects on neuromuscular function with BA supplementation in healthy subjects, along with the nature of neuromuscular function in PD, it may be a worthwhile strategy to explore in PD patients.

Therefore, the purpose of the present exploratory pilot study was to examine the effects of 4 wks of BA supplementation on physical performance and QOL measures in individuals diagnosed with PD. It was hypothesized that short-term supplementation with BA would improve all measures.

METHODS

Subjects

A total of 24 patients with PD were recruited from the Balance Disorders Clinic of the Tallahassee Memorial Hospital in Tallahassee, Florida and the surrounding areas. One participant was excluded due to physical limitations that led to an inability to complete physical testing at baseline. One participant did not continue after baseline testing due to unrelated health complications. One participant declined to be re-tested, and two other participants did not meet compliance for supplementation (>80%). Therefore, 19 participants completed the requirements necessary for the study. The primary care physician of each participant signed an assent letter for participation prior to any physical activity and exercise. Participants were excluded if they had unresolved, uncontrolled, or recent major medical issues, and/or musculoskeletal injuries. All subjects were able to independently ambulate (Stages 1-4) while those who required the use of an assistive device (Stage 5) were excluded. None of the subjects were supplementing with ergogenic supplements or compounds. All subjects were instructed to continue their normal medication regimen throughout the study with no changes other than the addition of BA or maltodextrin placebo (PL). All procedures were approved by the Florida State University Human Subjects Institutional Review Board in accordance with the Helinski Declaration, and all subjects gave written informed consent.

Procedures

The study was a double blind, placebo-controlled, pilot study designed to evaluate feasibility of the methods chosen and the effects of BA supplementation in a PD population. This was all performed on a small scale prior to scaling the study up for future direction. The subjects visited the Human Performance and Sports Nutrition Laboratory at the same time of day on two different occasions: (a) the initial baseline testing session (pre); and (b) the post-supplementation testing session (post). Both sessions were separated by 4 wks of BA supplementation. The time chosen was based upon the subjects' medication schedule, thus allowing the subjects to test during their most functional time frame, induced by medication and commonly referred to as the "on" phase. The subjects were instructed to take their normal medications (both PD-related and medications addressing other underlying medical conditions) with no adjustments prior to testing. Cardiovascular measures, anthropometric measures, maximal muscular and anaerobic power using a Wingate test, functional exercise capacity using the 6-min walk test, muscular strength, power, and fatigue using the BIODEX, and QOL using multiple questionnaires were tested during the pre and post visits.

After measuring voluntary knee extension peak torque on a dynamometer (Biodex Medical Systems, Shirley, New York, U.S.A.), the subjects were stratified by relative maximal isometric leg strength and randomly assigned to consume the same amount (4.8 $g \cdot d^{-1}$) of either slow-release CarnoSyn® BA (BA: n=9, age 68 ± 9 yrs; Natural Alternatives International, Inc., San Marcos, CA, U.S.A.) or a maltodextrin placebo (PL: n=10; age 68 ± 9 yrs; Natural Alternatives International, Inc., San Marcos, CA, U.S.A.) for 4 wks. The slow

release BA was used because paresthesia (tingling or burning sensation) is attenuated with this formulation (31). Four weeks of BA supplementation was used because this time frame has been found to elicit a significant increase in muscle carnosine levels and reduction in subjective levels of fatigue (18,20,28,29,31). The entire amount of color-matched and taste-matched supplement tablets required for the 28-d period (+2 d) was provided to each subject in opaque white bottles. Both groups consumed two 800 mg pills, 3 times·d^{-1} with meals (4.8 g·d^{-1}). On the last day of supplementation, the subjects were scheduled to return to the laboratory for post testing. Subjects were instructed to bring their supplement bottle and any remaining pills to the laboratory to verify compliance, which was determined as >80% supplement consumption.

After sitting quietly with both feet on the floor for 5 min, cardiovascular measurements were measured manually by the same research personnel. Systolic (SBP) and diastolic (DBP) blood pressures were measured twice and averaged using a standard sphygmomanometer (American Diagnostic Corp., Hauppauge, NY, U.S.A.) and stethoscope. Pulse rate was measured manually at the radial artery for 30 sec and then multiplied by two to obtain the beats·min^{-1}. Measurements of height (SECA, Hamburg, Germany) and body mass (Detecto, Brooklyn, NY, U.S.A.) were recorded without shoes and with minimal clothing to the nearest 0.1 cm and 0.1 kg, respectively.

Physical performance measures were conducted in this order: (a) Wingate Test to measure maximal muscular power and anaerobic power; (b) Six-Minute Walk Test to measure functional exercise capacity; and (c) multiple tests on a Biodex Machine to measure muscular strength, power, and fatigue.

Anaerobic capacity was measured using the Wingate Test on a plate-loaded and friction-braked cycle ergometer (Monark Ergomedic 874-E; Monark Exercise AB, Vansbro, Sweden), as previously described (3). Variables that were analyzed included peak torque, average peak torque, relative peak torque, total work, average power, acceleration time, deceleration time, and relative total work of flexion and extension. Briefly, after an un-weighted warm-up at a self-selected pace, and 3 sec prior to the addition of a weight equivalent to 7.5% of the subject's bodyweight body mass (kg) onto the weight basket of the cycle ergometer, the subject began to pedal as hard as they could for 30 sec while the number of pedal revolutions was counted. If at any time during the test the subject could not continue pedalling, the test ended immediately and was considered incomplete. Although the 30-sec Wingate Test is rarely performed in a Parkinson's population, it has been validated in an untrained, older population (26).

The Six-Minute Walk Test was used to measure the subject's functional exercise capacity, as previously described (24) and validated in a heart failure population (ICC = 0.91) (10). The subjects were told to perform one of two turning options on the testing area: (a) either a "tap and pivot" where the subject crossed the 100-ft mark and immediately pivoted; or (b) a "gentle turn" where the subject crossed the 100-ft mark and made a wide turn around the cone marker. The chosen method was recorded and used for both pre and post testing. Distance covered over the 6-min time frame was recorded.

To measure muscular strength, power, and fatigue, the subjects were placed in the upright seated position on a Biodex System 3 (Biodex Medical Systems, Shirley, New York, U.S.A.).

The seat height and position were adjusted in order to align the instrument's axis of rotation with each subject's dominant knee. Isokinetic 60°·sec^{-1} and 180°·sec^{-1} unilateral knee extension and flexion tests were conducted first. Five repetitions of consecutive maximal extensions and flexions were performed during each test, with a 1-min rest interval between tests. Following the isokinetic tests, a 60° isometric knee extension and flexion test was performed. This test required three maximal extensions and flexions against an immovable arm, with 10-sec rest periods between attempts. Finally, a 50-repetition unilateral knee extension and flexion fatigue test was performed at an isokinetic speed of 180°·sec^{-1}. Isokinetic tests have been substantiated with healthy, young individuals (60°·sec^{-1} ICC = 0.95; 180°·sec^{-1} ICC = 0.96) (12). Likewise, isometric tests were shown to be a reliable measure in a fibromyalgia population (ICC>0.9) (2).

Seven questionnaires were administered, including a Health History Questionnaire and a Physical Activity Questionnaire prior to testing, and the Unified Parkinson's Disease Rating Scale, the Schwab and England Activities of Daily Living, the Short Form 36 Health Survey, and the Beck's Depression Inventory at pre and post testing. A complete health history from each subject was collected and reviewed before testing began. A list of current medications and supplements was analyzed to determine that a BA-containing compound was not being consumed. Subjects were asked to report a 7-d recall of frequency, intensity, type, and duration of daily physical activities, exercise, and physical therapy in a questionnaire.

The Unified Parkinson's Disease Rating Scale (UPDRS) was used to monitor the severity and progression of PD impairment and disability (ICC = 0.9) (23). Of the five sections of the examination, Part II (a self-evaluation of the activities of daily living, consisting of 13 Likert scale questions) and Part III (motor evaluation performed by trained research personnel, consisting of 14 Likert scale questions) were evaluated. The values were totalled, with higher values indicating high impairment and disability.

The Schwab and England Activities of Daily Living questionnaire (25) was administered, which is a Likert scale of percentages used to measure the subject's ability to perform daily activities with speed and independence (ICC = 0.7) (8). A score of 100% indicates total independence, while 0% indicates complete dependence.

The Short Form 36 Health Survey (SF-36) has been validated in PD (ICC > 0.8) (27), and consists of 36 questions, yielding the subject's degree of health on eight different scales of functional health and well-being, psychometrically-based physical and mental health summary measures, and a preference-based health utility index (22).

Beck's Depression Inventory (BDI) has been validated in a PD population (ICC = 0.89) (32), and is a 21-item, self-report rating inventory that measures characteristic attitudes and symptoms of depression (4). A high score indicates more severe depression and related symptoms.

Dietary analysis was measured using a 3-d food record (two weekdays and one weekend day) before baseline testing and during the last week of supplementation. Subjects were asked to maintain and record their normal eating patterns and habits throughout the study. The Food Processor Dietary Analysis Software (Salem, OR, U.S.A.) was used to analyze dietary data. The same research technician entered all food logs. The logs were evaluated to

determine comparisons in calories, and grams of carbohydrate, fat, and protein between pre and post assessments.

Statistical Analyses

An a priori power analysis was performed using the outcome variable of physical working capacity at fatigue threshold (30), which revealed a need for approximately 10 subjects per group with a power of 0.80, α = 0.05, standard deviation = 10 (BA group), difference to detect = 20 (between groups) (JMP v.12, SAS, Cary NC). Data were first analyzed using a one-way analysis of variance (ANOVA) to examine possible group differences at baseline. A 2x2 (group: BA vs. PL; time: pre vs. post) repeated measures ANOVA was used to analyze changes in dependent variables. *Post hoc* analyses, using a Student's *t*-test, were conducted when a significant value was observed for group × time interactions. Significance was set at $P<0.05$ and all data are reported as means ± SD unless otherwise noted. SPSS Version 21 software (SPSS IBM, New York, U.S.A.) was used for all analyses.

RESULTS

Participants and Compliance

The majority of subjects (89%) supplemented with the required amount (≥80%) of their assigned supplement. There were no statistically significant differences between groups at baseline (Table 1). There were no reported side effects with supplement use.

Table 1. Descriptive Characteristics of the Subjects.

	BA	PL
Number of Subjects	9 (5 M, 4 F)	10 (8 M, 2 F)
Age (yrs)	68 ± 9	68 ± 9
Height (m)	1.69 ± 0.08	1.71 ± 0.01
Body Mass (kg)	78.4 ± 11.7	75.3 ± 10.4
BMI ($kg \cdot m^{-2}$)	27 ± 3	26 ± 4
Heart Rate ($beats \cdot min^{-1}$)	69 ± 6	74 ± 13
SBP (mmHg)	129 ± 9	123 ± 10
DBP (mmHg)	80 ± 7	71 ± 7
Average Daily Activity (min)	252 ± 148	301 ± 235

Values are mean ± SD. **BA** = beta alanine; **PL** = placebo; **BMI** = body mass index; **SBP** = systolic blood pressure; **DBP** = diastolic blood pressure; **M** = male; **F** = female. No variables were significantly different at baseline.

Cardiovascular Measurements

No significant differences were observed for resting heart rate. There were no significant group × time interactions for heart rate or SBP. A significant main effect of time was observed for SBP (BA: pre, 132 ± 12 mmHg vs. post, 127 ± 10 mmHg; PL: pre, 130 ± 10 mmHg vs. post, 124 ± 12 mmHg; P=0.05) and DBP (BA: pre, 82 ± 9 mmHg vs. post, 78 ± 8 mmHg; PL: pre, 80 ± 6 mmHg vs. post, 75 ± 9 mmHg; P=0.036).

Muscular Power, Strength and Endurance

Of the 19 subjects, 13 subjects (BA: n=6, 3 men, 3 women; PL: n=7, 6 men, 1 woman) completed the full 30-sec Wingate Test during both testing sessions. The remaining subjects could not complete the test due to an inability to maintain pedal movement throughout the 30-sec protocol. Individual responses of maximal wattage are depicted in Figure 1. Maximal wattage was increased in 63% of subjects in the BA group, while it was increased in 78% of subjects in the PL group (Figure 1). There were no statistically significant differences observed for any of the measures.

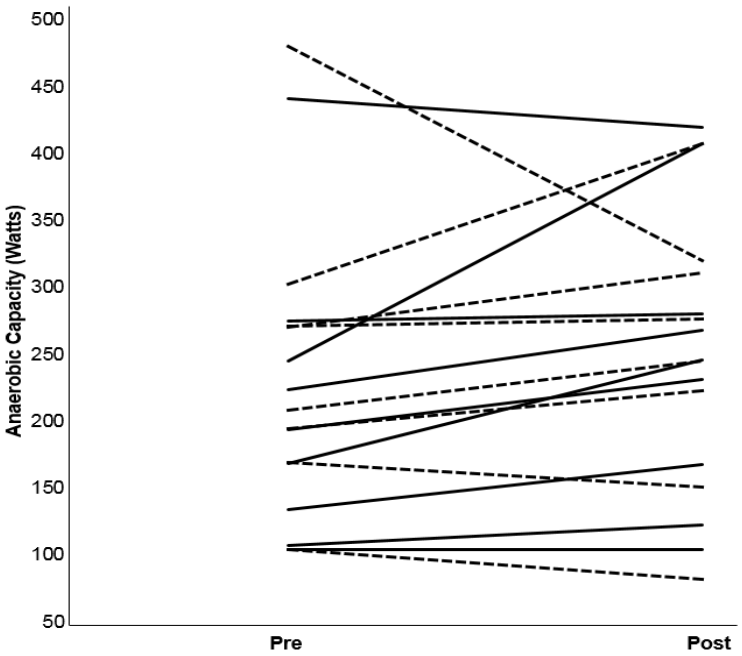

Figure 1. Individual Responses in Anaerobic Capacity with BA and PL Supplementation.

All subjects were able to complete the Six-Minute Walk Test. There were no group × time interactions. There was a significant main effect of time for an increase in distance covered (BA: pre, 437 ± 98 m vs. post, 473 ± 87 m; PL: pre, 461 ± 100 m vs. post, 500 ± 94 m; P=0.001).

There were no significant group × time interactions observed for the Isokinetic 60°·sec^{-1}, the Isokinetic 180°·sec^{-1}, or the Isometric 60º tests. Significant group x time effects were observed for relative total work during flexion (BA: -2.4% ∆; PL: +9.1% ∆; P=0.018) and acceleration time during extension (BA: +4.4 msec ∆; PL: -13.0 msec ∆; P=0.000) during the Isokinetic 180°·sec^{-1} Fatigue Test. A significant main effect of time was observed for peak torque during flexion (P=0.023); average peak torque during flexion (P=0.007); relative peak torque during flexion (P=0.028); total work during flexion (P=0.014); average power during both extension (P=0.023) and flexion (P=0.000); and deceleration time during extension (P=0.008) during the Isokinetic 60°·sec^{-1} test. During the Isokinetic 180°·sec^{-1} test, all variables demonstrated a main effect for time, with no significant main effects for group. A significant main effect of time was observed for peak torque during the away phase (P=0.024), average peak torque during the away phase (P=0.021), and relative average peak

torque during the away phase (P=0.034) during the Isometric 60º test. A significant main effect of time was observed for peak torque during extension (P=0.006), relative peak torque during extension (P=0.007), and acceleration time during extension (P=0.043) during the Isokinetic 180°·sec^{-1} Fatigue Test.

Dietary Analysis

A total of 17 food logs were analyzed (BA: n=8; PL: n=9). The remaining two participants, one from each group, did not return their 3-d food records for analysis. A significant group × time (P=0.05) effect was observed for absolute total protein intake. However, post hoc analysis revealed no statistically significant differences (BA: +10.5 g Δ, P=0.15; PL: -9.8 g Δ, P=0.18) (Table 2). No other significant differences were observed in any variable for either group.

Table 2. Dietary Analysis Before and After 4-Wks of BA or PL Supplementation in PD.

	BA (n=8) 5 M, 4 F			PL (n=9) 8 M, 2 F		
	Pre	**Post**	**%Δ**	**Pre**	**Post**	**%Δ**
Total Energy (kcal)	2598 ± 580	2694 ± 652	3.7	2325 ± 639	2227 ± 488	-4.2
Total CHO (g)	314 ± 58	302 ± 55	-3.8	293 ± 69	308 ± 99	5.1
Total PRO (g)[a]	89 ± 24	100 ± 28	12.4	90 ± 23	80 ± 24	-11.1
Total Fat (g)	109 ± 43	117 ± 45	7.3	93 ± 38	79 ± 26	-15.1
% CHO	50 ± 10	46 ± 9	-8.0	51 ± 5	55 ±11	7.8
% PRO	14 ± 3	15 ± 3	7.1	16 ± 3	14 ± 3	-12.5
% Fat	37 ± 8	38 ± 8	2.7	35 ± 5	32 ± 9	-8.6
Rel PRO (g/kgBW)	1.13 ± 0.27	1.29 ± .40	14.2	1.22 ± .29	1.10 ± .31	0.0

Values are mean ± SD; **M** = men; **F** = female; **BA** = beta alanine; **PL** = placebo; **CHO** = carbohydrate; **PRO** = protein; **a** = significant group x time effect P=0.05.

Quality of Life Questionnaires

There were no significant main effects of time or differences between the groups within any of the measures in the questionnaires.

In regards to physical activity, subjects were categorized as either sedentary with no reported exercise, additional physical activity, physical therapy (BA: n=3, 3 M, 0 F; PL: n=3, 2 M, 1 F), or physically active with reported regular exercise and/or additional physical activity (BA: n=6, 2 M, 4 F; PL: n=7, 6 M, 1 F). The subjects did not change their physical activity or exercise habits over the course of the study.

DISCUSSION

The main finding of this pilot study was that short-term BA supplementation did not improve markers of physical performance or QOL measures in individuals with PD. In fact, compared

to the placebo, in some cases, BA seemed to blunt positive changes in physical performance. It is convenient to consider BA as ergolytic in PD patients, but given the limitations of the present study this conclusion is speculative. Much more research is needed. Nevertheless, this is the first study to evaluate the potential effects of BA in PD patients.

Previous data indicate that BA may delay both central and peripheral fatigue (5), with these effects confirmed in older adults (30). Even so, although mechanistically there may be a theoretical basis for using the hydrogen buffering capacity of carnosine through supplementation with BA to combat disorder-related fatigue in PD, to date there is no empirical evidence suggesting that augmented hydrogen ion accumulation is the impetus of fatigue in this population. Thus, with indirect interpretation, it may be assumed that fatigue in PD is not the result of disordered hydrogen ion accumulation, given that it was not improved with short-term BA supplementation.

The metabolite directly responsible for the hydrogen-buffering capacity, carnosine, is increased when elderly subjects (60 to 80 yrs) were supplemented with 3.2 $g \cdot d^{-1}$ of BA for 12 wks (11). This increase in muscle carnosine with BA supplementation (independent of an exercise protocol) was accompanied by improvements in time to exhaustion in both incremental and constant-load treadmill tests, but not in functional tests such as timed-up-and-go and or timed-stands (11). Thus, it may be concluded that BA is capable of improving physical capacity in repetitive movements at higher intensities possibly through increased buffering capacity, but likely has no effect on functional movements of much shorter duration at lower intensities. This notion is widely accepted, and has been recently published in a position statement on BA supplementation and performance (31). These findings are consistent with other data in 55 to 92 yr old subjects supplemented with 2.4 $g \cdot d^{-1}$ of BA for 12 wks (30).

The lack of significant findings in the Wingate Test of muscular and anaerobic power may be due in part to the intensity required for the test, in combination with the subjects' disorder-related exacerbation of muscular function. Measures of physical functioning are directly related to Wingate performance in elderly adults (26). Patients with PD display poor physical functioning, and thus would be expected to perform poorly on a Wingate Test. Failure to meet the acceptable criteria for the test in most of the population in the current study conveys the importance of using an abbreviated, modified version of the Wingate Test in future studies, as has been used previously and verified (9). Likewise, natural variation may have been a factor of the different changes in pre and post measures between the BA and PL groups.

The higher protein intake in the BA group compared to the PL group (BA: pre, 89 ± 24 g, post, 100 ± 28 g; PL: pre, 90 ± 23 g, post, 80 ± 24 g, $P=0.045$) was interesting, as it juxtaposed the lack of changes in physical performance measures compared to the PL group. Despite the significant interaction for absolute total protein intake, there was only a 10 g protein difference (40 kcals), which likely has limited influence on physical performance measures. Even so, it is well documented that there is a relationship between protein consumption and lean mass in older adults (19). However, although individual evaluation of protein consumption showed relatively minimal changes, three subjects in both groups (6 total subjects) had an average increase or decrease in protein consumption of ≥30 g. Thus, these data may have been skewed significantly by subject recording error, which is common when utilizing self-reporting dietary logs (6).

Limitations in this Study

There are several limitations in this study. The current design excluded a familiarization day for two primary reasons. First, logistic limitations excluded the possibility of a third visit to the laboratory (i.e., transportation and parking). Second, compliance from our specific population is noted to be poor and, therefore, we aimed to minimize burden to the participants after direct instruction from the primary neurological physician. For the same reason, a shorter supplementation period (4 wks) was chosen as opposed to a 12-wk period that has been shown to be affective in elderly subjects (11). However, as little as 4 wks of supplementation has been shown to be effective in increasing intramuscular carnosine concentrations in healthy individuals (16).

Future research must include a familiarization phase of laboratory testing. Additionally, the sample size used was small (n=19). The number of subjects was chosen based on previous research using BA in elderly individuals (n=26) (11). With limited evidence of the effectiveness of exercise testing protocols in PD, it was difficult to effectively choose appropriate testing methods. Based upon the inability of most subjects with PD to perform the cycle ergometer tests, use of these exercise tests would not be recommended in future studies. Commonly reported difficulties of these tests were rapid fatigue of the legs and difficulty maintaining a fixed path of pedal rotation.

Additionally, there were no measurements of muscle carnosine concentrations, which should be incorporated in future studies. Lastly, incorporation of an exercise intervention with BA supplementation is paramount for any future research in this area, as the primary action of BA is providing a buffer for the changes in acidity within the muscle with intense, repetitive physical performance.

CONCLUSIONS

This exploratory pilot study was the first to evaluate the effects of short-term BA supplementation in subjects with PD (Stage 1-4 PD). Four weeks of BA supplementation, independent of an exercise intervention, did not significantly improve physical performance or quality of life in adults diagnosed with PD greater than a PL.

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