Published in final edited form as:
J Cardiovasc Nurs. 2013 ; 28(4): 380–386. doi:10.1097/JCN.0b013e31824af587.

The Effects of Smoking Status on Walking Ability and Health-related Quality-of-Life in Patients with Peripheral Arterial Disease

Cynthia Fritschi, PhD[1], **Eileen G. Collins, PhD**[1,2], **Susan O'Connell, MHA**[2], **Conor McBurney, BA**[2], **Jolene Butler, MS**[2], and **Lonnie Edwards, MD**[3]

[1]University of Illinois at Chicago, Department of Biobehavioral Health Sciences

[2]Edward Hines Jr. VA Hospital, Research and Development

[3]Edward Hines Jr. VA Hospital, Department of Cardiology

Introduction

Peripheral artery disease (PAD) is estimated to affect anywhere from 3–10% of the population, but that prevalence increases to 15–20%in the elderly, patients with diabetes, and those who smoke [1, 2] Smoking has been consistently cited as a leading risk factor for the development of PAD [3–6]. A systematic review of studies that measured the magnitude of the effect of smoking on developing PAD reported an average risk for developing symptomatic PAD in smokers to over twice that of non-smokers, and supported a dose-response association between smoking and PAD [4].

The prevalence of PAD increases with age, and is strongly associated with mobility loss and functional disability among the elderly [7]. Maintenance of physical activity, especially walking has been shown to reduce the rate of functional decline, and thus, reduce the risk for both morbidity and mortality in patients with PAD [8–10]. Walking is decreased in patients with PAD due to a variety of physiological factors; most notably intermittent claudication (IC) pain that begins at the onset of exercise and is only ameliorated by rest [11]. Intermittent claudication pain is common in patients with PAD and is thought to arise from localized decreases in calf muscle tissue oxygenation and increased metabolic waste build-up-usually in the calf muscles. As metabolic demand in the exercising calf muscle exceeds perfusion ability, pain occurs, which limits walking endurance[12].

Previous studies in patients with both PAD and intermittent claudication have explored the relationship between walking endurance and smoking status. Smoking has been associated with poorer measures of exercise capacity during treadmill testing, including peak oxygen uptake [13, 14] and earlier onset of claudication pain during walking [13–15]. The mechanisms by which smoking affects walking endurance are not well understood, but may be explained in part, by physiologic mechanisms, such as decreases in calf muscle hemoglobin saturation

Address for correspondence: Eileen G. Collins, Research Career Scientist and Professor, Edward Hines Jr. VA Hospital, 5000 South 5th Avenue, Hines, IL 60141. Tel: +1 708-202-3525; Fax: +1 708-202-3609; eileen.collins@va.gov.

There are no conflicts of interest to declare.

(StO2) kinetics. A literature search revealed few studies about the relationship between smoking status, calf muscle tissue oxygenation, and factors related to walking endurance, including claudication pain. In a study on the effects of smoking on calf muscle StO2, Afaq (2007) reported that smokers had significantly lower measures of tissue oxygenation (StO2) at 1 and 2 minutes into a walking bout and at peak claudication distance, despite having no differences in ankle-brachial index (ABI) levels or resting levels of StO2 than non-smokers [13].

There is also little known about the interrelationships between smoking status and health-related quality of life (HR-QoL) in patients with PAD. Poor HR-QoL in patients with PAD has been associated with shorter long-term survival [16]. In prior studies of the effects of PAD on quality of life, patients with PAD have consistently scored lower on all dimensions of the Short Form-36 than healthy controls or national norms [17, 18]. Both Myers and Izquierdo found relationships between measures of the SF-36 and objective measures of PAD, including ABI, and absolute or initial claudication distance. Regensteiner reported that patients with PAD described HR-QOL burdens similar to those patients with other cardiovascular diseases [19]. None of these studies specifically examined the effects of smoking status on the quality of life indicators.

We hypothesized that patients with PAD who currently smoked would have poorer walking ability, and reduced quality of life than patients with PAD who did not smoke. Therefore, the aim of our study was to explore the differences in walking endurance and PAD-related factors (pain, calf muscle hemoglobin saturation, HR-QoL) between smokers and non-smokers with PAD. Our primary physiologic outcomes included walking distance and onset of claudication pain in meters. Our primary HR-QOL outcomes included the SF-36 Mental and Physical Components Scores. We also explored other secondary variables as listed above.

Methods

We used a cross-sectional, descriptive design to compare differences in physiological and psychological factors between adults with PAD who smoked versus non-smokers. While we were not able to explore causal relationships between variables, use of the descriptive design was able to give us a preliminary view of factors related to walking ability. All study methods were approved by the Institutional Review Boards at the University of Illinois at Chicago and Edward Hines Jr. VA Medical Center.

Participants

Men and women over age 21 were recruited from a Midwestern Veterans Affairs and University Hospital, as well as through flyer distribution around communities located in an urban area. Participants were included in the study if they had a diagnosis of PAD, an ABI of <0.9 or evidence of stiff vessels as assessed by segmental Doppler studies, and gave a positive response on the Edinburgh Claudication Questionnaire (a scale used to evaluate lower extremity pain during walking and standing).

The ABI is the ratio of systolic blood pressure in the ankle to that in the arm. An ABI of ≤ 0.9 is considered diagnostic of PAD. The ABI was measured in both legs and the index from the most affected leg was used in the analyses. The ABI may be elevated due to arterial calcification, thus participants with known arterial calcification were included based on their response to the Edinburgh Claudication Questionnaire. Participants were excluded if they had ischemic ulcers or gangrene on the feet or legs; they were unable to walk on a treadmill, they had vascular surgery or angioplasty within the prior 6 months; they had class III or IV heart failure, they had COPD requiring supplemental oxygen, they had a positive stress test,

or they stopped a treadmill test for pain other than claudication pain. Participants were instructed to refrain from smoking on the day of the study.

Study Variables

Walking endurance (total distance in meters and distance walked at onset of claudication pain) and VO2peak were measured by symptom-limited progressive treadmill test. Subjects were exercised on a treadmill (Quinton) while wearing an ECG monitor. We used a gentle treadmill protocol that was developed for patients with peripheral vascular disease [20, 21]. Exercise began at 0% grade and a speed of 1.8 mph. Increases in percent grade occurred every 30 seconds, and, after the first six minutes, speed increased every three minutes. For participants who were unable to walk at 1.8 mph, the speed started at 1.5 or, in rare cases, 1.0 mph. Formulae were created to convert all walking parameters into distance in meters for standardization across all study participants.

Muscle tissue deoxygenation kinetics using near-infrared spectroscopy (NIRS)—Oxygen saturation of hemoglobin ($\%St0_2$) was measured in the gastrocnemius muscle during exercise testing using NIRS (InSpectra^{TM} Tissue Spectrometer, Hutchinson Technology, Hutchinson, MN). The InSpectra^{TM} Tissue Spectrometer is a non-invasive monitoring system that measures an approximated value of percent hemoglobin oxidation values in tissue ($\%StO_2$) based on spectrophotometric principles. The probe was placed on the medial belly of the gastrocnemius muscle of the leg with the lowest ankle brachial index at the point of greatest lower leg circumference during plantar flexion. The InSpectra^{TM} Tissue Spectrometer has been used in patients with PAD during walking, and successfully tracked the changes in oxygen saturation of the working calf muscles from rest to peak exertion [13, 22, 23].

Health-related Quality of Life

RAND Short Form-36 version 2 (SF-36 v2)—The Medical Outcomes Study/RAND Corporation's SF-36 v2 was used to measure perceived physical function and psychological well-being [24]. The SF-36 v2 is comprised of eight scales (physical functioning, role-physical, bodily pain, general health, vitality, social functioning, role emotional, and mental health), all of which have high levels of reliability and stability of scores when administered to groups of medically stable individuals [24].

Validity of the SF-36 has been supported in the PAD population. In a multivariate analysis, the present investigators demonstrated significant improvement in the physical function subscale in those assigned to a walking program using hand-held poles compared to the non-exercising control group (37% improvement, F = 10.10, $P = 0.003$) [21].

Subjective walking ability was measured using the Walking Impairment Questionnaire (WIQ). The WIQ is an 18-item scale used to evaluate the subject's self-reported difficulty in walking a defined distance (½ or less, 1, 2, or 3 city blocks), speed (walking one block slowly, normally, or quickly, or running or jogging one block), and stair climbing [25]. Responses are ranked on a 0 – 3 Likert-like scale (0 = did not do, 3 = no difficulty). In the current study, internal consistency reliability of the distance, speed, and stair subscales were supported (Cronbach's a = 0.92, 0.88, 0.90 respectively).

Depression Symptoms were measured with the Center for Epidemiological Studies-Depression Scale (CES-D). The CES-D is a 20-item measure designed to evaluate symptoms of depression in the community population. It is a valid and highly reliable instrument for identifying depression symptoms in the community [26]. Reliability testing resulted in a Cronbach's alpha of 0.86 for the current study.

Smoking Status was assessed by self-report of ever having smoked, current cigarette use and pack years; former smoking pack years and year of quitting smoking. Participants who currently smoked were asked to refrain from smoking on the day of the study.

Statistical Analyses

Descriptive statistics included measures of central tendency for continuous variables and frequencies for categorical variables. Independent t-tests were used to assess differences in physiologic and psychological differences between current smokers and non-smokers. A p-value of 0.0125 was considered significant after adjustment using Bonferroni corrections for the four primary outcomes. All statistics were run, using SPSS 18 ™, Chicago, Illinois.

Results

The sample included 105 adults (93% male), aged 70 ± 9.1 years with PAD. At the time of data collection, 36 of the 105 participants were currently smoking. Only nine participants reported never having smoked. Nearly half the participants had diabetes and one-third had some form of heart disease. The demographic and health characteristics are presented in Table 1.

Differences between smokers and non-smokers in measures of walking endurance and health-related quality-of-life

Primary physiologic outcomes:, walking endurance was poor, but similar in both participants who smoked and those who did not smoke. The smokers walked shorter treadmill distances than the non-smokers (375.5± 238.4 vs. 415.2 ± 229.6 meters), but the differences were not statistically significant. However, the time to onset of claudication pain during the treadmill test was different between groups. While there was no difference in pain level at peak exercise, the smokers had their first occurrence of pain at a shorter distance than the non-smokers (142.6 versus 247.7 meters, $p = 0.01$, equal variances not assumed). Amongst the secondary variables, the current smokers also had a steeper drop in calf muscle oxygenation from baseline to 2-minutes into the treadmill test than did the non-smokers (42.3 % vs. 33.0%, $p < 0.05$). The current smokers were younger than the non smokers (65.6 ± 7.7 vs. 72.3 ± 9.0 years, $p < 0.05$). Covariate analyses including age as a factor in the model did not change the relationship between smoking status and pain onset. There were no differences in other physiologic measures related to PAD, including ABI, nor any other measure of calf muscle tissue oxygenation (StO2).

There were significant differences in self-reported psychological health and quality of life between groups. Amongst our primary psychological and HR-QoL outcomes, the SF-36 composite score for mental health functioning from the SF 36 was significantly lower in the smokers than the non-smokers (48.5 ± 10.5 vs. 54.1 ≠ 10.3, $p < 0.0125$, equal variances assumed). There were no differences between groups in the composite physical components Score. Amongst the secondary psychological outcomes, the smokers had lower levels of self-reported physical functioning, lower levels of vitality, poorer social functioning, lower perceived emotional role functioning, and lower perceived mental health well-being. No differences were identified in self-perceived ability to walk a specified distance, walk fast, or climb stairs as measured by the WIQ. Depression symptoms as measured by the CES-D were higher in the smokers, but the difference did not reach statistical significance ($p = 0.054$)(Table 2). Again, covariance analyses did not find age to affect the relationship between smoking status and HR-QoL measures.

Discussion

When we looked at the differences between smokers and non-smokers in this exploratory study, we found that the smokers had an earlier onset of claudication pain during walking than non-smokers. The smokers also had a steeper decline in StO2 from baseline to 2-minutes during the walking bout than the non-smokers, although this finding was not significant. We did not find any differences in calf muscle StO2, walking endurance, ABI, or VO2 peak. In a study of exercise capacity in patients with claudication, Katzel et al. (2001) also reported younger age and earlier onset of pain in smokers. Their findings differed from ours in that they also found their smokers to have lower ABI and lower peak Vo2 values than the non-smokers, although the differences in peak VO2 were erased when they controlled for BMI. They did not measure StO2 in their study [15]. Afaq et al did examine the differences in calf muscle tissue StO2 between smokers and non-smokers [13]. They reported that the smokers had poorer StO2 at 1 and 2 minutes of exercise, as well as poorer StO2 at both initial and absolute claudication distances from non-smokers. Similar to our findings, Afaq reported the smokers and non-smokers had similar StO2 values at rest, but the smokers had steeper declines in StO2 from baseline to 1 minute of exercise. The smokers in our study also had steeper declines in StO2 during exercise, especially the decline in StO2 from baseline to 2-minutes, although these findings were not significant.

Calf muscle blood flow has been found to be reduced in smokers [27], however the mechanisms by which smoking, affects tissue oxygenation remain elusive and cannot be answered by this study. During treadmill walking, we noted a plateau in StO2 after the initial decrease at the onset of walking in all our participants; regardless of smoking status. This StO2 plateau has been noted in other studies of calf muscle oxygenation in patients with PAD during treadmill walking [13, 28]. Afaq hypothesized this was due to a compensatory, progressive increase in blood flow after the initial walking phase [13]. Tew (2009) measured calf blood flow during exercise and reported a progressive increase in total hemoglobin after the first minute of walking, thus supporting this hypothesis [28]. The steeper drop in StO2 among the smokers may have been a compensatory response to increase tissue oxygen extraction, thus allowing the smokers to maintain walking for a similar distance to non-smokers. These data suggest that cigarette smoking may confer a detrimental effect on calf muscle tissue oxygenation in patients who smoke as compared to patients who are non-smokers.

As noted, prior studies have reported reduced HR-QOL among patients with PAD [16–19], however, none have compared smokers to non-smokers with PAD. The findings from our study suggest that smoking may have an additive detrimental effect on both mental and physical domains of HR-QoL in patients with PAD. The non-smokers had much higher scores in vitality, physical and social functioning, their emotional role, and mental health than the smokers. These findings did not change when we looked at the presence of diabetes as a covariate. While smoking is a known precursor to the physiologic changes seen in PAD, the mechanisms by which smoking affects HR-QoL are yet unknown.

Limitations of the Study

One limitation of the study was the use of patient self report regarding smoking status and our inability to assess compliance with not smoking during the day of the study. All participants were instructed not to smoke on the day of the study, but compliance was monitored by self-report. In our study, the majority of patients were former smokers. The self-reported years since quitting smoking ranged from 0–58, with an average of 20.8 ± 15.9 years. There were too few patients who had no prior history of smoking, thus it was impossible to look for differences in physiologic and psychological factors between current, former, and never smokers. However, the percent of patients who have never smoked and

have PAD is consistently low among studies reporting smoking status data in patients with PAD. The use of a cross-sectional design limits our ability to make any causal connection between physiologic state and HR-QoL. Additionally, in this exploratory study, many of the significant findings involved secondary physiologic and psychological outcomes that were not adjusted for multiple comparisons.

In summary, smoking confers additional risks for disrupted tissue oxygenation during exercise above that of patients who have never smoked. Being a current smoker impairs mental HR-QoL, which is already low among patients with PAD.

What's New	Implications for Practice
• Patients with PAD who smoke have earlier onset of claudication pain than patients with PAD who do not smoke.	• Smoking cessation as part of the treatment plan for patients with PAD may aid in walking ability.
• Patients with PAD who smoke have much poorer measures of health-related quality of life than patients with PAD who do not currently smoke.	• Smoking cessation may be an important adjunctive therapy to improve quality of life in patients with PAD who smoke.

Acknowledgments

Funding Source and Conflicts of Interest: This research was supported by grants from the National Institute of Nursing Research (R01 NR8877-01A1: EG Collins, & K99NR012219: C Fritschi); and the Department of Veterans Affairs (Research Career Scientist: F7338-S), Hines, IL.

REFERENCES

1. Olin JW, Sealove BA. Peripheral artery disease: Current insight into the disease and its diagnosis and management. Mayo Clin Proc. 2010; 85(7):678–692. [PubMed: 20592174]
2. Norgren L, Hiatt WR, Dormandy JA, et al. Inter-society consensus for the management of peripheral arterial disease (TASC II). J Vasc Surg. 2007; 45(Suppl S):S5–S67. [PubMed: 17223489]
3. Agarwal S. The association of active and passive smoking with peripheral arterial disease: Results from NHANES 1999–2004. Angiology. 2009; 60(3):335–345. [PubMed: 19153101]
4. Willigendael EM, Teijink JA, Bartelink ML, et al. Influence of smoking on incidence and prevalence of peripheral arterial disease. J Vasc Surg. 2004; 40(6):1158–1165. [PubMed: 15622370]
5. Price JF, Mowbray PI, Lee AJ, Rumley A, Lowe GD, Fowkes FG. Relationship between smoking and cardiovascular risk factors in the development of peripheral arterial disease and coronary artery disease: Edinburgh artery study. Eur Heart J. 1999; 20(5):344–353. [PubMed: 10206381]
6. Fowkes FG, Housley E, Riemersma RA, et al. Smoking, lipids, glucose intolerance, and blood pressure as risk factors for peripheral atherosclerosis compared with ischemic heart disease in the edinburgh artery study. Am J Epidemiol. 1992; 135(4):331–340. [PubMed: 1550087]
7. McDermott MM, Liu K, Ferrucci L, et al. Decline in functional performance predicts later increased mobility loss and mortality in peripheral arterial disease. J Am Coll Cardiol. 2011; 57(8):962–970. [PubMed: 21329843]
8. Garg PK, Tian L, Criqui MH, et al. Physical activity during daily life and mortality in patients with peripheral arterial disease. Circulation. 2006; 114(3):242–248. [PubMed: 16818814]
9. McDermott MM, Liu K, Ferrucci L, et al. Physical performance in peripheral arterial disease: A slower rate of decline in patients who walk more. Ann Intern Med. 2006; 144(1):10–20. [PubMed: 16389250]

10. Gardner AW, Montgomery PS, Parker DE. Physical activity is a predictor of all-cause mortality in patients with intermittent claudication. J Vasc Surg. 2008; 47(1):117–122. [PubMed: 18178462]
11. Sieminski DJ, Gardner AW. The relationship between free-living daily physical activity and the severity of peripheral arterial occlusive disease. Vasc Med. 1997; 2(4):286–291. [PubMed: 9575600]
12. Brass EP, Hiatt WR, Green S. Skeletal muscle metabolic changes in peripheral arterial disease contribute to exercise intolerance: A point-counterpoint discussion. Vasc Med. 2004; 9(4):293–301. [PubMed: 15678622]
13. Afaq A, Montgomery PS, Scott KJ, Blevins SM, Whitsett TL, Gardner AW. The effect of current cigarette smoking on calf muscle hemoglobin oxygen saturation in patients with intermittent claudication. Vasc Med. 2007; 12(3):167–173. [PubMed: 17848472]
14. Gardner AW. The effect of cigarette smoking on exercise capacity in patients with intermittent claudication. Vasc Med. 1996; 1(3):181–186. [PubMed: 9546936]
15. Katzel LI, Sorkin JD, Powell CC, Gardner AW. Comorbidities and exercise capacity in older patients with intermittent claudication. Vasc Med. 2001; 6(3):157–162. [PubMed: 11789970]
16. Issa SM, Hoeks SE, Scholte op Reimer WJ, et al. Health-related quality of life predicts long-term survival in patients with peripheral artery disease. Vasc Med. 2010; 15(3):163–169. [PubMed: 20483986]
17. Myers SA, Johanning JM, Stergiou N, Lynch TG, Longo GM, Pipinos II. Claudication distances and the walking impairment questionnaire best describe the ambulatory limitations in patients with symptomatic peripheral arterial disease. J Vasc Surg. 2008; 47(3):550–555. [PubMed: 18207355]
18. Izquierdo-Porrera AM, Gardner AW, Bradham DD, et al. Relationship between objective measures of peripheral arterial disease severity to self-reported quality of life in older adults with intermittent claudication. J Vasc Surg. 2005; 41(4):625–630. [PubMed: 15874926]
19. Regensteiner JG, Hiatt WR, Coll JR, et al. The impact of peripheral arterial disease on health-related quality of life in the peripheral arterial disease awareness, risk, and treatment: New resources for survival (PARTNERS) program. Vasc Med. 2008; 13(1):15–24. [PubMed: 18372434]
20. Langbein WE, Collins EG, Orebaugh C, et al. Increasing exercise tolerance of persons limited by claudication pain using polestriding. J Vasc Surg. 2002; 35(5):887–893. [PubMed: 12021703]
21. Collins EG, Edwin Langbein W, Orebaugh C, et al. PoleStriding exercise and vitamin E for management of peripheral vascular disease. Med Sci Sports Exerc. 2003; 35(3):384–393. [PubMed: 12618567]
22. Bauer TA, Brass EP, Barstow TJ, Hiatt WR. Skeletal muscle StO2 kinetics are slowed during low work rate calf exercise in peripheral arterial disease. Eur J Appl Physiol. 2007; 100(2):143–151. [PubMed: 17310391]
23. Gardner AW, Parker DE, Webb N, Montgomery PS, Scott KJ, Blevins SM. Calf muscle hemoglobin oxygen saturation characteristics and exercise performance in patients with intermittent claudication. J Vasc Surg. 2008; 48(3):644–649. [PubMed: 18572363]
24. Ware, JE. SF-36 Physical and Mental Health Summary Scales: A User's Manual. Boston, MA: The Health Institute, New England Medical Center; 1994.
25. McDermott MM, Liu K, Guralnik JM, Martin GJ, Criqui MH, Greenland P. Measurement of walking endurance and walking velocity with questionnaire: Validation of the walking impairment questionnaire in men and women with peripheral arterial disease. J Vasc Surg. 1998; 28(6):1072–1081. [PubMed: 9845659]
26. Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. Applied Psychological Measurement. 1977; 1(3):385–401.
27. Gardner AW, Killewich LA, Montgomery PS, Katzel LI. Response to exercise rehabilitation in smoking and nonsmoking patients with intermittent claudication. J Vasc Surg. 2004; 39(3):531–538. [PubMed: 14981444]
28. Tew GA, Nawaz S, Blagojevic M, Zwierska I, Saxton JM. Physiological predictors of maximum treadmill walking performance in patients with intermittent claudication. Int J Sports Med. 2009; 30(6):467–472. [PubMed: 19214940]

Table 1

Demographic and Health Characteristics by Smoking Group

Characteristic	Non Smokers (n=69)		Current Smokers (n=36)	
	n	%	n	%
Gender				
Male	64	92.7	34	94.4
Female	5	7.3	2	5.6
Race				
Caucasian	57	82.6	27	75
African American	10	14.4	8	22.2
Asian	1	1.4	0	
Other	1	1.4	1	2.8
Hispanic ethnicity	5	7.2	1	2.8
Heart disease	21	30.4	12	33.3
Diabetes	34	49.2	34	94.4
Smoking pack years (M/SD)	44.0 ± 31.9		54.2 ± 33.2	
Age in years (M/SD)	72.3 ± 9.0		65.6 ± 7.7	

Table 2

Differences between non smokers and current smokers with PAD

	Non smokers (n=69) M ± SD	**Smokers (n=36) M ± SD**	**P value**
Primary Physiologic Measures			
Walking distance (meters)	*415.2 ± 229.6*	*375.5 ± 238.4*	*.408*
Claudication pain onset (meters)	*247.7 ± 226.2*	*142.6 ± 109.8*	***.010***
Secondary Physiologic Measures			
ABI	0.7352 ± 0.33	0.6735 ± 0.2	.264
Peak VO2	14.72 ± 3.2	14.44 ± 2.9	.679
StO_2 at baseline (%)	57.8 ± 18.7	62.9 ± 15.4	.171
StO_2 at peak (%)	17.5 ± 19.1	17.9 ± 20.6	.922
Drop in StO_2 from baseline to 2-minutes	33.0 ± 23.5	42.3 ± 19.7	.050
Calf pain at exercise peak	4.7 ± 3.5	5.5 ± 3.3	.249
Primary Psychological and Health-related Quality of Life Measures			
SF-36 Mental Components Score	*54.1 ± 10.3*	*48.5 ± 10.5*	***.010***
SF-36 Physical Components Score	*36.6 ± 7.4*	*34.8 ± 7.4*	*.242*
Secondary Psychological and Health-related Quality of Life Measures			
SF-36 Physical Functioning subscale	53.12 ± 20.8	42.5 ± 19.2	.012
SF-36 Role Physical Subscale	52.2 ± 24.5	43.8 ± 25.0	.100
SF-36 Bodily Pain Subscale	53.0 ± 19.2	54.7 ± 24.1	.143
SF-36 General Health Subscale	59.2 ± 18.7	52.3 ± 19.0	.734
SF-36 Vitality subscale	57.9 ± 18.2	44.3 ± 20.8	.001
Sf-36 Social Functioning subscale	79.2 ± 24.2	68.1 ± 29.3	.041
Sf-36 Role Emotional subscale	74.0 ± 28.7	60.0 ± 32.2	.024
Sf-36 Mental Health subscale	77.4 ± 18.2	69.2 ± 18.6	.032
CES-D	13.0 ± 9.6	16.9 ± 9.9	.054
WIQ Distance	0.3734 ± .29	0.3019 ± .24	.218
WIQ Speed	0.3296 ± .23	0.3013 ± .20	.515
WIQ Stairs	0.4700 ± .29	0.4236 ± .25	.418